Resuscitation and the Origins of Intensive/Critical Care Medicine

Ronald V Trubuhovich, ONZM
MD [UoA]; MB, ChB, BMedSc, BDS [all UNZ]; MSc [Oxon]; FRCA, FANZCA, FCICM

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The Australian and New Zealand Intensive Care Society (ANZICS).

Dr Ron Trubuhovich, Doctorate in Medicine graduate at age 90

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Dedication

In loving memory of my parents
Volento Trbuhovich, 1893–1965,
and
Violet Trbuhovich (née Cvitanovich), 1908–1975,
who sent me to St Peter's College, Auckland,
for the education they never had the chance of,
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Elizabeth has stood by me throughout my career and retirement. With patience and forbearance,
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List of Abbreviations

ANZCA	Australian and New Zealand College of Anaesthetists
AV	Artificial ventilation
BFSPPBU	Bennett flow-sensitive positive pressure breathing unit
BPPRA	Bennett positive-pressure respirator attachment
CCM	Critical care medicine
FR	Forced respiration
ICM	Intensive care medicine
ICU	Intensive care unit
INPV	Intermittent negative pressure ventilation
IPPV	Intermittent positive pressure ventilation
ITU	Intensive therapy unit
LACH	Los Angeles County [General] Hospital
m-IPPV	Manual IPPV
NDA	Nuffield Department of Anaesthetics
NPV	Negative pressure ventilation
NYFA	New York Foundling Asylum
OT	Operating theatre
Polio+	Life-threatening polio
PPV	Positive pressure ventilation
RHS	Royal Humane Society
UK	United Kingdom
US	United States
WHO	World Health Organization
WW II	World War II

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Abstract

Intensive care medicine (ICM) has been an independent specialty in hospital practice since its foundation in late 1953 at the Kommunehospital, Copenhagen. The person generally recognised for its introduction is Dr Bjørn Ibsen, an anaesthetist in Copenhagen at that time. Denmark was experiencing a severe epidemic of poliomyelitis in 1952–1953, during which treatment in Copenhagen of those seriously ill from it was provided at its Blegdam Hospital. Ibsen first attended there following the earlier disastrous epidemic period, July–August 1952, in which 27 of 31 ill patients afflicted with poliomyelitis complications perished, despite the best efforts of the Blegdam doctors.

The achievements of earlier pioneers of ICM are less well-recognised but are also important. Therefore, the purpose of this monograph is to provide a comprehensive account of their contributions to ICM. First, an introduction that briefly outlines steps in resuscitation prior to the late 19th century is presented. Then, two major themes are explored in the remaining chapters. The first considers three innovators, Drs Joseph O'Dwyer, George Fell and William Northrup, selected as pioneers of the fundamental treatment modes of ICM, which were necessary in the first instance for ensuring oxygenation through the patency of the airway and the adequacy of ventilation. The accomplishments of these doctors and of others, such as surgeon Rudolph Matas, have almost faded from memory over time, despite these pioneers' significant contributions to ICM. Thus, it is appropriate to honour pioneers of such significance in the history and development of this specialty. The persistence of negative pressure ventilation in treatment through the mid-1900s is assessed to include its undoubted assistance in treating ventilatory deficiency in acute poliomyelitis, although its relative success did hold back the introduction of positive pressure ventilators. A late-1930s Australasian outbreak of poliomyelitis is considered to highlight the usefulness of negative pressure ventilation respirators, as well as its drawbacks. In addition, the claims of early ICM as a treatment mode in barbiturate intoxication in Scandinavia are examined.

The second theme that is developed concerns the Danish polio epidemic in 1952–1953, with special consideration for Dr Bjørn Ibsen's role. Various aspects of articles written on the epidemic are examined to facilitate the correct documentation of facts, while the case for Ibsen's unit at the Kommunehospital being considered the true first intensive care unit (ICU) in the world is debated.

In this regard, the uptake of oxygen by haemoglobin in the blood, from ambient air breathed into the lungs through a patent airway, and the continued pumping of blood by the heart in normal rhythm through the circulatory system to deliver that oxygen to organs and cells, are necessary to maintain life. In tracking the antecedents of ICM or critical care medicine (CCM; the two terms are interchangeable), the first historical references appear to be those for resuscitation. ICM's true origins lie within the Age of Enlightenment in the 18th century.

On 27 August 1952, Dr Ibsen demonstrated, with a newly admitted, seriously ill patient, an anaesthetist's way of compensating for impaired ventilatory function, 'without machinery'. He showed that anaesthesiologists could apply AV of the lungs manually by using a method common in their workplace to compensate for impaired ventilatory function, which was frequently lethal, among the epidemic's sickest

patients. Later, on 21 December 1953 at Copenhagen's Kommunehospital, Ibsen's first patient came by transfer from a medical ward to his newly established ICU, a converted surgical recovery room, which now became a *multi*disciplinary or general ICU. Historically, as I discuss in this monograph, this was 'the first ICU in the world', although data from it were not published until 1958. Consideration of 'first in the world' claims for Ibsen's Kommunehospital ICU brings attention to apparent errors, inaccuracies and myths in the current understanding and documentation of the history of ICM/CCM. I have used primary sources as far as possible.

Prior to Ibsen's first ICU, there had been successful treatment at two institutions in Copenhagen for two discrete groupings of critically ill patients. These were:

1. Poisoned patients, especially those experiencing barbiturate overdosage. From 1 October 1949, the standardisation of treatment for patients with acute barbiturate intoxication was centralised to a dedicated department at Copenhagen's Bispebjerg Hospital, by psychiatrist Dr Carl Clemmesen, with documentation of improved outcomes.
2. Paralysed poliomyelitis victims with breathing problems, as already mentioned. In mid-1952 during a severe epidemic of poliomyelitis, Bjørn Ibsen introduced innovative treatment at Copenhagen's Blegdam Hospital (Blegdamshospital) for patients of epidemiologist chief HCA Lassen, who were critically ill from acute respiratory and/or neurological failure. From 27 August 1952, again in a specialised department, an anaesthetic, manual ventilation technique was employed on a large scale for patients with breathing failure. Its success was evidenced by the remarkable turnaround in mortality, which was more than halved.

In some minds, these two events might be claimed as representing the start of ICM practice, notwithstanding the 'limitation' that Dr Ibsen had identified. He had pointed out in 1966 that the groups were each of 'only one type of disease'. Such limitation would also apply to physician Dr Albert Bower's prior, long-term treatment of similar polio victims in Los Angeles in 1949 by employing Drinker mechanical respirators, some of which were modified for use as a hybrid of intermittent negative plus positive pressure ventilation.

This monograph, submitted as deriving from a body of published work, considers aspects of the Copenhagen topic in depth. My interest has been to try to understand the nature and antecedents of, and the sequel to, Ibsen's masterstroke. This interest has led to a study over a dozen years of the historically relevant medical literature, bringing improved clarity on the 1952 polio epidemic, and (as indicated), questioning the veracity of some current beliefs. I have been curious to identify information on the beginnings of ICM, and on those treatment methods used during the epidemic and afterwards in ICUs. Another aim was to identify personnel who can be considered pioneers but are marginally acknowledged for that role, and to record their effort. Among other conclusions, it has become clear that the topic has been relatively under-recorded and warranted further investigation and analysis. Several 19th–20th century innovators (especially Joseph O'Dwyer, George Fell and William Northrup, as well as John Forbes and Albert Bower) have not retained the reputation warranted by their pioneering methods. Hence, in this monograph, I include a detailed examination of their roles and achievements to address this issue.

Chapter Overview

The Introduction and the other chapters of my doctorate thesis and this monograph derive in part from original papers authored by me, RV Trubuhovich, their sole author, which have been published in medical journals but which I have now revised, corrected and expanded. The relevant title and journal of a paper are provided after the brief description in each paragraph in this overview.

The **Introduction** provides a brief account, setting the scene for notable resuscitation events in especially the two centuries prior to Bjørn Ibsen's formal foundation of ICM practice in Copenhagen's Kommunehospital in late 1953. Some of this work was originally published in:

- History of mouth-to-mouth rescue breathing Part 1. Crit Care Resusc. 2005 Sep;7(3):250–7.
- History of mouth-to-mouth rescue breathing Part 2: the 18th century. Crit Care Resusc. 2006 Jun;8(2):157–71.
- History of mouth-to-mouth ventilation Part 3: the 19th to mid-20th centuries and rediscovery. Crit Care Resusc. 2007 Jun;9(2):221–37.

Chapters 1 and 2 provide detailed studies on the pioneering of extended intralaryngeal/endotracheal intubation and of intermittent positive pressure ventilation (IPPV) by two Americans: Joseph O'Dwyer, who successfully designed and employed intratracheal tubes for laryngeal diphtheria from 1882; and George Fell, who employed 'forced respiration' (FR) from 1887 for life-threatening poisoning, by his system of IPPV via face mask or tracheostomy tube. Earlier sporadic endotracheal intubation by Jean Bouchut (reported in 1858), and Sir William Macewen, (reported in 1878), is also mentioned. See:

- 19th century pioneers of intensive therapy in North America Part 1: George Edward Fell. Crit Care Resusc. 2007 Dec;9(4):377–93.
- 19th century pioneers of intensive therapy in North America Part 2: Joseph O'Dwyer. Crit Care Resusc 2008 Jun;10(2):154–68.

Chapter 3 is devoted to O'Dwyer's combined Fell–O'Dwyer apparatus of 1891, which William Northrup successfully used for neurological disasters in 1894. In France, Theodore Tuffier and Louis Hallion overcame the great 'pneumothorax problem' of an open-chest operation by rhythmic positive pressure inflation, which allowed them to conduct successful intrathoracic operations in 1896. Likewise, in the United States, Frederick Parham and Rudolph Matas employed the Fell–O'Dwyer IPPV apparatus in 1898. However, World War I delayed advances in anaesthesiology suitable for adaptation to intensive care. See:

- 19th century pioneering of intensive therapy in North America Part 3: the Fell-O'Dwyer apparatus and William P Northrup. Crit Care Resusc. 2009 Mar;11(1):78–86.

Chapter 3 Supplement, a digression perhaps, is devoted to resolving the mystery of the identity of a 'Truehead of Galveston'[5] (United States), since his rhythmical inflating device for the apnoeic newborn would appear to have pioneered neonatal IPPV, around 1870 and onwards, even into the 20th century in Central Europe. The actual person became revealed with as close to certainty as could be obtained. See:

- Early artificial ventilation: the mystery of 'Truehead of Galveston' – was he Dr Charles William Trueheart? Crit Care Resusc. 2008 Dec;10(4):238–42.

Chapter 4 focuses on mechanical 'negative' pressure ventilating systems and the introduction of Philip Drinker's 'Iron Lung' that was highly favoured especially in the United States, from 1928. The chapter highlights the important Australian role in the 1937 design, in Adelaide, and the use of Both respirators (and also the Burstall respirator, in Melbourne) for respiratory insufficiency from poliomyelitis. The use of the Both machine became widespread (British 'Empire-wide', owing to the munificence of Lord Nuffield), but perhaps delayed the development of IPPV machines. See:

- Notable Australian contributions to the management of ventilatory failure in acute poliomyelitis. With special reference to the Both respirator and Dr John A Forbes. Crit Care Resusc. 2006 Dec;8(4):383–93.

Chapter 5 describes Albert Bower and V Ray Bennett's introduction and large-scale use of the IPPV attachment to intermittent negative pressure ventilation (INPV) machines ('respirators') in the poliomyelitis epidemic in Los Angeles during 1949, which thereby significantly reduced mortality for poliomyelitis patients there. See:

- On the very first, successful, long-term, large-scale use of IPPV. Albert Bower and V Ray Bennett: Los Angeles, 1948-1949.Crit Care Resusc. 2007 Mar;9(1):91–100.

Chapter 6 describes the efforts of the medical trio who pioneered the 'Scandinavian Method' of treatment of acute barbiturate poisoning. By November 1945, from Copenhagen's Bispebjerg Hospital, Aage Kirkegaard reported successful treatment of circulatory inadequacy from such poisoning, while in October 1949, Carl Clemmesen, who had long experience in treating barbiturate intoxication, established a Poisoning Centre there for the successful centralisation of treatment management, much of which he had applied for several decades. These measures at the Bispebjerg Hospital led to the establishment of an ICU (perhaps the second one) in October 1949. Then, in 1949–1950, Eric Nilsson at Lund introduced IPPV for the management of associated ventilatory failure. See:

- Carl Clemmesen and barbiturate intoxication – was there Intensive Therapy/Care before Copenhagen's Blegdam polio treatment in 1952? In: Cooper MG, Ball CM, Thirlwell JR, editors. Proc. 8th Internat Symp Hist Anaesth. 2013. Sydney: ASA; 2016. p. 696–8.

Chapter 7 recounts the events of 'Bjørn Ibsen's Day', 27 August 1952, when he adapted a manual anaesthetic system for treating polio breathing failure, which markedly reduced the epidemic's mortality rate from c.87% to c.11% in the last 18 seriously ill polio patients. See:

- In the beginning. The 1952-1953 Danish epidemic of poliomyelitis and Bjørn Ibsen. Crit Care Resusc. 2003; 5 Sep:227–30.
- August 26th at Copenhagen: 'Bjorn Ibsen's Day'; a significant event for anaesthesia'. Acta Anaesthesiol Scand. 2004;48(3):272–7. *[That 26th date needing correcting.]*

Chapter 8 presents an examination of statistics for the 1952–1953 Copenhagen polio epidemic to produce definitive figures from the variations in multiple official and semi-official reports. It is contended that the level of care provided for more than 300 seriously ill polio patients in their Blegdam Hospital areas also qualifies as intensive care. See:

- Further commentary on Denmark's 1952-53 poliomyelitis epidemic, especially concerning mortality; with a correction. Acta Anaesthesiol Scand. 2004;48(10):1310–15.

Chapter 9 is an extended summarising tribute to Dr Bjørn Ibsen, the recognised founder of ICM as a medical specialty, based on an obituary notice I wrote for the journal *Critical Care and Resuscitation* in 2007. It recapitulates some of the data given concerning the 1952–1953 Danish polio epidemic. See:

- (Obituary). Bjorn Ibsen: commemorating his life, 1915-2007. Crit Care Resusc. 2007 Dec;9(4):398–403.

Last, the **Conclusion** provides a historical perspective of the contributions of all these pioneers towards the development of the specialty of Intensive/Critical Care Medicine as we know it today.

Sincere gratitude is especially offered here to the multiple editors of the international journals listed in this overview for ensuring over a decade that my articles that they were prepared to publish were rendered into an acceptable format and their accuracy ensured.

Introduction

The first resuscitations are claimed to be those performed by Hebrew midwives during the Egyptian captivity (c.1300–1200 BCE) to revive apnoeic neonates.[1] However, it turns out that actual rescue breathing by the expired-air method was not explicitly described in the Old Testament Book of Exodus, or in the context of the often cited rejuvenations by Elijah and Elisha in the Second Book of Kings.[2] Instead, this belief seems to arise from nothing more than anecdote, embedded in a strong tradition,[3] old, revered and long-held. One of my aims in research into the origins of my speciality, ICM, has been to examine received wisdom by verifying source references, and this example is just one that I have examined.[4] The story of the history of ICM is remarkable, and my exploration into the work of those who sought to save the lives of people who were very ill, or indeed had just died, has left me with deep respect for their ingenuity, persistence and courage.

We can note for this Introduction that the Greek and Roman civilisations did not have a custom of attempting to resuscitate those experiencing cardiac or respiratory arrest. Their medical doctors had little knowledge of physiology, although they probably had some knowledge of gross anatomy. Nor did succeeding European nations foster this practice, and although tracheotomy was not unknown, wariness left it seldom attempted. Galen of Pergamon (129–c.216 CE), who taught that the left ventricle makes the innate heat to vitalise us, demonstrated that a dead animal's lungs could be ventilated by blowing through a tube sutured into the larynx but he did not attempt medical rescue after the sudden death of humans.[5[p389–98]] As regards China, Yu, Liu, Wang, Wang and Ao[6] reported that attempts at resuscitation in cases of hanging are described in ancient Chinese literature.

Reliable documentation on a practice later known as 'The Midwives' Secret', believed to have been continued throughout more than two millennia, is difficult to locate. Many authors have used such an expression since pre-Renaissance times, yet little is known or recorded from those times about the actions of midwives for the non-breathing newborn (or for those stillborn). In medieval Europe, two principal reasons hindered the development of resuscitation skills.[4] The first was poor knowledge of anatomy generally and poorer practical understanding of human physiology—dissections of the human body were forbidden during one and a half millennia, CE. The second was the dominance of certain religious ideas.

Resuscitation might have been seen as contrary to God's will, and therefore as an irreligious, lawless act. Further, there might be uncertainty about a body's being truly dead, without seeing some evidence of putrefaction;[7] also, there was reluctance to touch a dead body because of fears of contagion, or of religious or judicial reprisals. So, attempts to reverse sudden apparent death were not practised. But in the Middle East during the 11th century, polymath Ibn Sina (Avicenna), whose influence was pervasive for 500 years, did report on the use of a gold or silver tube to intubate the larynx.[8[p.161]]

In Renaissance times, Leonardo's unpublished drawings of various human and animal organs corrected some errors of anatomy made by Galen. By 1543, Andreas Vesalius, with access to dead bodies after centuries of prohibition, described an experiment in which he restored a pig to life by blowing down a reed inserted into an opening in its trachea.[9[p274]] In 1628, William Harvey published a landmark work[10] that

demonstrated the circulation of the blood and the true function of the heart. Robert Hooke showed in 1666[11] that the inflating air (delivered by bellows sutured into the trachea of a dog with its chest already opened) needed to be 'fresh' to resuscitate the animal, but this message appears not to have been widely taken up.[11] There was a lack of communication between scientists of those times, who were slowly acquiring physiological knowledge, and the public at large. By the 17th and 18th centuries, there was widespread fear, especially in France, of premature interment, as Winslow detailed in 1740,[7] furthering the need to ensure death absolute before burial.

Referring to times short of helpful documentation about resuscitation, Dagi[12] made an interesting comment in 1987. If resuscitation could sometimes reverse apparent death, one would expect the obligation to attempt resuscitation to be a civic duty.[12[p359]] But this was not the case, neither for physicians nor for laypeople, with the possible exception of 'midwives, colliers and watermen' all of whom apparently maintained a 'venerable tradition of resuscitation'[12[p359]] long before it attracted any academic or scientific interest. Without offering evidence, Sir Arthur Keith also asserted (in 1909), that 'the laity had practised methods of resuscitation since time immemorial'.[13[p746]] Nevertheless, documentation on those methods is sparse.

In 17th century Europe, Sebastian Weiss recorded the various methods for resuscitation he applied to people after their apparent drowning (reported in 1675).[14] These included stimulation by fumigation from tobacco smoke (a mode later reported as being used by certain Arcadian Natives in 1611).[15] An occasional report from Central Europe (e.g. by Borelli[16] or by Grubelius[17[p79-80]]) would indicate that the possibility of rescue efforts by people was known. Then, the Age of Enlightenment fostered attitudes of concern for one's fellow humans—attitudes extending to rescue, especially after drowning, or by suffocation as with hanging. Georg Detharding[18[p438–9]] was advising tracheotomy for rescue after drowning in 1714 (but until 1825, there were only 12 known survivals recorded after a tracheotomy). Then, in 1732, William Tossach[19] reintroduced mouth-to-mouth resuscitation (which might have had occasional use previously), an event in the presence of hundreds. The method was encouraged by John Fothergill,[20] who failed to inspire the London Royal Society, when he presented to this Society in 1745. Sir Arthur Keith stated: 'William Hunter spoke of mouth-to-mouth inflation as the method practised by the vulgar [i.e. the 'common people'] to restore stillborn children'.[13[p746]]

Resuscitation was promoted in France with numerous methods, such as attention to drying, warming and stimulation (following 'Philantrope' of Neuchatel,[21] reported in 1733), but these are measures mostly regarded now as ineffectual. Rectal fumigation by tobacco smoke was favoured from 1740 in continental Europe. It and other vigorous irritative and resuscitative methods were strongly promoted from Paris, initially by Winslow,[7] then René de Réaumur,[22] both in 1740, and later by Bruhier d'Ablaincourt, in 1742[23], and in 1745 and 1749,[24] with royal endorsement. Great reliance was placed on the anticipated stimulation of the intestines promoting revival, with or without artificial ventilation (AV) by bellows or expired air ventilation from mouth to mouth. Very occasionally, laryngeal or tracheal intubation, venesection or bronchotomy (see Footnote 1[1]) would be attempted. A 1746 booklet about treating the drowned by 'a Physician'[17]—who was probably Rowland Jackson[25]—has a useful summary of resuscitation methods and history.

[1] Footnote 1. At that time the terms 'bronchotomy', 'laryngotomy' and 'tracheotomy' were used interchangeably.[17p[52]]

In Amsterdam,[26] the formation of the first Humane Society in 1767 provided a lead that was then followed by a host of similar, new societies in other countries and cities, including Paris in 1771 and London in 1774. Metropolitan riverside rescue stations were installed alongside the Seine, and the Thames had 11. (London's first recorded rescue case was a successful revival by the Leyden jar method of electric shock.[27]) However, despite the influence of William Coggan and Thos Hawes, the use of mouth-to-mouth resuscitation was diminishing, except by laypeople, perhaps because it was all that they could try, although with fears of contagion. By the end of the 18th century, inflating bellows (considered inferior to mouth-to-mouth resuscitation for expired air ventilation by Fothergill,[20] back in 1745) were officially endorsed in 1782 by the Royal Humane Society (RHS), which, in early 19th century, completely abandoned mouth-to-mouth rescue breathing, for the expired air was considered poisonous and unfit to breathe. The discovery of oxygen and carbon dioxide had not led to increased resuscitation by this method. Notable names of the time include Monro secundus (reported in 1774),[28] Cullen[29] and Lord Cathcart (reported in 1774),[30] John Hunter (reported in 1776),[31] and Charles Kite[32] who devised a complete system for resuscitation in 1787. Carl Rafn and John Herholdt provide a comprehensive survey of much of the European experience until 1797.[33] Samuel Tissot[34] and Antoine Portal[35] were effective enthusiasts in France. Astley Cooper[36] (1768–1841) promoted AV of a kind by abdominal compression/relaxation cycles. For medical resuscitation of the newborn, William Smellie, 1762,[37[p226]] and Benjamin Pugh, 1754,[38] inflated the lungs by blowing down a small tube. Meanwhile, François Chaussier, 1806–1807,[39[p39–41]] used a laryngeal tube with bellows for inflating neonatal lungs. He advocated oxygen and recognised that the tongue could obstruct the airway.

Early in the 19th century, Benjamin Brodie's research on nicotine published in 1811[40] led to the abolition of the fumigation method with tobacco smoke. Brodie suggested that 'artificial respiration' would be of advantage for treatment of certain poisons which affect respiration.[40,41[p350]] But the 19th century has a poor record for effective ventilatory resuscitation, with the virtual abandonment of IPPV once understanding of the primary need for 'artificial respiration' was lost, other than by mid-century researchers such as John Erichsen,[42] who validated AV and oxygen in 1845, or John Snow,[43] who investigated resuscitation.

To some degree, the 1830–1856 period was a time of relative nihilism. Studies by Leroy d'Étiolles in 1826–1829[44] and Francois Magendie in 1829[45] resulted in the abandonment of intubation and of IPPV by bag inflation for fear of bursting the lungs with positive pressure, and the virtual loss of all forms of effective AV, leaving only feeble methods for lung inflation, such as John Dalrymple's version of long-bandage rolling, after Leroy's method.[46] Thus, there was no effective form of AV in use in Britain from 1837. Layperson resuscitation was essentially active warming and drying, as was performed in the RHS Receiving House, Hyde Park.[47] There were no instructions from the RHS for any form of AV before 1856, when a perturbed Marshall Hall, surgeon,[48] introduced immediate action with his Ready Method of negative pressure ventilation (NPV) through back pressures and repeated body turnings. He eradicated the contemporary use of the supine position because it obstructed the airway. Some dissatisfaction with his method resulted in the promotion of alternative methods, with arm–chest–body manoeuvring, but none as effective as ventilating by inflation with bellows or from mouth to mouth. Henry Silvester (in 1859), Benjamin Howard (in 1868), and later, Edward Sharpey-Schafer (in 1902), Holger Nielsen (in 1932) and also Frank Eve (in 1932), introduced their variants of NPV. Peter Karpovich lists these[49] among 100 methods.

During this time, mouth-to-mouth resuscitation was not abandoned by midwives, with startling results reported by Mrs Ann Newby in 1803,[50] Mrs Wigden in 1817, and her daughter Mary Wigden in 1857.[50] Thus, in 1856, more than 300 newborns were saved by that method at St George's Hospital, London.[51] Nevertheless, the basic problems for resuscitation in mid-19th century were the lack of a technique for safe oro-laryngeal intubation and the lack of effective positive pressure ventilation (PPV), which had been virtually abandoned since the 1830s. Bouchet's failure to establish the use of the orotracheal tube in 1858 in France,[52,53] Macewen's packing around such a tube[54,55] for tongue surgery in Scotland and, then, Joseph O'Dwyer's developing metal orotracheal tubes in the 1880s are discussed in Chapter 2.

Primitive NPV machines that were developed include John Dalziel's first tank in 1838, written of in Scotland, Alfred Jones's iron lung in the United States (US) in 1864, Ignez von Hauke's cuirass in Austria in 1874 and Eugène Woillez's spirophore in Paris in 1875 (all closely reported from original sources by Woollam[56]); other methods include Ferdinand Sauerbruch's differential pressure chamber at Breslau, in 1904.[57] These were eventually followed in 1928 by the successful Drinker tank ventilator (see Chapters 4 and 5), and in Australia by the Both and the Burstall tank respirators, and then the Burstall cuirass, in the later 1930s (see Chapter 4).

Circulation was now receiving some attention too, with James Blundell[58] first successfully transfusing blood in 1825 from her husband to a woman with postpartum haemorrhage. William O'Shaughnessy[59] started treatment for cholera (with some success) in 1832 by using intravenous salt solution, but the uptake of his principles was not widespread. At the end of the 19th century, Locke modified Ringer's solution with 1% glucose.

Jonas Balassa (in 1858)[60] made a simple tracheal stoma followed by chest compressions, successfully reviving within 15 minutes a woman said to be lifeless. In 1868, John Hill[61] reported that he had treated three patients in cardiac arrest from chloroform with careful external closed chest massage between 1860 and 1867. The later decades of the 19th century saw attempts at open chest cardiac massage, particularly after cardiac arrests with chloroform. McWilliam[62] recognised ventricular fibrillation as such in 1885–1887. The successful mid-European König–Maass method of open chest cardiac massage, reported in 1883 and 1891,[63–65] did not become established until the 20th century. Elsewhere, despite some successful open chest cardiac massage for arrests from chloroform, possibly *during* surgery, such treatments were performed in the first half of the 20th century only occasionally, since immediate, effective, open chest treatment was likely only during such surgery (or, although still less likely, in an environment of an operating theatre (OT) very close to hand).

It can be noted that closed chest cardiac massaging created little interest among surgeons dealing with a patient in cardiac arrest on the operating table, as they preferred the open method of intervention, which because of their skills, they were able to attempt. Later, in 1958, the closed chest cardiac massaging method with external cardiac massage was rediscovered by Guy Knickerbocker.[66] His method was confirmed through studies at Johns Hopkins Hospital, Baltimore, and then achieved universal adoption for resuscitation. Thereafter, Knickerbocker's method survived while that of König and Maas did not.

Significant 19th century milestones in the reintroduction of intermittent positive pressure ventilation

The late 19th-century list of important turning points comprises:

- 1885: Joseph O'Dwyer joined bellows to his personally invented tubes, for IPPV (Chapter 1).
- 1887: George Fell's reliable IPPV system saved 28 people after narcotic poisoning, etc (Chapter 2).
- 1888: Joseph O'Dwyer devised a combination apparatus, the Fell–O'Dwyer apparatus (Chapter 3).
- 1894: William Northrup reported eight cases using the Fell–O'Dwyer apparatus (Chapter 3).
- 1898: Rudolph Matas used the Fell–O'Dwyer apparatus for thoracic surgical anaesthesia (Chapter 3).

Then, the Dräger Pulmotor,[67] the first oxygen respiration device, became available by 1908 and was widely used by US firefighters, rescuers and lifesavers. IPPV was seen at that time as possibly applicable for treating drowning, asphyxia, intoxications and poisonings, convulsions, certain cerebral events and cardiac arrest.

The first decades of the 20th century

Before World War I, although anaesthetists had an interest in establishing PPV systems, multiple experimental PPV devices failed to become established. These are documented in the history section compiled by William Mushin, Rendell-Baker, Thompson and Mapleson in their comprehensive book *Automatic ventilation of the lungs*.[68] However, the US preference was for insufflation techniques, rather than the PPV methods of Fell, O'Dwyer and Northrup, or machines derived from their principles.[63–65] This war halted further progress with PPV. A historic curiosity, almost unnoticed during 1916, was that in Sweden, Giertz[69] had already advocated rhythmic insufflation AV for thoracic surgery.

The compressed air device of Robert Gesell and Joseph Erlanger (who were not anaesthetists) reported in 1913 and 1914[70] for artificial respiration by either the usual or the insufflation method might be an instance of an alternative device sought to cope with ventilatory problems that, ultimately after some decades, intensive care staff attended to.

Intermittent positive pressure ventilation in anaesthesia

In the **1920s,** long-term PPV was unavailable outside the OT, but the rapid advancements in the speciality of anaesthesia at this time led to the increased use of IPPV for selected patients undergoing surgery. Magill and Rowbotham's wide-bore endotracheal tube with 'bag-squeezing' systems for ether/cyclopropane apnoea in thoracic surgery was further developed by Guedel and Treweek and reported in 1934,[71] enabling IPPV, if needed, for abdominal surgery, while in 1932, Gale and Waters[72] developed a method of IPPV for thoracic surgery. So, by the early 1930s, anaesthesiologists had brought PPV to the OT for thoracic surgery, establishing bag-inflating techniques.

Increasing use of intermittent positive pressure ventilation

Obstetricians might breathe into devices applied directly to the face of apnoeic neonates (Pierce MacKenzie,[73] Edward Graber,[74] FA Alexander and Chas Martin,[75] Frank Rossiter [at Pittsburgh; reported in 1942[76]). In the US, outside the hospital, among those attending the suddenly apparently dead, the first reliance in cities was principally on help from firefighters. They were even called into hospitals—Claude Beck,[77] who in 1939 proposed a resuscitation squad in the hospitals, while an intern at Johns Hopkins (so possibly c.1923), was astounded at such dependency by a hospital (the tale is vividly recounted by David Leighninger[77[p259]]). Lifeguards such as the US Coast Guard Service were also depended upon for rescues.

After establishing clearance of the airway, rescuers could apply AV (as NPV) by the Schafer method, preferred in the United Kingdom (UK), or the Silvester method, preferred in the US, while those with a Dräger Pulmotor to hand after 1908 could try to establish PPV. For the American Medical Association, in the early 1940s, Bernard Ross[78] found a 13.9% survival rate among 1,633 victims, out of hospital and apnoeic at the start of treatment by trained life-saving crews.

While employment of IPPV by anaesthetists in the pre-World War II (WW II) OT was restricted, largely owing to the need for deep anaesthesia by ether or cyclopropane, which enabled open-chest thoracic surgery, *non*-anaesthetists made efforts over decades to devise positive pressure systems for use in certain conditions. This is perhaps exemplified in Marriott's five days of successful use of Erlinger's device in 1920,[79] at the designer's suggestion, for a patient with post-diphtheritic failure of respiratory musculature.

Notable others in the pre-history of ICM include Walter Dandy, who demonstrated in 1923 the benefits of concentrating neurosurgical patients postoperatively into a special care unit with close supervision.[80] Thus, 'two 2-bed rooms were used as a neurosurgical intensive care unit with at least one specially trained nurse in constant attendance'.[80[p492]]

Non-regular positive pressure ventilation efforts

Others employing non-regular positive pressure ventilatory means were those such as Poulton in 1936,[81] who treated the pulmonary oedema of left-heart failure with a primitive positive pressure device; Alvan Barach,[82] who during the late 1930s attempted to apply 5 cm-pressure nasal masks for medical patients with pneumonia and pulmonary oedema; Vladimir Negovskii (the 'Father of Reanimatology'),[83] who, in 1941 during WW II in Moscow, learned from battlefield resuscitation attempts and primitive IPPV and ICM measures; Massachusetts General Hospital staff, in 1942,[84] who with an emergency ICU for three weeks for survivors from the Cocoanut[sic] Grove Fire were spurring innovations in burn care; Lyman A Brewer,[85] in 1944 (in using Barach's positive pressure method), who at Cassino during WW II treated 'traumatic wet lung' in soldiers.

Anaesthesiologists and others who further introduced IPPV into anaesthetic and resuscitation practice were (in the English-speaking world) Ralph Waters;[86,87] Julius Comroe and Robert Dripps,[88] (both in the US); Robert Macintosh and Mushin (UK);[89] and the Edgewood (US) team (David Cooper,[90] Bruce Dill,[91] James Elam[92] and Peter Safar)[93], among many.

Progress in anaesthetic methods

Familiarity with IPPV during anaesthesia became routine after Cecil Gray's 1946 introduction of d-tubocurarine[94] to Britain (and Europe), which enabled lighter anaesthesia than was previously needed to achieve comparable muscular relaxation. Obligatory and compensatory PPV was produced through repetitive, rhythmic lung inflation by manual compression of the bag in the anaesthetic circuit.

Some harbingers of Intensive Care

Eventually, by 1949, Albert Bower and Ray Bennett (see Chapter 5) transformed some NPV Drinker tank respirators to provide supplemental IPPV for polio. In addition, Carl Clemmesen's 1949 provision of a psychiatric special care unit with introductory ICM, including Aage Kirkegaard's fluid resuscitation, for barbiturate and other intoxications (Chapter 6) provided centralised treatment for barbiturate poisoning in Copenhagen.

In Chapters 7–9, I will describe in some detail the role of Bjørn Ibsen, an anaesthetist, in extending the skills of this speciality from the operating room to the wards, to treat ventilatory failure during the Copenhagen polio epidemic. Before the Danish polio epidemic of **1952–1953**, there was no regular IPPV machine in regular use. Only towards the end of the epidemic were a few of them available in Denmark (the Engström and the Bang, as discussed in Chapter 7). The Engström was being further developed; it was not until 1958 that Ray Bennett's Model BA-4 Anesthesia Ventilator was established.

Conclusion

This introductory overview of the events and people antecedent to ICM will now be followed by chapters comprising closely detailed accounts of specific identities and rescue activities, more *immediately* preceding the establishment of the ICM specialty at Copenhagen in 1952–1953. These earlier preceding individuals who are hailed as pioneers appear to receive somewhat diminished notice in more recent times. They are selected here in acknowledgement of their individual roles and with the greatest respect for their achievements.

19th century pioneering of intensive therapy in North America

Chapter 1: Joseph O'Dwyer

Intubation ... as perfected through the conscientious labors of the self-sacrificing Joseph O'Dwyer whose name stands with those of Semmelweis and Crede as the greatest benefactor of infant life.

Fielding H Garrison (1929)[117[p613]]

Chapter 1: Table of Contents

1.1 Introduction

Joseph O'Dwyer, 1841–1898, was a distinguished New York medical figure who made important advances in treatment and was greatly admired for his personal qualities, both as a humanitarian and as a person with nobility of character. His progress can be followed through his publications,[95–102] articles about him and eulogies from his numerous admirers.[103–112] He has been described as 'immortalised by intubation'.[105[p164]] Within a decade of his death, he was revered by such statements as 'the most godlike character I have ever seen in man'[105[p164]] or 'American medicine has no more shining light'.[106[p326]]

1.2 A brief biography

Joseph O'Dwyer[104–106,111] (see Figure 1.1) was born on 12 October 1841 in Cleveland, Ohio, but spent his boyhood near London, Ontario. After two years under supervision, apprenticed to local medical practitioner, Dr Anderson, he entered the College of Physicians and Surgeons in New York, graduating in 1866 at the age of 25. He then worked for two years as 'sanitary superintendent'[104[p361]] at the New York City [Charity] Hospital on Blackwell's Island, attracting notice for his attention to duty and efficiency.[106] O'Dwyer then transferred to a general practice in New York with Dr Warren Schoonover.[106] By 1872,[104,107] he had gained appointment for the first of about 25 years on the medical staff of the Sisters of Charity New York Foundling Asylum (NYFA; see Figure 1.2), where he eventually became superintendent.[106[p345]] The NYFA's dependents numbered about 1,800, of whom approximately 600–700 were residents and 1,200 were 'wet-nursed or otherwise cared for outside'[102[p10]]; all were orphans or children of the poor who lacked access to medical care. O'Dwyer's stable New York practice included attendance at ultimately more than 3,000 obstetrical deliveries[104] in poor surroundings,[105] but by devoting so much of his professional life to the medical care of NYFA children, his obstetrical commitment came to be displaced by attention to the special problems arising from their diphtheria.[105]

In the last decades of the 19th century, diphtheria was atypical among infectious diseases in that it was becoming more prevalent.[108,113] O'Dwyer was appalled to see the way in which many children afflicted with diphtheria died at the NYFA, suffocated by a laryngeal pseudo-membrane (*ex* 'Gk *diphthera*... skin, hide': *Shorter Oxford English Dictionary*[114[p677]]). His efforts in devising methods to save them produced a satisfactory system of intralaryngeal intubation by the mid-1880s, and his retrospective article of 1896[102] illustrated the evolution of his intralaryngeal tubes (see Figure 1.3). Intervention became so successful that by 1894, there was a recovery rate of around 40% among 1,324 affected[112]—but diphtheria, a 'blood disease',[115[p542–3]] could still cause death in other ways, despite intubation.

By 1894, some physicians alleged that introducing calomel inhalations might produce a higher success rate than intubation.[116] Moreover, the need for intubation reduced within a decade of the development of Emil von Behring's antitoxin for diphtheria in 1890, which was available by 1894[113,101[p481],117[p584]] and then gradually adopted. O'Dwyer, later described as a pioneer in use of diphtheria serum as a remedy,[108] welcomed the development of the antitoxin and promoted its acceptance by using it in the correct dose.[106[p345–7]]

Figure 1.1: Dr Joseph O'Dwyer (1841–1898)

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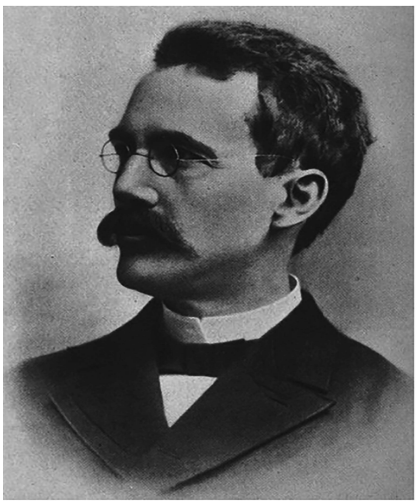


With thanks to Wellcome Images, Biomedical Collection, Wellcome Library of Medical History Collection, London, for this image.[118]

Figure 1.2: New York Foundling Asylum

The Sisters of Charity moved into the Foundling Asylum's original small house at 17th East 12th Street on 11 October 1868, and then in November 1873 occupied the new institution, above, on 68th Street. With thanks to the New York Foundling Hospital for this image.[119]

Figure 1.3: Evolution of O'Dwyer's intralaryngeal tubes

O'Dwyer's diagram entitled 'Tubes and instruments', illustrating the evolution of intubation without providing further identification. From above down are his introducer; an alternative introducer; a row of successively developed tubes, the first four bivalved, and then 'plain tubes' with progressive alterations in size and angulation of their heads; the remainder, also undesignated: an obturator; an extractor; and lowermost, a definitive O'Dwyer tube. With acknowledgement and thanks to the American Pediatric Society for this figure from Trans Am Pediatr Soc. 1896;8:20.[102]

O'Dwyer also developed a successful treatment in 1885 for chronic laryngeal stenosis in adults.[101] Then, in 1891, by using his airway intubating tubes in place of either the Fell face mask or the tracheotomy tube for delivering George Fell's 'FR', O'Dwyer[100] widened the application of IPPV beyond opiate and other poisoning, to include intracerebral disasters (as first reported by Dr William Perry Northrup in 1894[120]), followed by intrathoracic surgery in 1899.[121] The combination, generally called the Fell–O'Dwyer apparatus, came to include five sizes of interchangeable hollow tube heads, the appropriately sized one of which would be threaded distally onto the short intubation tube, which was then wedged into the glottal gap, thereby enabling IPPV without a tracheotomy and disposing of need to have the trachea tied off above a tracheotomy stoma when using Fell's apparatus. By 1896, at age 57, O'Dwyer's thoughts were turning to 'the possibility of a mechanical method of treating pneumonia', for which he had made 'many clinical observations'[103,106[p355],107] and to providing inflating apparatus for supplementing oxygen intake.

O'Dwyer's wife, Catherine Begg, his greatest supporter, had eight children, of whom four survived childhood.[107] After her death in 1888,[105] he slowly seemed to decline into mental and physical exhaustion. Then, 'In the first week of December [1897] thrombosis of certain cerebellar arteries took place and five weeks later death ensued from secondary meningitis'.[103[p122]]

(In a few documents, there are references to O'Dwyer as Joseph P O'Dwyer. On enquiry, in April 2008, the College of Physicians of Philadelphia advised: 'Joseph O'Dwyer, 1841–1898, the inventor of intubation, does not appear to have a middle name. There is also a Joseph Patrick O'Dwyer, 1869–1906.')

1.3 J O'Dwyer and diphtheria

When O'Dwyer joined the staff of the 'Foundling Hospital' (a term Northrup used at times[105]), 'diphtheria of the larynx'—then commonly known as 'croup'[105]—was the leading cause of infant mortality, with a death rate of 40%–50%.[106[p329],107] As yet, toxoid inoculation and even antitoxin lay further ahead. For several years, New York City had deaths from 'croup' annually numbering 700–1000,[98[p560]] and the survival rate was still very poor after about 200 tracheotomies had been conducted. When a diphtheritic airway was acutely obstructed, tracheotomy was the only available treatment to relieve asphyxia but was resorted to only in desperation. One statistic (quoted[111[p1]] without its source being identified) had the survival chances of victims with a tracheotomy, up to the age of 3–4 years, as only one in 10. Worse, from 1873 to 1880, tracheotomy performed in the NYFA's own 'croup room' for diphtheria did not save a single child from death.[98,106[p330]] At best then, in allowing 'the little sufferers to die easier'[102[p10]], tracheotomy provided, in effect, nothing more than what O'Dwyer later called 'a justifiable form of euthanasia'.[102[p10]] Yet, by the mid-1880s, tracheotomy results much better than the NYFA's drastic figures were being described by others; for example, in 1887, Charles Jennings claimed 17 recoveries after tracheotomy for 36 needing it.[97]

In 1879, the airway tubes that O'Dwyer was trying to modify for increasing 'the expulsive powers of the cough' were still solely tracheostomy tubes.[102[p402]] Their failure drove him to find a better treatment; later, he said the 'complete failure with tracheotomy extending over a period of several years [1869–1880] was the real incentive to the work'.[102[p10]] O'Dwyer, essentially a modest man,[106,112] reflected in his later years that he had been able to attack the problem successfully only by learning and developing systematic thinking … outside the beaten track … from the beginning to the end … without borrowed inspiration.[102]

In 1880, he decided to try an artificial oro-laryngeal channel through the larynx'[104[p361]] by designing metal tubes, about an inch long and placed below the glottis within the vestibule of the larynx, to completely tampon off the glottis. Rudolph Matas[122[p1375],123] referred to 'O'Dwyer's epoch-making observations (of an intralaryngeal tube) first published in January 1880'. He did not provide references for that, nor have I located any confirmatory paper. When O'Dwyer referred to 1880 in 1887, it was not in relation to his publishing, but only his 'operating'[97] and experiments.[102] Thomas Keys followed Matas in 1945 with the same '1880' for O'Dwyer's publishing.[124[p65]]

In 1882, William Northrup (1851–1935), then 10 years graduated, joined O'Dwyer as a research co-worker[105] at his own cold corner of the NYFA's autopsy room—'that lugubrious underground cell'[104[p362]]. Until 1882–1883, O'Dwyer was unaware of the mid-19th century failure in France of proposals[52,125] from Jean Antoine Eugène Bouchut (1818–1891) for intralaryngeal intubation (see Addendum 1.1A[52,53,125–129]). O'Dwyer's writings[95,102] indicate that his own intubations, ultimately successful, came from no flash-in-the-pan discovery, but prolonged, systematic, repeated investigation pursued over half a dozen years, combined with much practice on models and cadavers.

1.4 Causes of death in diphtheria

O'Dwyer's interventions could be successful only when the airway obstruction to be relieved was confined principally to the larynx. Even with intralaryngeal intubation, multiple factors could deny him success: pseudo-membrane formation in the airways beyond the larynx (fatally, at the bronchus, 57%; and 37% in the bronchioles[113[p5]]), producing untreatable asphyxia; or slough, coughed up from below**,** and fatally blocking the airway passages or inserted tubes; and disasters occurring with the laryngeal tube itself, during introduction or extraction, or on accidental extubation. The toxaemia of diphtheria, with or without concomitant airway problems, could also be the cause of death**.**

1.5 Development of intralaryngeal tubes and intubation

O'Dwyer's accounts clearly set out his struggles for clinical success during 1880–1885, with many heartbreaking failures,[95,102] and they are supplemented by Northrup's lucid chronological histories of his colleague's progress.[104,105] The writings of both indicate the extent of time O'Dwyer spent at the autopsy room to study airways for anatomical learning and to try out successive experimental designs and modifications, first, of intralaryngeal springs and, later, of tubes, long before conducting 'live' trials.

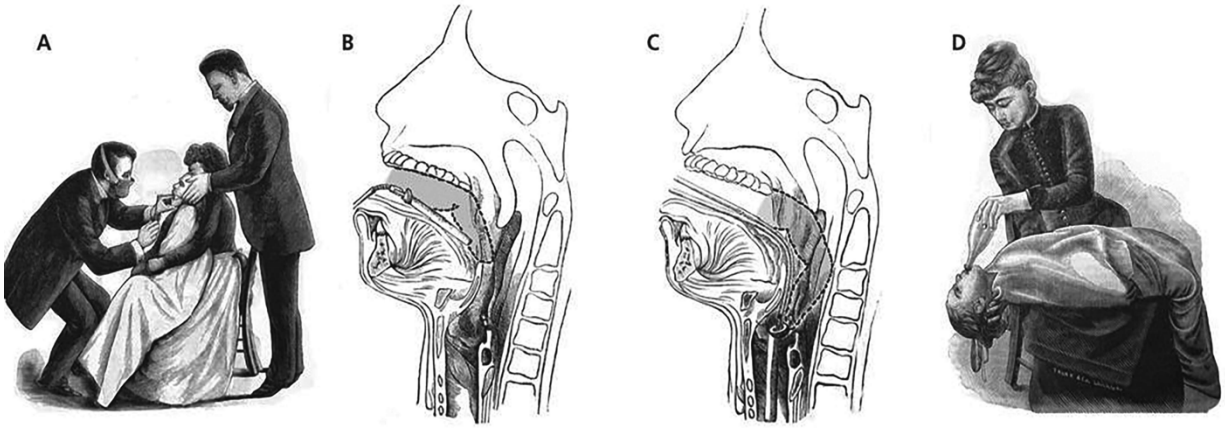
Initially, O'Dwyer's devices rested solely within the larynx.[102[p11]] He made numerous models by moulding putty into specimen larynxes and tried multiple versions of various lengths, shapes, sizes and appendages; and, after his first successes, made a whole succession of modifications to the size and shape of the tube heads to enable swallowing for feeding.[96] The number of tubes O'Dwyer must have had constructed and modified, before reaching satisfactory solutions to multiple problems, does seem amazing.

O'Dwyer started his clinical trials of intubation with a long 'prostatic catheter introduced via the nostril',[97[p318]] but quickly discarded it, progressing to a small, simple, laryngeal 'skeleton wired spring',[104[p361]] which he also soon found unsuitable. Various designs and lengths of laryngeal spring or speculum followed, and a

small bivalved device, which Northrup (and O'Dwyer also) often referred to not as a tube but still as a (laryngeal) spring. Initially, the various spring designs, as well as their introducers, were homemade by O'Dwyer's valued helper, the mechanically gifted house physician, Dr RE Chadbourne[105] whose recognition seems to have gained documentation rather late. The ancillary equipment of introducer (especially), mouth gag and extractor comprised *essential* equipment. After three years of experimentation, O'Dwyer had moved on from bivalved tubes to trying solid' *plain tubes* in 1883 and was conducting numerous experiments with an extensive range of successive modifications.[96] By then, he had expert instrument makers for his metallic designs that, from the beginning, represented a significant advance on Eugène Bouchut's tubes in Paris, 1858, which were cylindrical and short[95]. O'Dwyer illustrated multiple changes[102[p20]] and catalogued the tube-head changes.[96[p685]]

To effect the intubation of a conscious child, O'Dwyer and Northrup needed to use a very careful technique, described first in 1885 by O'Dwyer,[95] then briefly by Northrup,[130] then again by O'Dwyer[98] in 1887, and in considerable detail in 1888 by Frank Waxham,[131] whose remarkable book of the same year[132] had 45 engravings of the technique (Figure 1.4[132]). Gelfand's article in *Caduceus*[111] (a journal now defunct) has a series of historical drawings from the Dittrick Museum of Medical History, liberally illustrating O'Dwyer's careful intubating techniques and instrumentation. The Waxham illustration shows a child blanketed and firmly held for intubation, that is, without the benefit of anaesthesia.

Figure 1.4: Dr Waxham's drawings of techniques[131]



A. Child positioned for intubation, blanketed and firmly held.
B. O'Dwyer tube, held by an introducer, guided past the epiglottis.
C. Forefinger guiding the tube into the larynx.
D. Head-down position for feeding an intubated child.
With acknowledgement and thanks to the Br Med J. 1888, Sep 29;ii:716–9, for Waxham's Figures 7, 9, 10 and 5, respectively.[131]

1.6 Evolution of J O'Dwyer's intralaryngeal tube

1.6.1 The bivalved spring tube, or speculum

O'Dwyer's first tubes shaped for children, which he developed in 1880, were metallic, bivalved and ellipsoid front to back, with a narrow transverse diameter. The two valves were held together until the introducer (designed to guide insertion being performed by finger touch) was removed, when the valves sprang apart to grip the laryngeal walls.[104] 'A great deal of experiment was required to get the spring of the proper strength.'[104[p361]] A loop of braided silk able to run freely through the eyelet of the tube[130] was withdrawn only after the speculum was safely inserted (sometimes the silk was left attached, with the other end fitted around an ear[99[p36]]). An extractor could engage in the small slit in the tube's side. The length of the tube was at first a maximum of only 1.5 inches (3.8 cm) and then extended to 1.75–3.0 inches (4.4–7.6 cm), with a tube-top rubber collar or shoulder[102] of chloroform-softened gutta percha, 'applied layer after layer until a sufficient thickness was obtained'.[102[p11]] It prevented the tube from slipping further down the trachea.

After three years, the bivalved design was 'reluctantly given up as useless'[102[p12]] because, among its multiple flaws, O'Dwyer found swollen mucosa or pseudo-membrane progressively intruded between the separated blades into the airway, again 'obliterating the breathing space first obtained',[102[p11]] and providing 'the greatest difficulty and one that proved insurmountable'.[102[p11]] Soft tissue could also block the head's entry site into the tube, and similar intrusion into the slit for the extractor led to its being abandoned.

1.6.2 Clinical applications of the speculum

From an entry in the NYFA deaths book, the first intubation with a laryngeal spring/speculum was performed in October 1882[104] on a 4-year-old (awake) girl, relieving her asphyxia. However, the removal of the spring later led to relapse of the asphyxia after eight hours, and hence, tracheotomy was performed, but she died the following day from asphyxia caused by pseudo-membrane formation deeper in the lungs—'to finest bronchioles'.[104[p362]]

O'Dwyer did not make much fuss of the November 1882 recovery of a tracheotomy patient, the first since the Hospital's foundation back on 11 October 1869. Yet, that was a real triumph, as every previous 'operation' at the NYFA had ended in total failure. He did not reveal how long the bivalved tube was first in place, before the protracted wearing of a secondary[102] (tracheostomy) cannula for six months, with two laryngeal operations,[102] which eventually enabled final extubation. As laryngeal intubations became more frequent, a policy was adopted that patients who survived initial intubation and were judged to be the most likely to recover would subsequently receive a *secondary* tracheotomy.[102]

Intubations on two further patients failed to prevent their deaths. O'Dwyer performed the NYFA's third documented insertion of one under chloroform anaesthesia on 26 January 1883.[102[p12–3]] Within 10 minutes, the child had to be extubated to allow the extraction of pseudo-membrane that was too wide for ejection through the spring's lumen. 'Apparently dead',[104[p362]] she was resuscitated through artificial respiration (type unspecified) and then re-intubated with a larger spring, but extubated five hours later. Her condition

immediately worsened, but her mother refused to allow the tracheotomy then deemed essential, and the child died. (At this time, it was characteristic of these parents not to want tracheotomy or to give consent.) Springs were then given up. Nevertheless, the self-retaining bivalved tube, once inserted, had been 'always retained and gave prompt but transient relief to the dyspnoea'.[102[p11]] Later, in 1896, O'Dwyer assessed the situation at that stage: 'Tracheotomy was bad, but intubation so far was worse because it interfered so seriously with feeding'.[102[p12]] Understandably, such situations must have been exceedingly disheartening for O'Dwyer, his medical team, the nursing staff and the parents.

Contrary to widespread belief or expectation, these intubations, importantly, did demonstrate that the human larynx would tolerate a tube without damage, and that air could enter the lungs and secretions exit.[104] (Eugène Bouchut had already demonstrated these in 1858.[52,125])

1.6.3 Plain tubes

After three years of struggling with bivalve trials, O'Dwyer 'concluded to try instead a [*solid*] tube of plain oval form', at first small, 'about one inch long',[95[p145]] again with an introducer. The new tube was 'longer than the speculum, narrow, oval, or flattened laterally, having a collar at its upper end'.[104[p362]] The purpose of the collar was to rest on the vocal cord.[106[p334]] Its insertion was aided by the leading rounded head of an obturator, and again, an attached loop of braided silk thread. A tube extractor, improved in the summer of 1886,[102[p16]] could be engaged in a small posterior slit on the tube's top; Waxham later invented a valuable laryngeal forceps.[111[p29]] O'Dwyer stated:

> let us bear in mind that the backward curve, the blunt rounded head of such odd shape, the knobbed lower end, with the thin-walled, laterally compressed upper end (of the tube) were the results of long painstaking study and experiment, measurements and casts, trials, of models changing and changing for six years.[104[p363]]

Due credit must be given to O'Dwyer's instrument makers. Earlier tubes were from his unnamed 'little German' craftsman,[104] and the later ones, from H Keller of New York. Genuine 'O'Dwyer' sets were provided by Geo Tiemann & Co, New York,[130] while Chas Truax & Co, Chicago, manufactured Waxham's modification of O'Dwyer tubes,[132] with rubber epiglottis, which O'Dwyer demonstrated[96] (see Figure 1.5).

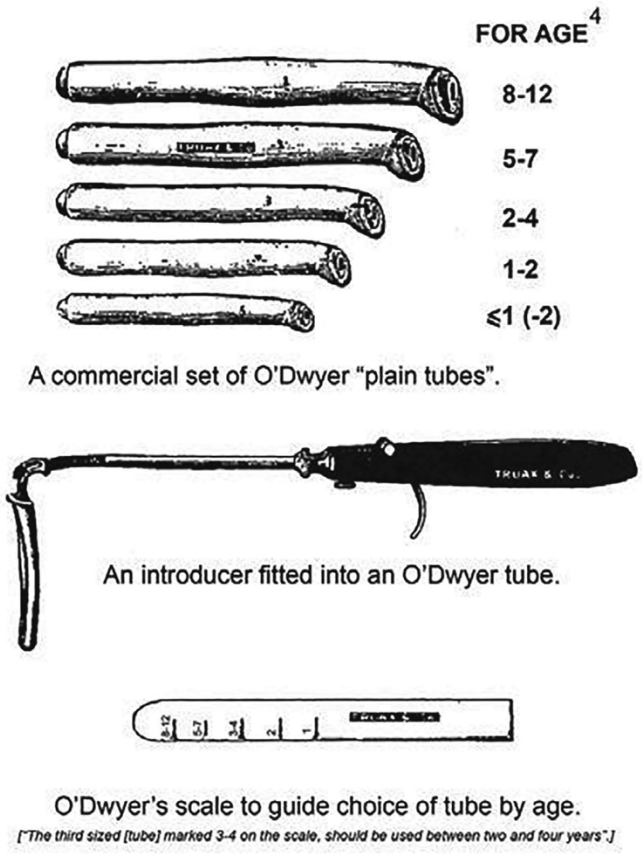
1.6.4 Physical aspects of plain tubes

Retention. An important spindle-shaped 'retaining-swell', finally evolved for the mid-tracheal part of the tube, proved best for retention (see Footnote 1.1[1]).

[1] Footnote 1.1. 'Longer tubes reaching nearly to the bifurcation were kept in place better, but after a cough the long tube ... had to be pushed back with the finger. A **second shoulder*** was then added to the tube, below the expansion or head at its upper extremity. This shoulder kept the tube from being repelled, but the abruptness of its upper border made extraction very difficult. The abrupt shoulder then gave way to a **gradually tapering enlargement***... [and the tube] ... when projected upward by coughing, will slip back into position by the pressure of the vocal bands on the sloping sides, aided by its weight. This **retaining swell** [at mid-tracheal position] is only made to hold the tubes loosely in the larynx, in order to permit of their easy expulsion in cases of sudden occlusion by masses of pseudo-membrane too large to pass through.' (*emphasis added)[133[p318]].

Difficulty in feeding intubated patients.[96] There was much concern over ensuring adequate nutrition. Eventually it was realised that despite various modifications to the tube-head 'no artificial device could overcome the difficulty of swallowing ... [although] ... when a tube is worn in the larynx for several weeks the ability to swallow perfectly was acquired'.[102[p16]] With the tube's head occupying the vestibule of the larynx, the epiglottis could close over it during swallowing.[97] Eventually head-down feeding (or the Casselberry method of 'swallowing uphill'[104,116]) was adopted[102] (see Figure 1.4D).

Figure 1.5: Truax set of five plain tubes



O'Dwyer 'mouth and throat instruments' showing a set of five tubes of graded sizes and an introducer, as well as the scale for choosing the tube for differing age ranges.[98] Although catalogued as O'Dwyer tubes, they were from Truax & Co, Chicago, and hence may have been Waxham modifications.

Pressure ulcerations.[102[p13]] Also troublesome, these occurred especially at the base of the epiglottis and within the cricoid ring (apparently, it was O'Dwyer who recognised that the cricoid had the narrowest calibre in the upper airway[104]). Extensive experimentation enabled the progressive reduction of the calibre of the tube, so much so that everyone marvelled it could still allow satisfactory breathing.[104]

Tube properties. With diphtheritic *distal obstruction* found around the end of many small tubes after death, the tube length was increased to within half an inch of the tracheal bifurcation—such tubes were 'never expelled'.[102[p13]] O'Dwyer started with (nickel–)steel tubes and obturators; the last modification was tubes made of 'hard rubber' (vulcanite), not metal, which proved best for the material of the tube head, concerning incrustation and corrosion. It could be boiled for disinfection, similarly to metal.[104] The initial models had imperfections, but later models were highly refined, wide, short tubes.[102[p.17–8]] ('Loose membrane or foreign body tubes'[116]). O'Dwyer developed short, very thin, metal tracheal tubes, three to four times wider than standard tubes, for extracting or breaking up a *pseudo-membrane*. These tubes, illustrated by Northrup[116[fig.2]] were left in place for only a few hours at a time and 'proved fairly successful in getting rid of false membrane when loose and not anchored below by extension into the bronchi'.[102[p18]]

1.6.5 Clinical applications of plain tubes

Northrup[102[p12]] quoted, 25 April 1884, from the NYFA's deaths book, the outcome of O'Dwyer's first trial with a plain tube, supplied to an infant aged '2 months and twenty-four days', with the breathing state relieved thereby to 'fairly good'. The infant then accepted almost a pint of milk; ease of breathing was 'retained until the child died sixteen hours later, free from any return of dyspnoea'[102[p12]]—presumably succumbing to diphtheria toxicity. The first mention of 'tube' is also made for that date in the same book.[104[p.362]] (Paluel Flagg, closely liaising with a son of O'Dwyer's who was also a medical doctor,[105[p562]] in 1909 and 1934,[110[p12]] states O'Dwyer 'first applied the "oval tube" on 23 April 1884',[110[p9]] but one might expect it to have been on 24 April.[104])

O'Dwyer then recorded on 21 May 1884 that his second patient, a girl aged about four years, was relieved.[102[p12]] She survived 67 hours of intubation and was extubated, but needed re-intubation after a further five hours, for which O'Dwyer had to administer chloroform to withdraw his (shielded) finger, leading him forthwith to construct a mouth gag. After seven days, she coughed the tube out, to become his first survivor with one of his plain tubes. O'Dwyer later hailed this success (rather than their 1882 survivor of intubation) as 'the first recovery in the history of intubation, and was, therefore, a very important event'.[102[p13]] Only after this triumph would O'Dwyer agree to consider operating on patients outside the hospital.[105] The newly coined word *intubation* 'became associated with the operation'.[104[p362]]

None of the next seven children intubated with this type of short tube survived.[102[p13]] For a boy of four years, O'Dwyer's third patient,[95] in whom dyspnoea returned after 24 hours following which he died, the tube did not extend sufficiently into the trachea and its distal end 'was found obstructed by a thick deposit of pseudo-membrane'.[95[p146]] The use of a new tube, without a hole/niche for the extractor to engage, produced only one more survivor from five further patients, in December 1884, and that was after a boy managed 10 days of intubation amid repeated perils to him.[102[p.13]] Thus, 'Intubation was now looking up. It had two recoveries [i.e. survivors] to its credit in five years and the prejudice against it was abating'.[102[p13]]

By 1885, although tubes long enough to reach clear to the [tracheal] bifurcation were placed in six patients, all had died. A further tube with a second retaining shoulder, below the cords for 10 patients, produced one survivor, 'the third recovery since the beginning of the experiments'.[102[p14]] These tubes were never expelled from the larynx. Multiple further design changes to deal with serious problems of feeding, ulceration sites and tracheal casts of membrane all took long periods of study and experimentation before O'Dwyer could publish two major lists of 50 patients in 1887[98] and 1888.[99]

1.6.6 Adult plain tubes

O'Dwyer could later state[102[p18]] 'at this time [December 1885] there was no such thing as an adult intubation tube' (incorrectly, actually; see Addendum 1.1B,[54,55,134,135] regarding William Macewen). But two years later, as published in vol. iv of the *Transactions of the 9th International Medical Congress,* Washington, 1887,[101[p1481]] he presented an 1885 patient, an 1886 one and other cases of treatment of adult chronic (syphilitic) stenosis of the larynx. After much careful study and measurement, his first adult set was constructed in multiple sizes for a woman with such a narrowing, but none of these tubes would penetrate the stenotic scarring, and even the largest of tubes for children was inserted only with traumatic difficulty. After the first sequence of laryngeal intubation followed by later extubation for this woman, a process repeated eight times during 18 days altogether, he increased the spells with successive indwelling tubes of progressively increasing size, enabling lengthening intervals between periodic reinsertions, such that he could report in 1894 (at Bristol, UK, where Brook referred to his own five cases[101[p1482]]) that by then, her freedom from re-stenosis had lasted five years.

By June 1887, O'Dwyer had intubated another woman 'over fifty times, leaving [the tube] in the larynx on several occasions as long as a week at a time';[96[p686]] further, another woman wore a tube continuously for more than 10 months. 'Other cases of chronic stenosis followed in quick succession. On the experience thus derived, the set of [10][101] adult tubes was constructed.'[102[p18]] The tubes were originally made of metal and then of hard rubber,[96] and were used later for the Fell–O'Dwyer apparatus for IPPV and anaesthesia (to be discussed in Chapter 3 of this North American series).

1.7 Further documented clinical applications

1.7.1 1880–1885: O'Dwyer's experiments with intubation

O'Dwyer reported his agonising 'experimental stage of intubation', from January 1880 to December 1885.[98[p557]] (Despite naming his starting date as January 1880, he wrote in August 1885 that he *'began experiments, almost five years* ago',[95[p145]] that is, not before August 1880.) During this whole *initial* period of developing and perfecting the tubes and accessory instruments,[99] O'Dwyer reported,[98,99] 'I operated on sixty-five cases of croup, sixty of them being in the New York Foundling Asylum, and had but nine recoveries, or not quite 14 per cent'.[98[p558]] Some died 'without any marked cyanosis' and others died with 'evidence of toxaemia' which had 'existed for some time'.[98[558]] O'Dwyer also reveals records missing: 'there is a break ... extending from August 1883, to April, 1884, which I cannot account for'.[102[p.12]]

After his third patient received a plain-tube insertion, referred to above, O'Dwyer admitted feeling obliged to present, and then to publish (in August 1885), his design of plain intubating cannulas, despite not wishing to do so without 'a greater degree of perfection'—as, with only one of three child patients surviving, and that notable success a single one after inventing 'tube after tube',[39[p158]] he assessed his work as still of a purely experimental character. Mushin and Rendell-Baker stated[39[p.158]] O'Dwyer's work was 'not recorded until a history of the Foundling Hospital was written in 1884'. Waxham later summarised his own parallel progress during 1885[136] (see Footnote 1.2[2]).

1.7.2 1885–1886: O'Dwyer's first 50 intubations in private practice

Between 5 December 1885 and 6 December 1886, O'Dwyer performed 50 intubations in a full year's private practice.[98] Survivors totalled 12 (24%), with their tubes averaging 5.3 days *in situ.* O'Dwyer supplied some specific case details, with the cause of death in 18 of the 38 considered an untreatable extension of the membrane to the bronchi, although no autopsies were performed. O'Dwyer's excellent, explicit paper clearly defines the skill needed for attempting intubation, and the seven dangers he identified with that procedure; his own expertise becomes most evident. He emphasised that in his practice he had 'never resorted to intubation until the symptoms of laryngeal obstruction were so urgent as to plainly indicate impending suffocation, unless the child were relieved by operation'.[98[558]] For another dyspnoeic four he was called to, he held off operating—and they survived.

Northrup analysed NYFA autopsies after laryngeal diphtheria and reported on 11 December 1886 on 87 recorded deaths of which in 56, the diphtheria began in the larynx. By the next June, he reported there were 20 more deaths, all from laryngeal diphtheria.[137[p.686]]

1.7.3 1886–1887: O'Dwyer's next 50 intubations in private practice

O'Dwyer reported his next 50 intubations in private patients, between 16 November (although possibly December) 1886 and 18 November 1887.[99] These patients were operated on, usually in consultation; on three occasions, O'Dwyer alone performed the intubation. Tubes were maintained *in situ* for 12 hours to four days; the first 15 patients died (how daunting that must have been), but overall, 15 survived, giving a 30% success rate. O'Dwyer details two severe cases for whom he considered he could withhold intubation,

[2] Footnote 1.2. Frank Waxham's 1901 article in the *Journal of the American Medical Association* with fascinating reminiscences[136] acknowledges the courtesy of O'Dwyer in providing instruments for Waxham to attempt intubations, at a time when the design of tubes was still crude and primitive. After first demonstrating a cadaveric intubation to the Chicago Medical Society on 20 March 1885, Waxham made live attempts. The first three patients (on 19 April, 23 April and 16 July) did not survive; but following five days of intubation from 15 September 1885 (less than 16 months after O'Dwyer's first success), he had his own first success, and then, by November, four recoveries out of 11 intubations. This early parallel work by Waxham in 1885 appears to be barely recognised in the literature (apart from by Gifford, for instance[109[p183]]). His engrossing paper clearly details the hazards amid which such pioneers worked—being reviled, threatened, attacked and needing to go armed when attending suffocated children.

who recovered after an emetic of turpeth mineral and bichloride of mercury (see Footnote 1.3[3]). One of these was also given steam vapour. O'Dwyer presents clinical details of seven patients severely ill and of several accidents, and he details the third of three instances of complete obstruction of the tube by a large tracheal cast, which resulted in sudden apnoeic death. Two out of 173 cases had their coughed-out tube enter their stomach. This 'analysis' paper describes a Heimlich-type innovative manoeuvre for expelling intra-laryngo-tracheal material when a tube prevents glottic closure.[99]

1.7.4 1885–1889: Reports to the Academy of Medicine of New York

O'Dwyer and Northrup were frequent contributors to meetings of the Academy of Medicine of New York, with documentation (including discussions) in New York's *Medical Record* and *Medical Journal*. O'Dwyer's expertise, and especially the intubation versus tracheotomy dilemma, attracted widespread interest, with commentators in the US from places as distant as California.

1.7.5 1894: Northrup, O'Dwyer and the British Medical Association

In July 1894, after almost a decade of providing treatment, Northrup and O'Dwyer presented their methods at the 62nd Annual Meeting of the British Medical Association in Bristol, UK. They spoke to the 'Sections of Laryngology and Otology and Diseases of Children' on laryngeal obstruction: Northrup on acute[116] patients and O'Dwyer on chronic.[101] Northrup reported the risk of inadvertently pushing down diphtheritic membrane during intubation and mentioned three occurrences during O'Dwyer's first 209 cases. (There, he also announced the first use of the Fell–O'Dwyer apparatus,[120] to be discussed in Chapter 3.)

1.7.6 1896: O'Dwyer's address to the American Pediatric Society

On 25 May 1896, two years before his death, O'Dwyer provided a detailed retrospective on his entire evolution of intubation in his presidential address to the 8th Annual Meeting of the American Pediatric Society.[102] Although others had intubated the larynx on sporadic occasions previously (well described by William Macewen[127]), O'Dwyer was the first to systematically develop a method he then used successfully on enough patients to establish its validity in the saving of lives.

[3] Footnote 1.3. (a) Turpeth mineral is mercury subsulphate ($HgSO_4.HgO$). (b) Bichloride of mercury ($HgCl_2$) is also known as mercury (II) chloride, mercuric chloride and corrosive sublimate. (c) At that time, before the severe toxicity of mercury was recognised, compounds of mercury were used for all sorts of diseases and conditions. (d) In his publications, Northrup[116] refers to the use of calomel (Hg_2Cl_2) also known as mercury (I) chloride and mercurous chloride. It was administered from a fumigating lamp in a closed cribtent. Its use, first publicly advocated by Dr. JC Corbin of Brooklyn, in 1881, was later taken up by Dr O'Dwyer in New York, and steadily gained favour. Northrup told the British Medical Association in 1894 that it allowed better and speedier recovery when used after an operative procedure, and quoted its successful use in hundreds of patients, but he still cautioned that the statistics available were insufficient to frame a strong argument. Later, in 1904, Northrup cited[105] calomel for promising early results for croup-laryngeal diphtheria and stated that it was thought to reduce mortality from 75 to 40%. I feel ambivalent about whether sufficient evidence was provided for calomel's real worth in laryngeal diphtheria.

1.8 Problems associated with intubation and tubes

O'Dwyer needed to perfect a 'blind' technique[95] for the intralaryngeal intubation of a conscious, terrified child, and then ensure the tube's maintenance *in situ*, which was usually needed for days. But other problems needed attention: determining an optimal design for tubes and obtaining the best material for fabricating 'tubage', as well as addressing those difficulties so well known in intensive care practice—keeping the tube's lumen patent; having the airway below the cords sealed off to enable feeding without aspiration; preventing self-extubation and ensuring the retention of the tube while still enabling its ready removal; providing humidification; plus coping with obstructive membranes coughed into the tube from the trachea and beyond. a feature specific to diphtheria.

1.8.1 O'Dwyer's personal problems and difficulties

Craig Gelfand's generously illustrated, fine account[111] describing the many battles O'Dwyer faced makes an interesting read, as do Northrup's two valedictions.[104,105] O'Dwyer's studies persisted with dogged tenacity,[122[p1375],123] despite disheartening clinical failures and heavy criticisms. Even when he could invoke his first few successes, he found initial refusal to accept his new method of treatment, or worse, opposition, not only from many colleagues but also among some NYFA staff. From Sister Rosalie, and the 'famed superior', Sister Irene,[106[p341,348]] came only support. To many, it seemed that the insertion of his tubes was torturing children already in severe pain from asphyxia and fearful of intubation. Some children were taken from the hospital to insalubrious homes to avoid it, and some 'out-children were not returned to the hospital for treatment'.[102[p12]]

Difficulties arose from strong professional scepticism or antagonism. Initially, experienced US tracheotomists, such as Dr Charles Jennings (who emphasised the 'personal equation' factor[97]) and Dr Max Stern,[138] could present, with moderation, arguments for and against intubation,[97,115] while seeing tracheotomy as equally or more effective. But Waxham, strongly advocating O'Dwyer's methods, modified O'Dwyer's tubes and reported his tube version as saving 269 lives after 1,000 intubations.[97]

O'Dwyer's drive for intubating came from the absolute failure of tracheotomy at the NYFA; yet, others were much more successful (see Footnote 1.4[4]). Controversy also ensued in Australia (see Addendum 1.2[134,139–145]), and Europe.[115] Meanwhile, attempted intubations by many unskilled practitioners brought discredit to O'Dwyer's method and blame for their failures reflected back onto O'Dwyer.[105] Cheap, carelessly imitative tubes in America and Europe (some with horrifying modifications[111]) brought all the problems he had spent a decade struggling to circumvent. Northrup was appalled to find (as Jeffreys Wood confirmed[143])—at O'Dwyer's request—that in the absence of a patent on O'Dwyer's tubes,[104] he could simply pick up in

[4] Footnote 1.4. Charles Clubbe[139] (presenting his own recovery rate of 42.5% for 120 tracheotomies) quoted Konigsberg's Surgical Clinic,1878–1882, for 49% recoveries from 123; Rankin at Munich reported 63% for 54; and Caselli, 72% for 18. Jennings had 17 recoveries with 36 tracheotomies, but none with 12 intubations.[97] Waxham[131] collated 1,072 intubations in the US, compared intubation versus tracheotomy by age groups and found that the highest intubation recovery rate was in children aged less than two years, with 15.6% surviving, versus Max Stern's 3% of this age group surviving after tracheotomy.[138] Stern advised intubation was always preferable for children aged less than 3½ years. Previously, for example, in the 1860s, 'English operators, unlike the French, continued to consider tracheotomy only as a last resort ... until death from apnoea was imminent'.[115[p544]] Despite their exploring O'Dwyer's methods, in their hands 'results were not encouraging'.[115[p547]] Anne Hardy provides a masterly account of tracheotomy versus intubation in the US and Eurpoe.[115]

London and different places samples of tubes sold by their best makers, and tubes made from models sent out from New York with O'Dwyer's approval; these tubes were a travesty and embodied every vice.[104] Some doctors would rush to buy an instrument set and try to insert a tube without any of the training on cadavers needed to become experienced, because of which they had all manner of accidents.[105] O'Dwyer had warned in 1887[97] that intubation may seem, but is not really, a simple operation. (In 1887[97[p31]] and again in 1896,[146[p136]] Northrup appeared to discount this caution; certainly, he failed to confirm it.)

Patients outside the NYFA could be sent there for O'Dwyer's personal management. But when an intubated child died by asphyxia from extension of the diphtheritic pseudo-membrane peripherally into the bronchi and beyond, many opponents were ready to blame the death on the tube or on O'Dwyer himself. The inexperienced had deaths during rough intubations, and also when extracting tubes, tubes could become obstructed. Accidents could happen with the tubes as may occur today. Nurses minding children's tubes became skilled, but they were not today's all-round trained intensive care nurses. One argument for advancing intubation over tracheotomy was that the former was more readily managed in a poor household! Thus, in 1887, Max Stern recommended intubating poor patients when they cannot obtain suitable nursing for a tracheotomy.[138] O'Dwyer's temperament made all setbacks personally devastating and debilitating, yet Gelfand considers him displaying 'a resilience and strength uncommon to most'.[111[p2]]

1.9 Declining need for intubation

1.9.1 Diphtheria antitoxin and immunising vaccine

Ironically, O'Dwyer's widest recognition, medical and lay, was eclipsed when the need for his tubes in diphtheria decreased or was even eliminated with improvements in the antitoxin. Diphtheria antitoxin, announced in Berlin in 1890 by Emil von Behring (1854–1917, the first Nobel Prize winner for Medicine in 1901) and Shibasaburo Kitasato,[113] was taken up enthusiastically by O'Dwyer himself, once it became available (the supply came from horses). Pierre-Paul Émile Roux of Paris also developed an effective diphtheria antitoxin independently and modified treatment, in 1894.[147] The antitoxin concentration in the first available doses was too low, at 50–100 antitoxin units, when 2,000–3,000 units were needed, causing many to repudiate it, but O'Dwyer persisted.[106[p346]] Antitoxin came to reduce the need for O'Dwyer tubes, even more so after Roux's nephew, Gason, effected the conversion of diphtheria toxin to toxoid, enabling the development of an immunising vaccine, followed by successful trials, in 1924.[113[p7]]

Waxham documented[136] the recovery of 27 of his first 100 intubated patients and 34, 40, 38 and 39 of a successive series of 100 patients (i.e. 332 deaths in 500 patients, a mortality rate of 66.4%) but only four deaths in his last 70 patients who were give antitoxin as well (a mortality rate of 6%). The distinguished 'virtual founder'[147,148] of American paediatrics, Abraham Jacobi *(*1830–1913), O'Dwyer's zealous convert to intubation, found that antitoxin use lowered the need for 'operation' (presumably intubation) in laryngeal diphtheria from 90% to less than 40%.[115[p548]]

1.9.2 Recognition for O'Dwyer's innovations

Eventually, O'Dwyer came to receive full recognition and honour, initiated by Abraham Jacobi at the (New York) 'State Medical Society, February, 1887'[104[FN-p363]] and then wider afield within the US, much of continental Europe, the UK (slowly there, mainly 'due to the faulty construction of the instruments that are made in England, after the pattern of O'Dwyer's earlier tubes' [143[p177]]) and Australia. The ovation O'Dwyer received at the IXth International Medical Congress, Washington, September 1887 (in such contrast to George Fell's reception there[149]) was for his efforts in developing his intubation system.[104] By 1894, at a Nuremberg research conference his intubating method could be credited with a recovery rate of about 40% for 1,324 diphtheria patients.[112] Two years later, he was the president of the American Pediatric Society.[102]

1.9.3 Widespread use of O'Dwyer tubes in North America and Europe

Reports of intubation used in diphtheria by other American clinicians were soon documented in issues of New York medical journals published in the later 1880s. O'Dwyer's influence continued into the 20th century, and he inspired the use of intralaryngeal tubes for diphtheria, as documented,[105,115] and its awareness seemed worldwide. How long O'Dwyer's own tubes continued in use for diphtheria is less clear. Anne Hardy has usefully documented their employment. They were widely used in North America (e.g. at Boston City Hospital, where, 'by 1900, primary intubation had entirely replaced tracheotomy'[115[p548]]); in European cities, such as Zurich,[109[p185]] Naples, Budapest, Munich and St Petersburg[105]; and also in Scandinavia.[115] However, in 'unconvinced'[115[p548]] European countries, such as Germany and Austro-Hungary, their usage was less until antitoxin 'helped make intubation rather than tracheotomy the preferred practice in Europe'.[115[p548]] Meanwhile, in England, tracheotomy had long remained a continuing option. Hardy also considers national medical cultures[115[p551]] when discussing the question: 'Why did American physicians adopt intubation [for laryngeal diphtheria] so swiftly, the Europeans more slowly, and the English hardly at all?' In the US, the O'Dwyer-type paediatric intubation sets were commercially available in 1920,[111[fig,p28]] and considerable use in diphtheria was documented in Melbourne in the mid-1920s[145] (see Addendum 1.2). For surgical anaesthesia, others used O'Dwyer's design of a longer tube developed for IPPV,[100] as will be discussed in Chapter 3. (The further detailed development into the 20th century of endotracheal tubes, with inflatable pilot cuffs, etc., is beyond the scope of this chapter but is neatly summarised in Alfred Lee and Atkinson's compilation *Synopsis of anaesthesia.*[150])

Eventually, during the 1920s, Ivan Magill (1888–1986) and Stanley Rowbotham (1890–1979), after initially using separate narrow tubes for inspiration and expiration, gradually developed a single-lumen, wide-bore, endotracheal tube for bi-directional gas flow, and it became the dominant form used for anaesthesia.[151] The same principle of a wide-bore tube had been introduced in Germany two decades earlier, by Franz Kunz in 1901, but this was largely overlooked.[151] Although O'Dwyer, supported by Northrup, was hailed for introducing his tubes into the intensive therapy of severe airway disease (i.e. croup and associated diseases[95]), he was also a pioneer in providing a means for the efficient application of Fell's FR principle in rescue IPPV.[120,146] In the US, others adapted this method for anaesthetic IPPV.[122,123] The Fell–O'Dwyer apparatus offered a workable solution for the great pneumothorax problem of thoracic surgery,[121] as will be discussed in Chapter 3.

1.10 Priority rights for intubation: Eugène Bouchut, William Macewen and Joseph O'Dwyer

> *'In science the credit goes to the man who convinces the world, not to the man to whom the idea first occurs.'*
>
> Francis Darwin,
> *Eugenics Review*,
> 1914.[152]

After the mid-19th century, these three pioneers (Bouchut, Macewen and O'Dwyer) each tackled the problem of diphtheritic *acute* stenosis of the larynx by blindly using a finger technique to insert a tube into the larynx, and perhaps the trachea also. Thus, the device O'Dwyer employed for his solution had been anticipated by two previous investigators[52,125–127,135] (see Addendum 1.1). Although Eugène Bouchut demonstrated in 1858 that a child's larynx could tolerate a metal tube for at least two days without damage to relieve acute laryngeal obstruction,[52] he faced stern opposition,[52,53,129] and, having lost his credibility, did not persist sufficiently to have his method accepted. Moreover, although Sir William Macewen at Glasgow successfully intubated the larynx for membranous croup in the 1870s,[134,135] he did not publicise this achievement widely or take steps to ensure its continuing uptake. He did intubate the adult larynx afterwards, using a skilled blind technique, but did not proceed further after three successes.[54,55,126,127]

O'Dwyer, often acclaimed with phrases such as 'immortalised by intubation'[105[p561]], and whose intubation success was considered his undisputed monument, has priority rights in terms of Francis Darwin's yardstick: he firmly popularised the method. Unlike Bouchut and Macewen, he continued undaunted, despite years of failure and criticism, until his method became established. Fine commercial *sets* of O'Dwyer and O'Dwyer-type tubes were used worldwide (components other than solely the tubes were essential), and apparently, his lessons (blessings[112]) applied even as late as the great diphtheria epidemic in Central Europe in 1945-46.[112] Macewen's meticulous writings refer to 19th century mid-European treatments,[126,127[p164]] by practitioners such as Friedrich Trendelenburg, Leopold Schrötter and his pupil, Wilhelm Hack, of *chronic* stenosis of the larynx, usually syphilitic and in adults, by dilatation with tubes. Following soon after, it was again O'Dwyer who effectively *publicised* his successful method of such treatment.

1.11 Postscript

In the extensive reading of O'Dwyer's published papers necessary for the preparation of this article, I do not recall seeing mention of the post-diphtheritic paralysis that can occur after the diphtheria has been thought no longer operative. This condition can affect the intercostal muscles of respiration, to affect the damaged heart further. The equipment of the times for positive pressure breathing may have been required, and McKim Marriott described using that of Gesell and Erlanger, at the suggestion of Dr Erlanger. It provided support to an affected girl aged 10 years, as needed for her weakened breathing for much of five days, until the paralysis dissipated. The apparatus used has been described in *Proceedings of the 26th Annual Meeting of the American Physiological Society*, 1913.[70,79]

Addendum 1.1: Further on priority rights for the introduction of intralaryngeal intubation

A. Jean Antoine Eugène Bouchut (1818–1891)

Figure 1.6: Dr Jean Antoine Eugène Bouchut

Jean Antoine Eugène Bouchut, Parisian paediatrician and 1858 laryngeal intubator for diphtheritic obstruction of the airway. With thanks to the Wellcome Library, London, for this image.[153]

In Paris, on 14 September 1858 the young paediatric physician Eugène Bouchut, *prof.-agregé* (associate professor),[52] addressed the Academy of Medicine on the trials he conducted with a silver, truncated, hollow cone, a little smaller than a common thimble'[52] (18–24 mm long; 6–15 mm wide):[125] with an introducer, he placed it 'on the point of a hollow sound'[126,127[p164]] into the larynx, first in a cadaver, and then for two children affected with diphtherite.[52] 'At the upper part of this tube there were a pair of rings, between which the vocal cords were supposed to rest and hold it in place'[106[p.338]] (a tube is illustrated by Gelfand[111[p7]]). An attached silk thread hangs out of the mouth.[52] The tube dilated the airway, to the great relief of the child, from paroxysms of suffocation.[52] After successful intubations in two diphtheritic children, one for 36 hours, the other for 42 hours, the cones were subsequently removed with the greatest ease.[52] The first child (although '*elle était guérie du croup* [she was cured of croup]'[125[p1161]]) did not survive the toxaemia and pneumonia. The *Lancet* of 2 October 1858, while regarding her death as a failure of Bouchut's method (If she truly had been cured of the croup problem, then surely the *Lancet* saw him unfairly?), did accept he had proved his laryngeal cone could be tolerated and could relieve the suffocation of diphtherite[52] (see Footnote 1.5[5]).

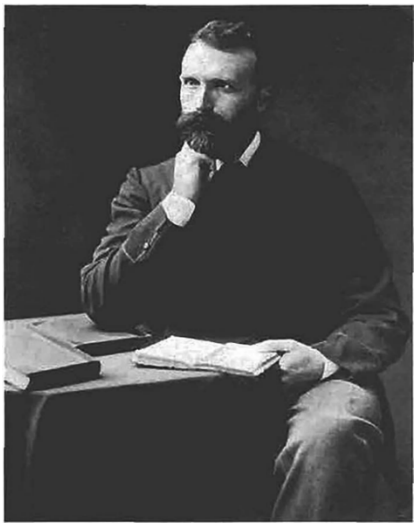
[5] Footnote 1.5. Nearly 30 years on, in New York's *Medical Record* of 15 January 1887,[128] an (anonymous) annotation on the early history of intubation of the larynx referred to the investigating committee's report as delivered in Paris two weeks after 14 September 1855, a year Gelfand also quoted, which surely must be a typographic error for 1858, the year of Bouchut's publication[125]; other writers concur for 1858. The Rapport of the investigating committee's decision,[53] released in Paris at the end of September 1858, obviously reached London for publication in the 2 October *Lancet* (Paris and London had a telegraph link from 1852).

Bouchut advised his colleagues that adopting his cone for intubation in (acute) laryngeal stenosis would avoid tracheotomy, but they wanted a committee to investigate his claim first. After two weeks of consideration, the commission, with the renowned tracheotomy exponent Armand Trousseau (1801–1867) as secretary, reported[53,128] that despite intubation having some virtues, tracheotomy was decidedly the 'principal' option of choice. With Bouchut's initiative 'quickly suppressed by the French Academy of Medicine at the instigation of Trousseau',[115[p547]] the older operation [tracheotomy] continued to dominate in France.

Further, in discussion following, Bouchut was most virulently and ungenerously attacked by others, as was his character and honour. In fact, his career was almost ruined, but he won a name in paediatrics through force of character and perseverance.[128] Not until 1882–1883 did O'Dwyer learn of the earlier failure of Bouchut's attempted promotion of intubations in Paris. Macewen states that of Bouchut's seven patients, five died and two were saved, but that was only by interposing a tracheotomy.[126,127[p.164],106[p.338]] It is not clearly stated whether these were patients of 1858, but when Bouchut reminisced in New York in 1887 concerning three recoveries in 10 paediatric cases, he indicated that they were the 1858 cases.[129] O'Dwyer, always insisting on finding his own way uninfluenced, steadfastly refused to study Bouchut's earlier tube: All he knew was that the tube was like a thimble and was never pronounced a success.[104]

B. Sir William Macewen (1848–1924)[134]

Figure 1.7: Sir William Macewen



William Macewen, renowned surgeon who, early in his career, 1870s, intubated the trachea for diphtheritic 'croup'.
With thanks to the Wellcome Library, London, for this image.[154]

William Macewen, famous surgeon and neurosurgeon, is also renowned for the four laryngeal intubations in Glasgow, which he labelled Cases I to IV (the first three in 1878, the fourth before July 1880, and all thereby before O'Dwyer's interventions), when he used orotracheal intubation for adults.

- In **Case I**,[54,126,127] he also packed off the pharynx for major oro-linguo-pharyngeal surgery: 'to occlude Haemorrhage from Larynx, and for administration of Anaesthetic', with postoperative

extubation 'when the haemorrhage had ceased and the patient regained consciousness'.[126[p122],127] He repeated this intubation similarly to successfully treat two other patients with acutely obstructed airways,[54,55,126,127] after which he recorded no further intubating attempts.

- In **Case II**,[54,126,127] concerning a man's glottis burnt from a hot potato, the tube was in situ during 36 hours, which included three intromissions, and was removed after 39 hours.[126[p123],127]
- In **Case III**,[54,126,127] one of 'acute oedema of the glottis following chronic laryngeal affection', the tube was *in situ* for 30 of the 35 hours total time, with temporary removals made every 12 hours.[126[p123],127]
- in **Case IV**, the surgical candidate had agreed to tube anaesthesia but removed the pre-inserted tube before tube anaesthesia could start; then, about 15 minutes after the house surgeon started a chloroform inhalation, this individual died suddenly.[126,127[p163]] Macewen's documentation[126,127] of each event in the *British Medical Journal* is very detailed; those in the *Glasgow Medical Journal*[54,55]are brief. He also gave careful reference to Bouchut's and others' earlier efforts, and cited two patients observed receiving Desault's nasotracheal intubations (1790s-type), with one survivor. Macewen regretted that learning opportunities for others did not follow on from these efforts. He also cited Dr Wilhelm Hack's patient at Freiburg, November 1878, who was relieved after an hour's *self*-intubation for acute on chronic 'oedema glottidis'.[126,127[p164]]

Neither Macewen's two papers reported to the Glasgow Pathological and Clinical Society in 1879 nor his fine two 1880 *British Medical Journal* articles mention diphtheria patients. But Dr James[134] informs us as follows: first, it was earlier, when Macewen was the medical superintendent of Glasgow's Belvidere Fever Hospital and faced with diphtheritic obstruction of the glottis, that he most probably formulated his ideas about laryngeal intubation as an alternative to tracheotomy, and that 1870 was the year when he was most active in this field of experimental work (preceding even O'Dwyer). From the title, 'The surgical treatment of croup and diphtheria by the introduction of tubes into the trachea through the mouth',[135] with which the *British Medical Journal* very briefly reported on Macewen's paper delivered to the International Medical Congress in London in 1881, there should be no doubt of his intubating earlier for diphtheritic laryngitis.

However, case numbers are not supplied, and as James says, 'Unfortunately, we do not know how many cases [of diphtheria] were treated this way [intubation of the larynx instead of tracheostomy]'.[134[p237]] Worse, ambiguous interpretation is possible (see Footnote 1.6[6]), even to suggest that Macewen had performed the procedure only *once* for membranous croup. Hence, although Macewen's 1881 London presentation may indicate he used intubation for that disorder in Glasgow, thereby also anticipating O'Dwyer,[134[p238]] more detailed evidence would be welcome. In addition, while Macewen does not appear to have persisted with treating infectious disease problems during his illustrious surgical career, neither does it seem his intubation writings[126,127] refer back to his using intubation for croup. James's article[134] illustrates Macewen's various tubes.

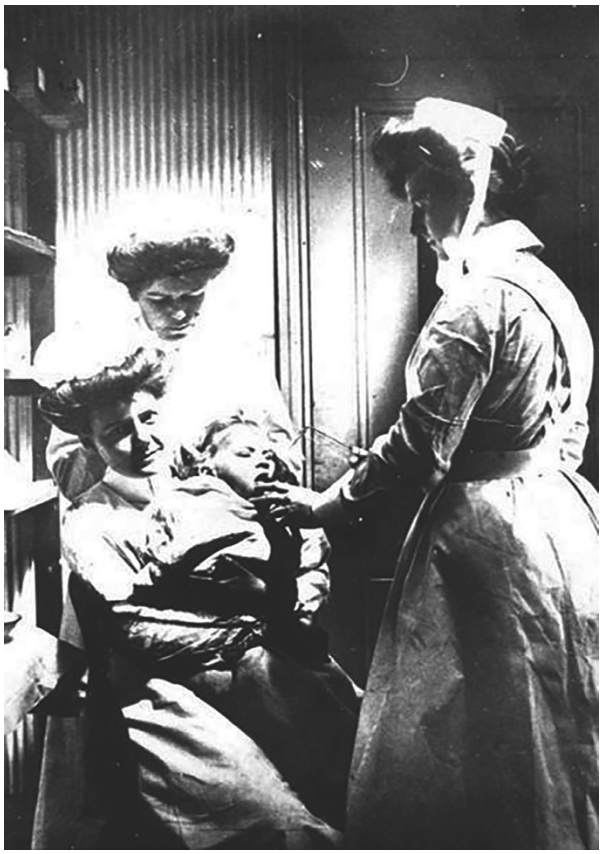
[6] Footnote 1.6. Consider: 'Dr Macewen related several cases in which he had introduced flexible [metallic, articulated] tubes into the trachea through the mouth, and gave details of one case of membranous croup in which their use had been attended by marked success. He showed a flexible silver tube, and some gum-elastic tubes; the latter he found the more satisfactory. Dr Robertson [of Glasgow] had seen some of Dr Macewen's cases, and ... had seen the fourth case and he found that the breathing took place freely through the tube'.[135[p523]] Was this fourth case Macewen's only one of croup, or was it the fourth case of all those he presented at London?[135]

Addendum 1.2: O'Dwyer tubes in Australasia

Dr Hacon, in 1889 in New Zealand, in his enthusiastic evaluation of his single laryngeal intubation for diphtheria,[140] seemed unaware of O'Dwyer tubes. Yet, in Australia the first publication[141] at the same time as the O'Dwyer-type intubation in diphtheria was proceeding, could be that from September 1890, featuring a Dr Hales with 38% success after 100 intubations—which Charles Clubbe deemed a bad record![141] In the same publication,[141] Lennox Browne claimed many successful cases, and Newmarch detailed his single case.

Controversy over the treatment of diphtheritic obstruction continued between two surgeons: intubation protagonist Jefferis Turner[142] at Brisbane Children's Hospital, and Charles Clubbe[139] at the Hospital for Sick Children (later the Royal Alexandra Hospital for Children), Sydney, favouring tracheotomy. At Sydney's 1892 Intercolonial Medical Congress of Australasia, Clubbe[139] presented recovery for 42.5% of 120 diphtheritic children needing tracheotomy, and Turner[142] presented his seven recoveries among 19 intubated patients (but among the dozen deaths, each of his last seven patients died).

Figure 1.8: Nurses at the Royal Alexandra Hospital for Children, Sydney



Nurses at the Royal Alexandra Hospital for Children, Sydney, attending a child with an O'Dwyer tube. With thanks to Michael Cooper of the Australian and New Zealand College of Anaesthetists, for supplying the photo taken by an unnamed photographer. The year in which this photograph was taken is unknown, but it is likely to have been taken in the early 20th century.[155]

Michael Cooper[156] (historian, Australian and New Zealand College of Anaesthetists) wrote to me in 2007 that Turner is supposed to have used the O'Dwyer tubes but does not actually mention it in his article, but then, neither does Clubbe name his tracheotomy tube, although I note in Turner's report[142] six possible indicators that he used O'Dwyer tubes.

Cooper advised that opinions (and therefore practice) differed significantly between the different states in Australia. Some authors presented balanced viewpoints, stating that intubation can always be followed by tracheotomy, if advisable,[142] and that tracheotomy and intubation should never be regarded as rivals, given that both are very valuable operations[143] (tracheotomy could provide valuable access for bronchial toileting). Wood[143] provides a thoughtful analysis.

Rod Westhorpe (historian, Australian and New Zealand College of Anaesthetists)[144] cites Scholes at the Fairfield Hospital, Melbourne, reporting a series of patients with laryngeal diphtheritic obstruction being treated there in the 1920s still with O'Dwyer tubes.[145] At the time of his book's second edition in 1927, a series totalling 1,127 patients had 16.3% non-survivors, compared with 58.7% deaths after 92 tracheotomies for the same condition. Scholes was taught the use of O'Dwyer tubes by Jefferis Turner, who was taught by O'Dwyer himself.

19th century pioneering of intensive therapy in North America

Chapter 2: George Edward Fell

It is no exaggeration to say that George Fell was the pioneer of long-term ventilatory assistance.

Michael Goerig, Filos and Ayisi[157]

Chapter 2: Table of Contents

2.1 Introduction

The beginnings of ICM during the early 1950s lay in meeting the need for ventilatory support in life-threatening impairment of breathing. The foundation of that service featured twin interventions: endotracheal intubation and IPPV. However, already well over half a century earlier during the 1880s, Joseph O'Dwyer and George Edward Fell identified the need to supply such interventions. They devised, introduced and further developed equipment for the purpose. That was three decades after Marshal Hall's 1856 strictures against *forcing methods*, in which bellows were used for AV, had led to these being virtually abandoned, while too often, various arm–chest manoeuvres used instead proved inadequate. After physician and engineer Dr George Fell of Buffalo, New York (1849–1918), failed to save the life of an opiate-poisoned patient using the popular Silvester resuscitation method, he resolved to try the AV method of bellows and a tracheotomy, which he had been employing in his animal laboratory. Following his first success in a landmark case in 1887, he better adapted his apparatus for use in humans and was soon succeeding with further difficult challenges. However, his reports of successful rescues to prestigious Washington conferences were met with derision in 1887, and indifference in 1893, although by the latter year he could detail that he had saved 28 individuals, mostly after opiate poisoning. He also switched to using face masks in place of creating a tracheotomy as simpler but found this produced a few complications.

The quotation heading this chapter certainly rings true, and we can acknowledge Fell and O'Dwyer as pioneers for anticipating the further development of IPPV. Although the invention of apparatus for intubation and for IPPV represented significant advances for anaesthesia, in the first instance these devices were designed for intensive therapy interventions on patients critically ill with life-threatening conditions.

Joseph O'Dwyer and George Fell have both been regarded as pioneers by other specialties: O'Dwyer by paediatricians and otolaryngologists, and Fell by anaesthetists, especially thoracic anaesthetists, and therefore by thoracic surgeons. Their ICM credentials could be better recognised within our own specialty, and we could reclaim them as ICM heroes. Their individual and joint stories may have been told before, especially O'Dwyer's, but apart from Fell's own publications, there appears to be limited writing on his work. Most texts of anaesthesia history make only perfunctory reference, if any, to either of them, paying fuller attention only occasionally,[124,157] usually in terms of that combination of apparatus bearing both their names (the Fell–O'Dwyer apparatus). I detail the pair's struggles and successes in the use of their equipment for conditions we treat today in the ICU. In addition, there is a third man to be honoured, William Perry Northrup, O'Dwyer's enthusiastic supporter, who was involved in the early use of the combination Fell–O'Dwyer apparatus for ICM-type cases. This second tribute is devoted to George Fell and describes his landmark IPPV case in 1887,[157] his other FR patients,[149,159–168] his practice and his place in ICM history. His achievements are summarised in Box 2.1.

2.2 George Fell: A brief biography

George Fell (see Figure 2.1[168]) although born in Chippewa, Ontario, Canada, on 10 July 1849,[169] initially qualified as an engineer at the University of Buffalo, New York. By age 20, he had placed the first crib of the great Buffalo Breakwater.[169] A decade later, he was an organiser of the first American Microscopical Congress, 1879, and a founding member of the American Society of Microscopists, and then its first

treasurer (noted as such by *Science*, *7* August1880)[170] and custodian for nine years. In addition to working as an assistant engineer on the Ontario–New York State International Bridge at Buffalo (1879), he graduated in medicine with highest honours[169] from the University of Buffalo in February 1882; his thesis was on the 'Histology of aneurismal clots'. In 1884, he was elected to the Chair of Physiology and Microscopy at Niagara University (1885–1895). From his experiments with dogs that had developed apnoea from an excess of anaesthetic agents, he became 'conversant'[158[p149]] with a method of applying IPPV with household bellows through a tracheotomy. Fell came to call the procedure 'forced respiration' (FR), 'to distinguish it from the ordinary methods of artificial respiration' (AV).[158[p147]] From that experience with mammals, Fell considered that 'if the respirations could be kept up by suitable means for a sufficient time to permit the elimination of the poison, life might be saved'[158[p149]]—the ICM tactic now used, *inter alia*, for opiate overdosage in humans.

Box 2.1. **Fell's achievements in forced respiration**

- Developed an apparatus for human forced respiration (FR), based on eight years' laboratory experience of animal vivisection.
- Demonstrated lives could be saved by FR when conventional methods of artificial respiration had failed.
- Advocated meticulous care and attention to the apparatus, kept in full preparedness for emergencies.
- Advocated supplying treatment onsite first, because time would be lost in transfer to hospital.[167[p171]]
- Emphasised teamwork and utilised groups of colleagues and students for cases requiring prolonged treatment.
- Substituted a face cup or face mask to enable FR without a tracheotomy to be used more readily by physicians and lay lifesavers.
- Advocated that every medical student in the country be required to possess the knowledge 'to utilize the apparatus'.[167[p170]]
- Advocated use of FR for breathing failure with a wider range of causes than opiate poisoning alone (e.g. for drowning or asphyxias)
- Foresaw application of FR for neurosurgery, and by adaptation of etherisation to the technique, in thoracic surgery.

Figure 2.1: Dr George Fell

Portrait of George Fell (1849–1918), of Buffalo City, New York, physician and engineer, who introduced his 'FR' for acute respiratory failure, July 1887. With thanks to Warren Publishers, of Buffalo City, New York, for this image from Buffalo Med J. 1915–16;71:296, Figure 4.[171]

Details of Fell's first case on 23 July 1887, with its saving of a human life,[158] represent a distinct advance. During the next 13 years, Fell documented in meticulous detail[149,159–168] his clinical rationales, his apparatus, his successes and his progress with FR (using what he called 'the Fell method') for more than 30 other patients, including some rescue efforts also reported by others (see Addenda 2.1 and 2.2). Nonetheless, he experienced disappointing setbacks from the severe criticism, and the great derision on presenting his pioneering case on 7 September 1887 at the 9th International Medical Congress held in Washington, DC[149[p326],163[p342]] By June 1891, although he could claim FR was 'at last beginning to be noticed both on this and the other side of the Atlantic' (i.e. Paris),[163[p342]] the uptake in his own country was limited, yet, his method was apparently well enough known in Buffalo. From his account, he was still highly frustrated at the time of the 1893 Pan-American Medical Congress, when he presented 28 case histories.[165A[p74]]

Fell documented two spectacular cases, one in 1896[166] and the other in 1899,[167] but then does not appear to feature again in medical literature until 1910, when he produced two articles,[168] one of which was readily available. Eight years later, Fell died after several years of bad health, with the causes variously given as paralysis,[169] or dilatation of the heart.[172] An obituary in a medical journal could claim, 'He gave his [FR] discovery to the cause of humanity and has made nothing from his work'.[169[p74]]

In addition to his pioneering medical work, Fell continued contributing to engineering projects, some amid controversy and others of great financial value to government agencies. These are mentioned in his obituary in the *Buffalo Medical Journal*[169] (as the journal that featured his original medical triumph was renamed). Fell was also inventive. In 1888, a New York State commission asked Fell to redesign Harold Brown's electric chair, which was as yet unused.[173] Because Fell's interest was a humane one, he conducted the preliminary scientific, animal vivisection experiments he considered necessary. After what

appeared to be a partially botched execution (on 6 August 1890), with Kemmler (the condemned man) found to be still breathing after the first jolt forcing a second application of electricity, Fell was one of the few observers who thought Kemmler did not suffer. Fell stated that although he would continue to defend the use of electrical execution, he could not entirely defend the manner in which Kemmler was executed.[174] His other accomplishments included the first 'simple, inexpensive practical submarine life-preserver using a face mask',[169[p74]] one 'whereby an individual may remain under water a considerable time without danger'.[172[p.485]]

2.3 Details of Fell's landmark intermittent positive pressure ventilation case

In 1886, people could freely purchase opiates. Three years later, Fell recorded that for 'the many cases of morphine poisoning, reported almost daily through the press, from San Francisco to Portland, ME, in which it has been frequently stated that every means was taken to save life (except forced respiration)',[161[p317]] physicians were starting to recognise that his FR could be life-saving**.** However, they 'generally fail to be forearmed'.[161[p317]] Three years later, he declared that 'the old methods [of artificial respiration] have failed' for drowning as well as for narcotic poisoning.[164[p130]]

On 22 June 1886, Fell along with other physicians attended the attempted resuscitation of a man who had taken an overdose of morphine, for whom conventional measures, including 'artificial respiration by Sylvester's [sic] method', failed to prevent 'the inevitable. At this time, I [Fell] felt keenly the inadequacy of the methods at our command, and then resolved, if opportunity ever offered, to make the operation ... which ... might have saved Mr Dyke's life'.[158[p147]] (Fell used 'making an operation' as his regular term for applying FR.) He wrote later that he made this resolution 'then and there',[160[p41]] at the time of the failed resuscitation of Mr Dyke, and 'thoroughly considered it, for fully [a] year'.[149[p326]]

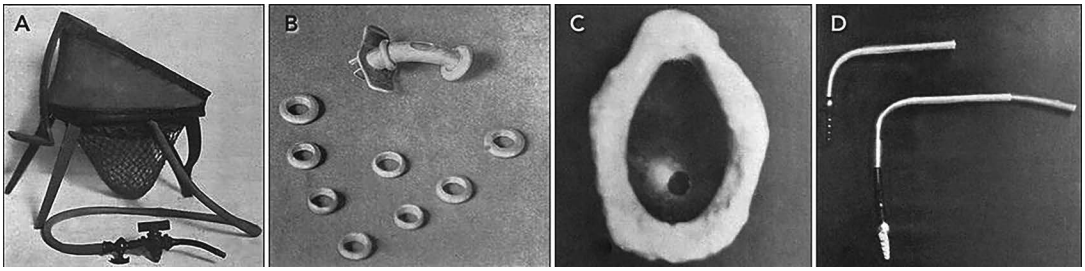
From his foundation Chair of Physiology and Microscopy in the Medical Department of Niagara University, over eight years,[162[p180]] Fell 'many times'[160[p38]] had practical experience with AV for anaesthetic-induced breathing failure in laboratory dogs, and also in FR for these dogs pre-terminally, with the thorax opened for exhibiting the thoracic viscera in action. The AV on dogs was by the FR method taught there for 20 years,[160[p38–9]] but Fell observed he had 'never heard it even hinted that a human life might be saved by the laboratory methods'.[162[p180]] His department's method of FR for animals required a simple fireside bellows, the nozzle of which was ligated into the opening made in the trachea.[160[p53]] Fell wrote that he always used foot bellows in the laboratory.[160[p55]] Rubber tubing connected the bellows to a cumbersome and weighty[160[p55]] 'one piece' unit, comprising a directional control valve and a large, brass tracheotomy tube.[168] (see Figure 2.2). The stopcock on the troublesome valve (illustrated in his 1910 article in *Surgery Gynecology and Obstetrics*[168]; Figure 2.2A) had to be turned by hand for each inspiration and again for each expiration, making it very difficult to avoid wrenching the trachea with each twist.

Although Fell was 'unable to find a case in which forced respiration has been used upon a human subject in opium poisoning',[158[p146]] he believed that it could be used. 'The prevailing opinion at that time was that the air vesicles of the lungs would not resist forcible mechanical measures in artificial respiration.'[166[p760]] Fell attributed such dogma to the currently held strictures of Marshall Hall, then 'the highest accepted

authority',[162[p180]] to avoid 'the use of 'bellows or any forcing instrument'[159[p352],176] (see Footnote 2.1[1]). Although it appears he made no actual advanced preparations, Fell mulled over his ideas of intervention for a year.[149[p326]] Then, on 23 July 1887, he was called to an alcoholic man deeply narcotised from opium poisoning. Fell's successful management produced a landmark case in the history of IPPV and ICM: he later claimed that the application of FR 'per tracheotomy in the treatment of narcotism from opium or morphia appears to be original with myself'.[160[p40]] His success, after artificial respiration by others present had failed, demonstrated the possibility of saving a life by FR without the feared destruction of the delicate vesicles of the lung (see Footnote 2.2[2]).

Fell frequently mentions the contrast between the passive FR patient, and the patient receiving manual artificial respiration, who he described as being 'tugged, squeezed, and rolled about, according to the method employed'[160[p38]].

Figure 2.2: Some of Fell's apparatus for FR (photographed by Fell personally)

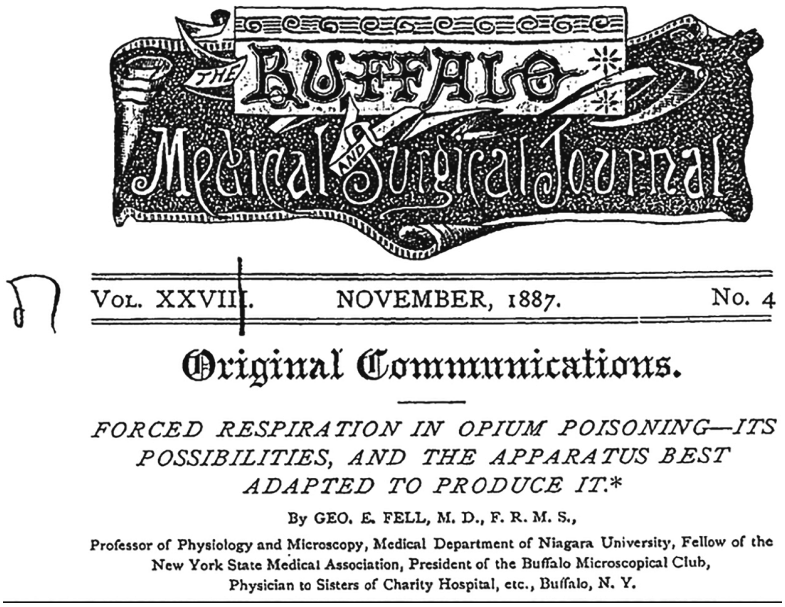


A. Fell's historic apparatus from his animal laboratory used in his landmark case of forced respiration, 1887.
B. Tracheotomy tube and airway-sealing tube rings.
C. Fell's first 'air cup or face mask'.[168]
D. Fell's endotracheal tube, a rubber tube loosely fitting into brass tubing (on the mid and right) compared with Joseph O'Dwyer's intralaryngeal brass tube with a conical screw-on tip of steel or vulcanite (on the left).
With thanks to the American College of Surgeons for these illustrations from Surg Gynec Obstet. 1910;10:578 (Fig.11), 580 (Fig.18), 577 (Fig.3A), 580 (Fig.16-7).[168]

[1] Footnote 2.1. Fell appeared almost to resent the thrall in which others held Marshal Hall's ideas for resuscitation; the feature he objected to particularly was the 'special stress upon the inadvisability of using any "forcing" measures or "instrument"'.[160[p37]] The resultant use of various methods of artificial respiration was too often ineffectual, short-lived because of a rescuer's exhaustion or unsuitable for longer-term use (e.g. when needed for more than a day, not just many hours). Later, although the method of artificial respiration used—usually Silvester's—was often ineffectual, Fell's method received only limited acceptance, even after he had demonstrated that it did succeed. However, Hall had emphasised worthy points,[177] such as onsite immediacy and the prone posture for airway protection, and his method had saved lives.

[2] Footnote 2.2. Fell, by directly quoting Horatio Wood (1841–1920) in one of his articles,[149[p325]] did concede that life had been saved by artificial respiration after poisoning. 'In 1875 (*Boston Medical Journal*, Vol. xxi.) Dr. John Ellis Blake reported a case of aconite poisoning in which, although there was no pulse for over three hours, "life was saved by artificial respiration [not by FR], with the use of oxygen"". However, this must be an erroneous reference. Volume xxi is for 1839–1840, Aug–Feb, while the index of 1875's volume xcii, Jan–Jun, has no aconite case nor any Dr JE Blake listed.

Figure 2.3: Title heading of Fell's landmark case history



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Original Communications.

FORCED RESPIRATION IN OPIUM POISONING—ITS POSSIBILITIES, AND THE APPARATUS BEST ADAPTED TO PRODUCE IT.*

By GEO. E. FELL, M. D., F. R. M. S.,

Professor of Physiology and Microscopy, Medical Department of Niagara University, Fellow of the New York State Medical Association, President of the Buffalo Microscopical Club, Physician to Sisters of Charity Hospital, etc., Buffalo, N. Y.

Title heading of Fell's article[158] on his first use of FR in Buffalo Med Surg J. Nov 1887;28(4):145. (The crossing-out mark that appears on repeat copies may suggest that a librarian had detected the volume number XXVIII is incorrect. My own librarian's investigation showed this volume's true number is 27, not 28, for issue 4 of Nov.1887.). With thanks to Warren Publishers of Buffalo, New York.

2.3.1 The history of Fell's Case 1

Fell and others attended 'Mr PB' about seven hours after the latter had ingested morphine (later estimated to be 20 grains [=1296 mg]) and some chloral.[158] Fell first tried simple measures, including dressing the patient and force-walking him in the cold air (a move also used for barbiturate poisoning, during 1940–1950), then resorted to Silvester's artificial respiration until he was too exhausted, and eventually stopped trying. With PB's respiratory rate and pulse respectively at five and 200 per minute, the doctors declared he would not survive; last rites were administered and the death certificate started in anticipation. Fell left after about five hours of care, in all. Later, Prof. (of Materia Medica) FR Campbell was called and found PB still alive, breathing once a minute, and hence had Fell wakened (after less than three hours' sleep). When they noted the patient's pupils at the near-final stage of the 'dilatation of asphyxia',[158[p149]] Campbell observed, 'We can do nothing more now'.[158[p149]]

Then, Fell offered to try the methods he knew from using them on dogs. He went to his nearby laboratory, returning with a 'tracheal tube covered with dog hair and blood',[158[p150]] fresh from 'electrical experiments [his electric chair ones?] on a dog ... a few days before this',[158[p150]] which he cleaned. With further assistance now available, he performed a low tracheotomy, during which (he later said) 'I felt that I was making the operation upon a cadaver and worked accordingly',[149[p326]] and also 'placing with the greatest difficulty'[158[p150]] a ligature around the trachea to seal off the upper airway from below. The patient was then very dusky, and Fell noted 'no respiratory effort had been made for some time'.[158[p151]] (One may wonder why the patient had not developed cardiac arrest after such prolonged apnoea, but Fell is very firm about his patient's clinical state.)

When FR was established (only after difficulty), PB's blood became 'more arterial',[158[p151]] but no spontaneous breathing attempts were detected for at least 30 minutes; then, 'after about two hours work ... natural respirations [gradually returned to] almost normal'.[158[p152]] But the patient could not sustain them adequately because of the handicap to breathing from the small opening in the side of the valve ('one-eighth by one-half inch or one-and-four-tenths by three-tenths mm').[158[p152]] 'Only a small portion of the expired air could pass through it, so that a large percentage had to escape by the side of the canula.'[168[p577]] Haemorrhaging from the stoma occurred because the patient had been moving about restlessly, but three soldiers living in the house restrained him. After 2.5 hours of FR, the 'cumbersome'[158[p153]] tracheal tube of the respiratory apparatus was changed for an 'ordinary'[158[p153]] tracheotomy tube (i.e. one with an inner cannula), and spontaneous breathing was allowed. The patient recovered, despite a relapse to breathing at a rate of six breaths per minute about 24 hours after the operation, and a bad attack of delirium tremens.

Fell's account is detailed. He later wrote, 'when I made my first operation it was with incomplete apparatus, and it was surprising on this account that I did not lose the case'.[149[p328]] Thus, Fell 'saved a life which [he] had thought there was no possibility of saving'.[149[p.326]] His comment, 'Had I failed it probably would have "settled" the question of forced respiration',[149[p.328]] can bring to mind what could have happened to Bjørn Ibsen on 27 August 1952 had his intervention of tracheotomy and manual IPPV for a moribund girl with breathing inadequacy from poliomyelitis failed (see Chapter 7).

2.3.2 Presentation to conferences and documentation

Within two months, Fell presented his case to an international audience in Washington (on 7 September 1887),[158] but received only humiliation, which he described later in 1891[149] (see Footnote 2.3[3]). Four months after this rescue, that is, in November 1887, Fell had his case history published[149] (see Figure 2.3), together with certain recommended changes necessary to repair the deficiencies of the animal apparatus. That is, 'The laboratory apparatus used at the time merely demonstrated the value and safety of the [FR] principle'.[166[p.760]]

[3] Footnote 2.3. At the 1887 Washington Congress, being

> 'acquainted with but very few individuals it was with the greatest difficulty that I had an opportunity to read my paper at all; and what was the most peculiar feature of the whole circumstance was, that, even among a class of men supposed to possess the highest medical knowledge, not any of them saw the point which presented in that first case of forced respiration, in which I breathed for a man two and one-half hours with a tube in his neck. They did not grasp that point. And I now make the statement, without fear of contradiction, that there was not a paper presented at the International Congress at Washington which had a farther reaching import, if to save a human life is desirable, than that little paper on 'Opium Poisoning', which I presented – a paper embodying in it demonstrations which would alter and advance one of the greatest medical practices of the day, a practice of wide application. It demonstrated what was not practically accepted in medicine before, that we could force air into the lungs for an almost unlimited period without danger to the delicate lung tissue.
>
> When I managed, however, to read my paper at Washington, they did me the kindness (?)[sic] not to publish it in the proceedings.'[149[p326–7]]

(One can wonder then, does Garrison's citing[117[p725]] of 'Fell: *Tr. Internat. Med. Cong*. Wash., 1887, I, 237' refer simply to the agenda?). In the 'brief discussion' following Fell's delivery, several physicians claimed to Fell that his FR treatment was not needed, or that Silvester's artificial respiration would have accomplished as much.[160[p37]]

From discussions with the organisers of the September 1887 Congress, Fell concluded:

> 'After I had saved my third life [December 1887], however, by forced respiration, and the world could not question methods which were so positive in their demonstrations, and so undeniably original, there not being a similar case on record, it was evident my paper was either not carefully read, or the principal point conveyed by it was not grasped by the members of the committee.'[149[p326–7]]

2.3.3 George Fell's conclusions

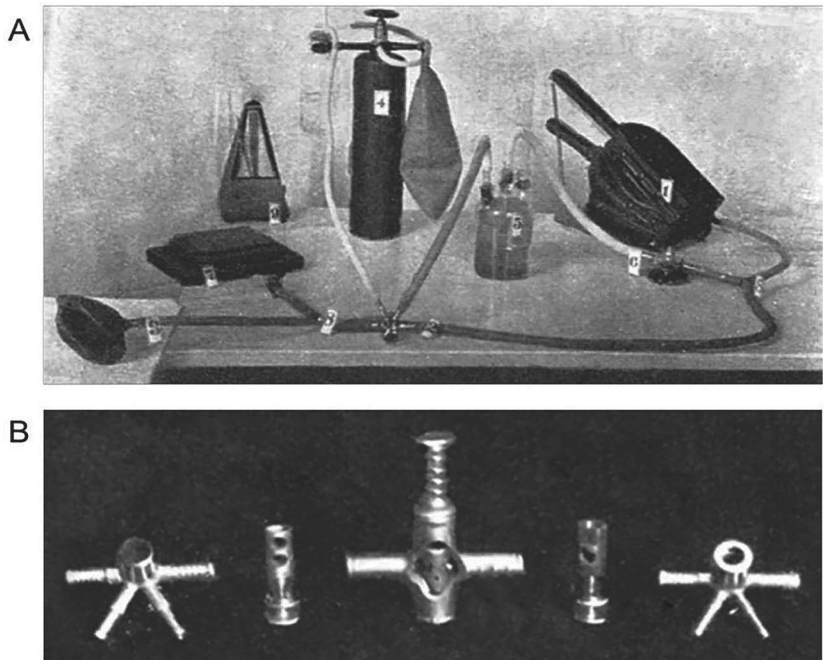
From his successful treatment, Fell arrived at certain conclusions:[158[p155–6]]

- Improvements to his animal apparatus, especially to the valve, were needed for using it on humans; he had them set out clearly by December 1888.[160[p53–60]] Also:
    - Cold air could be warmed by passage through a vessel of heated water.
    - A tracheal tube could be secured in place by annular corrugations or a rubber tampon.
- Pure oxygen is not necessary for breathing failure treated by FR (he later revoked this conclusion).
- Judgement is needed to prevent over-inflation of the lungs.
- The FR apparatus (without its air heater) can be fitted into a portable bag suitable for emergencies.
- Employing his apparatus with a tracheotomy offered, in his opinion, more positive results than it did with intubation of the larynx.
- FR should also be available for 'almost all varieties of conditions in which artificial respiration has been used, and with the certainty that it will be a more potent means of saving life than the latter'.

Fell described his refurbished apparatus (see Figure 2.4). as covering all requirements for FR by 'the most simple method possible'.[149[p328]] The bellows, 'with a rubber equalizer' (as he had used with the animal bellows[167[p171]]), allowed a *'steady,* instead of an interrupted or jerky column of air'; the air is 'rhythmically presented'[168[p574]] with 'proper periodic intermission'.[164[p131]] The cumbersome air-control valve was replaced by a two-way valve 'on the cornet-piston order', which could be opened or closed by the movement of the fingertip, offering 'absolutely no interference with the auto-respiration'.[168[p577]] With the valve, 'the air can pass in and out of the lungs at all times, except during the forcible inspiration'.[163[p344–5]] The stopcock of the valve now turned only one way. A new, flexible connecting tube inserted between the valve and the tracheotomy tube precluded the previous wrenching on the trachea from the need to twist the stopcock twice for each full manoeuvre to have a breath to pass in and out of the lung at all times.

An assistant could pump the bellows, worked by foot or hand power ('anyone can'![149[p328]]); the doctor would use a finger to control the valve for each insufflating breath, with (for example) two or three bellow movements both during the inspiration and the expiration time.[160[p57]] The 'gearing' measures to control the rate of breathing seem complicated (at least to this writer): for example, for FR Patient 5, Fell noted the 'bellows working at the rate of 108 movements per minute, patient by this means receiving 21 respirations to the minute'.[160[p52]]

Figure 2.4: Fell's modification of his 'FR' system for animals adapted for use with humans



A. Modified apparatus for 'forced respiration', comprising:
1. double bellows or air supply device; 2. air valves with oxygen and anaesthetic tubes;
3. air cup or face mask, intubation tube etc; 4. oxygen supply apparatus;
5. anaesthetic container; 6. rubber manometer;
7. three-way connections; and 8. Maelzel metronome.
B. Fell's air valve, 1887.
With thanks to the American College of Surgeons for these illustrations from Surg Gynec Obstet. 1910;10:577 (Fig.1–9), 577.[168]

2.4 Further use of FR

In the presence of four physicians on 10 December 1887,[160] Fell used 'his' apparatus (the animal set, now refined for use on humans) successfully for the first time[165[p76–FN]] for a 'difficult case' of laudanum poisoning; but only after refraining from intervention for as long as the others thought they could succeed with artificial respiration. 'Time was [first] given to demonstrate beyond question the uselessness of the artificial respiration [as cyanosis continued], until it was feared the patient might succumb before the FRs could be applied.'[159[p347–8]] Bleeding that was blocking the tracheal tube with some aspirated material necessitated a second ligature, around the trachea and tightened. Fell said that he was urged to discontinue the respirations because the case was considered hopeless. He did so at one stage, but 'The man was not dead, and we had to keep it up'.[160[p46]] After such long hours of work (14.5 hours of FR), Fell suggested that electromotive power be substituted for hand power in future.

In documenting this case in March 1888,[159] Fell emphasised the curious dissociation of returning consciousness, from a respiratory drive which did not match it. Presumably to gain extra evidence for his AV method, he labelled this FR patient—his second—as 'Case 3' of FR (see Footnote 2.4[4]). Fell called for his apparatus to be used in cases of drowning[159[p349-FN]] and then wrote 'we shall find it a favourable field' for the use of FR, again that year.[160[p64]]

With the completion of treatment in January 1888 of Fell's next overdose patient, his third successive difficult one, who needed 24 hours of FR, he had 'saved three human lives after all the usual methods had failed',[160[p38]] with 'all successful under most adverse conditions'. He wrote that the efforts for his 'second and third cases would surely have failed' had he not modified his original laboratory animal apparatus.[163[p343]] Fell estimated that the saving of the first three lives represented a US$23,000 boon for the insurance companies.[160[p50]]

2.4.1 George Fell's conclusions

Fell's professional report at the end of 1888,[160] in which he took stock of his patients until then (aged 18 days to 80 years), set out principles and practicalities worthy of an early intensivist:[160[p55]]

- the 10 essential principles he required for FR in man;
- the physical composition of his apparatus (see Footnote 2.5[5]);
- the need for suitable timing for intervention with FR, which is something more than a last resort compared with artificial respiration[160[p63]];
- the emphasis on the need for preparedness of equipment and teamwork in using it;
- the anticipated usefulness of FR for drowning (and almost all cases of asphyxia), if the opposition of the great majority of the medical fraternity to tracheotomy could be bypassed by oral intubation of the larynx or trachea; Fell carefully described the technique for intubation[160[p64]];
- the usefulness of FR in shock, demonstrated in his second and third applications of it. He implied that FR 'may give us results in shock to be attained by no other means, or may serve as an accessory to other methods to overcoming this bane of surgical progress'.[160[p65]] For his times, brave predictions indeed.

[4] Footnote 2.4. FR Case 2 (in Europe): Fell cited a successful Viennese instance of FR, on 21 September 1887, per tracheotomy for narcotic poisoning, soon reported in the *Lancet* of 15 October (within the section on 'The Medical Societies'[177[p776]] and not found readily), and then with more details in 1889 from Prof. Boehm.[161[p316]] Fell later appeared delighted at the Austrian success of tracheotomy with FR; but without actually claiming credit for it, Fell did not hesitate to state that the Vienna case occurred 'after the reports of my first case (read before a section of the Medical Congress at Washington [that September]) had appeared in the medical journals',[160[p44]] although Boehm did not mention either the Washington Congress or the *Buffalo Medical Journal*. Nor did Boehm write that he conducted the treatment himself, and thus, the rescue of the patient may have been achieved through the action of some other person. For that time, it is not clear whether this FR rescue was a unique incident in Europe or whether FR practice was better known there. Certainly, by June 1891 Fell could find 'his' FR being described in the reports of the Paris Academy of Medicine.[163[p342]] Further, by that year in the US, 'fourteen human lives which otherwise would have been sacrificed' were 'saved by several physicians who have utilised my methods'.[162[p176]]

[5] Footnote 2.5. Fell's FR apparatus now comprised:[160[p59]]

- air-forcing apparatus, laboratory or hand or foot bellows, with a rubber equaliser;
- air-warming apparatus, per a water-containing copper vessel, and an alcohol heating-lamp;
- air valve, now with a piston instead of a tap to inject a forced breath;
- tracheotomy tube and a set of sized, circumferential rings to seal off the upper airway;
- connecting rubber tubes allowing free movement and flexibility.
- Fell disposed of a second bellows being used for forced evacuation of the inflated lungs [as had been the method of John Hunter and others] as 'a grave defect',[165C[p123]] and he devised artificial lungs to study ventilation.[160[p57],168[Figs.13–14]]

After publishing his initial two successes,[158,159] Fell numbered and meticulously documented those subsequent FR patients of whom he had details, into successive groupings designated Patients 4–6 in December 1888,[160] Patients 7–10 in 1889,[161] Patients 11–22 in 1899 (by this time, with 'the saving of fourteen human lives'[162[p176]]), Patients 20–25 in 1892[164] (with now 19 lives saved), and from his presentation to the Pan-American Conference at Washington on 7 September 1893, Patients 1–28 (plus Patients 29–44 of other doctors) in 1894.[165]

Over a span of eight years, the journals featuring these case histories were progressively more prestigious: the hometown *Buffalo Medical Journal* was followed by the *Transactions of the New York State Medical Association* in 1888, 1889 and 1891, the *Archives of Pediatrics* in 1892 and then the *Journal of the American Medical Association* in 1891 and 1892. Later came the *Canada Medical Record* in 1894, the *Medical Record* (Philadelphia, PA) in 1896 and the *Medical Examiner* (NY) in 1899. Many of the publications appear to have excess material repeated from previous reporting, but often that would arise from articles being textual renditions of addresses delivered by Fell to societies. Thus, reports on FR Patients 4–6 were published in December 1888, with those on Patients 1–3 recapitulated, and the reports on Patients 1–10 were repeated with that on Patient 15 in 1891.[162]

Fell's dismal failure to convince his international audience at Washington in 1887 has been referred to; he was much more reticent about the reception of his 1893 presentation in the same city, the text of which in the *Canada Medical Record*[165A–C] was accompanied by his observation that 'the majority of the members of the Congress appear to know very little regarding the work accomplished by the methods described'.[165A[p74]] He did receive a few appropriate questions and a recommendation from 'the worthy president of this body',[165C[p127]] but recorded that 'No special recognition which has resulted in calling the method into general use has resulted'. In this article, Fell very fully describes the FR patients until then, Patients 1–28, with unhesitating recapitulation from earlier papers; he says he 'reported in detail simply to silence all doubters'.[165A[p74]] What he called 'my method' (first noted in 1891[162[p181]]), he changed to the 'Fell method' from 1893.

After 1894, Fell did not individually number the multiple patients in his next two articles: the first article in 1896,[166] describing the use of FR for instances of various anaesthetic overdoses (by ether, chloroform or nitrous oxide), and the second in 1899.[167] Each article contained a prolonged account of a spectacular and lengthy case. Perhaps the number of patients was becoming too unwieldy to describe each individually, as in May 1896 he wrote (without offering further evidence for the number):

> some one hundred human lives, which would certainly have been sacrificed by the methods at the command of the profession at the time, have been saved [since 10 December 1887] through FR. Most of these cases have been saved at my hands with few exceptions in the city of Buffalo.[166[p760]]

Fell referred to the 'saving of a human life' in all his articles, almost as if by rote, and although that may become mildly irritating, it does make his point. By 1899, he claimed FR had 'saved a few hundreds of human lives and is destined to save thousands'.[167[p170]]

Brief notes for some of the case histories (Patients 1–28) are shown in Addenda 2.1 and 2.2 (also see Footnote 2.6), together with accounts of the two remarkable cases of 1896 and 1899, respectively—remarkable considering their times and the level of medical knowledge then available for treating critical states. After a decade apparently without his writing further (see Addenda 2.1 and 2.2), what seems to be Fell's last FR medical report appeared in 1910.[168]

2.5 Critical illnesses in Fell's patients

The numbered FR patients referred to hereafter in this paragraph are described individually by Fell in reference and Footnote 2.6 (also see Addenda 2.1 and 2.2). Most of Fell's successes were with 'cases of opium narcosis',[163[p346]] and many patients had deteriorated to a truly desperate condition by the time Fell was called. Often, other medical attendants had quite written off the patient's chance of survival. Some patients had co-morbidity (in his reports, e.g. Patient 4 receiving FR), or were elderly (Patient 21, aged 78 years; Patient 19 [with a mercury bichloride, $HgCl_2$ fatality], aged 73 years; and Patient 5, aged 80 years).

All but one of the first 11 patients were poisoned by opium or its derivatives, and three of these (Patients 1 and 7 fatally, and 9) had near-terminal dilatation of the pupils. Some patients had advanced ventilatory failure with breathing rates of 1–5 breaths per minute (and breathing was described as absent for Patient 1); another, Patient 22, had Cheyne–Stokes breathing; and cyanosis was common. Others had more than ventilatory problems, such as shock in the instance of Fell's second success (Patient 3), haemorrhagic shock for Patient 4, some without detectable wrist pulses (Patients 4, 7, 10, 15, 17 and 22) and convulsions for two (Patients 6 and 21). Some of the problems resulted from lack of protection of the airway with the use of the face masks, and Fell described severe airway problems for Patients 8 and 21, and both the patients required ligatures to pull the tongue forward. Patient 18 had anuria, and others had oliguria.

The efforts of Fell and others at arousing the narcotised were those of the times:

- Emetics were given in the belief that vomiting would stimulate the 'benumbed' respiratory centre,[165A[p87]] where it was possible for the patient to swallow water and mustard with a teaspoon of salt. Otherwise, hypodermic apomorphine could be tried.
- Gastric lavage (for Patient 5) was given, and then, where possible, gastric fluids (even food).
- Repeated enemas were given.
- Other agents dismissed nowadays (except the last of these) might also be used:
    - 'Stimulants' such as coffee (about one strong pint for Patient 20 after gastric lavage[164[p132]]), strychnine, brandy, belladonna, atropia and digitalis.
    - 'Faradisation' (stimulation with an electric current), applied by a helpful colleague for Patient 15 (after Fell had already rescued her), but unfortunately fatally.
    - Blood-letting for Patient 23. Fell regretted he had not provided this to Patient 21, a 78-year-old person who had taken an overdose of gum-opium and had intense congestion of the encephalonic vessels.
    - Saline infusion via transfusion to Patient 4 for shock, but that brought later complications.

Misadventure seems to have caused death in at least three patients: Patients 6 and 19 (both irremediable from the prescription errors of others), and Patient 15; but for Patients 18 and 21, death was probably ill-fortune. The numbering of FR patients as in Addendum 2.1 is Fell's own.

An entry portal was provided for Fell's FR system to supply oxygen (from a cylinder) into the apparatus when that was needed, but its use was specified only for Patient 8, and for the two patients who received several days of FR (also see Addenda 2.1 and 2.2): Dr Henry Williams, in a 'desperate condition',[166[p762]] and Raymond Archer,[167] on whom the 'unquestioned practical demonstration of the value of the method' was in evidence[167[p169]]—although there was also the 'unnerving' complication of rupturing of the bellows during FR, which he wrote required an [estimated] 525,000 'movements'.[167[p171]] (Previously, '108 movements per minute ... provided the patient 21 respirations to the minute'.[160[p52]])

Fell reported that he was frequently questioned about lung complications, but he asserted, 'in not one of the many cases [with FR supplied for between two and 78 hours] has any lung complication of any consequence been produced'[167[p170]]—presumably referring to survivors—since 'no over-inflation of lungs is produced'.[167[p170]] Different complications occurred in Patients 18 and 21, as per Footnote 2.6.[6]

2.6 Fell on intubation versus tracheotomy for forced respiration

After Fell's experience of his first six FR patients, it must have seemed obvious to him that a factor strongly deterring other physicians from following his treatment method was that it required a tracheotomy which, in those times, many (understandably) would shrink from attempting, or would object to. 'Where tracheotomy is necessary, owing to the objection of the great majority of the medical fraternity to cutting operations, it may not be generally used.'[160[p64]] He pointed out early (in 1888) that in 'drowning [particularly] and many other cases' tracheotomy need not be performed, and FR 'may be applied by intubation of larynx or trachea'.[160[p64]] (This was still likely to be a difficult task for most doctors.) Then, he set out clearly the technique of intubating, using a tracheal tube of flexible rubber, and also how to secure it, but did not indicate whether he had already done that himself.

After Patient 15 (in March 1891), Fell declared that, based on his experience with a dozen living beings (they could hardly have been drowning victims only), he was not yet recommending intubation because it was not practicable in many cases owing to the difficulty of intubating the larynx. And he stated his belief, based on Patient 15, that 'in a long continued operation it is possible to breathe for the patient more easily and thoroughly by resorting to tracheotomy'.[149[p329]]

This contradicted the statements made by Prof. Horatio Wood of Philadelphia when promoting Fell's method to the Berlin International Medical Congress (25 August 1890).[149[p325]] There, Wood insisted that if there was failure with the usually satisfactory face mask (which he described as all that is necessary for artificial respiration—but only after he had switched to endorsing face masks instead of tracheotomy), then

[6] Footnote 2.6. Fell's 1894 article in the *Canada Medical Record*[165] supplies details of 28 FR cases, which include nine from other rescuers (numbers 2, 12, 13, 14, 16, 20, 25, (?)26 and 27), and brief notes on a further 16 (numbers 29–44) from Buffalo's Fitch Accident Hospital. The article's title includes 'the saving of twenty-eight human lives'. The 28 FR cases and these 28 survivors are not synonymous, as casual reading might expect. Among the FR Cases 1–28, there were 19 survivors, and the title's total of 28 can be arrived at only by adding the nine survivors from the Fitch series.

an intubation tube could replace it. Fell recognised the dangers of inflating the stomach and intestines when using a face mask ('too great pressure will distend the oesophagus and inflate the stomach and intestines'[165C[p125]]) and soon experienced serious consequences of that for Patient 18.

After Fell's first successful case, instead of continuing with his earlier ligature about the trachea to prevent the air from passing up the throat after the tracheotomy, he devised a ring[160[p56]] (in different sizes: 'larger or smaller'[163p345]) to attach near the tracheal end of the tracheotomy tube (Figure 3.3B). Because it fitted firmly, the ring also aided the retention of the tube and helped prevent the aspiration of stomach content, etc., into the lower airway—yet, fatally, such a ring did not stop aspiration in Fell's FR Patient 18 (1891). Joseph O'Dwyer had promoted laryngeal intubation for FR,[100] by replacing the tracheotomy tube of the Fell system with his own laryngeal tubes, thereby introducing the Fell–O'Dwyer apparatus, as will be described in Chapter 3 of this North American trilogy in medical history.

Among Fell's objections to intubation, as promoted by O'Dwyer for FR in opiate poisoning, was that it 'would prevent the imbibing of fluids, through which means we may most readily aid elimination of the poison', whereas 'tracheotomy offers more hope for our patient than intubation, as there is no interference with the passage of fluids to the stomach'.[163[p347]] (Further, it was advantageous against the danger of vomited material entering the larynx.) His viewpoint four years later was that:

> Many lives have been saved by it [face mask or cup] without necessitating the operation of tracheotomy and its use has indicated that intubation is seldom needed, although its value [presumably, of intubation] must not be lost sight of in forced respiration as I intimated in my first writings upon the subject.[166[p760]]

With his last publication of new case reports (1899), Fell was also backtracking on his earlier advocacy of intubation for drowning, as 'the time lost in attempted intubation in drowning cases would make the operation [of intubation] impracticable'[167[p169],] and asserted that the value of the face mask was a hundred times more.

Despite Fell's arguments, the Fell–O'Dwyer apparatus became established, and Fell obviously came to accept it, since he himself designed a rubber tube for it in 1908—one extending within the trachea and, as he saw it, less injurious than O'Dwyer's brass tube with its attached vulcanite tip, which did not pass distally beyond the larynx[168[p580]] (see Figure 2.2D). (But there seems to be no publication from Fell that mentions that he, or anyone else, used one of the Fell endotracheal tubes.) By 1910, Fell was prepared to rate an intubation tube equally alongside a tracheotomy tube and an 'air cup', as 'all of positive value in FR'.[168[p581]]

In 1892, by detailing nine points of improvement for his method,[163,164,165B] Fell three times rebutted 'Dr. John[sic] O'Dwyer of New York, [who] has given public utterance to the statement which Dr Wood first, and I think unwarrantably, urged'.[164[p130]] Wood had indicated at the Berlin Congress in 1890 that Fell's method was the same as that practised in laboratories on lower animals.[163] Wood escaped lightly, unlike the unfortunately savaged O'Dwyer. Notwithstanding Wood's remarks, Fell still wished to again pay his respects to him. But Fell seems to have missed noticing O'Dwyer's remarks repeated by his supporter, Northrup, in two of his publications, in 1894[120] and 1896.[146]

By 1893, Fell had changed his earlier opinions and had now largely abandoned any form of solely laryngeal intubation for FR, although he conceded[165C[p125]] that he had saved lives by performing a tracheotomy after the face mask failed in one or two cases because of heavy opiate narcotisation. And 'as to intubation, it may have its place in some cases of forced respiration, but to urge its value over the use of the face mask when the latter has accomplished so much, is unwarranted'.[165C[p125]]

2.6.1 Further on Fell's opinion on face mask versus tracheotomy for forced respiration

When managing Patients 8 and 9,[149[p329],165A[p82–3]] Fell noted that efficacious FR could be supplied—without a tracheotomy—if the tube (from his IPPV apparatus) was sealed into the mouth by closing the nostrils and compressing the mouth around the tube. This was first tried for a stillborn infant (Patient 8), and then for an adult male (Patient 9). Fell saw the possibilities of oral FR by face mask and first prepared one covering both mouth and nostrils (Figure 2C) to enable FR lasting four hours for Patient 11. Fell later revealed that the cup he had originally called the face mask *'*may be made of tin or hard rubber with the edges which are applied to the face or body of wax, which, by heating, will make it conform to the surface of the body'[168[p581]] (see Figure 2.2C). Thus, his face-mask system for FR[165C[p126]] came to comprise foot bellows, a rubber tube, an air control valve, further rubber tubing and then the face mask (see Footnote 2.7[7]).

Following the first patient (Patient 11) Fell treated using a face mask, Dr CR Vanderburgh treated Patients 12–14 successfully with face masks, and Fell treated Patients 15,17–19, 21, 22, 24 and 25.[165] However, after Fell re-oxygenated Patient 17 by face mask for three hours, she relapsed to cyanosis, pulselessness and inertness, all of which then indicated to him that there was no hope she would survive. That her condition then improved with FR 'by tracheotomy over that [FR] produced by the face-mask, was evident'.[162[p185],165A[p84]] After using a face mask for 11 hours for his next rescue (Patient 18), the amount of air (and oxygen) forced by FR into 'the stomach and bowels was so great as to markedly distend them, thus interfering to a certain extent with the inflation of the lungs'.[162[p186]] A tracheotomy then enabled ready inflation, and hence, after the procedure he handed over further treatment to a colleague and students; however, the patient vomited forcefully, aspirated vomitus and the labour of 18 hours was lost. After using an easily applied face mask for Patient 19, he used a tracheotomy for the next patient, but admitted making a mistake regarding Patient 21: Fell kept persisting with a face mask despite airway difficulty requiring a 'coarse' tongue ligature, before resorting to tracheotomy again after 11 hours, which proved a fatal delay.[162[p188]]

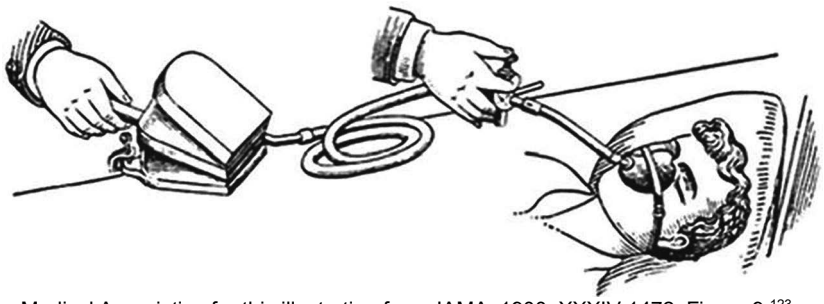
[7] Footnote 2.7. Regarding the use of the face mask to inflate the lungs, Fell wrote:[165C[p127]]

> For each three movements of bellows, press down piston of air valve, which permits the air to pass to the lungs, bulging out the cheeks, and produces an inspiration. If cyanosis does not pass away, make the inspiration a little longer. With the air valve you can absolutely control the outward or inward movement of the air, and by watching, if attempts at respiration should be made by the patient, you can materially assist them and change instantly from one to the other.

Thus, Fell's FR form of IPPV could convert to what today we could call 'assisted ventilation'.

For his very next patient (Patient 22), who had Cheyne–Stokes breathing, Fell indomitably proclaimed that the face mask had again demonstrated its great value in this typically appropriate case and continued using it. He emphasised that he had demonstrated that FR by use of his face masks had saved many lives with 'from two to ten hours work' *without* the need for a tracheotomy[167[p169]] (see Figure 2.5).

Figure 2.5: Fell's apparatus for inflating the lungs using a face mask



With thanks to the American Medical Association for this illustration from JAMA. 1900; XXXIV:1472, Figure 9.[123]

With the hindsight from a 21st century perspective, one can wonder how aware Fell was of the increased risks associated with lung inflation using a face mask, with the airway left unprotected. Possibly he discounted them because a face mask 'brings the operation [of FR] to that degree of simplicity that it may be readily utilized by physicians unwilling to make tracheotomy'.[165A[p83]] A few patients had to endure the typical consequences associated with use of a face mask without intubation: for example, as above,[162[p186]] having air forced into the stomach and intestines, with associated mortality—despite Fell's attempt to prevent this by a kind of 'reversed-Sellick' manoeuvre (Fell quoted *Agnew's Surgery*, vol. 3, p.88, as describing 'the larynx pressed back against the oesophagus'[160[p39]]). One cannot determine from the cases Fell's attended to personally the extent to which his persisting without a tracheotomy was his sheer determination to be 'right', or was because of his wish to encourage other practitioners to perform FR by their being spared the need for tracheotomy. Although an obstructing tongue was managed in multiple instances by a ligature to pull it forward, there is no mention of a jaw lift, or of the lateral rather than the supine positioning of the patient. Fell does state that extension of the neck was not a completely reliable solution, claiming his tongue ligature had corrected airway obstruction by the tongue in 'the few cases'.[163[p348]]

Fell developed genuine expertise in the FR method with a face mask, often for prolonged periods, enabling some impressive 'saves'. He regretted choices that proved fatally unwise (Patients 15, 18 and 21). But the adoption of his face mask would change a situation from one requiring an immediate tracheotomy on a moribund or dying patient while time was slipping away (with no lessening of the primary respiratory failure and hypoxia) to one where quickly starting FR using his face mask relieved the acute crisis, allowing a tracheotomy to be performed more safely.

2.6.2 Fell and mouth-to-mouth rescue breathing

Previously, I doubted whether Fell was aware of mouth-to-mouth ventilation,[36[p228]] but can now note that for a case of stillbirth (Patient 8) he stated,

> Previous to my arrival, the nurse had kept up the action of the heart by mouth-to-mouth insufflation. Cyanosis was extreme. As I did not wish to attempt tracheotomy, for a time I resorted to the same means. This not giving satisfactory results…[165A[p82]]

He obtained an immediate change by employing FR through a tube sealed into the mouth, and the success alerted Fell to the possibility of FR without tracheotomy. Obviously, both nurse and doctor were familiar with the mouth-to-mouth method. (The ever-optimistic Fell even tried to resuscitate an asystolic stillborn baby, on 18 June 1888.[160[p53]]) Fell makes no mention of mouth-to-mouth expired air ventilation in his 1910 list of artificial respiration methods.[168]

2.7 Fell's recruiting of collaborators, and his disappointments

It is obvious from Fell's writings that he did endeavour to spread his pioneering message. Once he achieved his own first five successes, he quickly advocated a practice that 'The only safe rule is to make the attempt [at rescue]'.[161[p318]] The case histories reveal that he could be called upon at any time to go to a patient needing ventilatory assistance ('I have always been ready to aid and assist anyone disposed to utilize the method' [164[p130]]), although after an overdose, the calls often came well after midnight. He was also prepared to lend his apparatus to experienced practitioners ('in Buffalo my apparatus has always been at the disposal of physicians whom I considered capable of using it'[161[p317]]). By the time of Patient 10 of 1889, the apparatus had become commercially available, with 'manufacture ... by a responsible firm'[161[p318]]), and Fell tried to ensure that the apparatus available be 'in the simplest manner'.[162[p180]] After two years, he reported, 'lives have been saved by several physicians who have used my methods', although FR 'has not yet come into use in [other] fields to which it is admirably adapted'.[162[p176–7]] (He indicated in 1896 that 'the Fell forced-artificial-respiration apparatus' was held at Buffalo's Fitch Accident Hospital.[166[p761]])

Fell clearly spelled out that although there was 'no impediment in the way of general adoption by the medical profession, no restriction on the manufacture of the apparatus by any instrument maker',[167[p169]] proper practice came 'only by the skilful use of an apparatus specifically adapted in detail for use upon man, and through practical knowledge which it has taken [Fell] some years to become acquainted with'. He criticised most fully any kind of FR by 'unskilled persons'[164[p130]] when the life of a human being hinged on it. What was needed was 'careful attention to the details of practical import ... not by slipshod methods which in the past have relegated this operation [FR] to oblivion and which methods some are now trying to revive'.[164[p131]] He criticised Wood for advocating '"cheaper apparatus" with common bellows',[163[p345]] but did concede its use for an emergency (although 'with a feeling of hesitancy') if nothing else was available.[162[p183]]

After the inspector of life-saving stations on the Great Lakes 'was much impressed by the results of my method per tracheotomy, but admitted that it not be put in operation by crews at life-saving stations'[165A[p83]], Fell advised that they 'can readily be instructed to use the method per face cup when it would be entirely

impractical without'[165A[p83] and 'the method per face mask makes it par excellence the only method which should be used at these stations'[165A[p83]]. He advocated that 'a ship's crew [also be] taught to utilise this valuable method of saving lives'.[165C[p122]]

The effective use of any apparatus required it to be available beforehand, yet 'physicians will generally fail to be forearmed, even if they have been forewarned'.[161[p317]] Although he reported two years later that 'lives have been saved by several physicians who have utilised my methods',[162[p176–7]] apparently that was for poisoning alone, not for other needs, such as drowning, asphyxia or traumatic shock. By 1891, he could muse that 'with only 5 or 6 physicians of the 90,000 in this country prepared, as far as I know, to use this method, it would hardly do to ... now retire on my laurels'.[162[p190]] Lamenting how many saveable lives had been lost from the inertia of physicians, in 1899 he was still ruefully commenting, 'That it [a rescue by FR] has not come into general use, can only be through the belief of medical practitioners that it will not accomplish all that I have claimed for it', although 'there is no impediment in the way of general adoption by the medical profession'.[167[p169]] One solution Fell proposed, which evidently fell on deaf ears, was that every medical student in the country be required to know how to use the Fell apparatus.[167[p170]]

Fell's publications repeatedly indicate his disappointment at not being able to penetrate the bulwark of medical conservatism, scepticism, opposition and criticism (e.g. of FR being 'unnecessary, useless and unjustifiable').[163[p342]] Many contemporaries could have been put off by the opinionated forthrightness of his messages with such an inconvenient truth—that there were lives out there that could be saved but were being let go because of timidity or the lack of willingness to intervene. But what would have seemed worse—it must have produced outright fury among his readers—was his 1899 aggressive accusation of the 'almost criminal negligence which has so fully taken possession of the medical profession regarding the value of the Fell method of forced respiration'.[167[p170]] Further, a medical teacher was 'committing a woeful sin of omission in turning out students disqualified to practice forced respiration as I have given it to the world'[165[p121]]—certainly strong criticisms. And while Fell knew that his method might seem sinful to those respecting Marshall Hall's views, he declared, 'So I am a medical sinner and a bad one, for I glory in my sin, which has now saved a few hundreds of human lives and is destined to save thousands'.[167[p170]]

When Fell needed physical help with his own cases, he could call on his students—it seems he formed a band of dedicated assistants, both physicians and students. For his third patient, his class of students aided him throughout the night[160[p49]]; six students helped for the 1899 patient, Archer,[167[p170]] 'an army' helped with Patient 18[162[p186]] and other doctors were often in attendance, to an extreme of a named 20 (plus his loyal team of six medical students) for his famous rescue of Dr Henry Williams in 1896.[166[p763]] A dozen hours of FR treatment using a face mask must have been very tiring for the operator, and Fell recognised that this human labour, needing substantial manual energy, should be supplemented for the physical task. Hence, he had a 'blower [which had the disadvantage of being noisy] for force purposes run by hand and crank power'.[167[p171]] It seems he was not able to realise on his 1888 suggestion to substitute electromotive power for hand power.[159[p353]]

2.8 Wider applications of the Fell method

Fell repeatedly stressed the possible wider applications of his method. Already with the reporting on his second success, Patient 3,[159] he was seeing the applicability of FR for drowning, and in late 1888, when reporting on FR Patients 1–6, he called for his apparatus to be so used and predicted that FR would be the most reliable agent 'in almost all cases of asphyxia, from whatever causes'.[160[p64]] From the usefulness demonstrated for shock in his second and third FR patients, he inferred FR *'*may give us results in shock to be attained by no other means, or may serve as an accessory to other methods of overcoming this bane of surgical progress'.[160[p65]] In 1891, after crediting FR with saving 15 lives, Fell was forecasting FR for *'*general use in cases of drowning, shock, the tiding over of critical cases, in asphyxia from whatever cause, as well as from narcotic poisons*'*.[149[p330]] As mentioned, it is unclear whether he ever was able to implement such uses himself. But meanwhile, Joseph O'Dwyer was developing early improvements on Fell's original design to widen its applicability[100] (Footnote 2.8[8]).

In March 1896, in the discussion that followed Fell's reporting the successful rescue in Buffalo of Dr Henry Williams—who required about 80 hours of FR for an opiate overdosage[166]—he received strong medical support (as well as a 'commendation for his unwavering appreciation of the value of his work') and the suggestion that the American Medical Association bring the attention of the US Government 'to the value of Dr Fell's method as a life-saving appliance'.[166[p763]] It appears that, during a time of what was called unjust and unreasonable conservatism in a progressive age, nothing worthwhile was taken up. But following the Williams report, Fell was advocating using FR for more venturesome possibilities he foresaw, 'never before contemplated in surgical procedures, such as the opening up of the thoracic cavities under conditions we dare not consider [without FR] ... and in conditions of shock, in drowning, and in many other contingencies'.[166[p763]] Note that this preceded Rudolph Matas's epiphany in 1897 ('thoracic surgery was on the eve of a revolutionary innovation'[122,123[p1469],179[p97]]) over the use of FR for intrathoracic surgery by the intralaryngeal route, to be elaborated on in Chapter 3.

2.9 Intracranial disasters and neurosurgery

In a further surgical field Fell had noted, without supplying the year for it, that London neurosurgeon Sir Victor Horsley (1857–1916) appreciated that with various intracranial disasters, the final common pathway to

> death is due to failure of respiration, and where death threatens from intracranial pressure[,] artificial respiration should be performed and the skull opened freely.[166[p763-FN]] So in many instances it is as important to perform forced respiration as if the case were that of drowning man.

[8] Footnote 2.8. By introducing laryngeal intubation instead of using either a tracheotomy or Fell's face mask, Joseph O'Dwyer refined Fell's original FR system into what he courteously named the 'Fell–O'Dwyer apparatus' (to be described in Chapter 3). The first documented case history[120] of its use for a patient appears to be William P Northrup's in 1894, followed the next year by a fuller description and illustration of the apparatus by James Voorhees.[178] By 1910 at least, Fell had come to accept the Fell–O'Dwyer apparatus with apparent good grace, while still referring to it as 'the so-called'.[168[p574]]

Fell believed that in many cases of brain surgery, altogether too little attention was given to support by artificial respiration, but now FR was available to open up new fields. He was at that time aware[166[p763-FN]] that Northrup had already applied IPPV for a neurosurgical patient, using the Fell–O'Dwyer apparatus, in 1894,[120] but Fell in his writings never seems to have quoted Northrup's 1896 series from Presbyterian Hospital of 10 critical interventions[120,146] with FR, using the same system (see Footnote 2.8).

2.10 Intrathoracic surgery

In June 1899, Fell was still lamenting that FR's 'utility in association with surgical operations has yet to come, and surely will do so'.[167[p169]] However, he must have felt satisfaction on learning that Frederick William Parham, 1856–1927 (after investigations by Rudolph Matas for him in May 1898),[121[p223]] had already taken up advice to use the Fell method of IPPV, first on 6 August 1898, with the anaesthesia being provided for intrathoracic surgery.[121] The apparatus was later modified by Matas himself, as reported in 1900.[122,123[p1472]] The first notification of Parham's pioneering use of the Fell–O'Dwyer apparatus for this intrathoracic operation seems to have been in a footnote[180[p434]] to an article just preceding Parham's definitive account.[121[p319–24]] Parham was most enthusiastic (also see Footnote 2.9[9]):

> the credit belongs to Dr Fell, of Buffalo, for giving to surgery an apparatus embodying in its practical evolution the principles so ardently urged by Fell and O'Dwyer. As far as I can see I am the first to demonstrate the value of this admirable apparatus ... in maintaining the respiration during operations of this kind.[121[p224–5]]

(It should be noted that Matas, as per Footnote 2.8, wrote a valuable historical survey[122,123] that included careful detailing of the experimental work by French [and other] investigators during the 1890s, and earlier, on anaesthetic systems to enable safe intrathoracic surgery, by 'insufflating the lung through an intralaryngeal tube'.[122,123[p1468]]). Parham wrote:

> The assistance rendered me ... was so striking that I can without hesitation indorse [sic] every word that has been said in its favour. Indeed, so imbued am I with its value that I believe no surgeon now would be justified in attempting thoracic resection without having the Fell–O'Dwyer apparatus to hand. I believe it will revolutionize this field of surgery, making possible operations in the chest that would otherwise be clearly too hazardous to be justified.[121[p348]]

[9] Footnote 2.9.[121] Chloroform was administered by 'the interne' [sic] for Parham to remove a large chondrosarcoma extending from the clavicle to the sixth rib. After atmospheric pressure almost completely collapsed the exposed lung, and the patient showed profound shock:

> Dr [J D] Bloom was requested to begin the use of the Fell-O'Dwyer apparatus. As soon as the tube was inserted and the apparatus working, the lung began to recover itself, and the man's condition at once improved. The respiration was now admirably maintained, so the operation was proceeded with.[121[p321]]
>
> The assistance rendered by the Fell-O'Dwyer apparatus was evident to all. Whenever there was any hitch in working the apparatus, the lung at once showed signs of collapsing, but when the apparatus was in working order, respiration was almost as regular as the normal breathing. I can imagine no better demonstration of the usefulness of this admirable device.[121[p322]]

Postoperative fevers and abscesses delayed the patient's successful hospital discharge until 7 November (i.e. in 4 months).

In discussing Parham's paper, Parker added, 'I believe use of the Fell–O'Dwyer apparatus will do a great deal to advancing this line of [thoracic] surgery'.[121[p367]]

2.11 Fell and his priority rights

Fell's attitude (possibly a smug one) to Prof. Boehm's account of FR Patient 2 has been recounted in Footnote 2.3. When he felt he was not receiving due credit as the innovator of FR, he did not hesitate to write forcefully: for medical pioneers, it is 'unjust, unfair and unthankful that credit should be held from those who are entitled to it'.[164[p132]] Fell could be read[162[p181],164[p132]] as being somewhat aggrieved with Dr Jean Baptiste Vincent Laborde for recommending FR to The Paris Academy of Medicine (as in its *Proceedings* dated 2 June 1891), without acknowledging 'Fell's Method'. Laborde had devised a face mask for treating chloroform narcosis, one 'to "all intents and purposes," similar to the one I [Fell] have been using for some years, and with which I have saved a number of lives'.[162[p181]] Laborde's announcement (wherein 'Dr Laborde speaks of his invention as novel'[162[p181]]) took place after reprints of Fell's articles (from the *Transactions of the New York State Medical Association*) had been distributed at the Paris Exhibition of 1888 (but were ignored there by the US Government's medical representative, so Fell claimed[165C[p122]]). While at the Berlin Congress in 1890, Prof. Horatio Wood had drawn attention to Fell's face mask, which Fell said he had been using for the previous two years. According to Fell, *'*At that time they took no notice of my work, but appropriated it, utilised it, and now claim originality for methods which I long ago utilised and first recommended in practical shape to the medical world.'[162[p182]] It seems Fell did not allow for the possibility of coincidental discovery, if that could have been an explanation—perhaps 'they' had read Galen?—although he did concede later 'It may be, however, that they did not see them' [Fell's publications, left at the Paris Exposition of 1888].[165C[p122]]

Fell asserts on multiple occasions that he could find no evidence of any other person treating opium narcosis by providing prolonged FR. Thus, from 1891:

> I looked up the home and foreign literature on the subject, Nothing was discovered which appeared to controvert the fact that I was justly entitled to the credit of being the first to systematically and practically solve the question of the value of forced respiration in the saving of a human life; or that I had demonstrated, as one physician puts it, 'that air can be forced into the lungs without any damage to them'.[162[p179]]

(But regarding the latter, had he not heard of, for instance, Charles Kite or James Curry, at least?) 'To be denied the credit ... of a method ... in the saving of human life is what I could not and will not quietly submit to'.[165B[p103]]

Fell, who claimed, 'I have probably had more systematic operations of forced respiration upon man than all the rest of the physicians of the world combined',[165C[p124]] was also intensely patriotic: he described, 'its practical introduction to the world from this side of the Atlantic',[162[p181]] and 'in its success an entirely American idea'.[165B[p103]] But although his apparatus met 'all the requirements' for FR for humans, and 'in the simplest manner'[162[p180]] for the future, he would 'not question that different mechanical devices [after his] might not be successfully used'.[165C[p123]]

2.11.1 What followed for George Fell?

Before 1895, Fell's publications had been either recapitulating in minute detail, or occasionally summarising, the cases of 28+ patients[165A–C] in which the Fell method had been used, but not every case he listed or described was one of his own. He produced two further articles before 1900, each including an extraordinary case of opiate poisoning: one, Patient 13, in 1896 needed 73.5 hours of FR (my calculation); Fell says 'several long intervals' within a time span he gives of 83 hours; then, another in 1899 needed 'over seventy-eight hours'.[167[p170]] After these, I can find no other medical publication of his until 1910.[168] Although that 1910 article contains no new FR experience, Fell gives an historical, well-illustrated perspective on his introduction of FR in 1887, and of the validation of his equipment for FR in his early cases. He also places the Fell method ('now termed by some the positive pressure method' [168[p573]]) in the context of the other methods of his time for AV. Therefore, one may wonder: Did he actually stop his practice of FR in the new century, and, if so, why? From what I have read about Fell for this period, William Mushin and Leslie Rendell-Baker seem the only historians to comment. They alone make the particular statement, 'Though he retained his interest in microscopy he settled down in Buffalo as an ear, nose and throat surgeon'.[39[p154]] His obituary notices[169,172] mention illness, which culminated in his death in 1918. The brief biographies available are unhelpful for describing these last six years.

An obvious inference is that, even after the evidence of the successes he defined so clearly in his case reports, in the face of an unequal struggle to establish acceptance of his method of treatment into wider emergency practice, it all proved too disheartening for him. From 1887 to 1900, Fell was undaunted, pugnacious (if not aggressive), critical and even accusatory in his attacks on conventional attitudes to new ideas. His rallying cries for the adoption of FR, and his suggestion it be used for critical conditions other than overdoses, failed against the attitudes and conservatism, 'prejudice or ignorance',[167[p170]] of his critics and the sceptics. He had offered a life-saving system; the world had largely ignored it. Perhaps after a dozen years of struggle and example, he just tossed in the medical towel. However, that would hardly accord with either the previous character or the spirit displayed by this man of vision, this pioneer. (How many in desperation today would attempt a *domiciliary* tracheotomy, where that was unavoidable, for a convulsing neonate, just 18 days old, 'without proper apparatus'? [149[p327]]) His record does show continuing consultation in large projects in the engineering world.[172] Perhaps he just went back to a field where he was better accepted, one perhaps without the jealousies he says he found in medicine. Yet hardly so—because, as outlined in a brief obituary, this fiery spirit found plenty of opposition to his critical viewpoints concerning engineering controversies too, in 1903 and 1910.[173]

After writing the above, I came across Fell's second 1910 article[181] published in a medical journal: seven *JAMA* pages concerning 'The currents at the easterly end of Lake Erie and head of Niagara River. Their influence on the sanitation of the city of Buffalo, NY'. Fell had delivered the report that year at the American Medical Association Annual Session 'in the Section on Preventive Medicine and Public Health'.[181[p828]] Again, we can see Fell's courage and confidence—bolstered by his 'former experience as a hydrographic engineer'—in his opposition 'in toto to the views of the great majority of our healing physicians'.[181[p830]] The article concerned the risks of typhoid from epidemic debris brought by 'spring freshets' entering Lake Erie, whence the water supply of Buffalo City was drawn. (Again, this was all carried out 'at considerable personal expense, which was never reimbursed'.[181[p830]]) With his characteristic tenacity, he performed experiments

(I cannot find the year) to validate his arguments, confounding his critics. His absence from recording clinical medicine is thus not surprising—and my admiration for all his achievements increases.

2.12 Summary: Primitive intensive care medicine

Fell's activities, especially those of the first dozen years, warrant recognition for his pioneering role in ICM. He dealt with critically ill patients, some with severe multisystem disorders. To save their lives when conventional treatments had failed required an extraordinary intervention of skilled personal attention, at times in prolonged application (up to a maximum of around 80 hours of FR of one occasion recorded). Fell took on cases of indisputable severity at any hour of the day or night, stayed with his patients until too exhausted to continue and developed teams to share the burden of hard physical work. He recorded case histories and their features carefully, published repeatedly and shared his knowledge and equipment generously. He was always widening his vision, was desperate to teach his methods to others and wanted the same messages taught in the curriculum of every medical college. Ultimately, he failed to establish a system of rescue and to have his method widely adopted by professionals and trained paramedics, despite long striving for its universal uptake.

Fell improved a system used for laboratory animals, adapting it and making it safe for humans. But he also tried to simplify his equipment, with the aim of increasing its application. In the US of his times, he seems to stand alone in finding an effective alternative to inadequate methods of ventilatory support by the then current 'artificial respiration' from arm–chest manoeuvres. While he saw the need to prevent the unnecessary waste of human lives from opiate poisoning, he was determined to extend rescue to other, potentially remediable, life-threatening conditions. He foresaw other applications of his principles and methods, including 'tiding over' in critical conditions, such as traumatic shock, asphyxia or near-drowning; and for anaesthesia. He invoked ICM principles that we are familiar with today. He advocated readiness of equipment for emergency intervention and action, and then for sustained continuation. It was only later that he came to see that his IPPV method would be useful in neurosurgery, and later again that it could help manage 'the pneumothorax problem' of intrathoracic surgery. Others took up Fell's principles for these problems (see Addendum 2.1).

For all of Fell's efforts and attempts to contribute to medical practice and publicise something new and worthwhile (as in Addendum 2.2), he generally met a stone wall of resistance from the ranks of conservative medicine. Fell, however irascible and 'prickly', deserves to be remembered with admiration for his sterling efforts and his successes. His plea for recognition of his priority right for utilising IPPV in the saving of lives is warranted. It bears repeating the claim in an obituary in a medical journal:

> He gave his [FR] discovery to the cause of humanity and has made nothing from his work.[169[p74]]

Surely, a 'Pioneer Intensivist'.

Addendum 2.1: Some aphorisms of George Fell

In his writings, Fell invoked ICM principles, many of which we acknowledge today.

1888

- With suitable apparatus, keep up the respirations until all the poison could be eliminated.[160[p43]]
- A physician was not justified in giving up until life became extinct.[160[p49]]
- The surgeon who manipulates the valve is therefore responsible for any over-distension of the air vesicles.[160[p57]]
- Forced respiration ... [is] something more than a *dernier ressort* compared with artificial respiration.[160[p63]]

1889

- The only safe rule is to make the attempt [at rescue] because it is difficult to state at what stage preceding death it will not prove valuable.[161[p318]]
- He who would attempt to save human life by forced respiration must be supplied beforehand with suitable apparatus.[161[p317]]

1891

- Never permit a human life to be sacrificed for want of FR when you can procure a rubber tube ... face mask ... bellows.[162[p183]]
- Provision made to exhaust air from the lungs ... complicates the apparatus, and ... is not in accord with physiological conditions[162[p178–9]] [and] very dangerous ... in the hand of an average physician in an emergency case.[165C[p123]]

1892

- In emergencies it is difficult to obtain proper apparatus [which] ... must be supplied beforehand.[164[p131]]
- Give careful attention to the details of practical importance ... not by slipshod methods.[163[p345]]
- The life of the patient is not out of danger until the poison is eliminated from the system.[163[p346]]

1894

- Medical opinion must be moulded so that it will be considered hazardous to attempt to save life without proper appliances being provided beforehand.[165C[p126]]

Addendum 2.2: Some individual case histories of forced respiration

It may be obvious that Fell's numbering system is retained for the FR cases as they are in his publications, because he included some FR patients who were not his own. To exclude them would produce confusion with numbering 'his' patients. The best general reference for the numbered patients is Fell's own 1894 report in the *Canada Medical Record*,[165] supplemented by his reports in the *Transactions of the New York State Medical Association*[162] and the *Archives of Pediatrics.*[163]

The following are case numbers Fell assigned:

- **1:** Showed that FR can inflate lungs to save lives, without causing damage to the lungs. Peri-tracheal ligation was performed.
- **3:** (Fell's second FR patient). After artificial respiration had failed for a 'difficult' case and even when Fell delayed FR to a late stage, and was 'urged to discontinue' as the patient was 'considered hopeless'.[160[p46]] FR prolonged for 14.5 hours was successful: 'accomplished only through the new apparatus [Fell] had specially devised for use upon human beings'.[166[p760]] The patient was very seriously ill for the next 3–4 days. Fell was already seeing indications for FR with the drowned.
- **4:** With employment of a team of students and physicians to supply FR for 24 hours, and recovery achieved after the patient was already given up 'to all appearances dead (so reported)',[160[p47]] and a coffin delivered. 'A physician was not justified in giving up until life became extinct.'[160[p49]] The patient had provided his own [suicidal] entrance wound for the tracheotomy tube.
- As mentioned, Fell claimed that the saving of three lives (nos. 1, 3 and 4) saved insurance companies $23,000 in life policies.[160[p50]]
- **5:** FR for longer than 14 hours failed to save an 80-year-old man who had taken an opiate overdose. Fell was called to him at the local hospital.
- **6:** FR per a tracheotomy to an infant aged 18 days failed after 4½ hours' delay in calling for Fell's assistance after an accidental massive morphine overdose (of 1 grain = 64.8 mg; circa 70x[160[p46]] [or even 80x[149[p327]]] the infant's dosage). FR following 'occasional gasping breaths',[160[p52]] per a 1/8-inch (external) catheter, via a tracheotomy (and an increasing series of larger tubing back to the air valve) for 3½ hours was called hard work. Death was by misadventure from a homeopathy physician.
- (A stillborn babe receiving FR per a 'catheter in the trachea' was undoubtedly dead.[160[p53]])
- **7:** FR per a tracheotomy for a man (hospitalised) with spasmodic respiration following opium poisoning, whose return of wrist pulses lasted only an hour before cardiac action finally stopped. Fell said, 'I waited too long'.[161[p318]]
- **8:** This case showed that FR without tracheotomy is possible, by using mouth-to-mouth inflation. FR supplied through a tube sealed off within the mouth of a stillborn (forceps delivery, a ruptured brain: a hopeless case), abandoned after 4–5 hours. The case indicated the need for 'a suitable mouth-piece'.[160[p49]]
- **9:** A dying opiated patient, with the dilated pupils of asphyxia, received successful preliminary FR inflation per a mouth tube, then tracheotomy and FR for 11 hours, with survival.
- **10:** The same patient again, heavily opiated (2 fl.oz. tinct. Opii [=591.5 mg of morphine] plus 5–10 grains morphine [=324–648 mg]); tracheotomy for FR of 14 hours, recovered. Patient was certified.

Note: Regular FR for Cases 9 (11 hours), 10 (14 hours) and 11 (4 hours) was a success, while the patient in Cases 9 and 10 led to the preparation of the face mask covering mouth and nostrils, providing new options.

- **15:** Morphine overdose; face mask proved superior to artificial respiration, nine hours' successful FR, but the patient died after a colleague's interfering attempts at faradisation. Thus, death by misadventure, another's mistake (not Fell's). 'I do not recommend as yet intubation because I think there are many cases, in fact I have seen many, where it is not practicable owing to the difficulty of intubating the larynx'.[149[p329]] Fell predicted using FR would be useful for disaster conditions other than poisoning.[149[p329]]
- **17:** Morphine overdosage, 15 grains (=972 mg); after three hours of face mask FR, the woman's state deteriorated to heart sounds being absent, and hence, at tracheotomy, it was considered that there was no hope of survival, but more effective FR (which was evident) enabled recovery after 12–14 hours total of FR.
- **18:** With FR by face mask (10.5 hours); ligature needed for the obstructing tongue; serious gaseous distension of stomach and bowels; anuria; Fell's first extended use of oxygen; then tracheotomy. But after FR totalling 18 hours' work, the patient vomited, aspirated the vomitus and died. Fell recommended 'something other than manual labour'[165A[p85]] for the amount of energy expended for FR.
- **19:** Wrongly dispensed corrosive sublimate swallowed by a 73-year-old woman; face mask FR enabled her to live two days, but death by misadventure, the mistake of another. The energy expended in respiring for a human being, which was considered excessive, indicated the need for mechanical aid.
- **21:** A woman aged 78 years with opium narcosis received FR for 11 hours per face mask; a ligature through tongue was needed, then tracheotomy, but she had status convulsions and died. Fell recognised his error of judgement in waiting too long before intervening with a tracheotomy.
- **22:** Took morphine, 11 grains (=712.8 mg); Cheyne–Stokes breathing, almost in asystole before Fell arrived; FR by face mask for four hours was successful.
- **23:** A prisoner with opium overdose had FR by face mask but was cyanosed for 30 minutes; recovered after four hours. Venesection performed. She died unexpectedly of heart failure a few days later. Fell declared: 'intense congestion of the encephalonic vessels'.[165A[p87]]
- **24:** Laudanum overdose. Doctors at Fitch Hospital declared there was no hope the patient would survive after 5½ hours of artificial respiration; the patient's wife demanded Fell attend, and thus, FR was brought in, the patient recovered, but stupor recurred for days at home owing to a stove leaking natural gas; the patient recovered rapidly when shifted. Fell insisted hospitals maintain their own equipment to treat by FR.
- **25:** (1892, when '19 human lives have been saved by this [FR] method'.)[164[p130]] When two general practitioners could not use the FR apparatus on a man, 'reported as hopeless' after morphine overdose, Fell's student nephew who was 'ill', did so successfully.[164[p130]]
- **26:** Subcutaneous morphine, total 2/3 grain (43.2 mg) for a woman's colic. Cyanosis, and hence, artificial respiration for 5½ hours, but cyanosis was so profound that Fell and the GP thought she would die before a tracheotomy; however, she recovered after 5¼ hours' FR.

- **27:** Malnourished woman, opium poisoning, with respiratory rate of 3–4 per minute and cyanosis. Fell was unavailable, and therefore, 'for ten mortal hours we used [his apparatus] continuously' (or occasionally), with recovery.[165B[p99]]
- **28:** 7-year-old boy with diphtheria and life in immediate danger (dilating pupils). Tracheotomy, then FR on six to seven occasions, but tenacious membranous casts of tubes and trachea developed. Died after two days, of 'exhaustion and heart failure'.[165B[p100]]
- **29 to 44:** 16 patients of the Fitch Accident Hospital,[165B,C] of whom Fell lists: seven poisoned with opium, three with cocaine, one with carbonic oxide (CO), one with rat poison, one with ether narcosis, two with trauma, one drowned and one unspecified. Very brief notes on treatment are presented by Drs John Parmenter and EL Ruffner. There were nine survivors; the seven deaths were from opium (three patients, with two after 'long lying', and one from heart failure), drowning, brain injury, uraemia (after CO), pneumonia ('injury from house falling on him').[165B[p101]]
- **Unnumbered** anaesthesia cases: with a need for FR after the agents ether, chloroform or nitrous oxide.[167]
- **Unnumbered:** The remarkable 1896 case of Dr Henry J Williams.[166] This was the method's most severe test in a desperate case of overdosing with multiple opiates. A tracheotomy tube quickly replaced the initial face mask, and oxygen was also supplied. There were multiple alarms from failure of Fell's repeated efforts during that time to re-establish reliable spontaneous breathing. Three cycles of FR were given over a period of four days and three nights. 'The total time during which the apparatus was in constant use was nearly eighty hours'.[166[p763]] The treating team comprised more than 20 named physicians, plus medical students. Fell wrote, 'This speaks volumes for the perfect working of the simple apparatus'.[166[p762]] Fell also referred to this case in other publications.[167[p168], 168[p576]]
- **Unnumbered:** The remarkable 1898 case of Raymond Archer'.[167] At first, he was gasping at the rate of one breath per minute, cyanosed and almost pulseless after 33 grains (=2,138 mg) of morphine. He was given FR at his home: first, per a face mask for two hours, then per tracheotomy. Archer survived after FR 'was kept up … for over seventy-eight hours, constituting the longest period in the history of medicine in which a human being was kept alive by artificial means of respiration'.[167[p170]] Archer's was another case where during 'a tonic convulsion'[167[p169]] the tracheal ring failed to prevent aspiration, mainly of water that had been placed in the stomach, but without disaster following. Fell also referred to this case in another publication.[168[p576]]

19th century pioneering of intensive therapy in North America

Chapter 3: The Fell–O'Dwyer apparatus and William P Northrup

Too much credit cannot be given Dr Northrup for his persistent advocacy of this valuable apparatus, the invention of his lamented and ingenious colleague, O'Dwyer.

Rudolph Matas [179[p97]]

Chapter 3: Table of Contents

3.1 Introductory overview

In the two previous chapters, I have described the successful pioneering towards the latter part of the 19th century in North America of methods to treat impaired respiratory function in certain medical conditions. Joseph O'Dwyer designed, developed and successfully used his intralaryngeal tubes for treating airway obstruction, mostly in diphtheritic acute laryngitis. Meanwhile, to compensate for impaired breathing, particularly from opium intoxication, his contemporary, George Fell, had reintroduced 'forced' AV by his own system, either through a somewhat unsatisfactory face mask or through a tracheostomy tube. O'Dwyer replaced Fell's two delivery options with a longer orotracheal tube. After early experimental efforts by Drs O'Dwyer and Northrup, a combined system of intubation and AV by IPPV was then used on the initiative of surgeon Rudolph Matas, which delivered both anaesthesia and lung inflations to enable safe and successful intrathoracic surgery, and was first executed in the US by Parham. The combined system became known as the 'Fell–O'Dwyer apparatus', the use of which was widened, especially by Northrup, to include treating apnoea from intracranial disasters. Although the apparatus was used beyond New York—for instance, in New Orleans especially by Bloom for treating neonatal apnoea—it is difficult to find other specific references. Matas and Bloom improved O'Dwyer's original system, but after Charles Elsberg's clinical success of using continuous insufflation anaesthesia for thoracic surgery in 1909, American anaesthetists came to prefer that system, thereby delaying the uptake of routine IPPV by at least about three more decades.

3.2 The transition

At the American Pediatric Society's meeting on 23 September 1891 in Washington, DC, O'Dwyer reported that for medical patients with various respiratory difficulties, he had made 'some experiments six or seven years ago at the New York Foundling Asylum [NYFA], using the laryngeal tube ... through which air was forced into the lungs by means of [serial inflations of] a rubber bag'.[100[p33,34]] In 1944, Paluel Flagg (1886–1970) later confirmed that this application 'had been in use in the New York Foundling Hospital for six years, for some of the time when it was still in the experimental stage'.[110[p12]] The years that O'Dwyer gave for supplying IPPV in this way, endorsed by Flagg,[110[p11–2]] preceded George Fell's 1887 introduction of his FR method (equivalent to IPPV).[158]

After his own early experiments with FR, O'Dwyer was impressed by the results Prof. Horatio Wood obtained in his animal laboratory from applying the Fell method,[100[p32–3]] as Wood had presented them to the 1890 International Medical Congress in Berlin. Wood considered the Fell method remarkable, in contrast to the inefficiency of resuscitation by the Sylvester and similar methods. By applying Fell's method, Wood had reversed asystole and 'apparent death' from two minutes of apnoea after administration of ether or chloroform ('the heart had therefore ceased to beat' [100[p33]]). O'Dwyer designed a set of intralaryngeal tubes (dated by Rod Calverley[151[p335]] as first happening in 1888) to replace the Fell system, apparently first announced by Northrup at Bristol, UK, in 1894 (*v.i.*). At that time, the system included either a tracheostomy tube or a face mask, and as aforementioned, O'Dwyer introduced the prototype of what later came to be

called the Fell–O'Dwyer apparatus, to paediatricians in Washington in 1891. He first documented his improvements[110] the following January, without illustrations (see Footnote 3.1[1])[163,168,182]

The first depiction of the apparatus (as in Figure 3.1) seems to have been in an 1895 paper[178] by James Voorhees, house physician at the Presbyterian Hospital in the City of New York (Figure 3.1). It shows a (now longer) intralaryngeal tube with a distal tip (i.e. head), which O'Dwyer had described as 'conical in shape ... to tampon the larynx below the vocal cords, so that no air can return beside it' [100[p31]]—that is, a seal was obtained without an inflatable cuff. (In contrast, Fell had secured, and sealed off, his tube in the trachea, either by 'annular corrugations or a rubber tampon'.[158[p155]])

Figure 3.1: An early model of the Fell–O'Dwyer apparatus

The system comprises

i) inflating [foot-]bellows,
ii) tubing for delivering inflating air or gases to,
iii) the entry one of the two ports on,
iv) the metal intralaryngeal cannula (showing a conical, laterally grooved, detachable head to wedge in the glottis for an air-tight fit during inspiration), and
v) the exit port for the operator's thumb-tip control of inspiratory-expiratory cycling.

A Tiemann's commercial box of tubes and heads is also shown.
With acknowledgement and thanks to the Presbyterian Hospital, New York, for this figure from their medical and surgical reports for 1896, included[146[p132]] in Dr Northrup's paper (Chapter XII).

[1] Footnote 3.1. As referred to in Part 1 of my series of papers,[182[p385]] O'Dwyer's paper prompted a verbal onslaught from George Fell,[163] perhaps from having the title's word 'improved' preceding 'method of artificial forcible respiration'. Fell, it would seem, saw this as a criticism, or, worse, as O'Dwyer taking over his apparatus. What seemed to annoy Fell particularly was O'Dwyer's misunderstanding that Fell's Method was the same as that practised in laboratories on lower animals.[163] Fell issued a comprehensive nine-point rebuttal of O'Dwyer's errors over the Fell system, emphasising that his own current system, improved for humans nearly four years earlier, was much superior to his initial animal apparatus that O'Dwyer had assumed Fell was still using. After 18 years, Fell was speaking more kindly of 'Dr John [sic] O'Dwyer whose memory we revere'.[168[p575]] (The courteous O'Dwyer appears never to have spoken of an 'O'Dwyer–Fell' apparatus).

Re nomenclature: On occasions, I have seen the term 'Fell–O'Dwyer apparatus' referring solely to an O'Dwyer intra-glottic tube, whereas the true Fell–O'Dwyer apparatus comprised an O'Dwyer tube plus connecting rubber tubing plus Fell inflating bellows.

O'Dwyer did not personally report further clinical use of IPPV, apart from a single instance of a patient, apnoeic but conscious: 'During the past winter [hence, either late 1890 or early 1891?], I tried the same instrument in one case with the bellows attached ... for several hours', as apparently the patient 'lost the power to breathe for himself'.[100[p34]] O'Dwyer advocated his apparatus for medical, ventilatory purposes: 'inspiratory muscles ... rendered temporarily as useless, ... puerperal and other forms [of] eclampsia, and also in acute pulmonary obstruction from various causes.'[100[p33]] It had been for the latter that O'Dwyer made his NYFA experiments, 6–7 years earlier.[100[p33–4]] And he quoted Fell's repeated demonstration of FR 'in a number of cases of opium poisoning successfully treated'.[100[p32]]

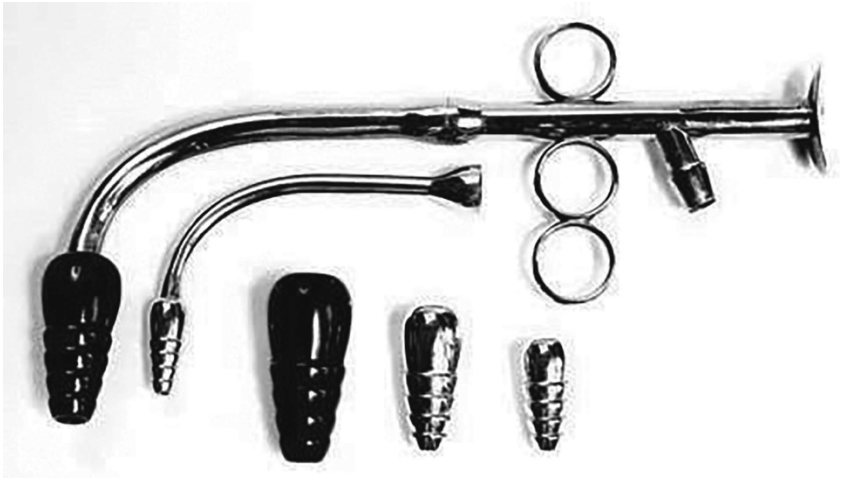
Although O'Dwyer's tubes were designed for the apparatus, he also recommended them as useful for control of the airway in certain surgical operations. Hence, originally, the new tubes were not only, as Rudolph Matas [1860–1957] said,[122,123[p1471]] for *non*-surgical conditions but also 'to prevent blood from entering the lower air-passages' during operations in or about the mouth.[100[p34]] William Northrup later reiterated this aspect.[146] (We can note that William Macewen had also designed and used oro-laryngeal tubes for that purpose in 1878.[124,126,127,183[p167]]) O'Dwyer's tubes were sufficiently wide-bore to be 'at the same time affording a free passage for the air to and from the lungs'[110[p34]] (see Footnote 3.2[2]).[100,102,120,178] O'Dwyer himself did not document either delivery of an anaesthetic by his improved system, or IPPV under surgical anaesthesia by that system. However, Keys noted that within two years of the description of the Fell–O'Dwyer apparatus, Karel Maydl in Prague had connected a Trendelenburg funnel to an O'Dwyer tube to modify it for 'satisfactory' oro-rhino-laryngeal anaesthesia.[124[p65]] Further, by 1896, Northrup stated: 'An anaesthetic may be administered through the intubation tube.'[146[p136]]

By the time of his 1896 retrospective, O'Dwyer was advocating the Fell bellows as the 'only ... means of producing efficient and at the same time prolonged artificial respiration' with the apparatus.[102[p18]] Surprisingly (and disappointingly), both Northrup's memorial address after O'Dwyer's death[104] and his 1904 address to graduates on O'Dwyer[105] (together with—nearer our own time—Gelfand in 1987[111] and Wiedemann in 1992[112]), while concentrating on O'Dwyer's struggles to establish satisfactory intralaryngeal intubation, do not appear to find room to mention the innovative Fell–O'Dwyer apparatus, or its application during anaesthesia, and that it was harm-free when used for 24 hours of IPPV.[120] Understandably perhaps, paediatric papers about O'Dwyer concentrate on his treatment for diphtheria.

[2] Footnote 3.2. For the definitive Fell–O'Dwyer apparatus,[100,120,178] O'Dwyer's metallic, oral intubating attachment had several sizes of detachable conical tips. Figure 2 shows an adult commercial set of three nickel-plated steel tube heads and two vulcanite heads, and the tubes onto which they were mounted. The laryngeal end was 'curved at right angles, on the distal end of which is a grooved cone which fits into the laryngeal socket'[178[p768]] (laterally) grooved 'to allow the vocal cords to aid in holding them down' [100[p32]] and designed to be of the right size to wedge itself into the larynx and prevent air from returning between it and the laryngeal wall.[120] The conical tip was graduated, so that each one of O'Dwyer's set of five heads would fit several size variations of the larynx.[102[p19]] The conical shape of the head allowed the tube to wedge between the cords into the larynx, thereby sealing off the respiratory tract, enabling IPPV and preventing the entry of blood, vomitus, etc., into the lungs.[100] Two long tubes (one adult size, one paediatric)[100] featured two ports proximally: one for entry of the inflating gas from a Fell system of bellows and rubber delivery tube; the other to be stopped intermittently by the thumb, which the operator could remove rhythmically, to act as a valve,[102] allowing intermittent exit of expired air. (Fell always emphasised the need to allow sufficient time for expiration.)

The Presbyterian Hospital of New York was the first to own and employ the apparatus.[146] O'Dwyer's colleague and admirer, William Perry Northrup[184] (see Footnote 3.3[3] and Figure 3.3), reported in 1894[120] on the first clinical use of the apparatus for one patient at that hospital. He also stated that this apparatus had been used several times at the Presbyterian Hospital and at the Foundling Asylum. It 'obviated the necessity of tracheotomy and removed the embarrassments of relaxed tongue and larynx'.[146[p128]] O'Dwyer's 1892 paper[100] simply refers to the Fell–O'Dwyer apparatus being at the NYFA, without providing details. Voorhees,[178] who, in 1895, mentioned the use of the apparatus for neurosurgical-type apnoea, documented two patients with severe morphine poisoning who survived through the use of this apparatus. In addition, Northrup reported on these three patients, and five additional Presbyterian Hospital patients, in an 1896 case series,[146] as described later.

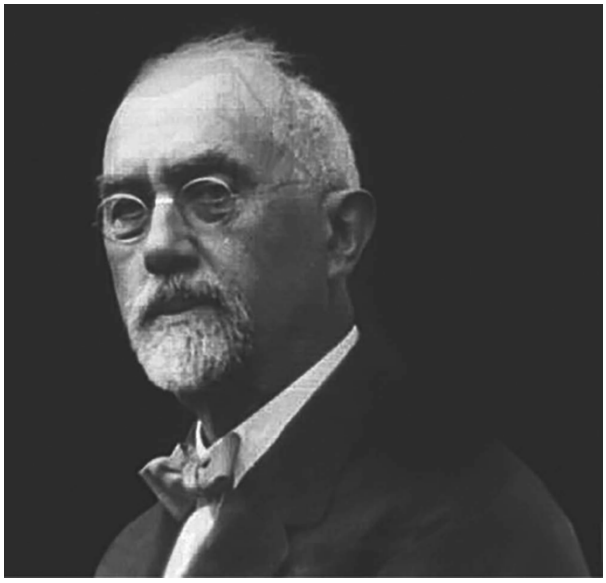
Figure 3.2: Cannula, tubes and heads for the Fell–O'Dwyer apparatus



A commercial set for the apparatus of two O'Dwyer tubes (one adult and one paediatric) and five exchangeable heads.
With thanks to Wellcome Images, Biomedical Collection, Wellcome Library of Medical History, London, for these illustrations.[185]

[3] Footnote 3.3. Paediatrician, physician, pathologist, William Perry Northrup (1851–1935)[184] was born near Syracuse, New York, graduated in 1868 from the College of Physicians and Surgeons, practised in New York City and then became a pathologist at the NYFA where he came under the influence of Joseph O'Dwyer. Later, he became an attending physician at the Presbyterian Hospital. He also features prominently in the other parts of this trilogy (Chapters 1 and 2).[183] An obituary notice regretted that his 'important work concerning the value of fresh air ... has been little heeded by the bulk of the profession'.[184[p32]] He was summarised as 'a man of general culture, a good physician, a wonderfully attractive lecturer, and teacher'.[184[p34]] His renowned humour was exemplified when presenting himself for a World War I field posting as 'Baby specialist, but I'll give up *infantry* and take to *adultery*'.[184[p32]]

Figure 3.3: Dr William Perry Northrup (1851–1935)



Northrup was a strong promoter of the Fell–O'Dwyer apparatus.
With acknowledgement and thanks to the National Library of Medicine, Bethesda MD, for this photograph.[186]

3.3 Historical context of the introduction of the Fell–O'Dwyer apparatus

3.3.1 The vision of Rudolph Matas

During the last decade of the 19th century, the oft-quoted admonition from Johannes Friedrich Dieffenbach (1792–1847) that the surgeon 'should halt at the pleura',[122[p1371],123] 'was religiously observed until comparatively recent years by the vast majority of surgeons when attempting the extirpation of tumors of the thoracic parietes'.[187[p411]] But '…thoracic surgery was on the eve of a revolutionary innovation':[122,123[p1469]] Matas[122,123,179,187] noted much experimental work carried out, especially during the 1890s, principally by European surgeons (many French), on direct, intralaryngeal inflation of the lungs using positive pressure (other than by the 1891 Fell–O'Dwyer apparatus). He saw that such a manoeuvre could resolve the great 'pneumothorax problem', which was otherwise inevitable when surgeons opened the chest, and that it 'would appear the same thought had occurred to those surgeons [listed by Matas,[122,123,180] see Footnote 3.4[4]][187–190] independently of each other and about the same time'.[187[p431]]

[4] Footnote 3.4. The Matas papers[122,123,179,187] supply us with many names, nowadays easily forgotten, of pioneers in intubation and lung inflation from the 18th century onwards. Matas acknowledged independent, experimental work (which was separate from the Fell–O'Dwyer apparatus) on direct intralaryngeal inflation of the lungs for the intrathoracic problem. Although this took place from 1896 to 1897 especially, it also went back at least to 1872. Matas[122,123[p1468]] cited successively Péan, 1872 (unsuccessfully); Lambotte; Délorme, 1897; Milton, 1897 (successfully, but only used for a goat[188,189]); 'Tuffier, Quénu, with their associates Hallion and Longuet, and Doyen'.[187[p430–1]] Even in 1902, Matas[179[p97]] could not see any safe, simple and reliable alternative to the Fell–O'Dwyer apparatus in Europe, except Doyen's, for which Matas stated no clinical use was documented. (Notes: Jules Péan, 1830–1898; Albin Lambotte, 1856–1912; Edmond Délorme, 1847–1929; Herbert Milton; Théodore-Marin Tuffier, 1857–1929; J W Hallion, 1862–1940; Eugène Doyen, 1859–1916; Eduard André Quénu, 1852–1933; F Longuet. Péan was immortalised in Henri Toulouse-Lautrec's painting, called 'Une Opération de Trachéotomie—but see the article by Aronson and Ramachandran on this[190]).

When Northrup first described a clinical use of the Fell–O'Dwyer apparatus at Bristol, 1894,[120] his further anticipation was that in peri-oral surgery, it is expected that the patient is capable of automatic (i.e. spontaneous) respiration, but the use of the Fell–O'Dwyer apparatus to deliver an anaesthetic with intubation also protects the airway. Matas realised, in reading the 1896 report from New York's Presbyterian Hospital on the use of the apparatus,[146] that it would enable intrathoracic surgery to proceed safely and effectively.

3.3.2 'Insufflation anaesthesia' techniques

Because of the inadequacies of 'insufflation anaesthesia' techniques[39[p13],151[p336]] (see Footnote 3.5),[5] this new IPPV option was most welcome, at a time when other solutions proposed involved more extreme manoeuvres.[57[Ch.VIII]] These included the differential pressure ventilating chamber that Willy Meyer was investigating, Ferdinand Sauerbruch's sub-atmospheric 'pressure chamber',[39[Ch.IV–V]] which he had introduced in the new century, and the positive-pressure head-boxes of Brauer and others.[39]

The practical employment of the proposal from Matas[121[p223]] enabled Frederick Parham to resect a chest wall sarcoma on 6 August 1898, in an operation both Matas[122,123] and Parham[121[p223–4]] described as revolutionary for thoracic surgery. They both emphasised their endorsement of Fell's principle of IPPV, but, of course, its application was rendered considerably safer by O'Dwyer's improvements. Yet, it seems the Fell–O'Dwyer apparatus was not adopted in Europe (see Footnote 3.6[6]).[57,175]

Matas[122,123[p1471]] found similarities to the Fell–O'Dwyer apparatus in Eugène Doyen's experimental system, reported in 1897, but was inclined to think Doyen was unaware of O'Dwyer's apparatus. In fact, it is Doyen's 'simple and reliable' system that Matas describes as 'the first finished model of an intubating and insufflating apparatus for the systematic application of this treatment as a preventive of pneumothorax [during thoracic surgery]'.[122,123[p1469]] Apparently though, Matas considers it was not used on humans.[179[p97]] In the 18th and 19th centuries, innovative suggestions often seemed to be published for their promotion, without the authors

[5] Footnote 3.5. Re 'Intralaryngeal insufflation': One has to read carefully to understand what each writer means by 'insufflation' (and contrast it with 'inhalation'). In Matas's time, the former word might be used in the broadest sense of getting a gas or powder into a body cavity, and he applied that expression's action to the Fell–O'Dwyer apparatus and also to tracheal insufflation with a bellows. However, flow with the Fell–O'Dwyer apparatus was not continuous but intermittent, and hence, it was really providing FR (IPPV). Matas's phrase in 1900 may surprise some of us today, familiar with Mushin and Rendell-Baker's[39] careful, more specific delineation (first from 1953, then repeated by Mushin, in 1963[175]) of continuous flow, as below, which derived from developments in the method for insufflation in 1907–1910 (to be described later). American anaesthetists, 1910–1926,[191[p4]] then came to favour a continuous insufflation method rather than the struggling, parallel developments attempted for PPV[39[p52–66]] apart from with the proven Fell–O'Dwyer apparatus. Insufflation was described thus:[39[p13]]

> a catheter, generally with a bore rather small compared with the trachea, is inserted down to the carina and a constant stream of gases blown through it, so that a pressure of about 10-20cm H_2O is registered in the manometer on the apparatus. The gases escape to the exterior between the tube and the trachea [compare the Fell–O'Dwyer apparatus: expiratory gas returned via the wide intralaryngeal tube].

Further, 'In effect the conditions produced are those of positive pressure spontaneous breathing', although apnoea may almost develop. With the inevitable respiratory acidaemia, practice every 2–3 minutes was 'to interrupt the flow ... at regular intervals, and to allow the lungs to collapse'. (Also see reference 39, pages 67–77.)

[6] Footnote 3.6. The communication lag that resulted in some medical advances made in one country (e.g. the US) being unknown in another (e.g. Germany) is apparent from surgeon Ferdinand Sauerbruch (1875–1951) writing (exaggeratedly, as events proved) in his intriguing autobiography: 'In 1902, not one doctor in the world knew how to operate with any hope of success through the chest wall ... pneumothorax killed the patient immediately'.[57[p35]] The same applied to the lack of some European knowledge in the US, such as about the direct laryngoscope of Alfred Kirstein (1863–1922) in Berlin, invented in 1895—a time when most surgeons (ignorant of O'Dwyer) shrank from attempting laryngeal intubation as impossibly difficult.[39[p48]]

having made their own prior clinical confirmation of them. Footnote 3.6[39,57] about international consultation also needs considering.

Mushin and Rendell-Baker, in their masterly, well-illustrated 1953 history, *The principles of thoracic anaesthesia past and present* (now reprinted by the Wood Library-Museum, 1991, as *Origins of thoracic anaesthesia,*[39] with contents untouched), describe what appears to be the pioneering intrathoracic operation with IPPV on a human: they cite Tuffier and Hallion for a successful partial-lung resection in 1896.[39[p46]] Using the anaesthetic technique of 'one of us' (so described by Tuffier and Hallion, in translation), with which they had previously experimented in animals, their technique produced rhythmic inflation with bellows and a type of positive end-expiratory pressure, all of which they called operative artificial respiration by insufflation (see Footnote 3.5). This was before Parham's 1898 thoracic operation using the Fell–O'Dwyer apparatus[39] (and also before the intended mediastinal operation of Herbert Milton, MRCS[Eng], on 25 January 1897 in Cairo, who was ready to apply IPPV per bellows via a tracheotomy for operating on his goat, but neither intervention was needed[188,189]).

From a historical viewpoint, Matas[122[p1375],123] regarded that a rhythmical inflating device for neonates employed in Berlin in 1870 (and documented only in German[192]) by a 'Dr Truehead' (truly, it would seem, Dr Trueheart,[193] who later returned to Galveston, Texas), was, in principle, a complicated anticipation of the Fell–O'Dwyer apparatus. Matas labelled the latter the second American (IPPV) invention, as he regarded 'Truehead's' system entitled to precedence over O'Dwyer's. See the discussion in the Supplement to Chapter 3.

It can be noted that in 1910, Fell asserted[168] that his own endolaryngeal tube for the Fell–O'Dwyer apparatus was superior to O'Dwyer's, which may indicate that Fell's was available and possibly used in some places in the US at that time. But I cannot find Fell's design of his own tube for the apparatus, in 1910,[168[p580]] documented elsewhere.

3.4 Clinical application of the Fell–O'Dwyer apparatus

O'Dwyer, while mentioning only a single, clinical, personal experience of using the Fell–O'Dwyer apparatus,[100[p34]] advocated its value for all forms of narcotic poisoning, by then 'amply demonstrated by Dr Fell', [100[p32]] and also for conditions involving 'inspiratory muscle ... rendered temporarily as useless, as if paralyzed by spasmodic contractions, such as strychnine poisoning, puerperal and other forms [of] eclampsia, and also [as already noted] in acute pulmonary obstruction from various causes'.[100[p33]] With experience of attending 3,000 obstetrical deliveries, O'Dwyer did not promote his IPPV system for apnoea in the newborn, as he considered mouth-to-mouth resuscitation 'amply sufficient' for that purpose.[100[p34]]

The Presbyterian Hospital of New York (at least) kept one Fell–O'Dwyer apparatus ready, and Northrup reported on its first clinical use there at the 1894 annual meeting of the British Medical Association in Bristol, UK.[120] He detailed the delivery of 25 hours of FR (supplied by multiple hands—expert ones, such as those of O'Dwyer and others) for a woman suddenly apnoeic from presumed intracranial hypertension, attributable to a cerebral tumour. FR was stopped after 24 hours, leading to asystole 12 minutes later; but further IPPV—or to continue with the expression favoured then, FR—restored her heartbeat until, an hour later,

FR was stopped because of her absolutely grave prognosis (see Footnote 3.7[7]). But even in 1896, Northrup was erroneously repeating that 'Dr Fell's method is identical with that employed in laboratories'.[146[p127]]

The specific use of the Fell–O'Dwyer apparatus at the NYFA was not found documented.

3.4.1 Northrup's clinical series with the Fell–O'Dwyer apparatus

In the *Medical and surgical reports of the Presbyterian Hospital in the City of New York* for 1896, Northrup reported the clinical application of the Fell–O'Dwyer apparatus there for the first series of eight adult cases published, headed by his single 1894 patient, already mentioned.[146] The apparatus must have been readily available for that first use when the house physician, without experience in any type of intubation, performed it successfully and promptly, enabling him to supply FR using the apparatus.

Northrup credits an 'HP [or] HS' with initiating the first seven interventions, and Dr O'Dwyer himself re-inserted the tube into the eighth patient.[146] Further documentation of any other use of the apparatus at the Presbyterian Hospital appears lacking, which leaves one wondering how much further it was applied there—or in other places. Northrup appeared to be the strong advocate for its use in intracranial disasters, with O'Dwyer participating, for example, for Patients I and VIII. Brief details of the eight patients are shown in Box 3.1.

Northrup concluded that the Fell–O'Dwyer apparatus:

- is efficient for prolonged artificial FR;
- is especially suitable for opium overdose—it definitely saved three lives;
- prolongs life (temporarily) in cerebral disasters with apnoea;
- offers great promise in operation theatres for sudden respiratory failure; and
- is a tactile technique of intubation that can be successful without previous practice (and presumably, without a laryngoscope; see Footnote 3.8[8]).

[7] Footnote 3.7. Flagg,[110[p12]] in later referring to presumably this same patient (identified by him as the one presented to the 'British Medical Society' in 1894), might cause confusion by his statement that the patient recovered and lived for quite a period in good health under Dr Flint's care, whereas Northrup clearly described that, if she was the same patient, an autopsy was performed after FR was stopped for her.

[8] Footnote 3.8. Voorhees had already claimed that his case reports showed how easy it was to use the tube and bellows.[178] This tube for the Fell–O'Dwyer apparatus was longer than the smaller O'Dwyer tube for diphtheria. O'Dwyer himself had expressed concerns over difficulties in intubating with the latter, in discussion at an 1887 New York meeting.[97[p318]] (But Northrup, speaking after him, reminded the select audience that they had, as he had also, 'in the aggregate used the tube several hundred times and had never met with untoward accidents'.[97[p319]])

Box 3.1. Clinical applications of Fell–O'Dwyer apparatus reported by William Northrup in 1896[146]

Patient I.	Brain tumour, apnoea; 25 hours FR, multiple rescuers; given up (O'Dwyer and Northrup attended).
Patient II.	Acute opium poisoning, deep coma, apnoea; about 3 hours of FR; recovered.
Patient III.	Ditto, breathing (4 breaths/min), cyanotic; not severe, only 20 minutes of FR; recovered.
Patient IV.	Cerebral haemorrhage, coma; trephined, breathing (4 breaths/min); 1–1½ hours of FR (abandoned?); died.
Patient V.	Brain trauma, coma, moribund, breathing (4 breaths/min); 8 hours of FR, but then became asystolic: died.
Patient VI.	Cerebral haemorrhage, coma, apnoea, operated; 6 hours of FR; died.
Patient VII.	Acute morphine poisoning (12 gr = 778 mg), breathing (4 breaths/min); intubated, 7¼ hours of FR; recovered.
Patient VIII.	Ditto (30 gr = 1.94 G), apnoeic, cyanosed; intubated twice for 9 hours total, FR time not given; recovered.

Four patients survived, but they were ones with opiate overdose alone, and not cerebral catastrophes.

Voorhees described Patients VII and VIII having 'very bad ones [poisonings], almost in extremis'.[178[p769]]

Northrup noted for Patient VII that the stomach tube and laryngeal tube were in position and in use simultaneously, and for Patient VIII, that it took an hour to get the stomach tube down the oesophagus, past the laryngeal tube.

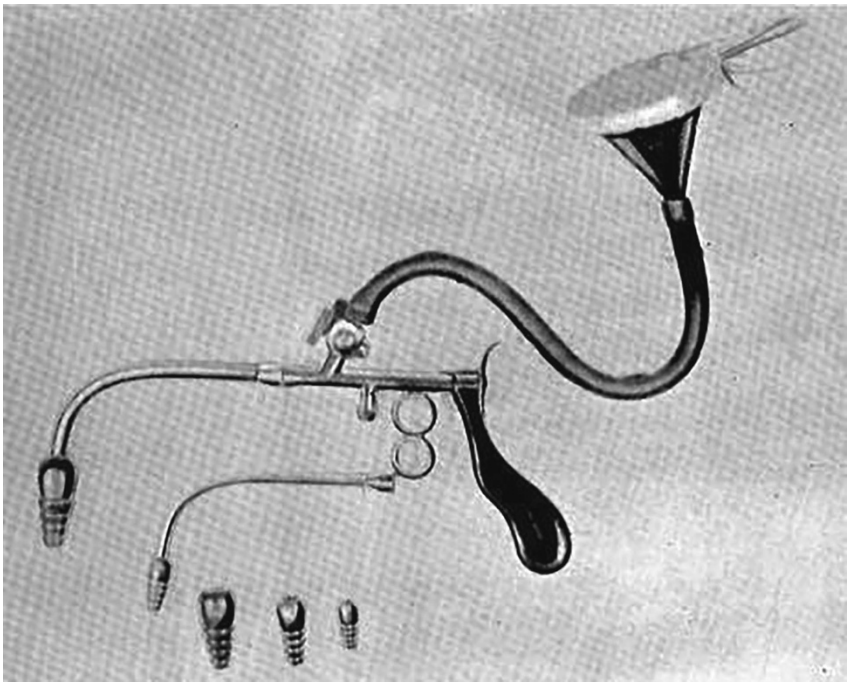
3.4.2 Further modifications to the Fell–O'Dwyer apparatus

While steadfastly maintaining the principles set forth for the FR form of IPPV, first by Fell, then O'Dwyer, subsequent developers modified the pump and other components, rather than continuing with a form identical with O'Dwyer's original Fell–O'Dwyer apparatus. Thus:

- 1900: A modification by Matas[122,123] enabled anaesthesia to be incorporated into IPPV when required (see Figure 3.4): a sidearm to the cannula had a rubber tube and funnel attached for administering the anaesthetic agent. The handle was now shaped like that of a pistol for better control, and a manometer enabled better control of inflating volumes.
- 1900: Dr J D Bloom's *'*adaptation of the O'Dwyer intubating canula for intraglottic insufflation in the treatment of asphyxia neonatorum' had 'a syringe rubber bulb with a valve at its free end to permit the entrance of air', illustrated by Matas (as his Figure 3).[122[p1374],123]
- 1900–1902: Bloom's modification of the original Fell–O'Dwyer apparatus had labour-saving additions (see Figure 3.5), all illustrated by Matas (as his Figure 10),[122,123[p1472]] such as a long lever for compressing the bellows, an in-line air filter and also a source of oxygen for inflation in asphyxia.

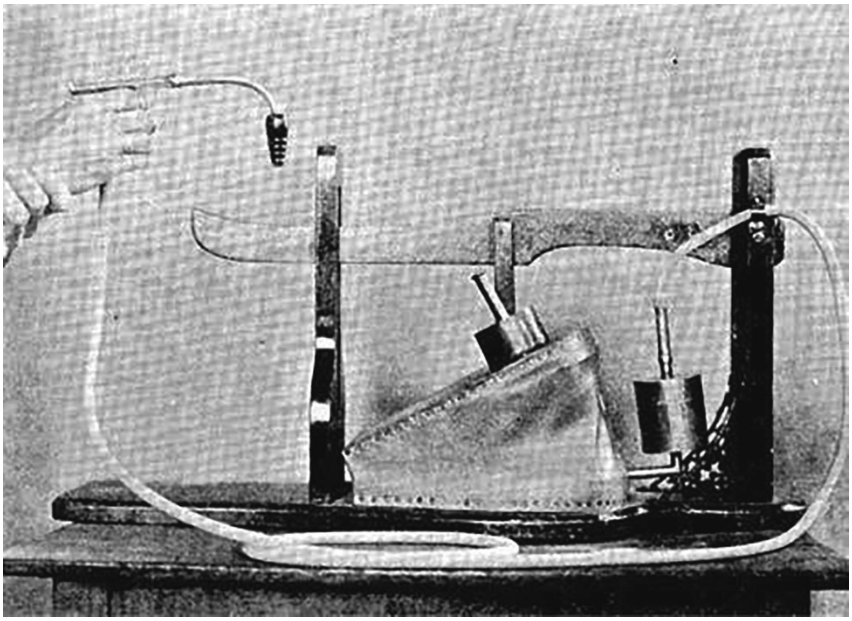
- 1902: Matas and Bloom, with the aid of engineer Dr John Smyth, while retaining O'Dwyer's intralaryngeal tubes, replaced the bellows, which they considered inadequate, with an 'experimental automatic respiratory apparatus' because of 'certain conditions met in surgery which differ radically from the conditions met in purely medical cases'.[179[fig.3,4]] Matas described its use only on a dog or on a cadaver, not live patients. In fact, Anthony Dobell noted (correctly?) that Matas apparently did not use the apparatus on patients himself.[189] (Matas is much revered in the US,[194] and William Osler spoke of him as 'the father of vascular surgery'[194[p885]]).

Figure 3.4: Matas's modification of the Fell–O'Dwyer apparatus for anaesthesia



Matas added a cone with an on–off tap to enable the passage of anaesthetic supplement into its delivery tube and also a pistol-shaped handle for easier control. When attached, bellows delivered inflating air, with or without anaesthetic gas.
With acknowledgement and thanks to the American Medical Association for this illustration from JAMA. 1900 Jun 9;34:1472, Figure 11.[123]

Figure 3.5: Bloom's modification of the Fell–O'Dwyer apparatus for anaesthesia



This model can inflate with oxygen. It also has a long lever to ease the exertion of compression of the bellows by foot (or hand), and a cylinder of filtering cotton in the circuit.
With acknowledgement and thanks to the American Medical Association for this illustration from JAMA. 1900 Jun 9;34:1472, Figure 10.[123]

3.4.3 Usage of the Fell–O'Dwyer apparatus by others

It is unclear not only how widespread was the use of the Fell–O'Dwyer apparatus, but also how much, or how little, unrecorded use was happening in the US, where acute opium poisoning was not uncommon. Were others not treating that, or other respiratory insufficiency such as those experienced by several of Northrup's neurosurgical-type patients, by any better method than arm and chest manoeuvres? Was Sir Victor Horsley's advice[166[p763]] not heeded for neurosurgery, or were Northrup's case-example treatments[146] not imitated by others? How much use for thoracic operations followed Parham's initial operation? If Matas had not written in 1899[187[p430]] that Bloom first introduced the Fell–O'Dwyer apparatus to the Charity Hospital of New Orleans, we would hardly have known of its presumed use[187] other than at New York or Buffalo, or by Matas and Parham elsewhere in New Orleans. Concerning *'*acute opium-poisoning, for which [the Fell–O'Dwyer apparatus] is admirably adapted' and 'used thus far most extensively,' Matas stated it had 'already saved several lives in the practice of [Bloom's] Hospital'.[187[p430]] And although Hutson and Vachon confirmed 'For years the Fell-O'Dwyer intubating canula had been used for the treatments of nonsurgical opium narcosis and of acute obstructive laryngitis in diphtheria', their further statement, 'as well as in the resuscitation of drowning victims around the country',[194[p888]] needs confirmation.

Instead of favouring the Fell–O'Dwyer apparatus, US anaesthetists preferred continuous intratracheal insufflation, which is considered[191[p15]] to have been introduced to anaesthesia in 1907 by Barthélemy and Dufou de Nancy[195] (see Footnote 3.9[9]), who documented it for a mandibular hemi-resection, even though its nature was assisted respiration, which is nowadays called ventilatory assist. Yet, Gillespie describes that these innovators were 'apparently unaware of the work previously done in Scotland, Austria, Germany, and Holland'.[191[p15]]

However, the use of continuous intratracheal insufflation flourished for thoracotomy, especially after the boost from its first successful clinical application on 20 February 1910[196] by Charles A Elsberg (1871–1948) (acting anaesthetist,[196[p27]] and subsequently neurosurgical pioneer), following on his own experimental work and that of others (see Footnote 3.10[10]). Elsberg's electrically powered clinical apparatus, in readiness at the patient's bedside,[196[p26]] already had its first reported usage in December 1909, for a medical patient, a myasthenic woman suddenly totally apnoeic. When deemed 'hopeless after six hours of continuous intratracheal insufflation', she was allowed to die.[196[p25–6],198[p495]]

3.4.4 Other intermittent positive pressure ventilation efforts for thoracic surgery

In the first decade of the new century, there were many other experimental efforts in the US (as well as in Europe where, however, Sauerbruch's influence dominated in the early decades) to produce a satisfactory IPPV system for thoracic surgery. This is well described in the authoritative and detailed accounts for the period from Mushin and Rendell-Baker.[39[Ch.VIII]] Innovative investigators before World War I included L Brauer, S Robinson, FT Murphy, W Meyer, F Kuhn, H Brat and V Schmeiden, NW Green and HH Janeway, Läwen and Sievers, GM Dorrance, JH and AB Dräger, M Tiegel, F Lotsch, S Bunnell and H Morriston Davies.[39[p63]]

3.5 Were Fell and O'Dwyer intensive therapy pioneers?

The answer needs to be 'yes' from both adult and paediatric standpoints. Both men produced appropriate instrumentation for their treatment by evolving methods, but for many years they had to battle disbelief, derision and opposition to establish their claims—Fell largely alone,[151] and O'Dwyer, with the strong support of colleague and key player Northrup, and through the enthusiastic adoption of his methods by others, such as Frank Waxham.[97,183[p162]]

[9] Footnote 3.9. Even Barthélemy and Dufou's original paper does not supply the forenames or initials of this pair. With each spontaneous breath of the patient, their endotracheal system[195] allowed ventilatory assist, from squeezing a hand-bulb ('*La soufflerie était actionée à chaque inspiration*') in circuit with a chloroform inhaler, thence to the endotracheal tube, to free the face area for surgery. Thus, it was intermittent inflation, hardly insufflation but IPPV, to maintain 'A constant partial distension of the lungs',[191[p4]] interrupted every 2–3 minutes.

[10] Footnote 3.10. In consecutive articles in the 1911 *Annals of Surgery* [52:23–9 and 30–3], acting anaesthetist Charles Elsberg (1871–1948),[196] and then surgeon Howard Lilienthal (1861–1946),[197] claimed that their operation of 20[196] or 21[197] February 1910, by employing Elsberg's modification of the Meltzer–Auer animal apparatus,[196,198] was the first case of thoracotomy in a human being under (ether) anaesthesia, for a foul, septic lung abscess[197] by continuous intratracheal insufflation of air (Meltzer), as Elsberg called it.[198] By means of foot bellows, his apparatus blew an (occasionally interrupted) air and ether stream at 15 mmHg pressure[196] through a tracheal catheter, of half the trachea's diameter; the method was then used for other patients.[196] Although Elsberg quoted his own and Alexis Carrel's experimental groundwork for 'the Meltzer method,' and although other originators were even earlier, he acknowledged physiologist Samuel Meltzer (1851–1920) and his physician son-in-law John Auer (1875–1948) for their work on curarised animals. (Without any normal or artificial rhythmical respiratory movements, the lungs were kept in continuous inspiratory distension, interrupted every 2–3 minutes.)

3.6 Summary

Initially, George Fell adapted a system from the animal laboratory, proving to be clinically successful in saving lives by [re-]introducing PPV.[158,165,168] He used it primarily for adults critically ill from opium poisoning, but also for a few cases of paediatric ventilatory failure, even in newborns. Fell strove valiantly by example, lectures and writings to popularise his successful Fell method. Chapter 2 documents his pioneering role.[182]

Joseph O'Dwyer took Fell's method and improved it by adapting his own airway tubes to it and thus increased its safety and widened its applications. The subsequent Fell–O'Dwyer apparatus further diversified the capabilities of that method by opening new fields in anaesthetic IPPV, most strikingly for thoracic surgery, and in rescue IPPV, such as for intracranial disasters.

However, what O'Dwyer is probably more famous for is his dedication to the problem of children dying from obstruction of the larynx by diphtheritic pseudo-membranes, at a time when the only possible alternative treatment by tracheotomy still left a very high mortality rate. O'Dwyer pioneered intubation directly through the larynx, but his significant success with that deadly disease came only after almost a decade of painstaking research to develop and refine his own method. Thus, where others had failed, O'Dwyer established the intensive care principle we use today of intubation for acute airway obstruction (which was such a relief for the fearful parents of O'Dwyer's patients, because it avoided the surgery for tracheotomy that they dreaded; see Footnote 3.11[11]). Yet, despite O'Dwyer being frequently described as 'the inventor of intubation' (e.g. by enthusiastic admirers such as JJ Walsh[106]), he was the *re*introducer.

Although the 'O'Dwyer principle' was established for diphtheria, the development of a diphtheria antitoxin, and then, a toxoid, early in the 20th century much reduced the need for his instrumentation. However, according to the Dittrick Medical History Center, Cleveland, Ohio, *his* intubation instruments did not disappear from the medical scene [in immunised communities] until the mid-20th Century.[112,199]

While Fell advocated the possibility of his own method for intrathoracic surgery,[194] O'Dwyer's invention and Northrup's publications both showed Matas the practical way. This resulted in the application of the Fell–O'Dwyer apparatus (although it had, as Matas put it, 'originated in other sources')[122[p1375],123] to prevent the problem of pneumothorax with open-chest surgery, unresolved hitherto, and to allow safer chest operations.

3.7 A final tribute to Joseph O'Dwyer

Rudolph Matas[122[p1375],123] summarised O'Dwyer's place thus:

> It is to O'Dwyer, therefore, that the greatest credit is due for establishing intubation in its present form, and it is due to his unswerving and indefatigable perseverance, patience and mechanical ingenuity that the present form of [Fell–O'Dwyer] apparatus has attained its marvellous efficiency.

[11] Footnote 3.11. Rudolph Matas compiled an impressive list[122[p1373],123] of dedicated, earlier intubators, from John Hunter and Monro Secundus onwards, including especially the numerous 19th century neonatal interventionists who intubated apnoeic newborns, defying the strictures, which was a reaction to the adverse findings of Leroy d'Étiolles, Magendie and A-M-C Duméril, 1827–1829.[36[p222],122[p1373],123]

Further: 'by similarly transforming the tracheotomy canula of the [Fell] insufflating apparatus ... for ... opium narcosis he has opened a new chapter of still greater interest and promise than that which his previous achievements had brought to a close'.[122[p1375]] (The meaning Matas implied for 'insufflating' was not the same as that of later anaesthetists.)

A last tribute to O'Dwyer is appropriate for his outstanding personal qualities,[104[p362]] which come through so readily in articles about him. Some are so adulatory as to seem hero-worshipping. Thus, within a decade of his death he was revered by such statements as Northrup's: 'the most godlike character I have ever seen in man',[105[p564]] and 'American medicine has no more shining light'.[106[p326]]

Again, from Northrup is that 'O'Dwyer left a memory among his colleagues of a purity of character, an uncompromising honesty and uprightness that was almost childlike'.[105[p562]] So, let Dr Northrup have the last words then: 'with this genius, there was all that goes to make a man'.[104[p164]]

19th century pioneering of intensive therapy in North America

Chapter 3 Supplement:
Early artificial ventilation:
The mystery of ‘Truehead of Galveston’.
Was he Dr Charles William Trueheart?

Chapter 3 Supplement: Table of Contents

3.8 Introduction

It seems surprising that medical literature from the US has only a single source for a novel device for AV in neonatal resuscitation. Dating from c.1870, its invention is attributed to 'Dr Truehead of Galveston, Texas'. The mystery of such a silence arises from separate misspellings of the inventor's name in the medical literature. I propose that he is correctly identified and named as Dr Charles William Trueheart (1837–1914), who was also of Galveston.

3.8.1 'Truehead of Galveston'

During an address to America's Southern Surgical and Gynecological Association in late 1898, Rudolph Matas[200[p65–6]] drew attention to a device enabling the supply of rhythmic AV during resuscitation of the apnoeic newborn. He referred to documentation of the use and construction of the device by a 'Dr Truehead of Galveston' in the German literature on obstetrics, 28 years previously.[192] Matas included this item of historical interest in his comprehensive 1900 article on 'Intralaryngeal insufflation' in the *Journal of the American Medical Association*[122[p1374–5],123] (see Figure S3.1). However, apart from the repetition of the Matas phrase 'Truehead of Galveston', first in 1933 by eminent anaesthesiologists Ralph Waters, Emery Andrew Rovenstine and Arthur Guedel,[201] and then in 1945 by medical librarian–historian Thomas Keys,[124] there appears to be no obvious medical record of any Dr Truehead, or any writings by such a person. In briefly referring to Truehead's apparatus only by quotation from Matas, these later writers shed no further light on the identity of its inventor.

That absence of further comment about such a ventilator caused me to try to identify 'Truehead', but I could find no medical Trueheads recorded, either in printed documents or on the internet. Initially, librarians at the Philson (Medical) Library, Auckland, were unable to locate the Truehead paper Matas had quoted. But on enquiry made to the Wood Library – Museum of Anesthesiology (in Schaumburg, Illinois), archivist Felicia Reilly suggested that 'Dr Truehead might really have been Dr Trueheart' (see Figure S3.2). An internet search revealed many Truehearts in Galveston and Texas—but no Trueheads.

3.8.2 Dr Charles William Trueheart of Galveston

Charles William Trueheart, born in Virginia on 27 February 1837, died in San Antonio, Texas, on 14 December 1914,[202] was the contemporary of any such 'Truehead', even to being in Berlin around 1870, the date Matas indicated Truehead was writing. Charles Trueheart is a notable figure in Texan history and, together with his older brother Henry Martyn Trueheart (1832–1914), was the subject of Edward B Williams's 1995 publication *Rebel Brothers, the Civil War Letters of the Truehearts* (see Figure S3.3).[203] This book provides some information about CW Trueheart's medical career.

Figure S3.1: Article in the *Journal of the American Medical Association* in 1900

The Journal of the
American Medical Association

VOL. XXXIV CHICAGO, ILLINOIS, JUNE 2, 1900. No. 22.

Original Articles.

INTRALARYNGEAL INSUFFLATION.

FOR THE RELIEF OF ACUTE SURGICAL PNEUMOTHORAX. ITS HISTORY AND METHODS WITH A DESCRIPTION OF THE LATEST DEVICES FOR THIS PURPOSE.

BY RUDOLPH MATAS, M.D.
NEW ORLEANS, LA.

From pp 1374-75: ....the apparatus described by Dr. Truehead, of Galveston, Texas, in 1869. (Ein Apparat Zur Kunstlichen Respiration bei Asphixia, Mitth. a.d. Sitz-Protok. der Gesselschaft f. Geburtsch. in Berlin, 1869-72, i., 154-156, 2 pl.) This rather complicated appliance was originally intended to insufflate air into the lungs by means of an intubating canula which was inserted into the glottis and larynx. There are at least two sizes of the intralaryngeal pieces, graded to suit the ages of the patients—chiefly newborn infants. The laryngeal piece is pyriform in shape, and is made to conform to the size and outline of the glottic orifice. It fits closely to the larynx, and its conical shape facilitates its tampon action. The mouthpiece is shaped like a curved catheter, and this is connected to a bellows which works on a vertical axis and automatically injects and aspirates air in and out of the trachea in a rhythmical fashion. As a true intubating canula and respiratory machine supplied by a bellows, it clearly anticipates—though it is a far more complicated way—the second American invention, with which we are familiarly acquainted as the Fell-O'Dwyer apparatus. Truehead's description is fully detailed and well illustrated in "Transactions of the Berlin Obstetrical Society" for 1869-72.

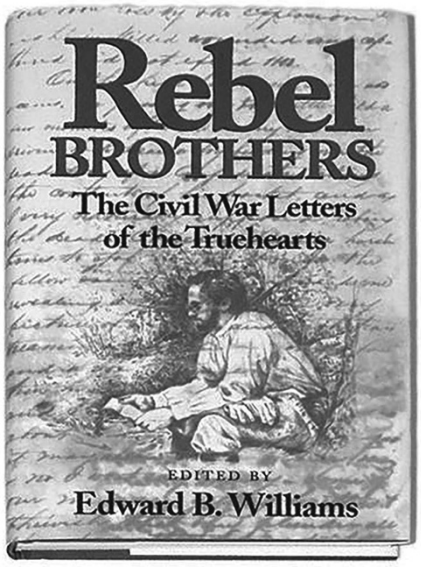
The passage from probably the best known of the publications of Rudolf Matas, about a 'Dr Truehead of Galveston', wherein he cited a paper presented by Truehead at Berlin. Mistakenly, Matas dates the paper as published in 1869 not 1870. He translates the title of the 'Truehead' paper from its German as 'An apparatus for artificial respiration in asphyxia' and refers to the publishing journal as *Transactions of the Berlin Obstetrical Society*.[122]

With acknowledgement and thanks to the American Medical Association for this illustration.

Figure S3.2: Dr Charles Trueheart

Dr Charles Trueheart, 1837–1914, at around 1866, when about aged 30 years.[204]

Figure S3.3: Dust cover of the book by Edward B Williams



With thanks to the Texas and A & M Press and the author, Edward B Williams, for this illustration of the dust cover of the book on the Trueheart brothers' American Civil War letters,1995.[203]

During the American Civil War, Trueheart functioned both as a soldier and as a medical attendant, while an undergraduate and then a graduate from March 1864 (see Footnote S3.1[1]). The account from Williams continues into post-war times:[203[p216]]

> For the next several years after returning to Galveston, Charles practiced medicine there. … [After his first wife's death, 1867, the year after their marriage] Charles continued his medical education, first at Bellevue Medical College in New York, and then in Europe at Göttingen, Germany [for 6 months[205]]; then [he studied a year and a half[205] at] Vienna, Austria; and [a year[205] at] Berlin. In 1870–1871 he served with the Germans during the Franco-Prussian War as a surgeon in their military hospitals. At the close of [that] war he returned to Galveston, where he resumed practice.

[1] Footnote S3.1. Williams states[203(p8–10)] that Trueheart started medical studies in 1858–1859, transferred to the University of Virginia in 1860 and served in a 'University Volunteers' military company from April/May 1861, until leaving university for private soldiering in a unit in the field in October 1861 (page xii). He participated in various campaigns, and then was a hospital steward from September 1862 until the following September, before returning to Richmond to complete his medical training by March 1864. He was assigned as an assistant surgeon to the 8th Alabama Volunteer Infantry Regiment. He transferred in December 1864 to the 1st regiment of Confederate Engineers to return to the war front, Petersburg VA. He surrendered with that unit at Appomattox on 9 April 1865, and then took part in the official surrender ceremony three days later.

Rick Cox reported in the *North San Antonio Times*, 11 March 1982, that reputedly, Charles 'brought the first fever thermometer to the United States upon his return from Germany' in 1871.[203[p216]]

In this regard, archivist Jodi Koste of Virginia Commonwealth University, Richmond, VA**,** advised (2008, personal communication):

> in 1872 he [Chas Trueheart] published an article on 'Conjunctivitis granulosa chronica treated by galvanization' that appeared in *Medical Record* (NY) 1872, volume 7, page 569. He listed himself as surgeon to 'The Eye, The Throat, and Ear Department' of Galveston City Hospital.

Later attainments in Trueheart's medical career are outlined online in *The handbook of Texas,*[202] while EB Williams lists engineering accomplishments (reminding us of Dr George Fell similarly practising activities[182[p390]] that were non-medical). Stephen Greenberg of the US National Institutes of Health's National Library of Medicine (NLM/NIH) unearthed for me Trueheart's dozen or so articles, but none of these is in German or concerned with artificial respiration. The Texas Physicians Historical Biographical Database[206] lists 23 Trueheart references, which include 10 of his papers.)

At this stage, I had reached stalemate as, apart from exceptional coincidences of time and place, there was no reliable indication that Trueheart was Matas's 'Truehead', an innovator in IPPV at Berlin around 1870.

3.8.3 'Herr Trueheard aus Galveston'

After considerable difficulty (because the author's correct name was not as Matas provided, and also because a 'Trueheard' seemed associated with the published German article; see Figure S3.4 for the journal), the author's name is first seen only in the fourth line of writing, a little down the article's front page, as in Figure S3.5. Librarians at the University of Auckland eventually located for me the article Matas quoted.[192] Unless Trueheart spoke in German, the article would seem to be a German translation of the English text of a lecture given by a 'Trueheard' (named as such without his initials in the publication) to an obstetrical conference in Berlin on 26 June 1870—a date three weeks before the outbreak of the Franco-Prussian War in which Trueheart served. On the publication of the text of the lecture two years later, authorship in the journal was attributed not to Truehead, as recorded by Matas, but to '*Trueheard aus Galveston*'. Based on this, Stephen Greenberg from the NLM/NIH, Bethesda MD, further suggested that the 'Truehead' I was enquiring about should be Trueheard. Obviously, searching was needed beyond 'Trueheart' to locate Matas's subject.

3.8.4 Trueheards in Texas

For seeking Trueheards at Galveston, the internet (Google Scholar) supplied a lone entry: in the *Sam and Bess Woolford papers for 1834-1979*, held by the University of Texas Library.[207] 'Lone'—except helpfully and one fortnight later, Australian and New Zealand College of Anaesthetist (ANZCA) librarian Jenny Jolley sent me another reference of the name Trueheard, from Michael Obladen's article on the history of neonatal resuscitation[208] coming coincidentally online on 9 July 2008. It was likely to be the first appearance of 'Trueheard' for many years. But when I saw that under 'Woolford' (on Google) were Trueheard forenames spelled as Henry Martyn—identical with those of Trueheart's brother, even to the less common spelling of Martyn—it dawned on me that this Trueheard was likely to be a Trueheart. Attempts by archivist Nikki Lynn Thomas at the University of Texas to locate the Woolford file's specific section were unproductive, and she believed that it is safe to assume that the Trueheard of the Woolford

Tales is, in fact, Henry Martyn Trueheart, as there is only one Henry Martyn of any importance that she could find in Texas history. Hence, in the Berlin publication, 'Trueheard aus Galveston' actually seems likely to be a misspelling for '[C W] Trueheart'. Felicia Reilly's original advice now appeared correct.

In personal communication to me in July 2008, Michael Obladen suggested that the name '"Trueheard aus Galveston" probably was misspelled in the proceedings of the Society of Obstetrics and Gynecology, and afterwards Knapp and others copied the misspelling from there. But this explanation is purely speculative of course'. My own conviction is strengthened that the Berlin Trueheard is a misspelling of (CW) Trueheart, who neatly fits the minimal life features known and attributable to Matas's Truehead. It has always seemed to me to be too great a coincidence that a Dr Trueheard and a Dr Trueheart, both from Galveston, went to Germany and Berlin around 1869, where Trueheard showed a resuscitating machine (Figure S3.6).

Figure S3.4: Copy of the front page of the 1872 German medical journal

Beiträge

zur

Geburtshülfe und Gynäkologie.

Herausgegeben

von der

Gesellschaft für Geburtshülfe

in Berlin.

I. Band.
Mit 15 Tafeln.

Berlin, 1872.
Verlag von August Hirschwald.
Unter den Linden No. 68.

From the *Proceedings of the Society of Obstetrics and Gynecology*, Berlin, published 1872.[209]
With acknowledgement and thanks to this Society.

Figure S3.5: The first page of the original article in the 1872 German medical journal featuring 'Trueheard auf Galveston' and his device

Nachtrag zur Sitzung vom 26. Juni 1870.

(Hierzu Taf. VIII., IX. u. X.)

Herr **Trueheard** aus **Galveston** hat nachstehende Beschreibung nebst Zeichnungen der von ihm am 26. Juni 1870 der Gesellschaft vorgelegten Instrumente eingesandt. (Uebersetzung aus dem Englischen).

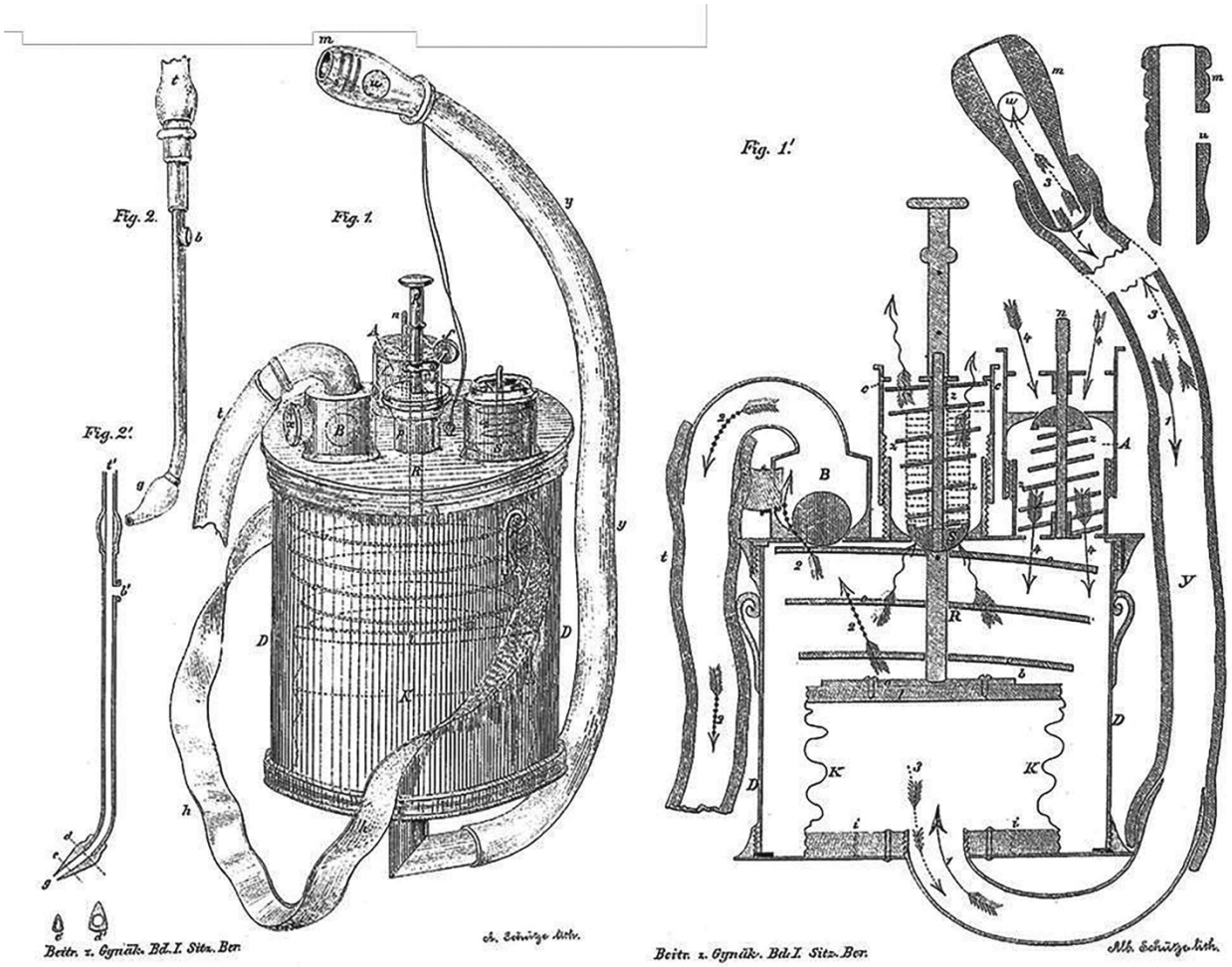
1.

Ein Apparat zur künstlichen Respiration bei Asphyxie.

(Taf. VIII. u. IX.)

From the *Proceedings of the Society of Obstetrics and Gynecology*, Berlin, published 1872.[209]
With acknowledgement and thanks to this Society.

Figure S3.6: Trueheart's apparatus in his original drawings, 1870



Matas wrote (in 1898,[200] and in 1900[122,123]): 'The rather complicated appliance was originally intended to insufflate air into the lungs by means of an intubating canula which was inserted into the glottis and larynx. There are at least two sizes of the intralaryngeal pieces, graded to suit the ages of the patients – chiefly newborn infants. The laryngeal piece is pyriform in shape, and is made to conform to the size and outline of the glottic orifice. It fits closely to the larynx, and its conical shape facilitates its tampon action. The mouthpiece is shaped like a curved catheter, and this is connected to a bellows which works on a vertical axis and automatically injects and aspirates air in and out of the trachea in a rhythmical fashion. As a true intubating canula and respiratory machine supplied by a bellows, it clearly anticipates – though it is a far more complicated way – the second American invention, with which we are familiarly acquainted as the Fell-O'Dwyer apparatus'.
Translation from the *Proceedings of the Society of Obstetrics and Gynecology*, Berlin (published 1872), for the *JAMA* of 2 June 1900; vol. xxxiv (no.22), p. 1375.[122]
With acknowledgement and thanks to this Society.

Thus, there is an 1872 article in German that documents a neonatal life-saving device, but, although a generously illustrated article, it provides minimal data on treatment. There are evidently no English-language articles that record its practical application. Michael Obladen's recent article[208] has an illustration of the device from a 1904 edition of Ludwig Knapp's book,[210] wherein it is pictured from Trueheard's original paper. In a personal communication in July 2008, Obladen did doubt

> that Adolf Gusserow (Berlin Chair of Obstetrics from 1878 to 1904) used 'Trueperson's' portable ventilator for neonates in his delivery room. [We communicated somewhat irreverently, about

'Trueperson'. With my apologies where due.] He was not too keen towards technical advances, and when the 1896 industrial exhibition closed (to which he had loaned eight pre-term infants believed chanceless) he did not buy Lion's incubators, despite the fact that six of the infants had survived. On the other hand, Ludwig Knapp's textbook of 1898 (*The apparent death of the neonate*) was so successful that a second edition appeared in 1904,[210] both of them referring to 'Trueheard's' respirator in text and figure. So, I assume the ventilator was used at least in 'Deutsche Frauenklinik zu Prag', where Knapp was professor and 1st assistant. Moreover, the device was manufactured by companies in two European capitals, Mattich in Berlin and Leiter in Vienna.

3.8.5 To summarise regarding the names

The evidence available to date appears to suggest strongly that:

- Matas's Truehead, in reality, was Charles William Trueheart.
- The Trueheard now deemed by me to be misspelled in the 1872 Berlin article illustrating a novel neonatal ventilator was not Trueheard but Trueheart.
- The Trueheard cited from the Berlin paper by Knapp in his 1904 book was Charles Trueheart.
- Henry Martyn Trueheard, said to be referred to in the Woolford papers at the University of Texas, was Henry Martyn Trueheart, Charles's brother.

I present a possible sequence to provide a logical explanation of the three names:

- Trueheart gave a talk in Berlin but its text, published after probable translation from an English text, had the final letter 't' of his name changed in error to 'd'.
- Another error arose when Matas quoted the article from the German text but omitted the final 'r' of Trueheard, giving the name Truehead (see Footnote S3.2[2]).
- Hence, Trueheard, correctly in my opinion, would need to be referred to as Trueheart, as will be employed hereafter.

3.8.6 Pioneering intensive therapy?

Matas regarded Trueheart's complicated IPPV device as an American first, as it anticipated in principle almost all the essential characteristics of O'Dwyer's later, but more simple, appliance.[122,123,200] Whatever use it was put to in Europe, so far it appears that this invention was not taken back to the US on Trueheart's return to Galveston in 1871, nor does Matas mention its use in the US. Yet, because the device featured in successive editions of Knapp's textbook in Germany—one over 30 years after the initial description—and

[2] Footnote S3.2. In a comment to me, Dr Tony Newson, FANZCA, wrote (personal communication, August 2008):

> The copperplate script that was popular before typewriters were introduced made misspelling so common, and a good example is an 'ed' *vs* an 'et', especially when the 'd' is made with a flourish. [Christopher 'QWERTY' Sholes patented his typewriter in June 1868.] Then of course, once an error gets into the system it just 'keeps on keeping on'.

Further, he added:

> Another example of misspelling [is] from 1846 when 'Medical' became misread as 'Musical'. This must have been from typescript since the *Spectator* (London) referred to J H Bigelow addressing a paper on ether to a Boston musical Society (actually the Boston Society for the Advancement of Medicine and the Arts).

because at the time it was still being manufactured in at least two European countries, it seems to have been known and used. Even if Trueheart was not a pioneer in his own country of neonatal IPPV by tube-to-infant's mouth, he still qualifies through his device as a pioneer, seemingly without any recognition at that time, for introducing his mode of neonatal intensive therapy. He is entitled to rescue from such oblivion and to be honoured accordingly. Indeed, Matas wrote that in the history of IPPV devices, he regarded Trueheart's rhythmical inflating device for neonates as entitled to precedence over O'Dwyer's.

An obvious difficulty is understanding why, after Trueheart's return to Galveston, he appeared so uninformative about his invention of a device with life-saving potential. Surely the medical fraternity would have welcomed it? But, so far, it seems that the US medical literature was bereft of mention of the invention until Matas described it (see Figure S3.1).

3.8.7 Conclusion

In conclusion, without having absolutely confirmatory evidence (a devastating hurricane, 'Ike', from 13 to 14 September 2008, ravaged Galveston the day after I sent four separate enquiries there about Trueheart, with the destruction therefrom precluding answers), there is, of course, conjecture in my contentions for which the evidence is inconclusive, but is, perhaps, reasonably convincing.

Charles William Trueheart appears to 'fit the bill' satisfactorily for him to be acknowledged as the 'Truehead of Galveston', who made a brief appearance in medical history as the deviser of an effective apparatus for neonatal resuscitation at its time of the 1870s, preceding both the George Fell and the Fell–O'Dwyer apparatus, which was manufactured and employed in Europe for several decades more.

20th century artificial ventilation before the Danish poliomyelitis epidemic

Chapter 4: Australasian management of the ventilatory failure of acute poliomyelitis

Chapter 4: Table of Contents

4.1 Introduction

Acute infectious poliomyelitis, or the Heine–Medin disease, was first systematically described in Europe in 1840 by Jakob Heine,[211] although the withered leg portrayed on an ancient Egyptian relief would suggest that the disease existed in antiquity (see Figure 4.1). Karl Oskar Medin's study, completed in 1891, was the first documentation of an epidemic of polio, at Stockholm in 1887,[211] while Australia's first epidemic recorded as polio[212] was at Port Lincoln, South Australia, in 1895 (Megan Hicks, Powerhouse Museum, Sydney, New South Wales, personal communication, 2006). During the first six decades of the 20th century, successive epidemics swept through populations that lacked an acquired immunity in many countries.[211] In New Zealand, there were epidemics in the years 1937–1938, 1947–1949, 1952–1953 and 1961.[213] There have been no further outbreaks since a mass vaccination program in 1962.

Children were affected more often than adults, and 'infantile paralysis' became, in the commonly used newspaper phrase, every parent's greatest dread. Before 1928, available methods were inadequate for supporting those with the paralytic breathing failure that can complicate polio, thereby putting those afflicted at high risk of death.

Figure 4.1: The 'Polio Stela'

The 'Stela of The Doorkeeper Roma to the (Syrian) Goddess Astarte', from her sanctuary at Memphis, Egypt, considered to be from the XVIII Dynasty reign of Amenophis III, circa 1403–1365 BC, is now in the Ny Carlsberg Glyptotek, København (inventory # AEIN 134). The withered right leg is generally regarded as resulting from polio.
Photographed by Ole Haupt; reproduced with permission of the Glyptotek.[214]

4.1.1 The Drinker respirator

At the Harvard School of Public Health in Boston in the US, Phillip Drinker (1894–1972) and Louis Shaw,[221] with help from Cecil Drinker,[222] designed and built a truly effective ventilating machine: their 'artificial respiration tank' was a body-enclosing sheet-iron cabinet (with the patient's head and neck protruding from the cabinet and sealed off from its interior), powered by an electric motor, which provided external, intermittent 'negative' (sub-atmospheric) pressure ventilation (INPV).[221] The initial use of this first-ever practical[222,223] INPV machine over 14–19 October 1928 extended the life of a dying child for 122 hours.[221] Warren E Collins Inc., Boston, undertook full production of the Drinker(–Collins) respirator (as it was called then, not a 'ventilator'), and an unknown reporter dubbed it the 'iron lung'[222] (a term of usage that Mushin and Faux referred to sardonically in the *Lancet* of 25 November 1944).[224] Although effective and life-saving, it was large, heavy (about 102 kg[222]), cumbersome and expensive: in the US, an adult machine cost about US$2000 in 1930[225[p1253]] (and £2000 to 'land' in Melbourne in 1936,[226[p6]]), while the cost in Europe in the mid-1950s was around £1500 sterling[227[p57]]). Apparently, the Emerson iron lung (from May 1931), subject of an acrimonious lawsuit over patent, was about half the Drinker's price. Infant-sized machines were also made,[225] although adult machines were used on children.

The 1937 polio epidemic reaching Australia brought a dire need for respirators. There were only 'a few' Drinkers in the country then, and they had to be sent to the US for servicing.[212] One had been imported to the Fairfield Hospital in 1936 to treat a patient with post-diphtheritic, bulbar-type paralysis.[215]

4.2 Respirators in Australia

A notable, eminently practical 1937 invention from Adelaide, South Australia, provided a realistic alternative to the Drinker. This chapter now attempts to pay tribute to the designers and manufacturers, Edward Both (died 1987) and his brother Donald (died 2005),[212,215] and to the later but probably foremost clinical user of their invention in Australia, Dr John Forbes (1920–1989) of Fairfield Hospital.[218,219] Lord Nuffield, William Morris (1877–1963), made an important contribution[228–230] by ensuring the manufacture and widespread availability of the Both respirator, free of charge, throughout 'the Empire' (the UK and Dominions). The independent contributions of Aubrey Burstall[216] (1902–1984) must also be recognised, although his cabinet respirator did not achieve the same Australia-wide use as the Both respirator.

4.2.1 Edward and Donald Both and Aubrey Burstall

When Australia's 1937 epidemic of poliomyelitis created an urgent need for extra ventilating machines to compensate for respiratory paralysis, Edward Both (the -o- in 'Both' as in 'moth'), an innovative Adelaide biomedical engineer, invented a wooden cabinet respirator[212,215] (see Figure 4.3) that could be made relatively quickly in quantity to meet the demand anticipated during an epidemic. His device, here called 'the Both', alleviated the problem at Adelaide's Northfield Infectious Diseases Hospital and others and was introduced into England in late 1938 during Both's visit to that country. Appreciating its merits, Lord Nuffield financed assembly-line production at the Morris Motors factory in Cowley, Oxford. Then, through the Nuffield Department of Anaesthetics in Oxford's Radcliffe Infirmary, he had the Both distributed throughout the Commonwealth, as a gift for treating ventilatory failure in polio—especially in children.

For the 1937 epidemic in Victoria, and according to the design of Melbourne University's Professor of Engineering, Aubrey Burstall, nearly 200 units of another wooden cabinet respirator were ultimately built. Some were installed at the Acute Respiratory Unit of the Queen's Memorial Hospital for Infectious Diseases Hospital at Fairfield, Victoria (hereafter referred to as Fairfield Hospital),[216] and then at other hospitals throughout Australia.[217]However, by the early 1950s, the Both had replaced Fairfield Hospital's 'Burstall', which since 1937 had functioned as Victoria's favoured respirator. Dr John Forbes at Fairfield became the foremost Australian clinician for expertise with the Both.

Before the advent of IPPV, the Both's usefulness had seen it tried for ventilatory failure in some non-polio conditions, but the uptake of that application was limited. Nonetheless, Nuffield's philanthropy with the Both ultimately furthered progress along the 20th-century pathway to ICM.

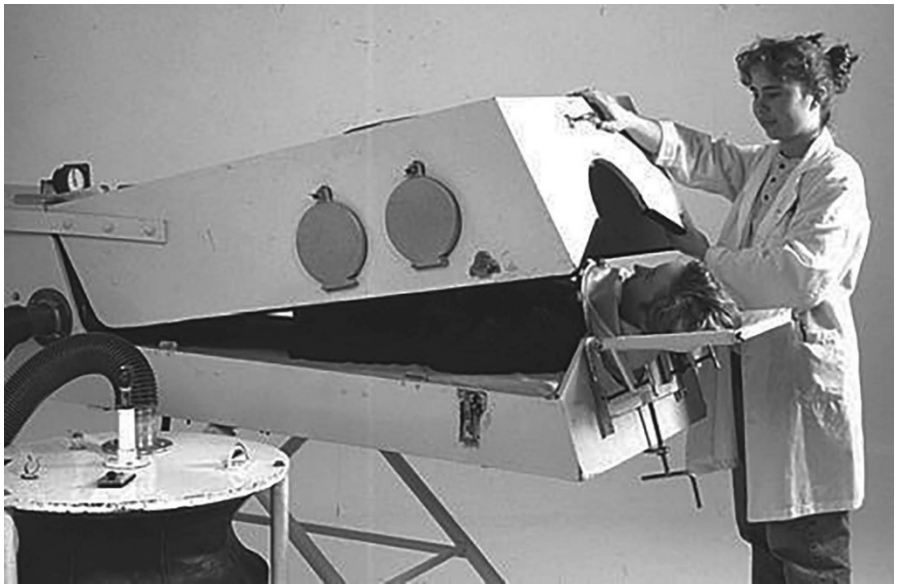
4.2.2 The Both team and their portable cabinet respirator[212]

Biomedical engineer Edward (Ted) Both developed a medical equipment laboratory at Adelaide University in the 1930s. With his wife Eileen and brother Donald (see Figure 4.2), he formed Both Equipment Limited. During the 1937 polio epidemic, in response to requests from South Australian health authorities for an alternative to the Drinker, the brothers designed and constructed their cabinet respirator within a few weeks[231] (see Figure 4.3). Although its cabinet was made of plywood, the Both respirator could not shake off the 'iron lung' nickname, used even by the Both brothers, it seems.[215]

Figure 4.2: Edward, Eileen and Donald Both

Edward Both (left) with his wife Eileen and younger brother Donald, photographed at his home, 1987.[232]
Photograph reproduced with the kind permission of Dr Richard Bailey, FANZCA.

Figure 4.3: The Both cabinet respirator



The 'alligator' design of the Both Portable Cabinet Respirator, in Auckland Hospital.[233]
Image courtesy of Alex Fraser and the hospital's Department of Medical Photography.

The Both tank worked in much the same way as the Drinker, but the incorporation of a bivalved design, hinging upwards the top section of the tank, allowed temporary access to the patient's body. According to Hicks, 'Working non-stop with the help of several other enthusiastic young men, the brothers produced sufficient machines to cope with the polio epidemic in South Australia'.[215[p3]] The GEC electrical motor to provide pressure changes was external to the cabinet, together with the compressor and bellows, and connected to it by a large flexible hosepipe. This wooden Both portable cabinet respirator, as it was named,[215] was considerably lighter than the Drinker, and its wheels made it mobile. It was quickly put into life-saving use at Adelaide's Northfield Infectious Diseases Hospital (with a few also at the Royal Adelaide Hospital [Stephen Hagley, FCICM, Royal Adelaide Hospital, retired, personal communication, 2001]), and then was taken up in multiple hospitals and respiration units in other states (such as the Royal Alexandra Hospital for Children at Sydney's Camperdown, NSW,[234] and in Western Australia and New Zealand).

Megan Hicks has noted that the Both was the commonest of the various respirators based on the Drinker and could be made quickly and relatively inexpensively.[212] The original Both, costing around £100,[226] was made only in Adelaide, but Both agencies were established later in Sydney and Melbourne (Richard Bailey, FANZCA, personal communication, 2006). The respirator underwent various improvements and was also 'copied in the workshops of several Australian hospitals',[212[p1]] as was a 'Nuffield–Both' at Prince Henry ('Coast') Hospital, NSW, in the 1940s.[212] Therefore, the Boths and facsimiles in Australian museums are not necessarily identical, as they could have been manufactured in different places at different times.[212] Even in 2006 in Australia and New Zealand, a few individuals with residual ventilatory incapacity used their own Both at home, unwilling to exchange a trusted and reliable long-time friend for a modern machine. Thus, in 2003, there were five in homes in Victoria[215] and one in NSW.[235] Such individuals were still partially dependent on their Both, for instance, during sleep[235] or for respiratory infections.

4.2.3 Prof. Aubrey Frederick Burstall

For the 1937 polio epidemic in Victoria, a different wooden cabinet respirator, the Burstall,[216,217] (see Figure 4.4) was used at the Acute Respiratory Unit of Fairfield Hospital, under the direction of the unit's 1932 founder, Dr Henry (Sandy) McLorinan.[219] This was purpose-designed locally by Aubrey Burstall, and six respirators could be coupled to a Burstall pulsator unit (35 of which were eventually made; see Footnote 4.1[1]).[236,237] Burstall cabinets were installed 'for use all over Australia'.[217[p611]] Burstall himself implies a total of nearly 200 at the end of 1937,[56[p.674],214] but Bryan Speed's count[216] (see Footnote 4.2[2]) does not indicate so many.

Later, towards the end of the epidemic (Christmas 1937), Burstall developed a simpler, neck-to-waistline 'jacket respirator',[56,217] a hammered-out, 6 lb (2.7 kg), aluminium, thoracic cuirass for respiratory support at the convalescent stage of polio ventilatory paralysis (see Figure 4.5). This jacket had its initial clinical use in the first week of February 1938 at Melbourne's Children's Hospital, and then soon after at Fairfield.[39]

[1] Footnote 4.1. Biographical Note. [See Carolyn Rasmussen's *Increasing momentum – engineering at the University of Melbourne*[236]]. Aubrey Frederick Burstall (PhD, Cambridge), newly arrived from England to the Chair of Engineering at Melbourne University, rapidly developed his solutions for treating ventilatory failure in polio during 1937, the year the University of Melbourne conferred on him a DSc, honoris causa. Burstall also designed gas producers for motor vehicles. His medical devices included a tiny, heat-regulated respirator for neonates at Royal Women's Hospital, Melbourne, and an aspirator, while his faculty supplied a Crash Team for beach rescues.[236] In 1946, Dean Burstall, after difficulties over challenging the dominance of civil engineering,[237] returned to the UK to the chair of Mechanical and Marine Engineering at the University of Durham. His books include the highly rated 1963 *History of mechanical engineering* and the 1968 *Simple working models of historic engines*.

[2] Footnote 4.2. Bryan Speed has determined [216[p5]] that at Fairfield Hospital's Acute Respiratory Unit:

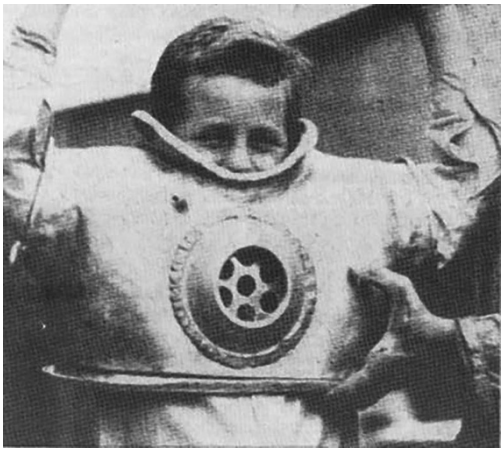
> The initial six respirators increased to 23, with up to 47 patients having to 'time share'. A further 36 'Burstall' respirators were distributed to regional centres in Victoria. A total of 1,275 patients were treated. Most were less than 14 years old, 140 had respiratory paralysis, 106 required respirator treatment and 37 of these died.

(Thus, the mortality rate was 35% among those ventilated.)

Figure 4.4: Burstall respirators at Fairfield Hospital

A rare photograph of Burstall respirators at Fairfield Hospital, 1937.[238]
Courtesy of Dr Bryan Speed, FANZCA, and the Fairfield Hospital Historical Collection.

Figure 4.5: Aubrey Burstall's cuirass jacket respirator, from his original 1938 article[217]



Reproduced with the kind permission of the British Medical Journal.

One Burstall cuirass could be connected to a pulsator, or many cuirasses to a cabinet respirator. The inventor documented the jacket's merits and demerits.[217] The Burstall apparatus provided sterling service for Victoria,[216] but unfortunately no examples appear to remain today. Despite the Burstall cuirass becoming a local success (it was also successful in England, as Prof. Macintosh later mentioned in his renowned 'Letter'[239] of 'explanation' to the *British Medical Journal* of 14 January 1939), it was through the Both's ubiquity—as well as its intrinsic advantages—that it became the Commonwealth's regular cabinet respirator, as will be discussed further in relation to their numbers distributed.

4.3 Lord Nuffield and the Both Respirator

In October 1938,[229] the professor of the Nuffield Department of Anaesthesia (NDA) at the (old) Radcliffe Infirmary, Oxford, was New Zealander Robert Macintosh (later Sir Robert; 1897–1989), who ensured that Lord Nuffield saw a film featuring a child whose life was saved in one of the newly invented 'lungs'.[229] Eileen Both described Nuffield as impressed with the Both's 'simplicity of operation and its design'.[215[p4]] Accordingly, Robert Jackson's biography of Nuffield[230] has him enquiring directly after viewing the film why more hospitals were not equipped with tanks. 'Expense' was Macintosh's simple answer.[239] Further, he commented that it seemed a dreadful state of affairs that children were dying because hospitals cannot get hold of iron lungs in time.[230] Nuffield then asked: 'If every hospital throughout the Empire had a "lung", is there a reasonable prospect of three lives being saved?' To Macintosh's reply, 'Undoubtedly they would', Nuffield responded, 'Well, I will give instructions immediately for a thousand to be made'.[239[p84]] (See Footnote 4.3.[3])

Jennifer Beinart states in her careful account[229[p43]] that after viewing the film, Lord Nuffield chanced a few days later upon a newspaper headline 'Iron lung arrives too late' for a young patient who might have been saved. The accompanying article stated (possibly incorrectly[228]) that there were only five iron lungs in all England. This led Nuffield to offering on 24 November 1938 'to make 5000 of them if necessary, at a cost of something like £500 000'.[229[p43]] Other doctors wanted Nuffield to be aware that further improvements could still be possible, but Nuffield was dismissive. The whole story has been clearly set out by Macintosh,[239] which Robert Jackson[230] does not reference in 1964 when quoting Nuffield for his well-attested statement:

> If I had waited for the perfect car, I should be bankrupt now. We must get on with the best possible model available now and improve on it as we go along. It seems a pity to think that some of these respirators will be used as coal scuttles, but it is more tragic still to think of the possibility of a life being lost through the failure on my part to spend £25 or £30.[230[p176],239]

A London County Council medical officer called Nuffield's providing iron lungs for 'all and sundry'[240[p35]] the height of folly.[230] But, trusting the advice of his friend Sir Robert, Nuffield himself laid out the line for the mass production of the Boths in a corner of his Motors Ltd. factory in Cowley, Oxford.[230] Until stopped by the war, production continued to supply every Commonwealth/Empire hospital still asking for Boths[228]— and Jackson said several thousand did so.[230] Jackson repeats the above cost (sterling) of a Both–Nuffield at about £25 each[230] (But note that Beinart [229[p43]] states the cost of 5,000 respirators as £500,000, that is, £100 each, and the caption to Figure 4.6 states £98 each).

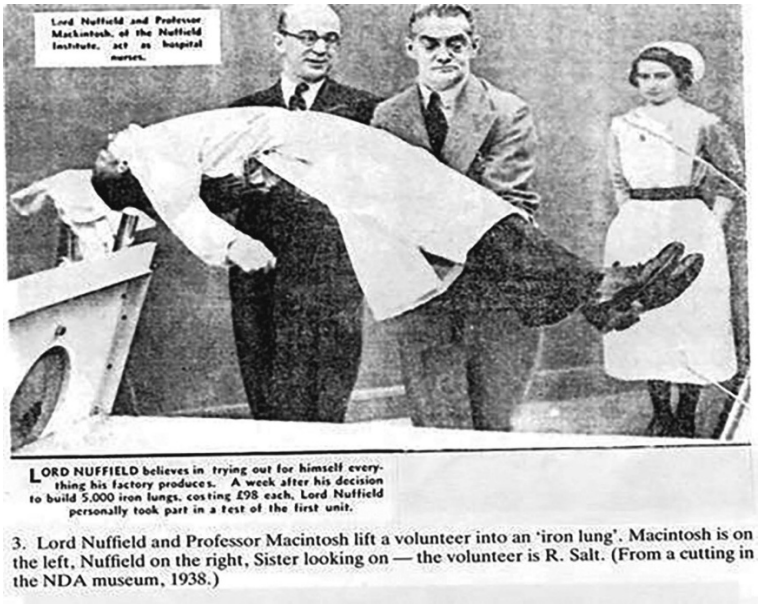
[3] Footnote 4.3. It was only with the near-completion of this work that I stumbled upon the documentation by Prof. Macintosh of his interaction with Lord Nuffield (see Section 4.4), leading to the latter's generous gift of Both respirators to hospitals in the Commonwealth. This 1½ pages account[239] appeared in the *British Medical Journal* of 14 January 1939, buried in the section headed Correspondence but under the sub-heading 'Mechanical Respirators', which were of much interest at that time. I had never heard about it, either from 'Prof. Mac' himself or from others, while I was in his Department of Anaesthetics at the Radcliffe Infirmary. This was such a surprise to me this I wrote to the two other New Zealand Nuffield Dominion Scholar doctors who had preceded me at the NDA, suggesting that, surely, they were aware of this letter even if I was not. But like myself, although they were well familiar with the happenings, neither remembered ever seeing or hearing of the said letter. The wry comment of my friend Prof. Paul Moon, notable New Zealand historian, on this chance, fortunate for me, finding was, 'What a lucky find at the last moment. Some sources simply refused to be discovered'.

The NDA was to distribute the Both–Nuffield respirator.[228] NDA Prof. Alex Crampton Smith records:

> in 1939 he [Nuffield] made the gift of a Both type 'iron-lung' to every hospital in the Commonwealth which asked for one ... The Nuffield Department helped to distribute the respirators and by demonstrations and films gave instructions in their use.[228[p52]]

Initially, Sir Frederick Menzies sharply criticised this act as a wanton waste of private benevolence,[240] but Macintosh was staunchly defensive against this opinion.[239] By Smith's later precise count,[241] at the end of March, 1939, in the British Isles, including the Services, there were 965 Both machines. (Smith also noted that there were 30 Drinker machines and 43 Bragg-Paul respirators.) At the end of 1939, the NDA could report that just over 1600 respirators had been allocated throughout the Empire and about 800 had been delivered[229] (versus 'just on 1800', as Prof. Peter Morris of the John Radcliffe Hospital, Oxford, assessed[215[p4]]). However, Beinart stated in 1987[229[p44]] that there is uncertainty about that number because the later NDA records of the total number of Both–Nuffield respirators supplied have not survived. Earlier, the *Lancet* reported in 1947 definitively (anonymously) on the numbers distributed. In Australia, sometimes Boths, a proportion of them coming from Cowley,[229] were called 'Nuffields' (see Footnote 4.4.[4])

Figure 4.6: Lord Nuffield (on the right) and Prof. Macintosh (on the left) with the first Both respirator at Oxford



3. Lord Nuffield and Professor Macintosh lift a volunteer into an 'iron lung'. Macintosh is on the left, Nuffield on the right, Sister looking on — the volunteer is R. Salt. (From a cutting in the NDA museum, 1938.)

The photo is likely to be from the *Oxford Times* newspaper. The upper of the two captions reads: 'Lord Nuffield believes in trying out for himself everything his factory produces. A week after his decision to build 5,000 iron lungs, costing £98 each, Lord Nuffield personally took part in a test of the first unit.'
The photograph is reproduced with the kind permission of the Nuffield Department of Anaesthetics, courtesy of Prof. Clive Hahn, from Jennifer Beinart's *A history of the Nuffield Department of Anaesthetics*, *Oxford, 1937-1987*.[229]

[4] Footnote 4.4. The Pioneer Park Museum in Griffith, NSW, has pointed out that the museum's Both Bros 'alligator'-style respirator (purchased by the Griffith community), unlike that donated by Lord Nuffield, which patients had to be slid in and out of, had a lid that worked on a counterweight and that this system was regarded as much more user-friendly. [226]

A later Notes and News report[242] in the *Lancet*, concerning where the Nuffield–Both respirators were distributed approximately produced these figures: UK and the Services, 750; Canada, 347; Australia, 198; India and Burma, 183; South Africa, 46; Eire, 40; New Zealand, 33; Newfoundland, 14; British hospitals abroad, 10; and elsewhere in the British Empire, 134; total, 1,755.

Significantly in ICM history, 'at one stroke' Nuffield's foresight with 'this equipment ... forced physicians to treat actively patients developing respiratory failure'.[68A[p199]] Ted Both, Prof. Macintosh and Lord Nuffield certainly earned gratitude. As William Mushin contended, the Both respirator functioned well to compensate for polio-paralysed respiratory muscles, and it did 'an enormous amount of good'.[229[p44]] Mushin, Rendell-Baker, Thompson and Mapleson saw the anaesthetists whom Nuffield caused to be involved[68A[p199]] as the natural operators for tank respirators:

> At one stroke a large section of the population working in British hospitals became familiar with this [Both] apparatus and, perhaps as importantly, Departments of Anaesthetics became recognised as the experts in its use and in the care of patients with acute respiratory difficulties.[68B[p210]]
>
> The excellent results obtained in these units stimulated major hospitals in many centres to establish 'respiratory units'.[68A[p215]]

None of the reliable positive pressure machines developed in Sweden since 1934 for AV during anaesthesia had received a trial for the long-term AV that polio victims might require. (And, of course, IPPV would also require satisfactory, cuffed intratracheal tubes.)

4.3.1 Non-polio use of negative pressure ventilation and the Both respirator

The 1928 Drinker iron lung represented a distinct advance over any existing machine providing respiratory assistance.[223] Not surprisingly, 'the application of the respirator to a wide range of conditions other than polio started almost as soon as the Drinker machine was invented'.[243[p462]] Philip Drinker and colleagues listed its application in 80 patients by June 1930[225,244] (Table 4.1). In 1934, this machine enabled the survival of five out of eight diphtheria patients with diaphragmatic paralysis.[245] In the 1930s to 1940s, innovators continued trying out mechanical NPV for numerous life-threatening disorders other than polio (Table 4.2).

The arrival of the Both provided a respirator which was, although at times inconvenient and for some perhaps claustrophobic,[246] less complicated than the Drinker for nursing procedures, and much cheaper (Morris 'Mini' versus 'Rolls-Royce' comparisons have been heard). A year after the 'Both–Nuffield' was introduced to Oxford, Robert Macintosh was assessing it for preventing postoperative respiratory complications.[246] Then, William Mushin and Nancie Faux[227] successfully used it in a trial with 24 patients to reduce post-operative morbidity. However, contemplating his attempts at postoperative, prophylactic NPV, Prof. Macintosh commented ruefully: 'The sound of iron lung was pretty sinister ... The surgeons' reputation could not stand for it' (personal communication, 1987).[224]

Table 4.1: Patients treated by negative pressure respirators in the eastern United States[244]

Indication	**28 October 1928 to June 1930[225]**	**"After 2 years"**
Neonatal asphyxia	Limited success	>30
Polio respiratory failure	2 of 7 survived	100
Coal gas/carbon monoxide poisoning	9 of 17 recovered	50
Overdose	3 of 5 recovered	
After scoliosis surgery	1 of 1 successful	
Drowning	1 of 1 recovered	7
Alcoholic coma	3 of 8 recovered	10
Post-diphtheritic paralysis	–	1
Total	**80 (46 adults, 6 children, 28 infants)**	**>198**

Table 4.2: Further applications of negative pressure ventilation to 1945 (table based on Gilbertson, 1995[243])

Year	**Indication**	**Author**	**Reference Site**
1930–1940s	Poliomyelitis	Gilbertson A[243]	*J Roy Soc Med* 88:459P–463P
1935, Jun 22	Drinker respirator in diphth. diaphragm-palsy	Mitman M, Begg N[245]	*Lancet* i:1438–40
1939, Apr 22	Paraldehyde poisoning	Macintosh RR[see246]	*Br Med J* i: 827
1941	Snake-bite poisoning	Linton R, Sarkar N[see247]	*Indian Med Gaz* 76: 92(−3)
1940, Dec 14	New Both use x 2 in poor risk, abdominal surgery	Macintosh RR[248]	*Lancet* ii: 745–6
1944, 25 Nov	Ditto use in 24 patients	Mushin WW, Faux N[224]	*Lancet* ii: 685–6
1942, 28 Feb	Diphth. diaphragm-palsy treated in Box respirator	Todesco J[249]	*Lancet* ii: 261
1944, 09 Dec	Myasthenia gravis	Bates J[250]	*Lancet* ii: 770
1944,28 Oct	Pulsator treatment of crush injury	Marshall DV[251]	*Lancet* ii: 562–3
1945	Crush injury of chest	Hagen K[252]	*J Bone Joint Surg* 27:330–4

Still, the Both machine had sufficient, continuing, non-conventional use in the UK such that a 1944 issue of the *Lancet* [253[p695]] could have it that 'in a little over five years the Both respirator, once described as a "white elephant", has produced persuasive reports of its use both as a life-saver and as a valuable adjunct to physical medicine', and also that 'use of the [Both] respirator to combat respiratory depression of barbiturate poisoning may now be said to be routine'. But does the literature support such a claim? It would not seem to have been documented.

The Nuffield model of the Both was modified and improved in various ways over the years,[215] and infant models were devised: Nuffield's original intention was that it was to be a gift primarily for children.[230] In the 1950s, various adaptations made to the Both were documented: for instance, in the UK the valuable modifications of RE Smith,[242] the Ministry of Health especially[254] and others,[255,256] and in Australia of John Forbes.[222] At the Royal Adelaide Hospital in October 1938, Boths were also used, albeit rarely, for acute respiratory failure from (unspecified) causes other than polio, the last time probably in the early 1950s with attempted resuscitation of a drowned person (Dr Stephen Hagley, personal communication, 2006).

4.3.2 Demise of intermittent negative pressure ventilation

Anaesthetist Bjørn Ibsen and colleagues, with Lassen, demonstrated in 1952–1953 at Blegdam Hospital, Copenhagen, that manual IPPV (m-IPPV) was effective, safe and successful on a large scale and for long-term use.[227,257[p.21–8],258] That led to the rapid development of multiple models of positive pressure ventilators in Europe and the UK.[68B] However, in Australasia the Both had continuing use during the 1950s (much in the same way that the Drinker was being continued within the US—where the other successful INPV machines to become prominent in use in critical illnesses were the Emerson and Van Bergen respirators). The successful use in the 1950s of the Both at Melbourne's Fairfield Hospital is examined in Section 4.5. Nevertheless, within the large nations of the British Commonwealth, IPPV was supplanting INPV (per the Both) by 1960 for any intensive-care-type application, if not necessarily for patients with chronic stage polio. But, until then, the Both machine had made a valuable life-saving contribution for nearly a quarter of a century in Australasia, the UK and elsewhere. Although several other inexpensive, quickly manufactured substitutes for the Drinker tank had been devised for INPV outside the US (or at least in the Commonwealth), it was the respirator invented by Edward Both, OBE, which predominated.

4.3.3 Afterthoughts

Certainly, Lord Nuffield's benevolence saved many lives. Ultimately, it also furthered progress along the pathway to ICM, a benefit he hardly envisaged. But the thought does arise: Could the very availability of the Both respirator have inadvertently delayed both the introduction of PPV into Australasia and the earlier promotion of ICM there? Australia and New Zealand received their supply of Boths just before the outbreak of WW II, and they were distributed to widely separated units, or stored centrally, in New Zealand for instance, at Wellington. They were soon called upon for employment during New Zealand's polio epidemic in 1942 (there were 24 deaths in the country in 1943). Later, into the 1940s, exploratory attempts were made at Oxford to introduce the Both for some specific clinical applications postoperatively, but were abandoned.[224,248] Although the Copenhagen demonstration of the efficacy of m-IPPV in the Danish epidemic was reported in the medical literature just four months into its employment there,[258] m-IPPV was not taken

up seriously in Australasia for half a dozen years, apart from a few isolated instances. Possibly the stores of available Boths, such a godsend when there were exceedingly few mechanical ventilators in Australasia, inhibited an exploratory, more aggressive swing to developing PPV. Perhaps not.

Meanwhile, in the 1950s Europe, especially Scandinavia and the UK, innovative designers, motivated initially by fears of further polio epidemics (until 1961, after which the Salk and then the Sabin vaccines brought the disease largely under control in the Western world), pressed ahead with the invention of various PPV machines.[68A] That venture had great spin-off in its application to non-polio conditions, especially tetanus. When the last polio epidemic arrived in Australasia in 1961 (see Footnote 4.5[5]),[259,260] the very limited number of PPV machines there ensured that the Boths, whatever their drawbacks, were undoubtedly saving lives. Jennifer Beinart discussed these issues, and questioned whether the Both was outdated in 1939.[229[p45]] But the way ahead was not shown until 1952–1953, when Bjørn Ibsen did precisely that (it can be noted that Anthony Gilbertson boldly claimed '"Intensive Care" did not start in 1952, it changed gear'[243[p463P]]).

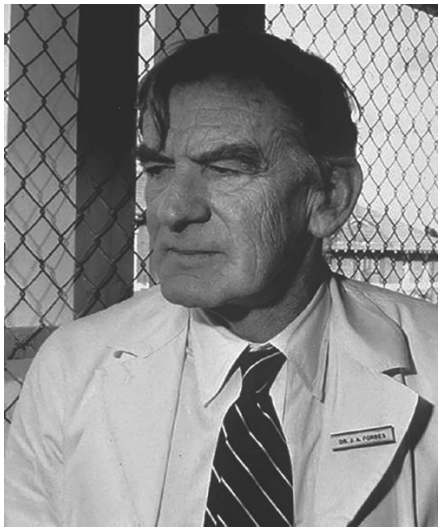
4.4 Dr John A Forbes and Fairfield Hospital, Victoria

John Alan Forbes (1920–1989; see Figure 4.7), one of the wartime 'Rats of Tobruk', qualified in medicine after demobilisation and, after a spell as a patient at Fairfield Hospital[220A] from 1950,[261] took up duties as registrar and senior medical officer in February 1953. He became deputy medical superintendent there the following year and, by January 1955, was reporting on the recent polio experience of Fairfield's respiratory unit, which had been under the overall supervision of Dr Henry (Sandy) McLorinan.[222] In 1956–1957, the unit was extensively refurbished and upgraded to be used as a modern respirator ward[220D] for providing excellent facilities for all types of respirator patients.[220E] When Dr McLorinan retired in 1961, Dr Forbes became medical superintendent. The tribute for the Royal Australasian College of Physicians by Mr Ian McDonald (FRACS)[261] states that Dr Forbes literally transformed Fairfield Hospital from a receiving centre for communicable diseases to a major university teaching and research establishment with an international reputation.

The Fairfield Hospital was founded in October 1904[220] and closed on 30 June 1996. In writing this chapter, I drew especially on articles in the March and December 2003 issues of *The HaMMer* (the Australian Health and Medicine Museums newsletter).

[5] Footnote 4.5. In New Zealand during the 1961 poliomyelitis epidemic, of the 304 cases admitted to Auckland Hospital, 85 went to its Infectious Diseases Unit or to the Acute Respiratory Unit. Sixteen of 17 bulbar and/or respiratory cases received IPPV (with a tracheostomy) when INPV was not appropriate or was inadequate. A single death gave a 6.25% mortality rate for this group.[259,260]

Figure 4.7: Dr John Forbes, AM



From the Fairfield Hospital Historical Collection,[262] courtesy of Dr Bryan Speed, FANZCA.

Despite the successful outcomes achieved by Dr Forbes and his team in treating the respiratory insufficiency of polio by INPV, and his two 1950s publications describing this treatment,[218,219] it is not mentioned in the tribute to Forbes by the Royal Australasian College of Physicians.[261] Possibly this was because his heavy involvement in treating acute polio lasted only a few years, compared with his time dealing with other infectious diseases. This College's measure of this outstanding man lies with his notable achievements in the following decades. After his earlier years treating polio and other infectious diseases, he made developments in immunology and conducted further research, later undertaking extensive philanthropic relief ventures in Ambon in Indonesia, and Vietnam.[261] OLDMEDLINE lists about 35 articles with Forbes as sole or joint author. The clinical and research titles are spread widely, principally over infectious diseases and epidemiology.

Treatment of polio at Fairfield, and mortality data[218,219]

Forbes's success between 1953 and 1956 in treating the breathing inadequacy of polio by artificial respiration (accompanied by tracheostomy in 28 of 71 patients) is impressive. For the 30 months from July 1954 to December 1956, of the 295 patients with paralytic polio, 85 had respiratory or pharyngeal paralysis (including adults and children). Of these 85, a total of 39 were artificially ventilated by tank respirator and a third of them with a tracheostomy (whereas 17 others with pharyngeal paralysis were not ventilated). The mortality rate among the 39 patients receiving artificial respiration was 21%) (whereas, for the 32 artificial respiration patients during the previous 12 months, the 11 deaths gave a rate of 34%). Of the eight deaths in 1954–1956, four occurred rapidly in the acute stage (as also happened in Copenhagen, 1952[227,257,258]) and four others were from severe, non-polio causes.[219] This death rate compares favourably with other NPV series, such as Lassen's 87% in a devastating first month of the 1952–1953 Danish epidemic.[227,257,258] Dr Bergman's reporting a mortality result for 1948 of 70% among 827 cases may reflect use of the Sahlin–Stille cuirass alone.[263] In the 1952 New Zealand epidemic, the death rate of 'respiratory cases' was 36 out

of 46, or 78%.[264] (But how many who died did not receive NPV? Compare Southland, New Zealand: deaths in bulbar and/or respiratory cases were 11 of 57, giving a 19% mortality.[259]).

The *Fairfield Annual Report, 1954–1955* (mid-year to mid-year),[220C] mentions the use of new chest respirators, presumably Boths, to replace longstanding Burstalls, but the changeover date before 30 June 1955 is not stated. Forbes introduced many additional changes to his Boths and placed considerable emphasis on the facility of a 'mechanical cough' mechanism (previously described in the US by John Affeldt, 1954[265]). After 2½–3 years' experience of the cough-type (Both) iron lung,[220F] which he described in detail[219] in 1958, he could state that the use of the mechanical cough mechanism was followed by a dramatic fall in the mortality rate of acute cases.[219] This had been noted earlier in the Medical Superintendent's Report of 1955.[220C]

Forbes made two further ventilatory improvements to his Boths: employing bellows of higher volume, and a larger intra-tank positive-pressure phase for thoracic and abdominal compression during expiration. In his opinion, his multiple measures all helped to reduce the mortality rate from the 34.4% of the previous 12 months (1953–1954). Apart from respiratory care and tracheostomy, other general measures, such as nutritional needs, fluid balance, infection control, nursing care and physiotherapy, also received careful attention. Forbes considered that 'chemotherapy' (antibiotics) had an important role in his unit, but one not to be abused.

4.4.2 John Forbes and negative versus positive pressure ventilation

John Forbes stated his firm belief that he had demonstrated INPV with his Both 'tank respirator' to be preferable not only to the use of cuirasses (this was indisputably so, as a cuirass had only 47–61% of a tank's efficiency[265]), but also to the use of PPV, and it was easier for nurses than managing IPPV (enabled better access for a patient's body-care).[219]

He quoted the Lassen–Ibsen polio mortality at Copenhagen during the 1952–1953 Danish epidemic as 51.3% of 232 intra-tracheal positive pressure cases. But such figures are not obvious in the reference that Forbes supplies. In addition, he wrote of *all* patients having a tracheostomy at Copenhagen—actually, 26[266] did not have tracheostomies among Lassen's 262 artificially ventilated Danish polio patients[267[p167]] (or among 277[227[p13]] at Lassen's other reference sites). Also, the lower mortality rate at Fairfield has to be considered in the light of the severity of the disease in Copenhagen, since Lassen had described the Danish epidemic as 'by far the worst ever recorded in Europe'.[267[p158]]

Lassen did not supply virus typing (apart from 20 of his patients studied, who all had type-1[227[p4]]), but Forbes points out that in his own cases the cerebral lesions were worse when the causative polio virus was type-3 rather than type-1 or 2.[218] Nevertheless, Lassen did further classify the Danish cases into six anatomico-clinical groups of differing severity.[227,266] Recently, the Copenhagen end-mortality has been re-estimated[266] as showing a death rate of only 11% among the last 18 patients, within a 42% overall epidemic mortality in patients whom Lassen classified as meeting his criteria for life-threatening poliomyelitis.[227,267] Reassessing their Blegdam cases after the epidemic, Lassen referred to improving results 'despite the constant severity of the cases throughout the whole epidemic period',[267[p158]] and hence, presumably such an assessment applied to the last 18 patients too.

4.4.3 Improvements to intermittent negative pressure ventilation, and mortality rate

The Medical Superintendent's Fairfield Report, 1956, claimed that the hospital, which had experience of all methods of artificial respiration, considered the tank type of respirator or iron lung to still be the most efficient machine.[220D] It would seem from the critical remarks John Forbes made about PPV that he distrusted it.[219] If he was using PPV, he would expect a higher mortality rate, the employment of a higher tracheostomy rate with the consequent inevitable tracheo-bronchitis, and unopposed intratracheal positive pressure producing problems of cuff trauma to the trachea and circulatory impairment. (In the early 1950s, there was widespread unease that IPPV unduly decreased venous return.) As Forbes quoted[218] Bower's 1954 textbook on treating acute polio at Los Angeles, presumably, he knew about Ray Bennett's IPP respirator attachment for their INPV machines,[268,269] although he never adopted it. But he did 'maintain' his patients when out of the respirator by intratracheal positive pressure oxygen.[219] The Bennett attachment did bring significantly better results for Albert Bower's team at Los Angeles (where, in 1949, the mortality rate among 130 'respirator cases' was 17%, compared with 62% in 1946, but see Footnote 4.6[268,269] regarding Bower's superior resources[6]); and was helpful for William O'Brien and associates[272] at Reno, Nevada (mortality rate of 24% over three years in a critical group of 51 ventilated patients). John Affeldt[265] at Hondo, California, invoking Bower, used a positive pressure attachment to the mouth or tracheostomy that significantly facilitated nursing care.

In New Zealand around this time, ventilatory support from tank respirators, cuirass respirators and positive pressure equipment was being advocated for respiratory centres that were to be established in the main hospitals. In addition, ventilatory equipment was to be stored in a central pool at Wellington.[273] Yet, Christopher Woollams has pointed out[56] that as early as 1938 in Britain, the Medical Research Council Committee had recommended respirators be loaned out, as required, from depots best sited at regional centres,[274] since the Both respirators were bulky and difficult to store.[56] But how well could such a system function to meet an immediate need—surely, it could be expected to fall short?

4.4.4 Early intensive care medicine in Australia: John Forbes and others

The unit probably regarded as Australia's first formal ICU—which is generally taken to be one using (at least) IPPV—was the one Victor Hercus had refurbished at Prince Henry Hospital, Sydney, in 1961–1962.[275] Claims could be made that before that pioneering, Forbes's NPV respiratory unit at Fairfield legitimately came close for claiming 'priority rights' in Australia. Thus, the Fairfield 1955–1956 Report[220D] states (Fairfield Hospital Annual Reports for a specified year were dated only to 30 June, but ran from 1 July of the previous year) that several severe tetanus patients had been successfully treated with relaxants and artificial respiration in a cough-type iron lung,[220F] while the 1957 Report states that in the last 15 cases of tetanus, there were only three deaths.[220E] Forbes's 1958 article[219] on his management of polio patients with

[6] Footnote 4.6. A comparison of resources: during the 1948–1949 polio epidemic, the resources available to Bower's Los Angeles unit enabled simultaneous treatment of patients by using 42 Drinker–Collins mechanical ventilating units, many with a Bennett positive pressure attachment, while 25 had physiological cams installed. The 42 units were in almost constant use,[267] providing IPPV. Compare Copenhagen: the epidemic starting with Lassen having no Drinkers, only one Emerson tank and six cuirass respirators.[227] The number of Both respirators available to Forbes would certainly be below Bower's total number of Drinkers. In his 1961 visit to Fairfield, Spence noted Forbes' unit 'equipped for the operation of 14 Tank (or Cabinet) respirators'.[270] These numbers do conflict with those Spence later gave at an address to the Australian and New Zealand Intensive Care Society (NSW) in 1986[271] of 25–30 Boths, of which 20 were occupied by chronic polio patients.

ventilatory failure also specifies treating patients with infective polyneuritis (also see Footnote 4.7[7]). Tracheotomy—but not endotracheal intubation—was readily performed by Dr John Forbes and his full-time assistant Dr Noel Bennett.[270]

For all these reasons it has been written that Forbes's unit 'must be credited as probably the first official ICU in Australia'[276[p4]] (see also Footnote 4.8[8]).[272,276] Properly however, such a term is reserved for IPPV units. Before 'true' ICUs became established in Australasia around 1960, there were occasional activities that warrant description as ICM. Thus, for treating tetanus, in July–October 1957, at the Royal Adelaide Hospital that did not have a formal ICU, nine patients of Maurice Sando and Graeme Marshall received curarisation and IPPV for controlling their tetanus, among whom five survived.[277] The same treatment had been provided earlier, such as by Patricia Wilson (1926–2015, became Mackay, OAM) in Melbourne in 1954 (personal communication, 2001), and later by others[278] there; by Dr I Schalit at the Royal Newcastle Hospital in 1954–1955[279]; and by Brian Dwyer in Sydney in 1956.[280] It can be noted that Dr Patricia Mackay's ANZCA Citation, 2000, mentions that she also treated head injuries, myasthenia gravis and polyneuritis (without specifying what means were used, although respiratory support would be expected).[281]

4.4.5 In conclusion, regarding Dr John Forbes

Concerning success in treating paralytic polio in the 1940s to 1960s it seems, with the limited information available, that one cannot make valid comparisons between polio respiration units in different countries. However, it is not unknown in ICM for a unit such as that of John Forbes, with a team superbly led by an inspiring leader and with organised, enthusiastic, experienced staff (especially nursing staff) and ready medical back-up (Forbes lived onsite) to achieve results not to be expected with limited or inferior facilities and resources (see Footnote 4.5). Forbes was imaginative and quickly responded to new ideas and advances in medical science.[261] It should occasion no surprise then to be informed that 'he treated patients with a wonderfully high and compassionate standard' (Barbara Rossall-Wynne, Curator, Fairfield Hospital Historical Collection, Austin Health, Melbourne, personal communication 2006).

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[7] Footnote 4.7. *Fairfield Annual Reports*: the year's table of total number and principal diseases treated, among which polio is prominent, has a large group (e.g. 2,156 of 4,405 admitted for 1953) labelled miscellaneous—including admission for General Division. Tetanus is not separated out from this group until 1957, with seven cases,[220E] and acute infective polyneuritis not until 1958, with 15 cases.[220F] Fresh polio admissions taper off in 1955–1960. Not until 1958—and for that year alone—do the reports provide data on the total of polio paralytic cases (168), cases with respiratory or pharyngeal paralysis (53) and cases requiring artificial respiration (22), and the mortality of paralytic cases (4 patients, 2%).

[8] Footnote 4.8. Regarding Forbes's Acute Respiratory Unit: a first-hand assessment lies within Matt Spence's extended and locally famous 1961 Report to the Department of Health in New Zealand,[270] written following his close scrutiny of the world's top [English-speaking] ICUs[261] during a 5-month overseas study tour. Fairfield's unit was the first overseas ICU that Spence saw, over a 6-day visit. Apart from some milder criticisms, Spence asserted:

> He [Forbes] has accumulated a vast experience in the management of respiratory inadequacy using tank respirators with a cough mechanism incorporated ... The respiratory unit at Fairfield Hospital receives mainly poliomyelitis, polyneuritis and tetanus patients. Respiratory inadequacy due to neurological lesions and respiratory disease has been treated. They do not receive surgical cases. The Unit operates very efficiently and all concerned are proficient at their duties. This is primarily due to organisation and close supervision by Dr Forbes and Dr Bennett, the permanency of the nursing staff and physiotherapy staff and the enthusiastic assistance of the hospital engineer in maintaining and effecting improvements to equipment.[270[p1–3]]

Dr Spence later added myasthenia gravis to the above list of conditions treated at Fairfield—and more. In his notoriously opinionated address at the ANZICS 6th Continuing Education Meeting, Newcastle, March 1986[271] (see also Dr Judson's criticism of it [276[p84]]), he asserted, 'Concentration of paralysing and convulsive diseases at Fairfield delayed the organisation of Intensive Care facilities in Melbourne Teaching and Acute Hospitals until the middle of 60's era of traffic accidents', etc.

20th century artificial ventilation before the Danish poliomyelitis epidemic

Chapter 5: On the very first, successful, long-term, large-scale use of intermittent positive pressure ventilation; Albert Bower and V Ray Bennett: Los Angeles, 1948–1949

Mechanical equipment plays a major role in caring for acute poliomyelitis patients with respiratory involvement. The purpose of this equipment is to keep as nearly normal as possible the physical and physiological environment for maintenance of life and recovery of the patient.

Albert Bower, Bennett, Dillon and Axelrod[268]

Chapter 5: Table of Contents

5.1 Introductory overview: The setting

In 2003, anaesthetists Preben Berthelsen and Michael Cronqvist identified the first ICU in the world as Bjørn Ibsen's 1953 multi-purpose ICU at Kommunehospital (Community Hospital) in Copenhagen. It used IPPV for ventilatory failure.[282] That conclusion by the two authors gave encouragement to determining whether Ibsen's unit provided the very first successful use on a large scale of extended IPPV, documented in English and recorded in the medical literature. It required studying treatment provided in the US during the preceding Los Angeles poliomyelitis (often hereafter, 'polio') epidemic of 1948–1949, [268,269,283–286] by the Communicable Diseases Service (see Footnote 5.1[1]) of the Los Angeles County General[288] Hospital (LACH), see Footnotes 5.1B and 5.2[2]), Boyle Heights, Eastern Los Angeles.[269,283–288] Such treatment was under the care of physician Albert G Bower (see Figure 5.1) with biomedical engineer V (for his unused first name 'Vivian') Ray Bennett (see Figure 5.2), and their medical, nursing and supporting services teams.

An unprecedented patient load for AV at LACH (294 respirator cases) in 1948 had arisen from a seasonal increase in poliomyelitis cases to near-epidemic proportions. Physician Albert Bower and his team had determined that respiratory acidosis was frequent in patients receiving INPV. With Bower's awareness of a previous high mortality rate during the standard treatment of polio ventilatory failure using Drinker–Collins respirators, that finding led to multiple advances in equipment technology at LACH. Most important was Ray Bennett's development (see Footnote 5.1) of the Bennett positive pressure respirator attachment (BPPRA), in use by September 1948, to convert an INPV machine, here the Drinker, into one also capable of supplying 'intratracheal' IPPV, supplementary to its NPV. That substantially augmented the total minute ventilation delivered. Bower and Bennett, together with their team, used this attachment for 73 of 1949's 130 respirator cases, to establish the first-ever, large-scale, long-term success of IPPV for respiratory failure in acute polio. In 1949, they demonstrated the superiority with IPPV supplemental to INPV alone, achieving a survival rate of 83.7% (108/129). This contrasted with the 21% survival rate in 1946 among the 38 patients ventilated that year.

A complete system of respiratory care was developed for polio victims at LACH by 1950, with levels of treatment and expertise from Bower and Bennett distinctly higher than was currently set at other known polio respiration units. These preceded the well-documented developments in Copenhagen in the early 1950s (described in Chapters 7–9). Extensive experience was obtained in one hospital by consistent medical staffing, working as a team. Bower and Bennett deserve greater recognition of their pioneering

[1] Footnote 5.1. Concerning nomenclature: There appears to be little consistency in the descriptive words used by Albert Bower and others to name the LACH department: whether Contagious[268] versus Communicable,[269,283–288] Service[268,269,283] versus Unit,[283,288] and Disease[269,283,288] versus Diseases.[268,284–286] For instance, within the 1950 two-part principal paper, the first part is recorded as from the 'Contagious Diseases Service'[268] and the second as from the 'Communicable Disease Service'.[269] Elsewhere, for the same year 1950, it was the 'Communicable Disease Section'.[287] The term 'Unit' does not seem to have been used other than in the years 1949 and 1954[283] (confusing!).

A. The word 'General' appears to be incorporated within the LACH name only once, that is, in West and Bower's study.[288]

B. Bower and colleagues usually write about the Bennett inventions with the name Bennett bracketed.

C. The term 'Resp. Patient', as used by Bower, Bennett, Dillon and Axelrod in their Table V,[269[p687]] can confuse as to its precise meaning: whether to take it as meaning respirator or respiratory. His classification of 'Respirator Patients' included **some** non-ventilated (also see, Footnote 5.2 and Table 5.1 in this chapter).

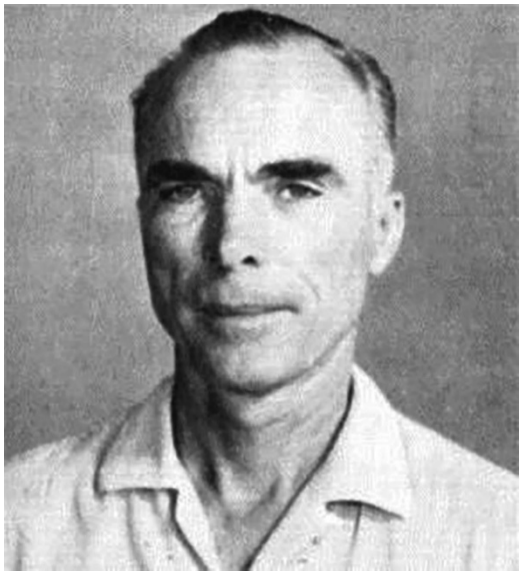
[2] Footnote 5.2. One notes that in Bower and Bennett's accounts, a patient's being classified as a 'respirator case'[269[p687]] did not necessarily indicate AV would be provided. Thus, although all the Resp. Patients in 1949 were ventilated, adequate equipment was not available for all (1948) acute cases.

merit than they currently receive in the written history of ICM. An attempt to show the justification for that is made herewith. Mention is made of improvements in equipment.

Figure 5.1: Dr Albert G Bower

Albert Bower, MD, was Clinical Professor of Medicine at the University of Southern California and Chief of Staff of the Communicable Disease Unit,[283] Los Angeles County Hospital.
Reproduced with thanks to photographer Kourken and the National Library of Medicine, National Institutes of Health, Bethesda, MD.[289]

Figure 5.2: V Ray Bennett



V Ray Bennett was Consulting Engineer, V Ray Bennett and Associates Inc, Los Angeles, CA.
His photo is reproduced with the kind permission of Blackwell Publishing, Oxford, UK.[290]

This chapter will describe some features of these pioneering achievements. Because its major interest is the treatment of acute ventilatory failure in the Los Angeles polio patients by AV, other acute aspects of their treatment of acutely ill, polio patients will not be pursued, unless they are relevant to the leading theme.

5.2 Poliomyelitis at Los Angeles County Hospital, 1946–1948

The Communicable Diseases Service of LACH provided a major receiving centre for patients with acute poliomyelitis. The basic respirator used there at that time was the Drinker–Collins cabinet respirator, a standard 'negative pressure' body-type tank; later, some Drinkers were supplied in large and in junior sizes.[283[p149–50]] There was also at least one Emerson respirator.[267[p693]]

5.2.1 Year 1946

Experience arising from the influx of patients proved salutary: Bower, Bennett, Dillon and Axelrod classified 48 of the 1,284 acute polio admissions as 'respirator patients', that is, with ventilatory insufficiency[269[p687]] (see Footnote 5.1B[269[p687],FN-2]). The mortality rate of these 48 patients (including eight 'bulbar patients' who were not ventilated) was 79%; among the 40 who were treated with INPV, 30 deaths still gave a mortality rate of 75%.[269[p687]] To what extent the AV treatment was apportioned between Drinker versus cuirass respirators is not recorded: the latter were mentioned, but without data.[283[p150]] It is evident that tracheotomy was used freely when indicated,[268[p574],288] although, as Bower recognised later, probably not every time that it should have been.[269[p687]]

Curarisation (per d-tubocurarine, as 'Intocostrin' [Squibb])[283[p44],288[p254]] was first studied for polio patients at LACH on 17 August 1946,[287] and was employed where needed to ensure synchronisation between patient and respirator.[286[p264],287,288] In addition to what appears to have been exemplary respiratory therapy[284–286, 288] and treatment of complications, appropriate attention was paid to routine supportive care, such as monitoring circulatory status, ensuring adequate nutrition and correcting biochemical abnormalities, especially of potassium ion.[283,284–286,288]

5.2.2 Year 1947

Total admissions the next year at LACH eased to 402, but two-thirds of the 21 'respirator patients' still died (it was not stated how many of these received AV). The deaths represented 4.1% of the LACH total year's polio patients, comparable with the 3.8% fatality rate for 1946.[269[p687]]

Dr Bower and team member Dr Harold West later wrote in 1949, that they were convinced their treatment (with meticulous respiratory care, often with a tracheotomy) during 1946 and 1947 saved about half the patients of a type that died earlier (i.e. those requiring AV).[288]

5.2.3 Year 1948

That year's epidemic, 'unprecedented in our experience as to the number and virulence of the cases',[268[p561]] gave a patient load of 294 'respirator patients', among the 3,094 with acute polio.[269[p687]] The high mortality expected to accompany the epidemic impelled director Bower, 'dissatisfied with the performance and action of the standard tank-respirator',[286[p262]] 'to devise additional equipment hitherto unavailable to meet the exigencies arising during the epidemic'.[286[p264]]

5.2.4 Clinical studies about September 1948

Early in 1948, Bower and colleagues conducted a study of their patients to reveal facts, 'startling in their implications'.[286[p262]] Acutely ill patients placed in INPV respirators could later be found in a state of profound respiratory acidosis (see Footnote 5.2); these included 30 patients of the 294 respirator cases in 1948, for whom standard ventilatory measures for eliminating excess carbon dioxide had failed.[269[p691]]

Further, Bower found that 'In most cases with so-called respiratory center involvement, ... it is remarkable how rapidly measures which provide a clear airway and adequate breathing can clearly show the error of this diagnosis'.[283[p3]] Also, in a patient presenting either with convulsions or coma, 'anoxia or pulmonary acidosis from too much retained CO_2 ... must be prevented or corrected to rule out encephalitis'.[285[p189]] William Frank maintained that 'most so-called encephalitic signs disappear when respiratory inadequacy is corrected',[283[p17]] as I discussed further in 2004[266] and now in Chapter 8, and as others had.[257,266,291] However, electro-encephalography suggested that clinically undetected encephalitis occurred more frequently than believed.[285[p188]]

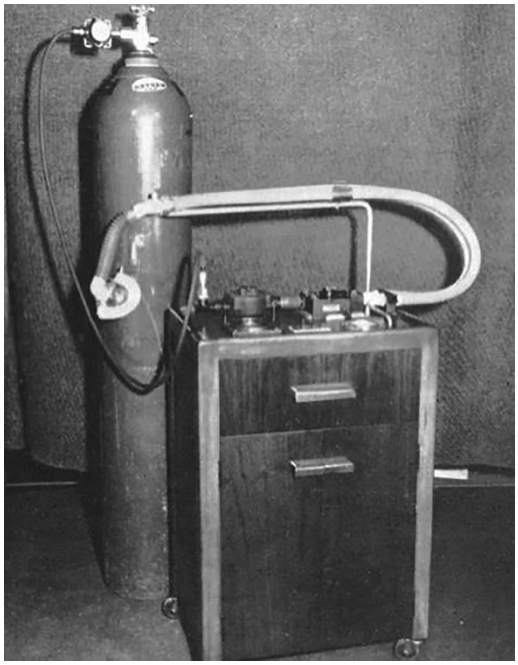
5.3 Bennett's achievements as regards newly designed equipment

5.3.1 Bennett resuscitator: Bennett flow-sensitive positive pressure breathing unit

Multiple respiratory devices already invented by Ray Bennett were being used clinically. His mobile Bennett flow-sensitive positive pressure breathing unit (BFSPPBU), 'in clinical use for several years prior' to 1948, was a 'completely developed instrument',[268[p564]] which was different from, and an improvement upon, resuscitators available hitherto (see Figure 5.3).

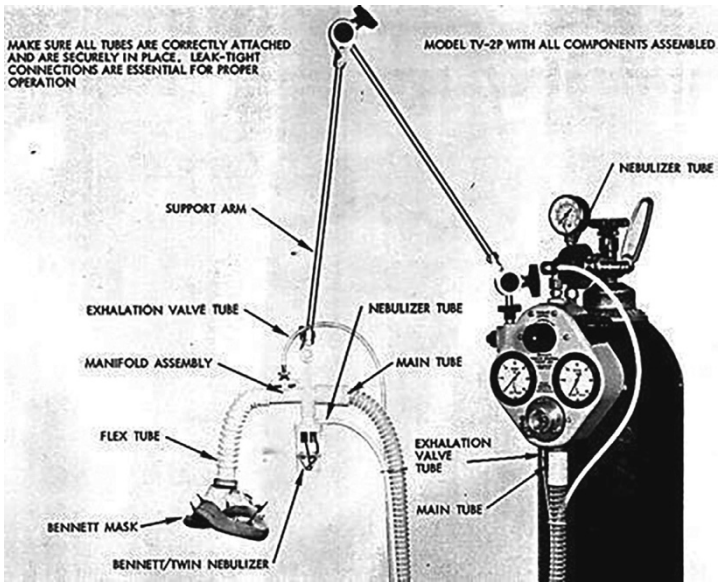
It was used extensively at LACH, mainly for respiratory emergencies or during tracheotomy, or for short-term relief. Two units were in constant service there in 1948 and later.[286[p262]] Hurley Motley and Joseph Tomashefski,[292] following experience with the MSA 'Pneophore' (for AV) from the mid-1940s, had switched to Bennett's TV-2P, as in Figure 5.4 (they appreciated the valuable properties of the Bennett flow-sensitive cycling valve [which was easy to clean, so it did not stick; there was no rebreathing] and of the compensated pressure exhalation valve [rapidly removed, readily cleaned]) to deliver bronchodilators by intermittent positive pressure breathing for patients with chronic respiratory disease.[283[p110],289] Bennett's resuscitator, the 'BFSPPBU', provided automatic cycling by pneumatic timing accumulators,[268[p565]] but also allowed the patient to take control of breathing; it was run electrically from mains power (115 volts AC) or battery (24 volts DC); by hand pump or by high flow compressed gases (c.15 L/min).[283[p152]]

Figure 5.3: The (Bennett) flow-sensitive positive pressure breathing unit**, BFSPPBU**



The (Bennett) flow-sensitive positive pressure breathing unit featured the **Bennett Model TV-2P respirator.**
Reproduced with due acknowledgement to Ray Wallace, Albert Bower and their early publishers:[268,269,284–286] the Los Angeles County Medical Association [for references 268 and 269]; and the Northwest Medical Publishing Association, Seattle, WA [for references 284–286].

Figure 5.4: The Bennett TV-2P respirator



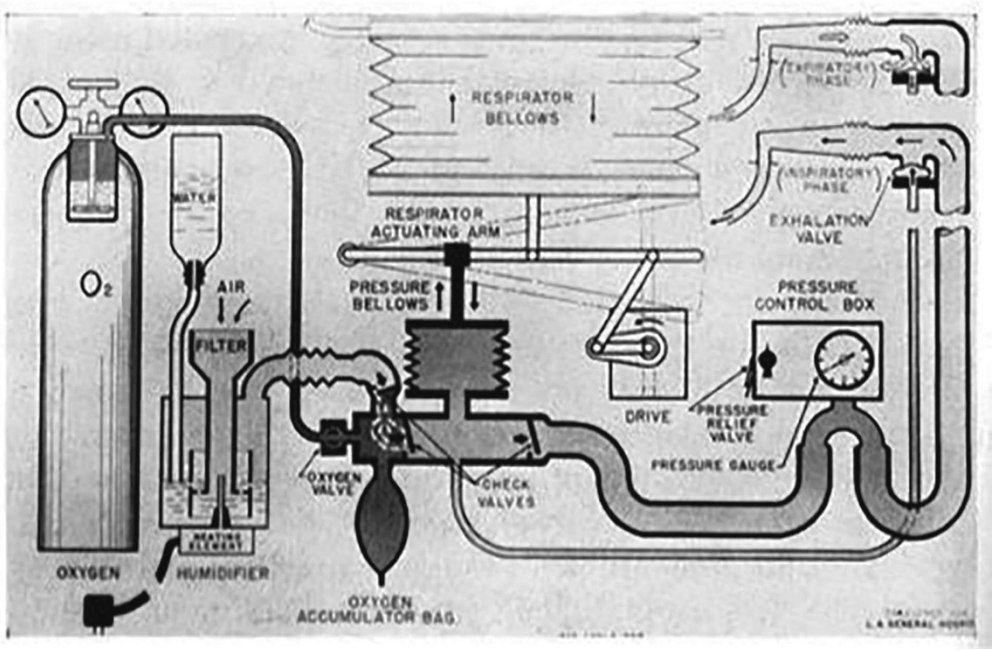
This apparatus was later incorporated into the (Bennett) flow-sensitive positive pressure breathing unit.
Reproduced with special thanks to Bob Ommen, Dr Warren Sanborn and the Puritan-Bennett Corporation staff.[293]

5.3.2 Bennett (intermittent) positive pressure respirator attachment

Bennett's BFSPPBU resuscitator was needed to help meet the demand for AV at LACH during 1948. But it proved less practical for prolonged IPPV than his later positive pressure attachment—even less than did NPV (see Bower[268[p566]] and Clarence Dail[283[p152]]). Thus, as Elizabeth Austin noted, the BFSPPBU 'was not used routinely for prolonged patient care'[283[p112]]). At Dr H West's request, Bennett and the collaborating medical engineering team 'quickly accomplished'[286[p264]] an adaptation from the BFSPPBU, known as the BPPRA, fitted as an accessory to the standard Drinker–Collins tank respirator to augment the minute volume delivered[268[p561–3],286[p264]] (see Figures 5.5 and 5.6). It was powered by the motor of the NPV tank. PPV could be applied via a mask covering mouth and nose, or via Bower's own newly devised tracheostomy adaptor.[286[p262]] Bennett's special exhalation valve was installed adjacent to the mask or tracheostomy connector.[268[p565]] The bellows attachment (see Figures 5.5 and 5.6) was 'easily installed without stopping the respirator, requiring only a few minutes'.[283[p156],286[p262]] Bower described it as 'ingenious with its simplicity'.[286[p264]]

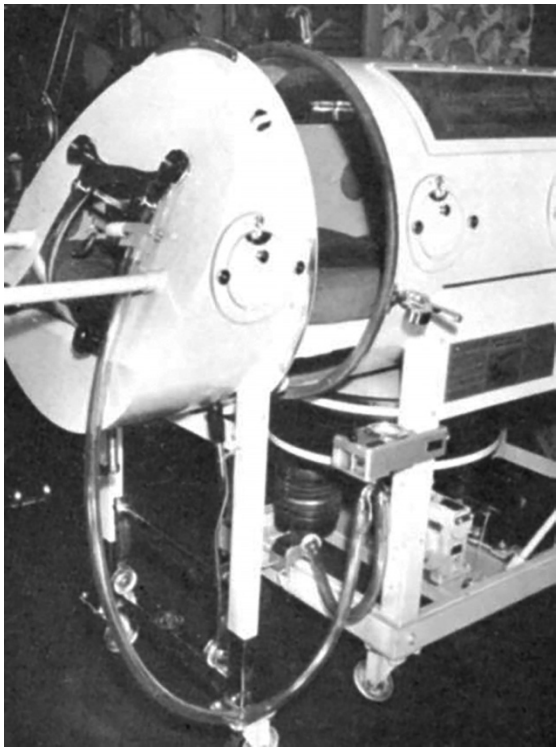
In a regulated dual action,[286[p264]] the BPPRA provided IPPV (together with humidification and, when required, oxygen and/or helium[286[p264]]) down the intratracheal passage to supplement the respirator's externally applied NPV. Bower called this the 'combined pressure' mode and saw it as 'maintaining ventilation under almost any condition'![268[p562]] It ensured a more effective ventilating volume than that from NPV alone, while allowing reduction in the intermittent negative pressure used. Typically, a negative-pressure mode with a range of −21 to −27cmH_2O was reduced to −10 to −15cmH_2O for the combined mode, which increased the tidal volume. For example, for Case 50, changing from the tank pressure of −18cmH_2O by negative pressure to a combined pressure difference of 18, derived from 9 positive pressure with 9 negative pressure, produced a rise in tidal volume from 300 to 400 cc (mL), while a tidal volume of 450 cc followed 18 positive pressure alone.[269[p690]]

Figure 5.5: The (Bennett) positive pressure respirator attachment, **BPPRA**



A diagrammatic layout of the functional parts of the (Bennett) positive pressure respirator attachment, including the optional humidifier attachment.
Reproduced with due acknowledgement to V Ray Bennett and his earlier publishers,[268,269,284–286] and with many thanks to Warren Sanborn and Puritan-Bennett Corporation staff.

Figure 5.6: The (Bennett) positive pressure respirator, **BPPRA**



Front view of the (Bennett) positive pressure respirator attachment mounted to a Drinker–Collins respirator.
Reproduced with due acknowledgement to V Ray Bennett, Albert Bower and their earlier publishers.[268,269,284–286]

For such needs as taking the patient out of the respirator, IPPV could completely replace INPV (thus, the 'positive pressure' mode), but for short periods only, even though IPPV on its own could provide greater ventilation than NPV.[268[p561],269[p691]] Certainly, the Drinker plus the BPPRA was in no way an IPPV machine in the way the 1951 Engström constant volume ventilator was, but a system of an INPV tank incorporating a device which, by delivery of a *sustained* pressure of gas at a positive pressure of 5–20 cmH_2O, rather than a set volume,[269[p691]] supplied supplemental IPPV 'in exact synchronisation with the respirator's cycle' (per 'positive pressure to the patient's lungs during the respirator's negative pressure phase, and complete release of pressure during the expiratory phase into atmosphere at zero pressure' [268[p561]]).

In Tables VI–XII of Bower, Bennett, Dillon and Axelrod's article,[269] they indicate the size of the positive pressure and negative pressure components comprising their 'Total Effective Pressure cmH_2O' in the combined pressure mode. The ranges in the studies vary: examples listed for patients were (plus)15 with (minus)17 to make a 'total effective pressure' of 32 cmH_2O[269[p690]]; or +7 with −7 to give 14 cmH_2O; or +10 with −16 to give 26 cmH_2O. Because the tables cover only some of the patients studied, one cannot determine for the combined pressure mode how often AV was essentially INPV augmented by IPPV, or essentially IPPV to a maximum of 20cmH_2O augmented by INPV. (The authors do describe IPPV as providing more effective AV, and hence, presumably, they would favour it.) The LACH experience appears to have been the first use of IPPV on a large scale, either as combined pressure or positive pressure alone.

Application of Bennett positive pressure respirator attachment[268,269,283–286]

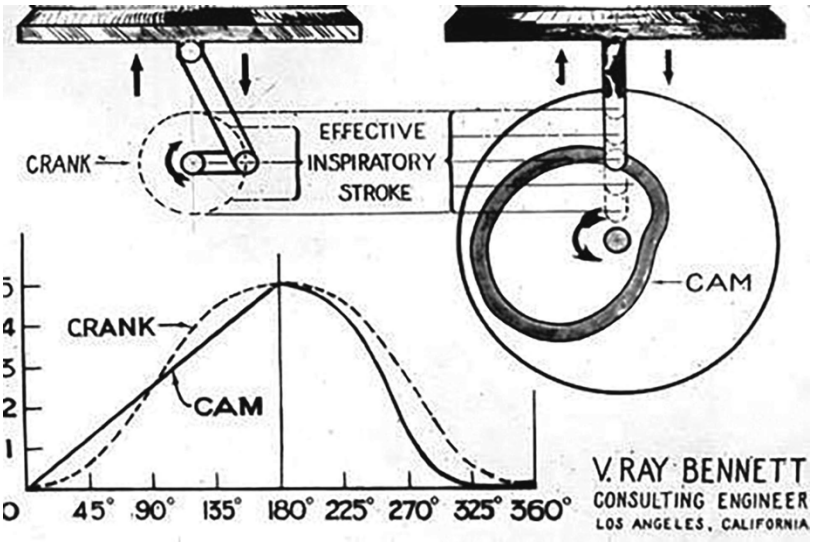
During October 1948 and throughout 1949, the LACH Service had use of 42 BPPRAs mounted on Drinker respirators. (I did not see it stated whether there were further Drinker respirators on site.) With 79 of the 130 'respirator patients' of 1949 needing a respirator (and 89, a tracheostomy), the BPPRAs were 'in almost constant use'.[268[p563]] The authors do not state the relative frequency of employing the combined pressure mode to deliver AV, versus either positive pressure or negative pressure modes singly. The BPPRA was also used at other hospitals on the US West Coast,[268[p563]] such as Rancho Los Amigos Hospital (Downey, California), and at Washoe Medical Center (Reno, Nevada) by William O'Brien and associates.[283[p234]]

The BPPRA was later developed for sole use without a conventional tank-type respirator, enabling a patient, if so able, to move around the bed.[268[p572]] But need was being foreseen to develop a 'completely new respirator design', post epidemic.[268[p582]]

5.3.4 The rotary cam

Bennett adapted a rotary cam actuation to 25 of the 42 Drinker machines, in place of the mechanism for shank actuation with its fixed sine-wave flow pattern[268[p567–8],283[p162]] (see Figure 5.7). The cam, able to produce almost any type of desired pressure pattern, was adapted for a slower, steady rate of pressure rise and a longer flow during inspiration—nearer to the ideal. It allowed lower peak pressures (which are 'more physiologically desirable'), and an inspiratory flow pattern (which could be varied), providing inflation that was more uniform, for better alveolar aeration.[283[p110]]

Figure 5.7: The rotary cam



A diagrammatic comparison of the cam versus a crank for the actuation of the Drinker–Collins respirator.
Reproduced with due acknowledgement to V Ray Bennett and his earlier publishers,[268,269,284–286] and with many thanks to Warren Sanborn and the Puritan-Bennett Corporation staff.

During expiration, the cam allowed sudden release of pressure against ambient air with a rapid expiratory flow followed by a somewhat prolonged pre-inspiratory phase, both operating to cardiovascular advantage.[283[p109]] (Two cases of circulatory impairment were recorded, attributed to 'positive pressure effects'.[269[p691]]) When the BPPRA was used in a cam-actuated respirator, 'greater advantages [were] obtained both in total ventilation and distribution of ventilation in the lungs', compared with that from the crank-actuated respirator with its typical sine-wave pressure pattern.[268[p570]] Thus Table 5.1 reveals greater ventilator efficiency in tidal air increase in the year with use of the cam—between 11% and 43%, and on average, 30%—in a group of patients studied in immense detail.[269[p708–10]]

Table 5.1: Comparison of outcomes for 1946 and 1949, inclusive*

Year	Patient's classification	Total cases	Survivors	Died
1946	'Respiratory'**	48	10 (21%)	38 (79%)
1946	Ventilated	38	8 (21%)	†30 (79%)
1949	'Respiratory'	130 (1 DOA)	108 (83%)	22 (17%)
1949	Ventilated	129	108 (84%)	21 (16%)

***** Derived from Bower, Bennett, Dillon and Axelrod[269[p687]] DOA is Dead on Arrival.
****** See Footnotes 5.1D and 5.2 herein
† The authors point out that this figure still represents 62% of 'Resp. Patients'.

In addition, many other equipment inventions or improvements were designed and employed for a complete system of safe respiratory care (listed in Addenda 5.1 and 5.2).

5.3.5 Outcomes with use of Bennett positive pressure respirator attachment

The caseload of 'respirator patients' at LACH was 294 in 1948, and 130 in 1949. Use of the BPPRA after September 1948 'virtually eliminated pulmonary acidosis in our cases and diminished or dried up secretions'.[286[p264]] Although some results from before October 1948 were available for the principal articles published,[268,269] records were complete only for the succeeding year. By 1949, the AV from a combined (or a positive, or a negative) pressure ventilatory mode was 'of significant clinical value for at least 130 acute respirator patients',[268[p563]] 82 of whom recovered to be completely free of a respirator. The total deaths in 1949 among these was 22, including one victim who was dead on arrival, a 17.9% case-fatality rate (which became 2.0% among the total of 1,128 polio patients admitted in 1949). This 1949 mortality of 17% contrasts with the 79% for patients ventilated by INPV in 1946 (see Table 1.1), although Bower notes that 'if actual 1946 respirator deaths only are used, the respirator mortality remains high [62%]' for 'Resp Cases'.[269[p687]]

Apart from its use for the acute respiratory failure of polio, the PPV apparatus provided adequate ventilation during respiratory failure emergencies, to take the patient out of the ventilator for nursing procedures, and for tracheotomy, medical treatments or transfer.

5.4 Ventilation meter

To assist a patient's impaired breathing, the medical engineering team developed a comprehensive system of respiratory and safety devices, most of which are listed with some details in Addendum 5.1.

It appears that the tool used by Bower, Bennett, Dillon and Axelrod , which was key for the assessment and for the successful, efficient control of patient ventilation, was the reliable Bennett 'respiratory ventilation meter': a positive-displacement type, with low resistance to flow and with low inertia characteristics.[268[p566–7]] This enabled 'actual breathing measurements in serial fashion'[283[p231]] of the volume of successive breaths or of vital capacity, at an accuracy always more than 95% and usually exceeding 97%.[268[p567]] It was used in comprehensive, detailed progressive ventilation studies,[268] which reported 'only a portion of the data obtained'; many early tests were discarded as unreliable, and only significant and accurate data from 'extensive' tests were documented.[269[p694]]

Data from the employment of the ventilatory meter:

- indicated when there was need for intervention with AV (the studies could 'strongly point to the importance of early measures to stop the progressive drop in respiratory function');[269[p700]]
- revealed and validated the different modes of AV undertaken;
- testified to the degree of the meter's own usefulness, and the reliance that came to be placed on it for ensuring effective AV; and
- contributed immensely to the dramatic fall in mortality rates.

The use of the meter enabled the effective control of the AV being delivered. Perhaps Bower and colleagues underestimated how critical was the role of the ventilation meter. From a study of their publications, it seems that in the overall clinical context, their ensuring of such careful control—'ventilation must be determined (i.e. individualised) for each patient'[283[p114]]—helps explain a drop in respirator mortality. That reduction would seem greater than could happen solely from the changeover in AV methods to allow IPPV supplementation. By the time of publication of the LACH book in 1954,[283] wider credit was given to the use of the meter.

It is interesting to note the specialist team's statement, which, in later experience, could appear optimistic: 'Maximum inspiration rather than maximum expiration was measured ... to prevent contamination of the working parts of the meter when used on contagious cases'.[269[p694]]

5.5 Breadth of changes at Los Angeles County Hospital

Regarding the management of polio patients, the documentation from LACH clearly demonstrates that all aspects associated with respiratory care were assessed completely, that the requirements for successful AV were determined, and that equipment redesigns were undertaken, achieved and then applied clinically.[269] It is admirable for how comprehensively the problem areas were identified in a complete working system, down to the smallest levels (e.g. for locking-type electric cord plugs and safety guards over the power switch), and then safeguarded to produce the best apparatus. Safety was also ensured: the ventilating apparatus had adequate alarms. Measures for the comfort of patients were also emphasised,

such as eliminating vibration and noise. In this total rethink, attention to redesigning extended to all equipment either already in use (e.g. tracheostomy tubes, all suction apparatus, collars and mattresses) or hitherto not available, as well as to nursing practices (e.g. early regular suctioning of the respiratory tree).

Such meticulous attention to detail in an acute respiratory unit during the last years of the 1940s certainly predated the care we prided ourselves on for ICM when ICUs were becoming more widely established a decade later. By 1954, Bower was again referring to his Communicable Diseases *Unit*[283[p1]]—which was, by the team's greatly improved results in 1949, recognisably a genuine respiration unit for acute polio and deserves greater recognition (see Footnote 5.3[3]).

Studies by Los Angeles team[269]

In their studies, the Los Angeles team obtained 'consistent and conclusive data' from 77 respirator cases, to show 'consistent and significant trends'.[269[p687]] After carefully designing, constructing and then studying the equipment in action, the authors documented 'data obtained on 77 respirator cases', and 'ventilation tests ... made on 222 patients',[269[p693]] mostly from 1949 and some from back to September 1948. Principally, the studies measured the effects of numerous factors on tidal volume and vital capacity. They investigated aspects such as the effects of different respirators, different modes of AV, combinations and pressures of AV, and different levels of paralysis and pulmonary care; the results occupied 16 pages of their 1950 Part 2 article,[269] with 27 tables and seven graphs. For instance, they found that the effect of the Trendelenburg position 'on the ventilation of respirator patients' was to cause an average drop of 20% less effective ventilation after 15 minutes.[269[pp704,712]]

These events took place a few years before the large-scale IPPV success of Ibsen and his team at Copenhagen, during Denmark's 1952–1953 polio epidemic.[257,258,291] There, the Danes applied IPPV manually by bag ventilation to a number (possibly as many as 277[266]) more than twice the number of patients machine-ventilated at Los Angeles in 1949. It does not seem well known that Bower and his colleagues, from the respirator cases they studied at LACH, achieved results comparable with those of Ibsen and his colleagues (throughout 1952–1953).[257] Ibsen studied the LACH articles before 25–26 August 1952 and hence was aware of the Americans' conclusions.[257,291]

The LACH findings indicated: [269[p688–92]]

- Underventilation was not uncommon with INPV (30 instances among 294 respirator cases were detected in 1948,[269[p691]] before extra IPPV was introduced); and underventilation allowed carbon dioxide to accumulate.
- INPV alone did not adequately ventilate some patients**:** they always had respiratory acidosis with INPV.

[3] Footnote 5.3. When Dr Matthew Spence of Auckland Hospital's Acute Respiratory Unit appraised the unit in 1961, Dr AG Bower was no longer Chief of Staff. In criticism, Matt Spence noted[270] marked hyperventilation, uncuffed metal tracheotomy tubes and the lack of a special respiratory regime(n), and was surprised that the type of AV used for poliomyelitis, polyneuritis and tetanus was still combined pressure. (He noted curarisation was employed as needed.) There were about 20 tetanus cases annually, but:

> the mortality rate was disturbing at this hospital, reaching 70% in the last two years. The methods of treatment [of tetanus] are basically similar to those at the Department of Critical Care Medicine at Auckland Hospital except for the management of respiratory insufficiency.[270[p45]]

- From routinely using the ventilation meter in respirator patients in 1949, no instances of underventilation were detected.
- IPPV provided better AV than did INPV, and IPPV could augment INPV.
- The administration of oxygen to under-ventilated patients might correct cyanosis but did not diminish carbon dioxide accumulation.
- For an index of ventilatory adequacy, reliance on either elevated carbon dioxide-combining-power, or elevated venous plasma bicarbonate, was incorrect without a simultaneous pH. For these patients, the elevations usually represented metabolic alkalosis compensatory for hypoventilation.

5.5.2 Documentation from Los Angeles County Hospital principals

OLDMEDLINE lists 35 articles with Bower himself as the first author or co-author. (PubMed lists five 1949 infectious diseases papers, two of them concerning polio.)

- West's 1949 article with Bower[288] established their case for tracheotomy, when needed, and forecast their detailed respiratory care.
- Bower published his 1949 address to the Oregon State Medical Society, 'A concept of poliomyelitis based on observations and treatment of 6000 cases in a 4 year period', in three parts between February and April 1950.[284–286]
- This chapter's reference 286 provided concisely the first documentation of the changed ventilatory methods at LACH. Relevant to this chapter is Bower's description of the process they worked through to achieve a solution regarding ventilation when tracheotomy alone did not correct respiratory problems.
- Bower and Ray Bennett, with colleagues Dillon and Axelrod (see Footnote 5.4[4]) reported their findings at considerable length and in extensive detail in the two articles describing their investigation of the care and treatment of poliomyelitis patients: Part 1 in October 1950[268] concerned the equipment they developed; and Part 2 in November 1950[269] concerned the case statistics for their clinical patients.
- By 1954, Bower, as editor, had the LACH methods summarised in the book *Diagnosis and treatment of the acute phase of poliomyelitis and its complications*.[283] He supplied only the first chapter (10 pages); numerous others contributed, including Elizabeth Austin (four chapters), and Clarence Dail and Seymour Cohen (two each). This was more of a how-to-do-it book and was short on detailed data on the number of patients treated and their survival/mortality rates.
- Dail, together with Bower and Bennett, had also written earlier in May 1950 of respiratory deficiencies in polio.[294]

[4] Footnote 5.4. Bower (and his three co-authors) carefully acknowledged associates or colleagues: Drs J Affeldt, E Austin, A Chaney, C Dail, J Chudnoff, L Fisher, W Frank, J Huntsman, E Knouf, H West, and RN W Gerling, plus resident staff, [268,269] and also R Denton and S Cohen.[283]

5.6 Comment

Despite such documentation, the Los Angeles success does not appear to have received the level of recognition warranted. Three of Bower's articles were quoted by Howe in a 1952 microbiology textbook.[211] Although Lassen cited one article of Bower and colleagues in both his 1955 World Health Organization (WHO) article[267] and his 1956 book,[227] each time with guarded approval, he was more concerned about describing the equipment improvements, rather than the results achieved at LACH (see Footnote 5.5[5]).

Ibsen, eventually in 1966,[295] and then again in 1975,[257] was generous in acknowledging his debt to Bower, although one might see his comment 'This I consider pioneer work in intensive therapy but only related to one disease'.[295[p279]] to be mildly begrudging. But Bower appears to have been ignored by Rendell-Baker and colleagues in the historical section of the 1969 second edition of the authoritative book *Automatic ventilation of the lungs* from William Mushin, Rendell-Baker, Thompson and Mapleson., and is minimally referred to in the 1980 third edition. ('Bennett and physicians used [his TV-2P respirator] to good effect during a polio epidemic in Los Angeles. Bennett pioneered in the use of IPP[V] for this and other acute and chronic respiratory problems').[68[p209]]

Two other authors who quote in textbooks the pair of articles by Bower, Bennett, Dillon and Axelrod[268,269] (Colice, in his reference 157 in a comprehensive documentation of the development of IPPV,[296[p27]] and Mörch, minimally in the extensive 'History of mechanical ventilation',[297[p22]] with its 26 pages of references), do so to mention Bennett's apparatus without any recognition—as appears, on inspection—of the clinical triumph achieved at LACH. Wackers[291] finally provides the acknowledgement owed to Bower, Bennett, Dillon and Axelrod for their LACH success, and in this century, these pioneers have been lauded in the journal *Critical Care and Resuscitation*.[298] In Pontoppidan's 2003 history of the ICU at the Massachusetts General Hospital,[299[p154]] he emphasised the triumph of the LACH with its eventual 17% mortality rate for '1949', as per this Chapter 5's Table 1, p.113. Yet in total, there does seem to be relative obscurity for these pioneers.

5.7 Some puzzles

Why is the pioneering work of Bower, Bennett and others at LACH, which established such a strong case for PPV and preceded some of Ibsen's comparable findings, not better recognised and referenced in its historical context? Perhaps because the distribution of the relatively new journal was not yet wide enough—by the time papers from Bower and his colleagues were published in *Annals of Western Medicine and Surgery*, 1950, the journal had reached only volume 4.[268,269] PubMed does not list it after October 1952. By

[5] Footnote 5.5. At Blegdam Hospital, Copenhagen, in July–August 1952,[258] before Ibsen revolutionised treatment there,[257] a large proportion of the ventilatory early (Danish) deaths (87%) would surely have been prevented had Lassen been aware of, and acted upon, Bower's article[291] (discussed in Chapters 7–9). But even when Ibsen—finally called in to a disastrous situation over 25–27 August 1952—showed him Bower's articles, a sceptical Lassen could not accept Bower's conclusions, although fortunately he let Ibsen proceed.[291] Ibsen had immediately recognised the flawed interpretations being made at the Blegdam Hospital concerning carbon dioxide content and metabolic alkalosis (see Chapter 7.2), over which he had come to the same diagnostic conclusion of respiratory acidosis as had Bower in 1948.[257,291] At LACH, Bower had decided to try Bennett's IPPV as well as INPV. (Any positive pressure used hitherto at LACH had been either intra-tank,[269[p707]] applied during expiration; or hand-IPPV during anaesthesia for tracheotomy.[287]) Bower's rationale was that intratracheal PPV would 'dry up secretions and remove excess carbon dioxide'.[286[p264]] (Bower records 1.5–2.7 litres of respiratory secretions aspirated per 24 hrs.) Any PPV apparatus available to them at that time was found inadequate: it 'interfered with free expiration and reduced tidal ventilation and the removal of excess CO_2 from the lung'.[286[p264]]

contrast, Bower's preceding revelatory articles in *Northwest Medicine*[284–286] (of which PubMed has no listing after March 1973) featured within a volume of high number (49). Probably it is unlikely that the mass of data they published[268,269] would ever have been reported with detail so space-occupying (thereby expensively), in more highly profiled, widely circulated journals.

Nor is it at all obvious that of Bower and colleagues' articles were well known within the US itself. Therefore, it is hardly surprising that news of Bower's methods, as presented in his 1949 address to the Oregon State Medical Society, did not reach the ears of Ibsen in that same year, who was then a budding thoracic surgeon who had switched to training in anaesthesia in Boston.[295[p281]] Returning to Denmark, Ibsen, 'consultant to the largest medical library in Denmark',[257[p22]] discovered the LACH articles at Copenhagen: 'Bower and Bennett I saw in the library' (Bjørn Ibsen, personal communication, 2003). Realising that the LACH team had demonstrated the benefits of IPPV for severe polio, Ibsen, when back at Copenhagen and likely aware of the disaster of poliomyelitis treatment there, wrote directly to Bower requesting a copy of his papers be sent to him,[291] before suggesting to Lassen (in charge of treatment for the Blegdam Hospital's polio patients in respiratory failure) that Bower's methods were favourable. Although Lassen was unconvinced of the merits of the treatment, he did 'allow' Ibsen to go ahead with m-IPPV, with a dramatic result.[257] Ibsen always freely acknowledged his debt to Bower and colleagues[257,291]

However, Bower and colleagues did not foresee that IPPV might actually replace tank respirators altogether.[300] Perhaps, a lack of knowledge about the findings at Los Angeles played a role in the persistence with INPV (instead of changing to IPPV) in the US for so long into the 1950s? By contrast, IPPV was quickly adopted in Europe after the lead from Ibsen and his colleagues.[68[p209]] The Danish epidemic was over by May 1953.[298] In the succeeding September, a polio victim was treated with IPPV at Oxford, UK, with Radcliffe A & B respiration pumps by Alex Crampton Smith and JM Spalding,[301] while another was treated at Ham Green, Bristol, UK, with James Macrae's 1953 Clevedon respirator.[300] Stockholm, ready for the epidemic of 1953 with Engström and other Swedish IPPV ventilators,[302[p63]] achieved a 70% survival rate success among 89 respirator patients.[302[p111]]

5.8 Summary

Greater acknowledgement should be made of the ventilatory achievements of Albert Bower and his teams in the Contagious Diseases Service at LACH in the 1948–1949 polio epidemic. There, the first long-term, large-scale innovative use of IPPV supplementing INPV proved successful. Without diminishing the tremendous credit due to Bjørn Ibsen and his colleagues, the Los Angeles pioneering, although on a smaller scale than that which took place at Copenhagen in 1952–1953, did occur earlier. The marked reduction in the mortality rate of LACH's ventilated patients, which was associated with the clinical expertise developed in the Service, together with the remarkable range of respiratory equipment and ICU-type equipment developed onsite, especially from the notable Ray Bennett, all testify to the excellence of the team, as can be seen in their documentation. Numerous studies, conducted in parallel with treatment, provided a scientific basis for their achievements.

It is evident from the documentation supplied by the LACH that these achievements place the LACH Contagious Diseases Service at the forefront of respiration units established at that time (although there does not appear to be any such claim obvious from LACH itself). Moreover, although the participants provided relatively complete documentation of their work, these virtues have not been widely recognised. The opportunity exists now to ensure that they are.

Addendum 5.1: Equipment developed by Ray Bennett[268[p561–82],283[p147–73]]

Ray Bennett (1922–1996) was already famous from WW II days for his BR-X2 resuscitator, which provided intermittent breaths of high-pressure oxygen to pilots in unpressurised aircraft involved in high altitude research.[68] He developed a flow-sensitive valve for the resuscitator in 1944. His mobile BR-X2 resuscitator for emergency IPPV of any degree required time-cycled use; for long term use, it was less practical than a respirator. After WW II, Bennett's attention to the problems of ventilated polio patients at LACH resulted in the following developments:

- the BPPRA, which was used with a mask, or with Bower's adaptor to a tracheotomy tube;
- the humidifier attachment for the BPPRA, which supplied 80% relative humidity (but at room temperature);
- the respiratory ventilation meter, which gave >95% accuracy and was used in all the 1949–1950 ventilatory studies;
- the (Bennett) flow-sensitive pressure breathing unit, which was in clinical use for several years before 1948;
- a physiological cam for the respirator, which increased ventilatory depth and patient comfort for a negative pressure lesser than that applied by a Drinker, and could be made to produce almost any pressure pattern;
- a mobile, motorised unit (a BPPRA unit in a cabinet), which was not flow-sensitive and provided fixed-cycle IPPV,[268[p572]] but to be connected in 'selected cases' only (as it was unsuitable for long-term use), via a mask or preferably an adaptor to a tracheotomy tube;
- an oxygen cylinder warning signal device, which sounded at 10 minutes residual oxygen;
- a respirator-pressure warning signal, which flashed red and was designed to be as foolproof as possible;
- an air-pressurising unit (electrical) to supply filtered air for the (Bennett) flow-sensitive pressure breathing unit;
- other advances in instrumentation:
    - improved respirator collar;
    - improved tracheotomy tubes, adaptors and accessories, and improved suctioning equipment;
    - positive pressure adaptor for patients due for bronchoscopy;
    - oxygen catheter for tracheostomy tubes and new smaller-celled pulsating pneumatic respirator mattress; and
- multiple miscellaneous 'developmental' improvements in the respirator itself.[268[p582]]

(The Puritan Compressed Gas Corporation, [which originated in the Parker B Francis Company of 1913], acquired V Ray Bennett & Assoc., 1956, hence the name Puritan-Bennett Corporation).

Addendum 5.2: Bower and colleagues' approach for physiological research[269[p688]]

Bower and colleagues used the following approach for their studies:

- a femoral arterial line (indwelling) for sampling, which was usually performed in duplicate;
- two laboratories, which enabled cross-checking of results;
- arterial oxygen saturation per (Van Slyke method of) vols% (with whole blood);
- assessment of respiratory acidosis from:
    - pH per a Beckman meter; with
    - CO_2 values by using two different laboratory methods:
        - (Van Slyke method of) total carbon dioxide, or
        - after removal of carbon dioxide from plasma, back-titration to original pH, which gave bicarbonate vols % of plasma.

(This method of assessing respiratory acidosis anticipated the Blegdam method, post 27 August 1952. Before that, Lassen was determining only the total carbon dioxide content, as the only pH electrodes available to him were too large for practical use until Poul Astrup soon acquired Radiometer's pH mini-electrode.[257,291]) By 1954, oximeters and infrared analysers for carbon dioxide were being used.

20th century artificial ventilation before the Danish poliomyelitis epidemic

Chapter 6: Pioneering intensive care medicine by the 'Scandinavian Method' of treatment for acute barbiturate poisoning

Chapter 6: Table of Contents

6.1 Introductory overview

Between the 1920s and the mid-1950s, barbiturates were the sedative–hypnotic agents most used in clinical practice. Their ready availability and narrow therapeutic margin accounted for disturbingly high rates of acute poisoning, whether suicidal or accidental. Until the late 1940s, medical treatment of poisoning was relatively ineffective, with mortality subsequently high, not only from the effects of coma, respiratory depression and cardiovascular shock with renal impairment, but also during the 1930s to 1940s, from complications due to the heavy use in treatment of analeptic stimulating agents. The incidence of barbiturate intoxication increased substantially following WW II. This chapter details the development of what soon became known as the 'Scandinavian Method' of treatment, which contributed substantially to the earliest establishment of ICUs and to the practice and methods of ICM.

Three medical names stand out for pioneering the Scandinavian Method of treatment. Successively,

- Aage Kirkegaard of Denmark, psychiatrist, for introducing effective anti-shock therapy with intravenous fluids;
- Eric Nilsson of Sweden, anaesthetist, for introducing anaesthesiologic principles including m-IPPV into clinical practice; and
- Carl Clemmesen of Denmark, psychiatrist, for introducing the centralisation of seriously poisoned patients in a dedicated unit.

Clemmesen's Intoxication Unit opened at the Bispebjerg Hospital, Copenhagen, on 1 October 1949. Bjørn Ibsen, the pioneer of ICUs, suggested it was the *initial* ICU, while noting, however, that it supplied intensive therapy for only one type of disorder (as would Lassen's Blegdam Hospital unit during Denmark's 1952–1953 polio epidemic). The claim to the first ICU is discussed in the Addendum to Chapter 8. Treatment for barbiturate poisoning during the 1950s in some other Scandinavian hospitals will also be briefly considered in Addendum 6.1 to this chapter.

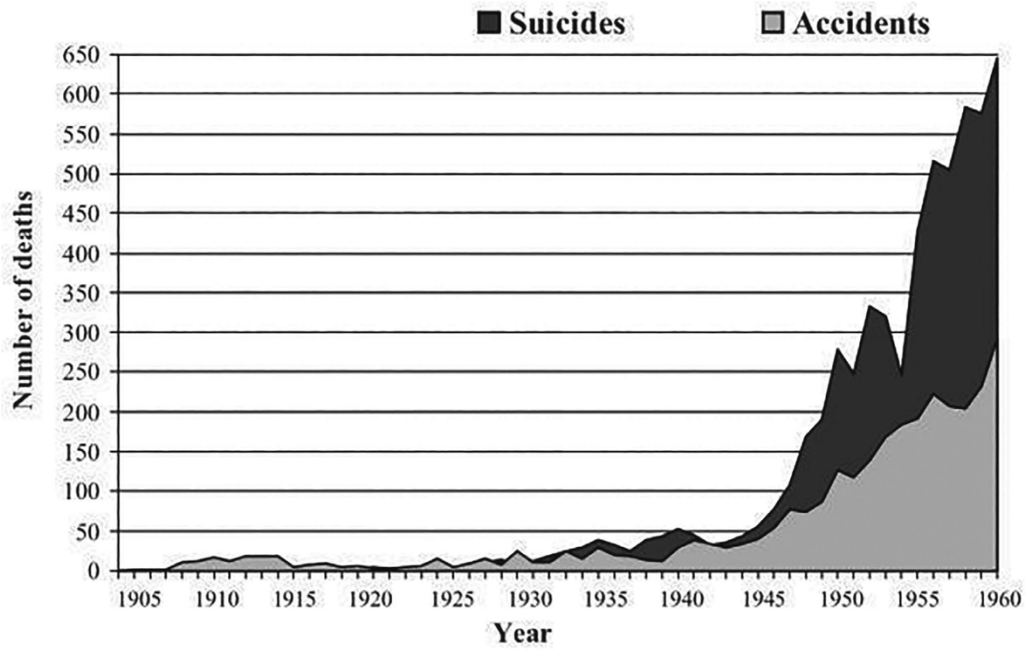
6.1.1 Mortality from barbiturates

In 1903, Emil Fischer and Josef von Mering recognised the sedative/hypnotic properties of barbital ('Veronal'®),[303] but cautioned that although no indications of adverse side-effects had been found, further research was required. From the 1920s to the mid-1950s, 'practically the only drugs used as sedatives and hypnotics were barbiturates'.[304[p14–5]] Such ready availability ensured the prominence of barbiturates in self-poisoning. López-Muñoz's graph of deaths from barbiturate poisoning in England and Wales in 1905–1960[305] (per the *Registrar-General's Statistical Review for England and Wales*[306] and as illustrated in Figure 6.1) demonstrated that death in the early years of barbiturate usage resulted typically from 'accidents', whereas from the 1930s, suicide became the prime causative factor. Intoxication from barbiturates in Scandinavia after WW II was enormous.[307] Thus, annual barbiturate deaths at that time numbered somewhat over 400 in Denmark but not fully 100 in Sweden.[308] In 2010, in the US, barbiturates were designated still the drugs of choice in geriatric suicide.[309]

6.1.2 Manifestations of barbiturate intoxication

Severe barbiturate intoxication produces deep coma and has life-threatening complications. Such coma allows the tongue musculature to relax, which obstructs the airway, and it impairs protective laryngeal and tracheal reflexes, thereby leading to retained secretions, atelectasis, pulmonary aspiration and respiratory infection. Respiration is affected early,[310] with hypoventilation—at worst, apnoea—producing hypoxaemia and respiratory acidosis. The barbiturate-induced dilatation of arterioles and venules with the depression of myocardial contractility can cause circulatory shock with subsequent renal impairment, all of which are potentially life-threatening.[310] In this condition, meeting immediate resuscitation needs becomes vital.

Figure 6.1: Deaths from barbiturates



Deaths from an overdose of barbiturates, by accident or by suicide, in England and Wales during the period 1905–1960 (*Registrar-General's Statistical Review for England and Wales*).
Adapted from Glatt (1962) by Prof. Francisco López-Muñoz, Ucha-Udabe and Alamo[305]
Reproduced with thanks to Prof. Francisco López-Muñoz, Ucha-Udabe and Alamo.

6.2 Scandinavian pioneering in management

The worldwide problem of acute barbiturate poisoning was well documented in Scandinavia from the early 1930s, especially at Copenhagen's Kommunehospital (Municipal Hospital), Bispebjerg Hospital, and Rigshospital (State Hospital), and later, in Sweden at the University Hospital, Lund, followed by the Southern and Karolinska Hospitals, Stockholm. This chapter recognises the pioneers who introduced effective treatment through what soon came to be known as the Scandinavian Method. This chapter will focus on Bispebjerg psychiatrist Dr Aage Kirkegaard's contribution of anti-shock fluid therapy; on his psychiatrist

colleague Dr Carl Clemmesen's policy of 'centralisation' at the Bispebjerg, and on his multiple detailed papers; and on Dr Eric Nilsson, anaesthetist of Lund, who was foremost in effectively introducing anaesthesiologic principles into treatment.

6.2.1 Progression of earlier treatment methods for severe acute barbiturate intoxication

Before specific attempts to counteract the effects of barbiturate poisoning, general hospital care during the 1920s to 1930s involved supportive medical and nursing attention for the unconscious patient (positioning, turning, suctioning, hydration, etc). For severe intoxication, these limited measures proved inadequate or ineffective in controlling the life-threatening consequences for respiratory, cardiovascular, neurologic and renal/metabolic functions, and too many lives were lost.

Carl Clemmesen (see Figure 6.2) asserted that by 1930, Danish treatment was anything but passive.[311,312] From long involvement in caring for poisoned patients, his relevant publications (following his first paper in 1932,[313] at least 15 concerned barbiturates,[311] which are referenced herein: five in English, three in Danish) showed him initially continuing the 'energetic' methods of the 1920s: 'massive' gastric lavage, followed by mild stimulation (e.g. 'one ml camphor oil, coffeine [sic], or digitalis alternately every second or third hour and ... subcutaneous saline').[311[p209]] After the dangers from lavage were demonstrated,[314] only emptying of the stomach by aspiration was occasionally undertaken.[315] A futile gastric 'charcoal period'[311[p210]] of treatment, also hazardous,[307] lasted only from 1940 to 1945.[311]

Figure 6.2: Dr Carl Clemmesen

Dr Carl Clemmesen (1899–1966), Copenhagen psychiatrist at the Community Hospital in 1932–1938 and at the Bispebjerg Hospital in 1938–1966.
From the Medical Museion, Københavns Universitet, with thanks to Ion Meyer.[316]

Within the period October 1928 – June 1930, New York's Bellevue Hospital pioneered respiratory compensation after barbital intoxication, by INPV, employing a Drinker respirator,[317,318] possibly with success, although the actual number of barbiturate cases was not stated.

At Copenhagen's Kommunehospital, psychiatrist Max Schmidt, rationalising that it was natural and obvious to try this respirator on patients with impending respiratory paralysis,[319] tested the INPV respirator of August Krogh (1874–1949, Denmark's 1920 Nobel laureate for 'Physiology or Medicine').[320] In 1931, Schmidt ventilated four sedative-intoxicated patients at the Community Hospital,[319] but the outcomes were still fatal. Once INPV was abandoned, it had no similar application recorded in Scandinavia until Kirkegaard's efforts in 1944.[321]

With lives still being lost, Clemmesen, who also was at Copenhagen's Kommunehospital, reported investigating analeptics, first with a large dose of nikethamide/Coramine®, especially to stimulate the respiratory centre.[322] Although transient lightening could occur, results were not revolutionary. Thus, among 33 patients with severe poisoning due to sedatives, only 14 died (i.e. 'only' 42%).[311] With nikethamide providing no ready answer, many other stimulants were tried: various pentylenetetrazoles (e.g. Metrazol), geastimol, picrotoxin, etc.[311] But whatever respiratory stimulation was achieved, all these agents could bring risks such as convulsions, pulmonary aspiration and hyperthermia. Clemmesen and Nilsson noted that during this era, intensive central stimulatory therapy did not decrease mortality below 20%.[307] Yet, psychiatrists battled on hopefully over about 15 years with analeptics.[311] To limit the frequently fatal cryptogenic hyperpyrexia induced by analeptics,[311] their usage was gradually decreased,[323] although it was only by 1949–1950 that they were formally declared abandoned.[311]

Elsewhere in Denmark, traditional methods of gastric lavage and charcoal, neostigmine and fluid therapy continued, such as at University Medical Clinic, Aarhus.[324] There, during treatment of 193 patients for barbiturate poisoning with total mortality at 20.7%, John Riishede conducted an analeptic trial from 1 November 1946 to 1 June 49: nikethamide for 61 patients initially, then amphetamine for the next 132. He established lower mortality with amphetamine: 9% died (21% of severe cases); contrasting with nikethamide: 44% died (63% of severe cases).[324] Again, before their final abandonment during the late 1950s, some creeping reintroduction of less drastic stimulatory agents was tried before their final abandonment in the late 1950s.[325,326]

Concerning the airway, most clinicians outside the OT, such as psychiatrists, were probably unaware of the value of endotracheal intubation (until 16 November 1950, anaesthesia was without specialty status in Denmark; and perhaps before 1946 in Sweden, since the first academic unit and the Swedish anaesthesia society, 'Narkosläkarklubben', were founded in 1946). In barbiturate poisoning, treatment with tracheotomy—either to protect lower airways or to access them—appears undocumented around this time.

In 1944–1945, Kirkegaard's laboratory experiments to extend survival time endorsed a combination of AV and fluid therapy for treating respiratory paresis in rats poisoned with barbiturates.[327–329] Yet, his 1944 employment of INPV solely by cuirass among 52 comatose human patients produced only a limited number of survivors in a non-randomised comparison: three from 23 ventilated, compared with six from 29 treated along conventional lines and not ventilated.[321] In fact:

> Kirkegaard's patients were treated with the respirator if one was available and if personnel who knew how to operate the apparatus were on duty ... From the description of the way the respirator treatment was carried out, it is obvious that very few patients were in fact ventilated. (Dr Preben Berthelsen, personal communication, November 2005)

Kirkegaard mentioned neither tracheotomy nor endotracheal tubes in his paper. He concluded that barbiturate deaths in humans were more commonly due to secondary circulatory shock than to the respiratory failure demonstrated among his rats.[327] It can be noted that a 1944 *Lancet* Annotation reported the Both respirator as being employed routinely[253] for barbiturate poisoning in Britain, but I can find no documented evidence for that claim.

Figure 6.3: Dr Aage Kirkegaard

Dr Aage Kirkegaard (1914–1992), Copenhagen psychiatrist at the Bispebjerg Hospital, Copenhagen (and 1936 hockey Olympian). From the Medical Museion, Københavns Universitet, with thanks to Ion Meyer.[330]

6.2.2 Introduction of effective treatment for barbiturate-induced circulatory failure

The dominant cause of (barbiturate) death before 1945 was peripheral circulatory failure.[307] Kirkegaard's experimental work (with Georg Brun's methods[328,329]) established that concentrated re-dissolved dry serum was effective in treating this extremely serious complication.[311] Studying 16 deaths among 80 Bispebjerg patients who were severely poisoned,[329] he devised the revolutionary[331] introduction of effective treatment for circulatory shock and tissue anoxia.[327,329] In November 1945, Kirkegaard presented to the Danish Psychiatric Society a series of cases of severe barbiturate poisoning; these patients recovered after the shock had been treated with intravenous injections of this serum.[311,312] Clemmesen, whom Ibsen credits with initiating Kirkegaard into that work in 1943,[295] declared that these results were the first real progress.[312] In 1946, clinical outcomes at Bispebjerg Hospital were regarded as improved greatly[311] (without further

elaboration), while countrywide, Denmark's mortality with 'barbiturate-etc'[332] agents (the term is explained in the next paragraph) fell: thus, 25% in 1945 became 17% for 1947. Then, from 1948, Denmark's 'barbiturate-etc' mortality was 14%, including Copenhagen's (which was separated out for the first time) at 12%. See Clemmesen's 'Table 1'.[311] Later, Clemmesen regretted that 'the work was hesitant and the results minimal ... [prior to adopting this] ... right line'.[311[p213]] Nilsson also acknowledged and consistently carried out[308] Kirkegaard's effective anti-shock therapy during 1949–1950.

This historical review adopts the expression 'barbiturate-etc.' to cover the sedative poisoning agents under discussion. In relevant Danish papers, there can be imprecision or confusion in distinguishing what proportion is solely barbiturate (although not with Myschetzky,[332] who, in 1958, tabled exact numbers comprising 72% of the Bispebjerg narcotic poisonings as being barbiturate). Further, for Clemmesen's reduplicating of the Danish Department of Health's numbers over the years 1945–1951, poisonings copied into his 1954 'Table'[323] were titled as 'barbiturates, morphine, etc.', but in his 1959 'Table 1',[311] the description was for 'Narcotics, Morphine etc.' (see Table 6.1). At times, titles of papers highlight 'barbiturate' while the text's range of 'hypnotic' poisonings includes other sedatives, etc.[331] This did not occur with Myschetzky,[332] as already mentioned in this paragraph.

Table 6.1: Danish 'Poisonings with Narcotics, Morphine, etc* 1945-1957'

OCTOBER 1959 DANISH MEDICAL BULLETIN

Table 1.
Cases of Poisoning with Narcotics, Morphine etc. Admitted to Hospitals in 1945 - 1957.

	Capital: Copenhagen, Frederiksberg & Gentofte		*Remainder of Denmark*		*Entire Country*		*Lethality as Percentage of Cases Admitted*		
Year	No. Cases	No. Deaths	No. Cases	No. Deaths	No. Cases	No. Deaths	Copenhagen	Denmark excl. Copenhagen	Total
1945		134		235	1509	369			24.5
1946		121		248	1488	369			24.8
1947		122		234	2122	356			16.8
1948	802	96	1612	234	2414	330	12.0	14.5	13.7
1949	1041	63	1749	180	2790	243	6.1	10.3	8.7
1950	1288	48	1637	135	2925	183	3.7	8.2	6.3
1951	1276	21	1725	90	3001	111	1.6	5.2	3.7
1952	1115	25	1777	86	2892	111	2.2	4.8	3.8
1953	1790	37	2044	79	3834	110	2.1	3.9	3.0
1954	1807	41	2166	88	3973	129	2.3	4.1	3.2
1955	1913	32	2318	64	4231	96	1.7	2.8	2.3
1956	1837	20	2123	48	3960	68	1.1	2.3	1.7
1957	1942	25	2171	46	4113	71	1.3	2.1	1.7

Dr Clemmesen duplicated in his 1959 article this table[311] of Denmark's Department of Health statistics* (compare: Clemmesen's text using the wording, 'cases of barbituric acid and morphine poisoning'[311]).

6.2.3 Regular respiratory measures

Clemmesen cited 1947 as the year for regular respiratory measures: 'continuous oxygen therapy ... with special attention [undefined] to keeping the air passages clear'.[311[p84]] Initially, oxygen was administered with 4 or 5% carbon dioxide as carbogen; later, oxygen alone was delivered, by mask, nasopharyngeal catheter or hollow tongue depressor.[333] Toileting helped clearance of the airway. Dr Per Lous introduced daily bronchoscopies at the Rigshospital in Copenhagen, for patients with pulmonary atelectasis and cyanosis after barbiturate intoxication.[334] (Two patients were intubated with cuffed endotracheal tubes—but this was for short periods, only to measure the spontaneous ventilation.) No clear evidence emerges to indicate use then in poisoning of either tracheotomy or endotracheal intubation. Later however, in 1952–1956, both were documented as options, that is, in Myschetzky's phrasing, unchanged principles of treatment for that period.[332]

6.3 Centralisation for treatment

As already acknowledged, Kirkegaard's 1945 anti-shock innovation was well established, and the importance of airway clearance and oxygen had been recognised in 1947. With Copenhagen's treatment needs becoming more costly, and also challenging,[311] by 1947, Clemmesen[312] was advocating the centralising of all serious barbiturate or other intoxication patients for treatment. Hence, a formalised Intoxication Centre was inaugurated within the Department of Psychiatry at Bispebjerg Hospital on 1 October 1949.[333]

However, at the Clinic of Internal Medicine, University Hospital, Lund, in April–September 1949, anaesthetist Eric Nilsson with physician Bendt Eyrich had already been defining and applying anaesthesiologic principles for treating barbiturate-intoxicated patients.[335] The next year, Nilsson transferred to (was allowed to stay at[308]) the Bispebjerg unit and introduced his non-stimulating form of treatment for barbiturate poisoning during February to March and July to August 1950.[308] That year, barbiturate-etc. mortality was further reduced: Denmark's fell to 6.3%, Copenhagen's 3.7% (Clemmesen's Table 1[311]). By March 1951, Stockholm's Southern Hospital [Södersjukhus] could claim to be following the Nilsson/Eyrich treatment scheme.[336]

The Bispebjerg's Intoxication Centre was a 4-room/9-bed unit[323,333] dedicated to centralisation for serious poisonings (until February 1971, when it was moved to the acute medical ward at Bispebjerg Hospital[337]). Clemmesen and Bie quickly reported on the Centre (April 1950[333]), to demonstrate the advantages of standardised practice,[311] for the Municipality and County of Copenhagen.[312] *S*taffing had psychiatrists in charge, with departmental registrars and specially trained nurses.[323] Anaesthetist Eric Nilsson attended for a part of 1950,[308] and, as required, psychiatrists could call on anaesthetists in particular[312] as well as laryngologists[338] and renal specialists.

All physicians and rescue services were indoctrinated[339] towards getting seriously poisoned patients rapidly to the Intoxication Centre, wherever feasible. The criterion for admission was poisoning so severe as to cause unconsciousness.[323] Once established, the Centre treated approximately the severe half[311] of Copenhagen's poisoned patients. (Copenhagen city's population was then about 1,200,000.[323]) For the later period, 1952–1956, about 30% of 1,290 patients were unconscious for more than 24 hours.[332]

6.3.1 Improvement in survival

Centralisation, together with the standardised measures employed by experienced staff (with Swedish input[340]), resulted in a remarkable reduction in barbiturate and morphine mortality. Clemmesen could report 12 patients, aged 70–85 years, all recovering from what he described as severe poisoning with barbiturates with unconsciousness for 24-144 hours.[341] Requirement for ventilatory treatment was not specified.

The reduced mortality was demonstrated in Clemmesen's tables, at first up to 1951,[323] and then up to 1957.[311] Despite the 'Capital' (listed in Table 6.1, herein) annual admission rate rising from 802 patients in 1948 to 1,942 in 1957, Copenhagen's 1948 mortality rate of 12.0% had fallen to 1.3% by 1957.[311] Suspending the heavy deployment of analeptic agents, at Nilsson's advocacy, helped produce this result; that barbiturates then were currently shorter-acting, contributed.[332,342] The Copenhagen Centre's success was achieved through a mainstay of specially trained nurses,[337] providing expert nursing care and physiotherapy.[323]

6.4 Bispebjerg mortality during 1950–1953

Bispebjerg psychiatrist Andrej Myschetzky's analysis of serious barbiturate complications and mortality showed 109 deaths,[343] but he did not supply the admission total. About one-third each was attributed to respiratory and cardiac complications; about one-sixth each to renal complications and protracted shock (see Table 6.2, herein). Myschetzky's own 'Table 2'[343] reveals that the 33 who died underwent an unexplained respiratory paralysis as the most important addition to the actual cause of death, while 58 had a pre-existing severe disability.

Table 6.2: 'Causes of death in acute barbituric acid poisoning' at Copenhagen Intoxication Centre, 1950–1953[343]

Cause of Death	1950–1953	1950	1951	1952	1953	Total
Respiratory*	c.⅓	15	4	5	11	35
Cardiovascular**	c.⅓	12	7	8	10	37
Renal	c.1/6	4	4	3	5	16
Protracted shock	c.1/6	7	3	1	6	17
Other		0	1	1	2	4
Total no. of deaths	c.1/6	38	19	18	34	109

* per Aspiration: 4[11%]; atelectasis: 8[23%]; pneumonia: 23[66%], but was 'virulent' during 1952–1959.

**per Pulmonary oedema: 27[74%]; pulmonary embolism: 6[16%]; coronary occlusion: 4[11%].

Total number of admissions is not given (but per annum: ~800[315] or ~'800–979'[311])
For ~½ of total: 'serious somatic conditions of one sort or another'; ~⅓ of total were aged >60 years.

6.4.1 Bispebjerg mortality for the period for 1952/3 to 1956

Myschetzky asserted that the basic principles of treatment at the Centre remained unchanged throughout the period.[332] His careful study of details for 1952–1956 noted 3,150 patients poisoned with barbiturates, comprising 72.6% of the 4,339 'narcotic' total[332] (which included 11% from carbon monoxide poisoning). Respiratory paralysis after barbiturates occurred in 61 patients, with death following in 29 (both numbers were included within a total of 43 'narcotic' deaths among a total of 101 'paralysis' patients). However, with opiates alone, death following breathing paralysis occurred in only one of 15 patients. Barbiturates accounted for 40% of deaths in those older than 60 years.[332] But, by this time, newly available methods were being introduced, such as haemodialysis, noradrenaline, bemegride and nalorphine.[331,332]

6.4.2 Some non-Danish treatment centres

A similar centralising unit in the 1950s in England, the North-East Metropolitan Regional Barbiturate Unit at Oldchurch Hospital, Romford, appears to be the British first,[340] and effective treatment became well established in America.[339] Locket and Angus's 1952 report from Glasgow[344] claimed complete recovery from barbiturate poisoning for a single patient after administering artificial respiration for the first hour or so; 83 others received entirely conservative supportive care among whom two died, which was probably unavoidable. This chapter's Addendum 6.1 provides brief details of certain Swedish units in 1944–1959. A special department for centralisation, established on 19 September 1955 at Södersjukhuset, Stockholm, was documented as not exclusively for intoxications.[345]

6.5 Utilising anaesthesiologic principles

In the early days of ICM, the noteworthy skills of an anaesthetist included endotracheal intubation plus IPPV and circulatory support. Outside the OT, barbiturate-poisoned patients presenting with airway problems, breathing inadequacy and circulatory depression thereby became prime candidates for the anaesthesiologist's attention. At Lund, Eric Nilsson brought this innovation into the University Hospital's Medical Division in 1949.[308,335] The following February, he took his skills to Copenhagen's Intoxication Centre where the uptake of Kirkegaard's 1945 regimen of adequate shock therapy for barbiturate-induced shock and renal impairment, plus oxygen and airway attention, had already improved outcomes.[323,341]

Alerted from the experience of anaesthesiologists with overdoses from anaesthetic agents, Nilsson emphasised the danger in barbiturate-induced coma of serious hypoxia in the cerebrum,[335] whether from airway obstruction, inadequate ventilation or areflexia (loss of cough). Further, administering analeptics only worsened brain and other hypoxia. Hence, urgency to prevent or correct hypoxia was paramount. At Lund, 1949, full respiratory care was prescribed.[335,346] Airway patency, if not established by tracheo-bronchial toilet, necessitated naso/intra-tracheal intubation to facilitate clearance and to enable energetic or artificial respiration with pure oxygen through the air passage, which had been rendered safe.[335]

Figure 6.4: Dr Eric [‘Nilla’] Nilsson

Dr Eric [‘Nilla’] Nilsson (1915–2008), Prof. of Anaesthesiolgy at Lund, and Foundation Editor of Acta Anaesthesiologica Scandinavica. With thanks to the Southern Swedish Medical History Society and Prof. Berndt Ehinger.[347]

It would appear that Nilsson was not using the term ‘artificial respiration’ solely for INPV/IPPV but also for oxygen alone, when directed into an endotracheal tube. Tracheotomy was performed as necessary[346]; m-IPPV was unspecified. For very shallow breathing, once airway integrity and oxygen supply were established and if the respiratory centre was tentatively proven responsive to carbon dioxide, douches of carbon dioxide/air were pulsed once each hour (e.g. for three of Nilsson’s first four Lund patients) to bring about temporary hyperventilation, primarily to prevent atelectasis.[335] Table 6.3[348,349] summarises the evolution of the Scandinavian Method.

Table 6.3: Phases of evolving treatment for barbiturate poisoning (additionally to general supportive care)

1. Attempted compensatory respiratory support by INPV 1931, Copenhagen: failure with Krogh respirator[319] 1944, Copenhagen: failure with cuirass respirator[321]
2. 1932, Stimulation by high-dose nikethamide,[322] and then other analeptics
3. Abandoning of unprofitable treatments: 1942, Gastric lavage[323] 1944, Gastric instillation of carbon particles [after 5 years of efforts[311]] 1949, Analeptics[333] [after about 15+ yrs, but not completely] 1950, Carbon dioxide applications: CO_2-in-O_2/air[308]
4. 1945, Anti-shock treatment with serum therapy[327] [later: blood & dextran[333]]
5. 1947, Oxygen therapy, initially as Carbogen[323]
6. 1947, Patency of the airway given 'special emphasis'[323]* toileting, 'even' endotracheal intubation[323]
7. 'Anaesthesiologic Principles' introduced: 1949 [Apr], Eric Nilsson's methods[335] incl. intratracheal intubation as required.[333] 1950, Daily cooperation of the Bispebjerg anaesthetists[311] 1950, Waters-type technique[341] of artificial respiration for apnoea—opiates [possibly only?] initially[333]
8. Centralisation Principle for treatment 1949 [Oct], Carl Clemmesen's Bispebjerg 'Intoxication Unit'[333]
9. Expedited elimination of barbiturate: 1951, Diuresis: forced/osmotic[312]/blood lavage[348] 1952, Haemodialysis[349]
10. Vasoconstrictors for circulatory support: pre-1955, weaker agents: ephedrine, amphetamine[323] 1955, 1July, effective agent: noradrenaline[331,332]

*Prior to 1947, neither tracheostomy nor endotracheal intubation appear recorded for respiratory obstruction in barbiturate poisoning.

6.6 Indicators of early intensive therapy at Lund and Copenhagen

6.6.1 At Lund

It can be deduced that foremost, it was Eric Nilsson who inaugurated the Scandinavian Method of treatment at the University Hospital, Lund, with Case No.1 (leading a series of five), on 18 April 1949: with no gastric lavage, no analeptics, but nasotracheal intubation, airway toilet, oxygen, hourly hyperventilation 'douches' of carbon dioxide and air, and appropriate (Kirkegaard-type) fluid therapy.[335] By taking his methods after these 1949 cases to Clemmesen's newly established Intoxication Centre (over February to March and July to August, 1950), Nilsson thereby recruited 141 individuals treated at Bispebjerg for barbiturate poisoning

into his doctoral thesis study. Combined with a total of 35 such individuals treated at Lund, from 1 January 1949 to 31 October 1950,[308] these completed the sample for his thesis, which was published in early 1951.

Nilsson's thesis set the standard for the Scandinavian Method. Its Chapter V also defined his principles, together with notes, some comprehensive, on the clinical details of the 87 patients poisoned with barbiturates he classified as severe, among 129 unconscious (71 of them for >24hrs), within the total of 176 patients. The complications due to poisoning were peripheral circulatory collapse in 38 patients; a few developed pulmonary oedema, five developed pneumonia (with a single death) and three had serious renal complications.[308]

Among 176 total barbiturate admissions, deaths numbered three for a 1.7% mortality rate (or if including one never-unconscious patient who died from pulmonary embolism, 2.3% for '3 + 1'). Nilsson's 87 patients whom he classified as severe had a death rate of 3.4%, while mortality among those 71 who were unconscious for more than 24 hours was 4.2%.[308]

In America, rapid appreciation of Nilsson's principles for respiratory management, as per his thesis, was evident in a summarising editorial in the *JAMA* issue of October 1951.[350]

Nilsson considered that rather than physicians caring for severe poisoning, 'it is only natural that [anaesthesiologists] are precisely those who should be more and more entrusted with looking after patients poisoned with narcotic or hypnotic drugs'[308[p91]] rather than those still seeking magical antidotes for reversing barbiturate respiratory paresis (and hopeful of an equivalent, nalorphine-type, mode?). From Nilsson's thesis caseload, Patient No. 24[308] required two hours of active AV (his single case instance) for total apnoea induced by barbiturate (blood level still 10.3 mg% at 16 hours post admission), possibly enhanced by morphine, with recovery following.[308] Only '3 + 1'[305] patients died but their observation charts on Nilsson's thesis pages 42 and 74–8 show that until death, spontaneous breathing continued throughout. Despite Nilsson's claim elsewhere for all attempts of therapy,[308] here, his own words do not appear to indicate that 'all' interventions were employed to compensate for every breathing inadequacy.

Later, in 1966 Nilsson was quite unequivocal about promoting scrupulous IPPV (mechanical) for patients with insufficient respiration[346] from severe barbiturate poisoning. (He referred to Astrup's micro-method and other blood gas determinations.) Nyköping's Jan Bergström, also in 1966, described intubation as needed for 173 (20.2%) among his 856 comatose patients, without numbering the occasions on which IPPV was administered if required.[338]

6.6.2 At Copenhagen

The Bispebjerg Intoxication Centre, a dedicated unit for treatment of suicides with hypnotic poisons, with intense clinical observation and active multisystem interventions indicating nascent intensive care/therapy, was soon reported on by Clemmesen and Bie in April 1950.[333] Clemmesen provided details until 1952 in Danish,[341] and then from 1954[323] in English,[307,311,312] for the range of intensive support for poisoned patients, emphasising full routine observations, laboratory monitoring, and attention to cerebral, circulatory, renal and respiratory systems (with emphasis on oxygen and airway care), plus prophylactic antibiotics.

Owing to the critical importance of preventing shock in barbiturate poisoning, Clemmesen advocated Kirkegaard-style circulatory support with blood products and infusion of dextran, etc.[341] As presumably was happening at Lund Hospital, the development of a Department of Anaesthesia at Bispebjerg Hospital[311] enabled its daily co-operation with the newly established Intoxication Unit, which proved to be a great advantage.

Arising from the Copenhagen Intoxication Centre's organisation and its management of barbiturate poisoning patients, Bjørn Ibsen, in 1966, generously lauded it as the first intensive therapy unit (ITU), although he noted that only one type of disease was being treated.[295] (Ibsen's own ITU, started in December 1953, was nominated the first truly multisystem ITU.[281]) Complications from a primary disorder, such as severe barbiturate poisoning, certainly would have needed and received further ITU-type care not available in conventional wards. Bispebjerg psychiatrist Asger Louw defined these measures explicitly in 1958,[331] reinforcing the viewpoints Clemmesen expressed many times.

(Around the same time, Gösta von Reis provided detailed Swedish-language retrospectives from Stockholm's Södersjukhuset', in 1957[345] and 1960.[351])

6.6.3 Respiratory insufficiency and ventilatory support

There were apparently some inadequacies in Clemmesen's practice. While apnoea became recognised as needing immediate compensatory measures, active intervention for significant hypoventilation and hypercarbia seems to have developed more slowly. In April 1950, Clemmesen and Bie, while treating with oxygenation and intratracheal intubation according to Nilsson's instructions,[333] clearly stated (although they omitted this from the summary in English) that '*tilfælde af respirationslammels ... gives kunstig respiration med ren ilt gennem »to and fro aggregat«* [In case of respiratory paralysis ... artificial respiration with pure oxygen is given through a "to and fro unit"]'.[333[p504]] Thus, for rescuing 10 apnoeic (among 29) morphine overdose patients in 1951 (all of whom survived), Clemmesen had nominated the Waters (canister) system,[341] but in his English summary, his naming of that specific tool was then replaced by the single word 'treated'.

That same year five died from respiratory paresis among 19 deaths from hypnotics,[341] without the agents identified and without the employment of AV. (Among the other deaths, two occurred on admission and the remaining 12 from various causes.) If barbiturates were involved here, were anaesthetists not? Clemmesen had declared that in poisoning with barbiturates, respiratory paresis occurs much later and is much more serious but, fortunately, is rare.[341] While he had not documented any Bispebjerg patients poisoned with barbiturates receiving this Blegdam Hospital type of m-IPPV, his response to hypnotic-induced respiratory insufficiency, preceding frank apnoea, seems limited. Although respiration in poisoned (barbiturate?) patients was so severely weakened that it could not be recorded using a spirometer[311] and also when reporting similarly for solely patients poisoned with barbiturates,[312] Clemmesen again omitted describing compensatory intervention with AV.

In 1954, at Copenhagen's Blegdam Hospital, with 25 patients receiving oxygen treatment after narcotic poisoning, 45 analyses showed an alveolar pCO_2 >50 mmHg in 70% of the cases.[352] (Quantities assessed were alveolar CO_2-tension [pCO_2], the arterial O_2-saturation, and the pH of the plasma and the CO_2-

combining power of the blood.) Asmussen and Larsen thereby suggested that the therapy, in such cases, should include artificial respiration to normalise the pCO_2.[352] But using blood gas analysis in intoxications does not appear greatly reported in Scandinavian papers from the later 1950s and early 1960s, apart from by Clemmesen and Nilsson.[307]

6.6.4 Mid-1950s: A lingering attraction to stimulatory agents

Almost four years after centralisation, although Clemmesen wrote that ordinarily, stimulation was abandoned,[323] he advised that only moderately stimulatory geastimol or amphetamine may be indicated.[323] Although he acknowledged that such agents were very seldom effective in severe (barbiturate) cases,[323] he reported success. During September 1955 to April 1956, he administered bemegride/amiphenazole to seven patients poisoned with barbiturates with respiratory paralysis and total apnoea, which were eliminated and respiration permanently restored to normal during or shortly after such administration.[298] This does contrast with the earlier outcomes in 101 patients with respiratory paralysis in 1952–1956. Among these were (i) 61 poisoned with barbiturates alone, of whom 30 died; (ii) 16 more poisoned with barbiturates + morphine, etc., of whom five died.[332] Again, was assistance sought from anaesthesiologists? Yet, even in 1958, Myschetzky still discussed treatment at the Centre with bemegride alone.[332]

Throughout the 1950s, after first announcing the abandoning of ‘dangerous’ analeptics, Clemmesen seemed reluctant to embrace the IPPV mode but appeared ever hopeful that moderately stimulating agents, such as bemegride, would restore ventilatory function. This same attitude is evident with some colleagues (for example Myschetzky, as in 1958,[332] but not in 1964[315]), but not with others (Louw, in 1958[331]). Only Clemmesen’s last paper in 1966[312] omits this recommendation, although even then he does not discuss ventilatory support. One may wonder: How much anaesthetist assistance was actually sought?

6.6.5 Reservations concerning Dr Clemmesen’s employment of ventilatory support

I confess difficulty in understanding Clemmesen’s seemingly limited recognition of pre-apnoeic consequences from hypoventilation due to barbiturates (contrast his adequately treating opiate apnoea[323,333]), or of its compensatory management by AV. It can be noted that in Australia, in early 1950, Norman James had advocated his self-designed ventilator’s suitability for IPPV usage in severe barbiturate poisoning,[353] but he did not document its actual employment. In the 1960s, at Auckland Hospital’s ICU, those seriously unconscious from barbiturate overdosage received liberal circulatory and ventilatory support (whereas about half of Ideström and von Reis’s patients, as they reported in 1951, were not unconscious[354]). Possibly, some Scandinavian intervention around the early 1950s may have waited for the breathing to become obviously inadequate, before ventilatory support was employed. Compare the judiciously interventionist 1960s ICUs: (Matt Spence’s) at Auckland Hospital or, as detailed by Barry Baker,[355] at Royal Brisbane Hospital.

Despite Clemmesen’s detailed writings, there is difficulty pinpointing the first occasion on which he employed m-IPPV for barbiturate hypoventilation or apnoea. Before 1953, use would hardly have been with ventilators, and unlikely even then: ‘mechanical ventilators would have been in short supply or non-existent for use in wards, and anaesthetists may have been reluctant to lose them from OTs to the wards’ (B Baker, personal

communication, 2013). So, for very serious but rare[311] barbiturate apnoea, in 1959, Clemmesen's most effective preparation for treatment, was still bemegride—after he characterised it as not a genuine antidote. He did not indicate employing the obvious alternative of ventilatory support, or furnish data, successful or otherwise. Although bemegride was still favoured in his 1961 joint paper with Nilsson,[307] its recommendation was followed immediately by the heading, 'Artificial ventilation – A new approach to control', and the assertion that it was best to use a mechanical ventilator.[307] Was this perhaps the co-author's contribution? Although Clemmesen's aim was to maintain physiological conditions to the extent possible during the period of unconsciousness,[323] no active intervention for barbiturate-induced hypoventilation was described. Clemmesen's final paper, in 1966,[312] while omitting bemegride, still did not recommend ventilatory management beyond solely determining blood gas analysis (well available by that time) to provide diagnostic aids if signs of failure develop. Meanwhile, it recommended that therapy must be directed against the shock as well as pulmonary affection.[312]

Willy Dam (earlier, a Bispebjerg anaesthetist), with James Eckenhoff of Philadelphia, PA, was quite forthright in endorsing ventilatory support for serious poisoning, in 1956.[339] Bispebjerg psychiatrist Louw wrote similarly in 1958 that in respiratory paresis, artificial respiration and insufflation of oxygen under positive pressure are employed.[331] By 1964, Myschetzky agreed that artificial mechanical ventilation is to be used in cases of respiratory insufficiency (also note his statement that central stimulants are never to be used.[315])

6.7 Conclusion

In severe acute barbiturate intoxication, Eric Nilsson's treatment with multisystem intensive care/therapy by the system commonly called the Scandinavian Method, first from Lund, on 18 April 1949,[335] and then from his time at Copenhagen, first in February 1950,[308] produced a decided reduction in mortality. The principles gained widespread acceptance, initially in Scandinavian centres. Carl Clemmesen's initiative from 1 October 1949[333] was of centralisation to a dedicated unit in Copenhagen providing special care for patients with multisystem complications of 'barbiturate-etc.' intoxications. This earned acknowledgement from the respected Bjørn Ibsen for early, if limited, intensive therapy.[295] Yet, Ibsen's tribute overlooks recognition owed to Nilsson's prior employment of anaesthesiologic principles, which included m-IPPV. Moreover, even earlier, in 1945, Aage Kirkegaard's success at Copenhagen from introducing effective anti-shock therapy made a substantial contribution. Thus, these three pioneers each merit esteem for effective innovations.

It is not completely clear who, around 1949–1950, successfully pioneered IPPV for respiratory insufficiency in barbiturate intoxication. Nilsson (with Bendt Eyrich), in early 1949, advocated energetic artificial respiration for treating increasing respiratory paralysis.[335] Early among his large 1949–1950 thesis caseload, Nilsson listed his single example of barbiturate (plus morphine) apnoea, for which he supplied short-term m-IPPV.[308] In 1952, Clemmesen first documented m-IPPV for the previous year's 10 instances of opiate apnoea (after earlier advocating, in April 1950, a Waters to-and-fro system for respiratory paralysis), but without specifying IPPV for either hypoventilation or apnoea from barbiturate poisoning.[341] Norman James's viewpoint, in early 1950, also warrants recognition.[353]

Although a legitimate claim for introducing IPPV as an intensive care mode of practice is not entirely certain, credit most likely goes to Eric Nilsson.

Addendum 6.1: Details of treatment for barbiturate poisoning in Sweden after 1943, as documented from four units

The 1940s reviews from the Medical Departments of two Stockholm hospitals reveal no descriptions of respiratory cardiovascular treatment.

1944–1948: Stockholm's **Karolinska Sjukhuset** with 83 barbiturate poisonings (six deaths = 7%); among them, 31 (i.e. 37%) severe poisonings (six deaths = 19% of these 'severe'). Treatment was mainly analeptic; no ventilatory assistance appears reported by Böttiger.[356] See Tables VI and VII in his paper.[356]

1946–1950: There were 350 patients at Stockholm's **Södersjukhuset**, and almost half were listed as unconscious (18 deaths = 5%). No treatment methods for sufficiently ill patients are mentioned by Ideström and von Reis.[354]

1949: A study of 16 patients poisoned with barbiturates at Södersjukhuset concentrated on barbiturate pharmacodynamics, without describing clinical treatment of respiratory or cardiovascular impairment.[357]

March 1951: The Södersjukhus team could then claim to follow Nilsson/Eyrich methods, specifying the vital components. Their respiratory control included intubation, bronchial toilet, administration of oxygen and, when needed, AV[336] (but the last was not stated as actually used).

Following early-1951 publication of Nilsson's thesis,[308] by the following December, Ideström and von Reis had re-endorsed the March 1951 commitment from the Södersjukhus to the acceptance of Nilsson's treatment of poisonings and wrote that a change to anaesthetic techniques had occurred (but that stimulants should not yet be abandoned[354]). Circulatory support came via intravenous fluid and blood, and ephedrine as required.

1950–1954: At **Centrallasarett, Jönköping**, Birger Herner reported that improved [Bispebjerg] therapy limited mortality to 3.5% among 118 acute patients poisoned with barbiturates.[342]

1949: **Örebro Hospital**'s Wilhelm Ohlsson, described his blood lavage[358,359] regimen of forced fluids and mercurial diuretics employed in medical ward treatment for about 30 poisoned patients annually (aiming to eliminate poisoning to reduce the duration of unconsciousness, particularly that from longer-acting barbiturates). Although he invoked the Nilsson/Eyrich treatment, he had no IPPV available to prevent the single hypoventilation fatality.[356]

1949–1953: Karolinska's survey showed 173 barbiturate poisonings, and deaths of 7 (=4%); 71 (=41%) of the 173 cases were moderately severe (seven deaths among 71 = 10%).[356] But ventilatory dysfunction was not mentioned.

While Clemmesen in Copenhagen was recommending bemegride/amiphenazol,[323,325] many Swedish physicians, continuing their apparent reluctance to abandon stimulants, sought agents that were more acceptable, or else, were alleged antidotes. Thus, von Reis reported in 1956 (although with some hesitation)

the use of malyzol and amphenazole for 16 patients and the difficulties accompanying their employment.[360] However, Nilsson's influence was prevailing: von Reis had abandoned analeptics by 1957.

1955: Böttiger declared[356] that the Scandinavian Method of care for barbiturate intoxications with airway/oxygenation, anti-shock and infection prevention was now being utilised, along with rejection of central analeptics (principally bemegride/amiphenazole). Later, and remarkably among the Karolinska's 1954 to 1958 period, mortality was nil when there were 311 barbiturate poisonings (with 53% labelled as severe or moderate'[361]). For the limited period of 1 September 1955 to 31 May 1957, treatment included bemegride (with valuable stimulation of respiration) as adjuvant to a clear airway and anti-shock therapy, for 120 patients (16% severe, 41% moderately severe). With nil deaths, this trial proved no advantage either with or without bemegride.

Böttiger/Östman advocated: 'If the respiration is not adequate when the air-way (sic) is free ... [and if trying Bemegride] ... to start respiration ... is not successful, artificial respiration should be started'.[361[p442]]By this time, tracheotomy was often used earlier and tracheotomy in the US for barbiturate poisoning appears first documented for three Bellevue (New York) patients in 1951.[362]

von Reis (see Figure 6.5) credited Clemmesen's example for the establishment of the Södersjukhus unit,[345] a unit following the Clemmesen and Nilsson guiding principles (which included tracheotomy when required). His account of treatment of barbiturate-intoxicated patients by the Scandinavian Method revealed[345] that up to 1957, of the 470 then poisoned, six died, and hence, the mortality rate was 1.3% (84 were comatose >1day). Respirator need for 17 apnoeic patients (4%) was mostly short-lived. Bemegride was used, and antibiotics administered. Noradrenaline provided useful vasoconstriction.[282,346,363]

Figure 6.5: Prof. Gösta von Reis

Prof. von Reis (1911–1977), Asst Prof. of Neurology 1951, Karolinska Institutet and a pioneer of intensive care in Sweden, at Södersjukhuset.[364]

Further, von Reis's fuller 1960 review[351] provided an extended account of methods employed for about 250 annual barbiturate intoxications monitored. For the 1,164 admitted over 11 September 1955 to 11 April 1960, in all, 299 were in a coma for more than a day; 43 comas lasted for 5–21 days. Apnoea occurred 98 times [8.4% of admissions], necessitating AV, and nurses were partially replaced by medical students, who served as breathing assistants. Tracheotomies numbered 82 (7%); fatalities 19 (2%); half were from respiratory causes, and four were embolic.[351]

The Danish poliomyelitis epidemic, 1952–1953

Chapter 7: Copenhagen, 27 August 1952: ‘Bjørn Ibsen’s Day’

Chapter 7: Table of Contents

7.1 Introduction

An event well honoured in the history of anaesthesia took place in Boston, MA, on 16 October 1846. It was at the Massachusetts General Hospital there that dentist William Thomas Green Morton, who was also studying medicine at the time, successfully demonstrated the use of ether for anaesthesia. This enabled the surgical operation for the removal of a tumour from a patient's jaw to proceed pain-free.

Another historic event for anaesthesia took place on Wednesday, 27 August 1952, with Danish freelance anaesthetist Bjørn Ibsen.[227,257,258,282,291,365–367] This was at Copenhagen's Blegdam Hospital (Blegdamshospital) during Denmark's 1952–1953 epidemic of acute poliomyelitis[227,257,258,282,291,365–367] (hereafter called 'polio'). The death toll among victims was catastrophic.[227,257,258,291,365] Clinicians treating the victims by established methods were being overwhelmed by the numbers of critically ill patients.[227,258]

The belief in the Department of Communicable Diseases at Blegdam Hospital at the time was that polio victims died when virus infection of the brain overwhelmed the patient. The clinical picture of cyanosis, hypertension, hyperthermia, respiratory distress and death, despite the use of negative pressure respirators (tank or cuirass), was caused by the encephalitic state. In that situation, nothing more could be done.[366[p504]]

On that day in August[258] (see Addendum 7.1), in the face of considerable scepticism,[291,365,366] Dr Ibsen demonstrated that he could provide an alternative treatment for patients with severe respiratory complications of poliomyelitis. He achieved success by using a technique of ventilatory management that anaesthetists employed at times in their regular work in the OT.[257]

7.2 Group meeting at Blegdam Hospital on 25, August 1952

At the earnest suggestion of Dr Mogens Bjørneboe, senior registrar in the Department of Communicable Diseases, to the chief epidemiologist, Prof. Henry Cai Alexander Lassen, Dr Bjørn Ibsen (although 'just an anaesthetist'; it was only in 1950 that anaesthesiology had become recognised as a medical specialty in Denmark.[291[p422]]) was reluctantly called in to join a consultation with representatives of medical specialties over ventilatory problems arising with the epidemic. (Dr Preben Berthelson[366[p504]] has lucidly explained the possible career risk to Bjørneboe for the audacity of dissenting with the accepted view and suggesting that an anaesthetist with no previous experience of polio might be able to help.) The group meeting was held at the hospital on Monday, 25 August.

Later, Ibsen confirmed in an interview[367suppl[p29]] that that he had

> explained to the group that the whole mystery lay in the fact that patients with shortness of breath lack oxygen. You eventually become cyanotic and get blue lips and fingernails. If you start treating the cyanosis only with oxygen through nasal catheter – which was quite common at that time – the consequence was that the patient did not ventilate enough, which eventually resulted in hypercapnia … with the corresponding symptoms. I pointed out to Lassen and his colleagues that the goal should be to normalise the level of the carbon dioxide and then maintain the level low.

His advice was to tracheostomise the patients and use IPPV afterwards. It was agreed to give him the opportunity to demonstrate his suggested treatment.

After the group meeting, Ibsen made his own careful further investigations[257,291] at the hospital, including studying autopsy findings on four recently dead polio patients. It became obvious to him that the treating clinicians did not appreciate that many of the patients receiving AV were dying because of the inadequacy of the method of AV delivered to them to compensate for impaired breathing. Further, what the involved doctors firmly believed to be 'metabolic alkalosis', Ibsen recognised (on the basis of his own research experience with Dr HC Engell on patients intra-operatively) was a result of carbon dioxide 'accumulation'[291[p425]] (itself an index of underventilation), which worsened when doctors supplied supplemental oxygen to affected patients.[257,291,365]

It is noteworthy that in Ibsen's last documented interview, in 2006,[367suppl[p29]] he acknowledged his full awareness that Bjørneboe, even 'at the end of July, early August', had already been considering the possibility of the bag-ventilation treatment he knew from his previous experience with Ibsen concerning an infant with tetanus earlier that year.[366]

7.3 The landmark event at the Blegdam Hospital, on 27 August 1952 (in brief)

On 27 August 1952, two mornings later, in an episode that became high drama as it unfolded,[257,291,368] Ibsen was presented with Vivi Ebert (whose name is in the public domain[282,367,368]), a hyperpyrexic girl of 12 yrs admitted from her home the previous day, who was experiencing the effects of acute polio, with all four limbs paralysed, and was desperately ill from the respiratory consequences of hypoventilation with atelectasis of the left lung. She was 'drowning in her own secretions'[258[p14]] (actual pharyngeal paralysis does not appear to be mentioned). Lassen selected the young girl as a test patient for Ibsen, who was to be allowed 'permission' to try to rescue her[365[S119]] 'without the help of a respirator'.[257[p23]] (In describing events in later papers, Ibsen is usually brief,[369[p72]] but in Lassen's 1956 book on the epidemic, less so.[229[p14–5]])

Otolaryngologist Dr Falbe-Hansen (with thanks for personal communication, Dr P Berthelsen, 2009) performed a tracheotomy operation first, for placement of a cuffed intratracheal tube, under local anaesthesia.[367suppl[p35]] This procedure started from **11.15 am** (clock times for the following précis are taken freely from the supplementary material in Dr Reisner-Sénélar's article[367suppl[p35–41]]). Ibsen remarked on the loss of a great deal of time during this operation. The hospital notes were entered closely thereafter, often every few minutes, and they detailed unequivocally the patient's struggles and the difficulties for her treating doctors. Immediately after the tracheotomy, her bronchi could not be aspirated satisfactorily and her own breathing, worsened by bronchospasm and her struggling, was inadequate; she required supplemental oxygen.

Ibsen took over treatment, by **12.10 pm** supplying manual(m)-IPPV, although with difficulty, by 'bagging' her (with a Waters to-and-fro carbon dioxide absorber system), together with 100% oxygen (the only options available were that or 80%). Improvement was temporary only, since stopping oxygen to aspirate her bronchi resulted in Vivi becoming desaturated with cyanosis, restlessness and jerking, and then, by

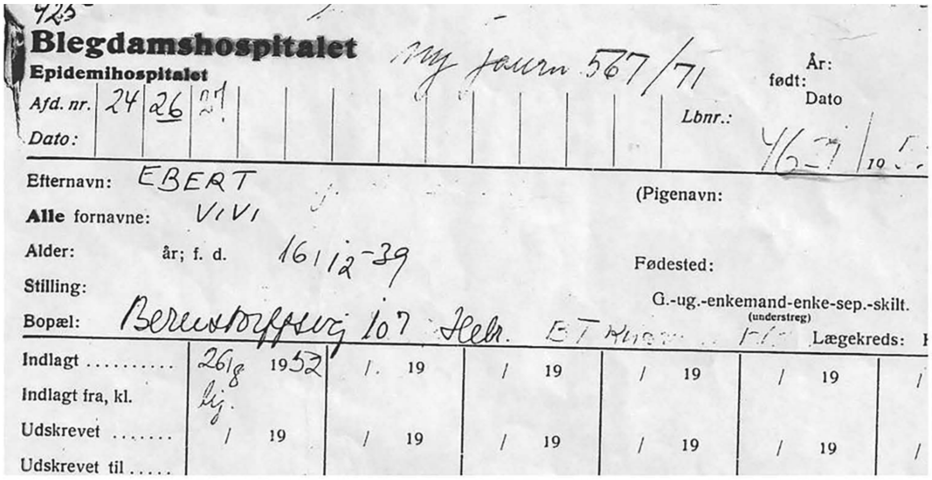
12.57 pm she was unconscious. Hypertension (e.g. SBP of 150 mmHg) was also apparent. Despite active support of her breathing starting again at **12.57 pm**, she was restless with struggling (spasms and agitation), with her situation becoming desperate. Ibsen then started administering 100 mg of thiopentone to her at **1.17 pm**. Other doctors who had gathered to observe his 'new' method, thinking they had seen failure enough, had been beginning to disperse. But by gaining control with that agent, Ibsen could manually ventilate her adequately by the bag, to reverse her markedly adverse clinical signs. Observers drifting back were astonished, 'incredulous'.[365[S119]] Ibsen reflected later on the control achieved by this treatment of her ventilation and agitation: 'That I could save the patient's life with such a simple method, was one of the most incredible moments of my life.'[367suppl[p30]]

There were further difficult times ahead for this patient, but she survived and eventually left hospital with respiratory problems for the rest of her shortened life.

7.4 Documentation of further events of 27 August 1952

From this stage onwards, and in a correction to a 2004 account in which I wrote[368] as if Ibsen's masterstroke with thiopentone and IPPV had ended the difficulties of managing Vivi's breathing problems (and as can be found similarly in some of the other accounts of this day), the following amplification is needed to try to supply the facts correctly and to demonstrate ongoing difficulties. Dr Louise Reisner-Sénélar[367suppl] located, transcribed and then translated into English the hospital's original clinical notes of that day's happenings (many entries are difficult to read, as shown in Figure 7.1, and some are in Ibsen's handwriting, even if not always signed). The results for hundreds of measurements made were entered. They are published in the public domain as supplementary material,[367suppl] which (together with a similar copy of case-note pages kindly sent to me by Dr Preben Berthelsen), reveal my own naivety in making certain assumptions earlier. These detailed hospital records (which, because of their brevity, which is likely to be obligatory, may seem written a little cryptically at times) now make available the definitive story of the eventful further happenings of 27 August 1952. In details for the interview Dr Ibsen gave to Dr Reisner-Sénélar on 16 July 2006, his recall of the event during the interview is comparable, as reported in the supplementary material.[367suppl[p29–30]] A few brief selections from the available records follow.

Figure 7.1: Extracts from hospital records



Blegdamshospitalet
Epidemihospitalet

Afd. nr.	24	26	27					Lbnr.:
Dato:								

Efternavn: EBERT (Pigenavn:

Alle fornavne: VIVI

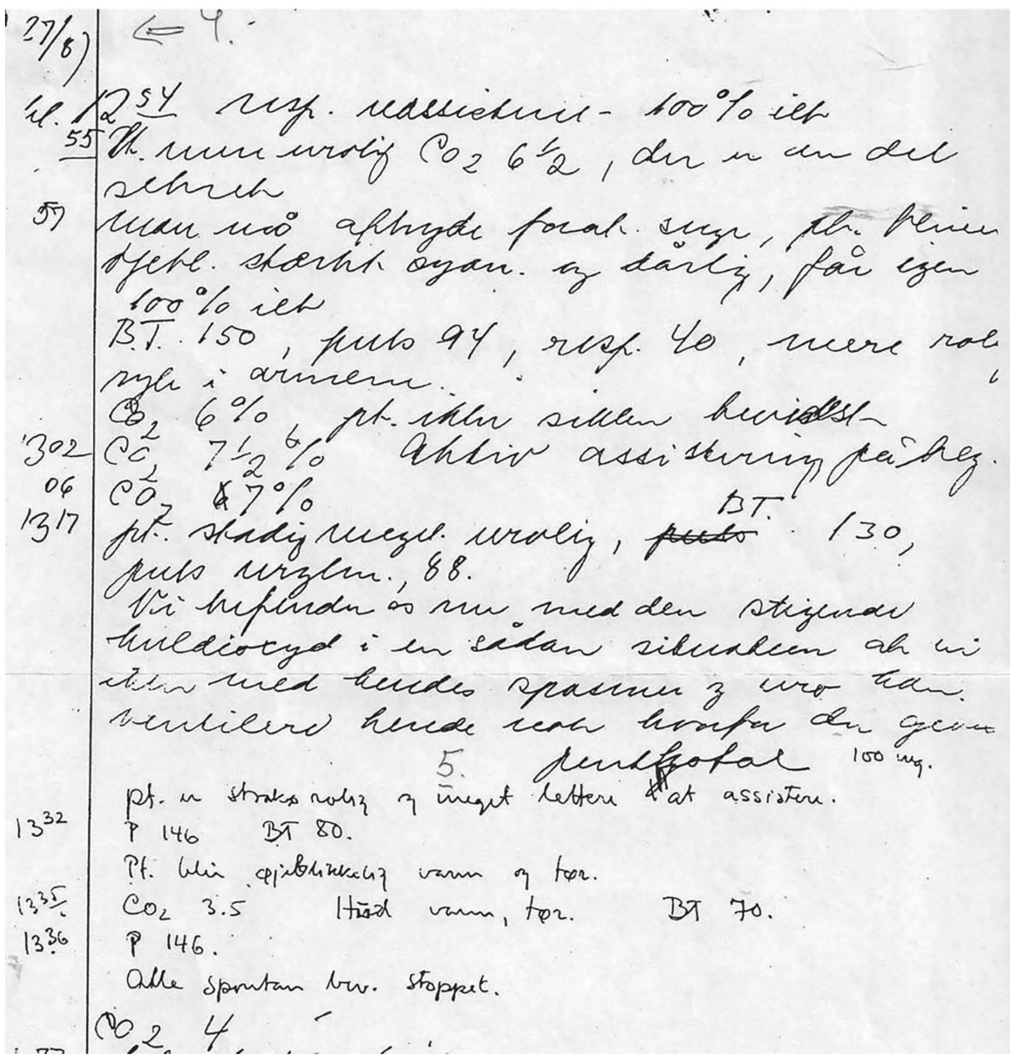
Alder: år; f. d. 16/12-39 Fødested:

Stilling: G.-ug.-enkemand-enke-sep.-skilt. (understreg)

Bopæl: Bernstorffsvej 107 Hell. Lægekreds:

År: født: Dato

Indlagt	26/8 1952	/ 19	/ 19	/ 19	/ 19
Indlagt fra, kl.					
Udskrevet	/ 19	/ 19	/ 19	/ 19	/ 19
Udskrevet til					



27/8

12⁵⁴ resp. uassisteret – 100% ilt
55 Pt. nu urolig CO_2 6½, der er en del sekret
57 man må afbryde forat. sug., pt. [illegible] 100% ilt
BT. 150, puls 94, resp. 40, [illegible]
CO_2 6%, pt. [illegible]
13⁰² CO_2 7½% Aktiv assistering påbeg.
06 CO_2 7%
13¹⁷ pt. stadig meget urolig, BT. 130, puls uregelm., 88.
Vi befinder os nu med den stigende kuldioxyd i en sådan situation at vi ikke med hendes spasmer og uro kan ventilere hende [illegible]
5. pentothal 100 mg.
pt. er straks rolig og meget lettere at assistere.
13³² P 146 BT 80.
Pt. blir øjeblikkelig varm og tør.
13³⁵ CO_2 3.5 Hud varm, tør. BT 70.
13³⁶ P 146.
Alle spontan bev. stoppet.
CO_2 4

Duplicates of pages of the hospital record of Patient Vivi. The lower one demonstrates the likely difficulty in deciphering correctly.[367]

When I wrote to Prof. Ibsen in 2003, he was ill and unable to answer my questions (see this chapter's Addendum 7.2).

Following events, 27–28 August 1952[367,367suppl[p37–41]]

When the patient was awake again at **1.38 pm,** spontaneous breathing had restarted, and for '1.45 pm', Ibsen then wrote in the clinical record about injecting another dose of 'Pentothal' (thiopentone), 100 mg; further doses were given as required (5.09 … 7.50 ... [?, *v.i.*] 8.25 pm). Twice in the 2006 interview, Ibsen referred to this intervention as administering phenobarbital, not Pentothal (see the supplementary material in Dr Reisner-Sénélar's article[367suppl]). He said: 'What was dramatic was when I administered 100mg Phenobarbital to a dying patient, I managed to ventilate her and eventually saved her'.[367suppl[p33]] The hospital notes, including one in his writing, confirm that it was not phenobarbital but 'Pentothal'.

Even Ibsen's successful strategy of anaesthetising with thiopentone a tracheotomised patient, Vivi, and then supplying m-IPPV for remedying her immediate difficulties, as described above, did not end that day's problems. Multiple episodes of high concern or real difficulty can be seen recorded at intervals until the next morning. It would be easy to expect from some written accounts (including my own,[368] previously) that after the first anaesthetising of Vivi and the supplying of m-IPPV, progress with ventilating and oxygenating her was straightforward. That proved not to be the case. Ibsen did have the benefit, and certainly with advantage for guidance, of being able to determine carbon dioxide levels with his Brinkman's (prototype) Carbovisor, his research experience with which had made him knowledgeable about carbon dioxide levels. (Ibsen was able to demonstrate to physicians the effect of carbon dioxide in expired air 'led through an indicator solution, 0.005% bromothymol blue',[291[p424]] with colour changing being controlled continuously by m-IPPV to differing levels[291[p426]]). In addition, he could obtain oxygen saturation of haemoglobin (SO_2) with a Millikan oximeter (a photo-electric ear attachment from WW II days), as well as measurements of pH and the bicarbonate level. During the afternoon of 27 August, he recorded multiple occasions needing attention to desaturation, hypercarbia and hypertension.[367suppl[p35–41]]

Some difficult-to-decipher observations on a scanned copy of the first two sheets of the original hospital recordings for that day, first provided in photocopy to Dr Berthelsen, have also been reproduced by Dr Reisner-Sénélar in her thesis[367suppl[p43–5]] (my thanks to both doctors). See Figure 7.1.

Some further details include the following:[367[p36–42]]

During early afternoon (of 27 August 1952), at **2.18 pm**, Ibsen noted a state of apnoea with almost controlled breathing. (The times listed are all taken from the supplementary material[367suppl] in Dr Reisner-Sénélar's article.) Then, at **4.26 pm,** Vivi was eventually transferred back to a body-cuirass respirator, but at **4.30** and **4.40 pm** was recorded to be not in synchronisation with the respirator, while at **4.55 pm** extrasystoles were detected. Ibsen intervened with bag ventilation once more for the signs of carbon dioxide accumulation, and again reversed her deterioration. When she relapsed yet again, with cyanosis (desaturation to 54% at **5.06 pm**, which supplemental oxygen did improve), Pentothal 100 mg was again given at **5.09 pm**; then, as it is written, '*Phenol* [?] *10mg*' at **6.18 pm**, and 'we go onto pressure ventilation with oxygen (+own breathing)'. Ibsen noted at **7.17 pm** that the left lung was insufficiently ventilated by the cuirass respirator, causing this deterioration. Further Pentothal, 100 mg, was given to allow blood gas sampling: pH 7.39. Then

followed the removal of the respirator at **8.02 pm,** and instead, m-IPPV was effected at **8.12 pm.** More Pentothal was used at **7.25 pm** (probably **8.25 pm** was meant). The records state spontaneous respiration at **11.30 pm,** while 'removal of the absorber at **00.12 am**' could indicate that m-IPPV was otherwise being continued intermittently overnight, when the SBP was (100 mmHg up to) a maximum at 200 mm. Supplementary oxygen was needed.

At **04.30 am** (28 August), Ibsen's handwriting recording SBP at 150 mmHg showed he was still in attendance. Her temperature still stayed high at **06.40 am.** The summary at that time was 'The patient has been ventilated with atmospheric air and intermittent positive pressure … The patient's condition is satisfactory'.

On 28 August, at an unspecified doctor's early morning check visit (the notes have only 'B.J.' entered as their author, but those are the first two letters of Bjørneboe's name), this doctor could record that by then the patient's condition was satisfactory. Then, at **06.40 am** that morning, it was recorded that Vivi was perfectly calm, warm and dry, and was being ventilated with atmospheric air and intermittent positive pressure.

An assessment of Vivi's next days is then given:[367suppl[p42]]

> In the following days the patient remained relatively stable, except for a further attempt to connect her to the respirator (i.e. the cuirass). The attempt failed. The condition of the patient worsened immediately and the doctors had to turn back to manual positive pressure ventilation.

In brief, once that method of m-IPPV treatment was established satisfactorily for Vivi, it was continued thereafter, and, as it became possible, was also supplied as needed for other patients in the hospital. One day after Vivi's episode, Poul Astrup obtained a small electrode from Radiometer enabling him to measure pH directly in blood from the patient.[291[p427]]

According to Louise Reisner-Sénélar (personal communication, 2006), Vivi Ebert remained a respiratory cripple the rest of her short life. Until January 1953, she was manually ventilated 24/7 and then maintained in hospital for several years. She eventually married, becoming Vivi Andersen, but died in 1971 from sepsis and respiratory insufficiency.

Reisner-Sénélar also said: 'When I last interviewed Ibsen he did not mention having ever seen his 'index patient' again. I am sure [he] would have … if he had in fact met Vivi after their initial encounters during her many months of treatment at Blegdam'.

7.6 Sequel

Bjørn Ibsen's demonstration established a new treatment for the respiratory complications of polio by m-IPPV/tracheostomy. Lassen now 'energetically dedicated'[291[p427]] himself to that method, it becoming immediately accepted at the Blegdam Hospital (of 500 beds), to which many of Denmark's suspected polio cases were brought. Ibsen wrote that 'after eight days a big organization was working'.[257[p25]] Some patients in Denmark were being treated peripherally, such as in Jylland (Jutland) at Skive County Hospital, where the Bang PPV machine was developed.[320,370] Despite all the associated logistical difficulties arising principally from the sheer numbers requiring rescue treatment at Copenhagen, the Blegdam's new method

was used throughout the epidemic wherever it was available and required. At the height of the epidemic, more than 70 patients needed bag ventilation for 24 hours a day.[291[p427]] The workforce, recruited continuously to supply manual ventilation, exceeded 1,000 in the first months.[258[p40]] It eventually comprised virtually all of Copenhagen's medical students, and later, in November[291[p427]] dental students too[366[p505]] and some nurses. In total, about 1,500 altogether applied treatment over 165,000 hours. The role of the students was more than squeezing the bag and included monitoring equipment and assessing the patient. Anaesthesiologists[371[p788]] (from all the Copenhagen hospitals and another 20–30 from the WHO training centre in Copenhagen) supervised their labours, and the patients received round-the-clock skilled nursing attention.[369,371,372] Lassen estimated the initial three months of this new system of treatment to have saved 100 lives before Christmas 1952.[258[p40]]

Later in the epidemic, mechanical ventilators came to play a partial role[227[66–72]] (starting with Engström's single, volume-controlled ventilator, a 'mechanical student'[291[p428]]), but could be used less frequently than manual PPV. There was a great boost to the devising and development of new ventilators, as other countries became worried about a polio epidemic (Louise Reisner-Sénélar, personal communication, 2006).

7.7 Further considerations

7.7.1 Personalities

HCA Lassen,[227,258,267,373] Bjørn Ibsen,[227,257,369,371–373] and Poul Astrup[365,374,375] are perhaps the best-known names of those involved in these aspects of the epidemic. Others include Erik Wainø Andersen,[371] and Mogens Bjørneboe, Frits Neukirch, J Pedersen, and T Søttrup (all in reference 227); but a magnificent team effort and organisation by Lassen, a masterful organiser of personnel (doctors, nurses, students and back-up), were essential to the ultimate success. Lassen was actually acknowledged as 'dammed intelligent' by a generous Ibsen to Berthelsen 2002 (personal communication, 2017).

It is to Bjørn Ibsen particularly that acknowledgement and honour are due for the introduction of alternative methods of treatment that led to the relatively successful management of the critically ill during the Danish epidemic. Ibsen himself continued contributing to the Blegdam ventilatory workforce.

(A side issue: how concentrated Ibsen's own attendance and assistance was at Blegdam after 27/28 August does not appear reported. But he certainly did attend after the first days. Concerning his continuing contributions to the Blegdam after 27 August, Geoffrey Spencer, during a Wellcome Trust Centre Witness Seminar, repeated an allegation (source not stated), 'It is claimed that Ibsen never returned to the polio unit' after setting up the test patient and then leaving.[376[p10]] However, Berthelsen confirmed to me (personal communication, 2011), that Ibsen continued to be involved, and quoted Ibsen's invoice to the Municipality of Copenhagen for 10,000 Kr., approx. £1,000, 'for services rendered').

The method of manual inflation of the lungs with an anaesthetic insufflating bag had been used in the OT by anaesthetists since before 1934,[377] and had occasionally been employed in the treatment of critical illness.[68,243,276,296[p27],377] Recognition also needs to be given to the prior successful use of supplemental

PPV in a polio epidemic, [268,269] the success of which was unfortunately not widely publicised or known. That was achieved at Los Angeles in 1948–1949 by Albert Bower, Ray Bennett, John Dillon and Bernard Axelrod, whom Ibsen always acknowledged in his papers—for example, in 1975.[257] (Also, as in the sixth line in Prof. Ibsen's first note on p151, and again, in Chapter 8 of this monograph.)

The above paragraphs include updating, in the light of Dr Reisner-Sénélar's information from interviewing Prof. Ibsen, 2006, as documented in her thesis and in the supplementary material.[367,367suppl] There is also Dr Preben Berthelsen's 2014 powerful paper on setting the record straight,[366] about treatment roles during the Copenhagen epidemic. Ibsen always appeared to downplay what he considered undue praise for his innovations, as pointed out by Berthelsen and Conqvist: it was 'really not such a big deal' that he established the first true ICU at Copenhagen's Kommunehospital in 1953,[282[p1192]] and he did not report on that until after five years, and then in a local journal, which was not prominent.[378] In the light of subsequent developments it was a big deal, surely!

Certainly Prof. Ibsen showed personal awareness of the best means available, with correct appreciation of the biochemical background and its management, which could be used at that time to try to cope with the influx of polio patients with ventilatory insufficiency—when the currently available mechanical system of INPV treatment was ineffective in stopping patients with ventilatory failure from dying. Wackers emphasised the importance of Ibsen's reintroduction of tracheotomy to help control retained secretions,[291[p425–6]] considering Lassen's dismissal of it originally: Lassen had argued that previously in 1948–1950, all polio patients at the Blegdam 'treated by tracheostomy and with respirators died, whereas treatment with respirators alone had a somewhat better prognosis'.[291[p47;Table II]] The success of the change in the treatment method also indicated that prior Blegdam clinicians did not appreciate the nature of the biochemical disturbance from the worsening of carbon dioxide accumulation, owing to their misguided belief that the problem was that of metabolic alkalosis.

7.7.2 Documentation

After just over three months of the new treatment, Prof. Lassen, Chief Epidemiologist at the Blegdam, provided a Preliminary Report to the *Lancet* of 3 January 1953.[258] It is not to Lassen's credit that the journal's editor needed to insist Lassen should not omit but must name Ibsen in the paper he was submitting.[366[p505]] Ibsen, as he later stated in a presentation to the Royal Society of Medicine, London, wrote more generously of 'enthusiastic encouragement from Professor Lassen'.[369[p47]] In the 3 January 1954 *Lancet* article's single mention of Ibsen, all that Lassen eventually stated was 'At this point we consulted our anaesthetist colleague, Dr B Ibsen, and on Aug. 27…' This was after 27 among the 31 seriously ill had died.[258[p38]] (Later, in 1953 Lassen refused to support applications by Ibsen for promotion elsewhere, and explained himself thus: 'battles are won by generals and not foot soldiers' [as Ibsen 'enlightened' Berthelsen, 2002].[366[p506]])

Prof. Lassen wrote in 1955[267] and edited in 1956[227] the two major retrospective accounts of many aspects of this epidemic. Whereas his works provide multiple statistical data for the epidemic, Dr Ibsen's writings have been concerned principally with events of that significant day, and arising out of it, the important consequences for anaesthesiology and for ICM. On 16 October 1953, Ibsen delivered an account of his own role to the Royal Society of Medicine in London, with his 'venture: to dare to speak up for the

anaesthesiologist's point of view'. This address was documented the following year, both in the Royal Society's journal of January 1954[369] and in the inaugural issue of the *Danish Medical Bulletin* in March 1954[372] (which also contained Lassen's own account given to The Royal Medico-Chirurgical Society of Glasgow on 23 October 1953[379]); again, in the 1956 book on the epidemic (which Lassen edited[227]), and then again, with extra biographical detail supplied, in 1975.[257] The latter description, and particularly Wackers' 1994 precise retelling, comprehensively and lucidly[291] describe more fully the conversion of the 27 August event from seeming failure to the triumph it eventually became. The success was reinforced in the 1998 summary of events by Severinghaus, Astrup and Murray,[365] among notable others. Prof. West adds a further dimension in viewing the epidemic as providing extraordinary challenges in applied physiology. He commented: 'An important development was the translation of the new knowledge from departments of physiology to the clinical setting. In many respects, this period was therefore the beginning of modern clinical respiratory physiology.'[380[p424]]

(Does that comment from Prof. West acknowledge Ibsen and Engell's research adequately? Prof. West refers to preceding recognition by Nielsen, 1946 in *Ugeskr Læger,* vol.108, of carbon dioxide elevation causing severe respiratory acidosis).

In terms of documentation from original descriptions, Dr Reisner-Sénélar's 2009 PhD thesis[367] for which she conducted an interview[367suppl] (in Danish) with an almost 91-year-old Prof. Ibsen on 16 July 2006, a year before his death, is a historical report that has singular value. She also had located the original, unpublished patient (hospital) record of Vivi, including a minute-by-minute transcription of the dramatic hours when Ibsen fought for Vivi's life.[367suppl]

(Dr Berthelsen had sought these earlier from the Public Archives at the Town Hall at Copenhagen, but officials could not locate them for him then, as it appeared unknown to them that they were already loaned [personal communication, 2006]). Dr Reisner-Sénélar's thesis, in German, has been available on the German State Library website, but she generously sent me her own translation in English (personal communication, 2011). However, she did provide some supplementary material for general attention. This material includes a very detailed bedside account of documentation for the events of 27–28 August,[367suppl] as excerpted for the description above, with medical comments on, and numerous recordings made from, the patient Vivi. It also includes her 2006 interview[367suppl] with Prof. Ibsen (*'*a very impressive man ... though very humble' [personal communication, 2011]). These publications now make unique historical documents.

After all, then, it is the more remarkable that—despite the important consequences for anaesthesiology and for ICM—the 50th anniversary itself of 'Ibsen's day', and largely, it would seem of the whole epidemic, appeared to slip past relatively unmarked in the medical literature in English during 2002–2003. An acknowledgement of a 50th anniversary did appear in two medical specialty journals, the premier *Acta Anaesthesiologica Scandinavica* journal[381] and the Australasian *Critical Care and Resuscitation*.[298] In addition, there was a referral to '50 years ago' in the 2003 statement of the UK's Intensive Care Society on the 'Evolution of intensive care in the UK'[382] from its origins at Copenhagen.

7.7.3 The immediate significance of 'Ibsen's Day'

For the remainder of the epidemic, the employment at the Blegdam of Ibsen's method, m-IPPV—familiar to the other anaesthetists who became involved,[371] and to anaesthetists elsewhere[68]—made continuous, prolonged, controlled PPV on a large scale a success in Europe for the first time. For some patients, m-IPPV was supplied for up to three months, and occasionally even more.[227] With the boost for the development of improved *mechanical* ventilators, the widespread adoption of their use in place of the m-IPPV method became the accepted means for controlling ventilatory failure. The commercial development of large numbers of effective machines for IPPV was accelerated in Europe, spurred on by the somewhat fearful anticipation (Mushin's 'winds of alarm') of immediate, further outbreaks of polio there.[291,296]

The advances in treatment learned during this epidemic provided clear starting points for the ICM discipline as practised today, seven decades later.[282,298]

A new era in medical practice had begun.

7.8 Sequels to the 27 August event

The following resulted from the methods of treatment employed during the epidemic and from the organisation of that treatment, all of which received wide publicity.

7.8.1 For anaesthesia

The skills of anaesthetists extended beyond safeguarding the adequacy of the airway and of ventilation, because of their understanding of, and competence in, the treatment of acute insufficiency of the circulation and of shock, and in resuscitation, with attention to fluid, electrolyte and acid–base balance, renal function, and nutrition. Around that time, the field of acid–base analysis, essential for the proper management of IPPV, was rapidly developing,[365,375] to be followed in a few years by electrometric pCO_2 measurement (from Richard Stow in 1954,[383,384] with improvements soon following on from John Severinghaus[385]); then, in a few years, polarographic pO_2 measurement (from Leland Clark's miniaturised oxygen electrode in 1956[365,375,386]). These advances, all fundamental to the practice of ICM, were obviously important for anaesthesia also.[68]

Some anaesthetists were emboldened to move out of their workplace of the OT, taking their treatment methods with them to wider applications. This led to the evolution of a variant anaesthetist, at times a new 'species', the anaesthetist who became an intensivist. Thus, in a few years the roles anaesthetists undertook varied from those typically working only in conventional anaesthesia, to some sharing their time between OTs and ICUs, to a smaller number who became full-time clinicians dedicated solely to servicing ICUs. Some physicians also became intensivists.

7.8.2 For intensive care medicine

There were two major sequels. First, the foundation was laid for what was eventually to become a new specialty.[257,277,365,375] All previous brave efforts at providing various forms of Intensive Therapy[68,243,276,296,377] had not yet established it as an accepted option of treatment for the critically ill. Second, a lead was given to the concept of the formal ICU[257,282,365,375] (even if it was only one patient treated in the first ICU—at that time officially called the Anaesthesia Observation Unit[282]—in 1953, from 9 December), with dedicated nursing teams now being established. In his 1958 paper, Ibsen was still referring to his ICU as an Anaesthesiologic Observation Unit. When all patients with respiratory problems were collected in a special department,[282] with a multidisciplinary team on hand, the advantages soon became obvious at the Blegdam. Ibsen firmly advocated establishing such units.[68,282,365,368]

Other consequences of the events of 27 August 1952 and its aftermath included the confirmation of PPV as the mode of AV being demonstrably superior for reliability and effectiveness, and the death knell for NPV.[68,368] Both factors changed the approach to the technique of artificial respiration globally.[258]

7.8.3 For anaesthetists in intensive care medicine

Initially, anaesthetists were the natural group for the intensive care pioneers to have come from,[68,387] but occasionally they were from a few other specialties. By the end of the 1950s, more anaesthetists (but comprising only a small proportion of the anaesthetic community) were progressing along one of two alternative pathways into Intensive Care. These anaesthetists were likely to be working full-time in public hospital practice. Interestingly, the journal *Anaesthesia* devoted an issue to ICUs as early as 1960.[387] The reports to the Second Congress of the World Federation of Societies of Anaesthesiologists that it published comprise presentations of the experience of anaesthetists in early ICUs in Toronto (by Fairley HB), Baltimore (by Safar P), Stockholm (by Norlander P et al) and Southampton (by Pearce DJ).[387]

One stream was sharing their working time between both anaesthetic and intensive care workplaces, a system rapidly developed in Scandinavia as well as other places in Europe, including the UK, and in North America. For the anaesthetist-intensivist (or physician-intensivist) practising in dual locations, ICM became a super-specialty. A clinical role shared between anaesthesiology and another specialty, here ICM, has been the tradition in Scandinavia, and the case for that was argued strongly in the journal *Acta Anaesthesiologica Scandinavica.*[282,388–390] Generally, confining clinical activity to intensive care practice alone has not been the characteristic Scandinavian way.

Another stream was for practitioners from one primary specialty (typically anaesthesiology), who were developing a commitment solely to what was becoming the specialty of ICM. Therefore, the full-time intensivist was coming onto the scene, elsewhere in Europe (if not in Scandinavia or Britain), in North America (e.g. Pittsburgh, Winnipeg) and in Australia and New Zealand.[276,391] From the beginning of the 1960s, such a dedicated specialist was no longer a complete rarity, and formal ICUs were becoming established.[257,276,387]

Trainees in ICM today, when specialised training and qualification can be obtained in many countries, do not necessarily come to its practice with a prior qualification in another specialty. For more than a dozen years into the 21st century, it has been possible to gain an intensive care qualification in Europe and the UK. Training for what became the Australasian Fellowship in Intensive Care Medicine (now seen as Fellowship of the College of Intensive Care Medicine or FCICM), started in the late 1970s. Currently, it requires an arduous six-year course,[392] training in which can start on completion of one year of postgraduate general hospital appointments. The history of the formation and institution of training and accreditation procedures of the College of Intensive Care Medicine of Australia and New Zealand, 2009, has recently been reviewed in depth by Prof. Baker.[393]

In Australia and New Zealand, the number of practising specialist intensivists is still distributed inadequately. Outside the major metropolitan centres in Australasia, there likely is reliance on anaesthetic and other specialist colleagues.

In the climate of today's educational and vocational opportunities, it is easy to overlook or forget the contribution that pioneering anaesthetists made to developing and establishing the new specialty. The history of this polio epidemic could be an eye-opener to today's younger intensivists of just what ICM owes to those founding anaesthetists. Thus, in the US, there were anaesthetists such as Peter Safar and Henning Pontoppidan and, in Toronto, Barrie Fairley, as well as others elsewhere. Anaesthetists, from their practice in the OT and from familiarity with resuscitation, but often in the face of scepticism and even antagonism, brought basic skills in life support, which were more than those of simply compensating for underventilation or inadequate airways, or for treatment of circulatory shock. In consultation with other specialties when appropriate, they extended their involvement in other body systems of patients, whether such patients were medical, surgical (including post-traumatic) or paediatric (the pioneering Auckland experience of the first 40 years has been documented).[394] For instance, just consider the developments over the past 60 years in the non-surgical aspects of therapy for cerebral trauma.[395] Measures include those for elevated intracranial pressure, assurance of cerebral oxygenation and control of hypercarbia.

7.9 In conclusion

On 27 August 1952, Dr Bjørn Ibsen successfully took outside the OT a simple anaesthetic method of manual AV of the lungs, for the treatment of a critically ill polio patient, then having his methods employed successfully in the same hospital for a prolonged time on a large scale, he launched a therapeutic revolution. This was in association with his attention to circulatory support and other critical factors. That auspicious day was significant in the development of both anaesthesia and ICM.

Addendum 7.1: Comments on variations in the date of Bjørn Ibsen's demonstration of manual intermittent positive pressure ventilation

A. As can now be confirmed from the hospital records of patient Vivi Ebert, the date of Prof. Ibsen's significant 1952 intervention was 27 August.

1. **Dr Bjørn Ibsen** himself does not appear to provide a precise date in his papers. However, both for his retrospective of 1975[257] and for his 2006 interview, his long-term memory was mildly defective in this respect, where he stated (as recorded in the supplementary material in Dr Reisner-Sénélar's article[367suppl[p4]]) that he was called in to a meeting of clinicians 'on a Saturday', with the decision then made that he was 'to demonstrate [his] theory on the following Monday on a suitable patient'. The hospital records of the patient correctly confirm Monday (25 August) instead of Ibsen's Saturday, and Wednesday (27 August) instead of Ibsen's Monday. Ibsen[257] goes straight on to describe the demonstration event (of two days later) in a way that could easily be misinterpreted as meaning it followed on the next day, or even the same day; whereas it was in fact two days later, on 27 August.
2. **Prof. Lassen** has two principal sites at which he dates Bjørn Ibsen's Day, (it, truly of 27 August 1952):

    a. His most quoted work,[258[p40]] the Preliminary Report (*Lancet* of 3 January 1953) states correctly 'on August 27th the first patient received the treatment'.
    b. He repeats this date in his treatise in the 1955 WHO publication on poliomyelitis,[267] where he states on p158 'from 27 August an emergency method was introduced'. But on the last page, 209, he supplies a date of 26 August (that actual day on which Vivi was admitted to hospital) for 'when the first case was treated'. It must be allowed that this wording may be referring not to Ibsen's IPPV the next day, but (unknown to me) to cuirass-NPV being supplied to Vivi upon her being admitted—if it ever was; I have not found it to be documented. The cuirass respirator certainly was available for her in the treatment room on 27 August after Ibsen supplied m-IPPV, and thus, *possibly* it was likely before, and hence could have been also after her admission the day before, too, but that does not appear in reports. Item B.1.a later in this list does not support that suggestion.
    c. **Wackers** in his detailed 1994 *Acta Anaesthesiologica Scandinavica* paper[291] repeats the 27 August date, as it was in Lassen's earlier leads (see item A.2.a and b), and also the papers of Berthesen[282,366] and of others.

B. In some sites, this date is ***other than*** the 27 August now given in this chapter, as 26 August.

1. Lassen revises to a date of **26 August** in

    a. the first issue of the *Danish Medical Bulletin* ('The introduction of bag ventilation on August 26').[379[p7]]
    b. his WHO publication[266] (as explained in item A.2.b of this list), on p209. (Is it a typo, since on p158 he had preferred August 27?)

   c. his 1956 book on the epidemic, *Management of life-threatening poliomyelitis*, in Introductory Remarks, has 'and on August 26 the first patient was treated with the method which soon became our method of choice in patients with impairment'.[227[pxi]]

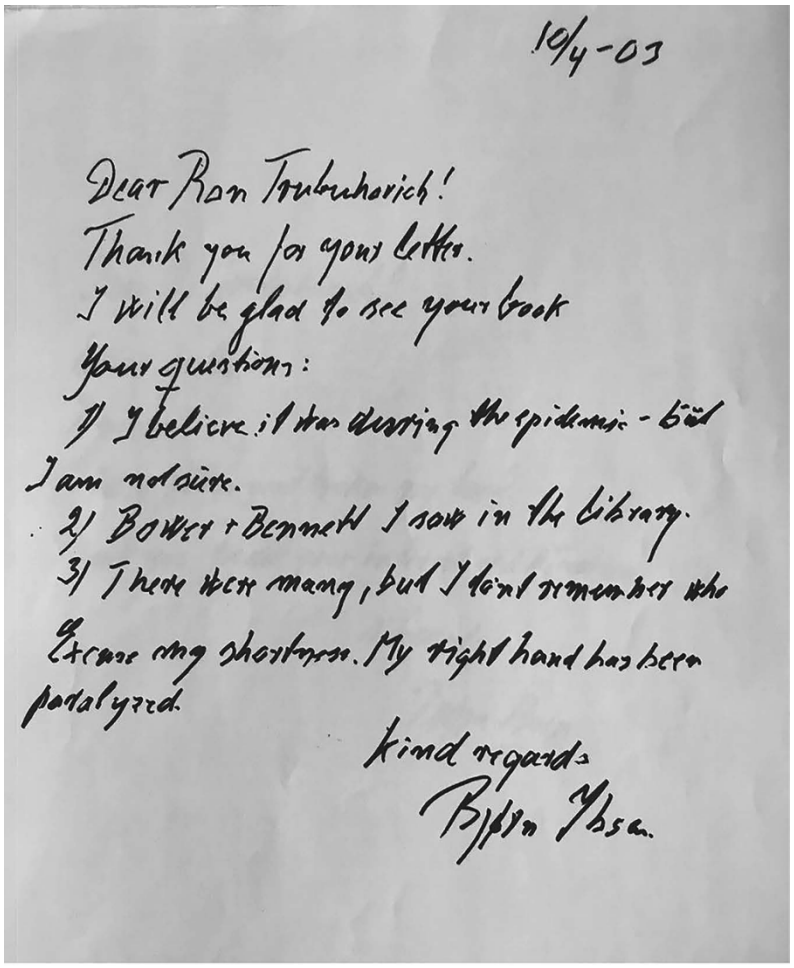
2. Other authors in the same 1956 book,[227] all under Lassen's editorship, give 26 August as the introductory date.

   a. Neukirch and Søttrup[227] repeatedly state 26 August 1952 as the date for the introduction of 'New Methods of Treatment' (p147, and especially p150)
   b. Bjørneboe and colleagues report, 'from 26 August 1952 the therapeutic regime was altered'. [227[p19]]

3. Andersen and Ibsen[371[p786]] refer to 'up to August 26' (noted as such, since it is not 'up to the 27th').
4. Astrup, in his 1986 book with Severinghaus, *The history of blood gases, acids and bases*, stated (although he was an eyewitness to the morning's event) the demonstration occurred on the following day, which would thereby need to be the one after the day on which Ibsen was called in to the meeting of clinicians, including Astrup (which took place on Monday, 25 August).
5. Severinghaus, Astrup and Murray, in their otherwise admirable 1998 paper, firmly state a date of 'the next day, 26 August'.[365[S119]] The supplementary material in Dr Reisner-Sénélar's article has in its first line (a typo?), 'The first patient that Ibsen treated with the new method on August 26th1952'; that is, Tuesday's date was mistakenly written for one day earlier than that of the actual Wednesday event.[367suppl[p1]]

Finally, research by Dr Preben Berthelsen, Copenhagen anaesthesiologist, unearthed a photocopy of the original hospital records for patient Vivi's admission (a copy of which he kindly forwarded to this author). He reported (personal communication), 'She was admitted on August 26th 1952 – and Ibsen treated her on August 27th'. Hence, Wednesday, 27 August 1952 was always indisputable as the correct date for Bjørn Ibsen's historic introduction into Denmark of a new method for treating acute respiratory failure in life-threatening polio (polio+).

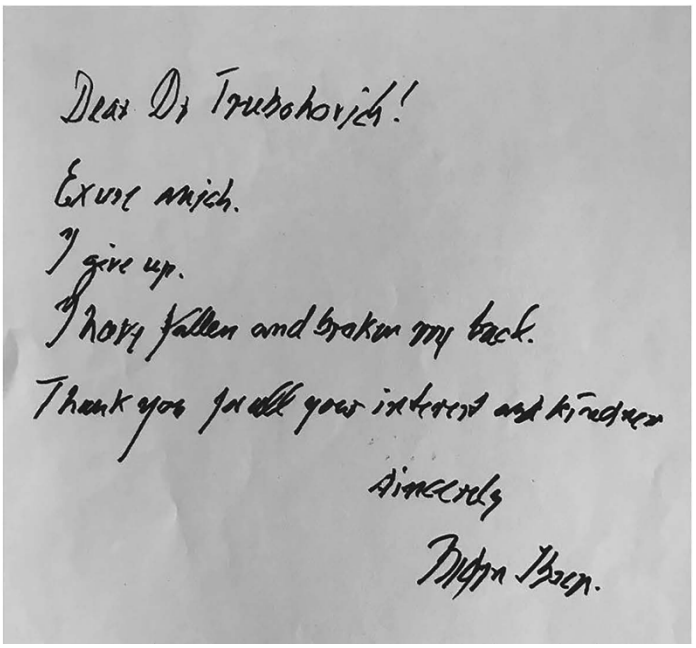
Addendum 7.2: Correspondence received from Prof. Ibsen



10/4-03

Dear Ron Trubachovich!
Thank you for your letter.
I will be glad to see your book
Your questions:
1) I believe it was during the epidemic - but I am not sure.
2) Bower + Bennett I saw in the library.
3) There were many, but I don't remember the
Excuse my shortness. My right hand has been paralyzed.

kind regards
Björn Ibsen.



Dear Dr Trubachovich!

Excuse mich.
I give up.
I have fallen and broken my back.
Thank you for all your interest and kindness

sincerely
Björn Ibsen.

The Danish poliomyelitis epidemic, 1952–1953

Chapter 8: Further commentary on Denmark's 1952–1953 poliomyelitis epidemic

Chapter 8: Table of Contents

8.1 Overview

In this chapter, I consider in greater depth certain aspects of the 1952–1953 Copenhagen poliomyelitis epidemic already discussed in Chapter 7:

1. Dr Bjørn Ibsen's method of treatment of the sickest patients at the Blegdam Hospital during the epidemic: although successful, it could have failed at the first demonstration.
2. The roles played by ventilatory failure versus gross neurologic destruction in causing deaths, that is, whether the cause of death in polio, as asserted at times, was inevitably ventilatory rather than, on occasion, cerebral. With pharyngeal paralysis without that protection aided by tracheotomy, there also was respiratory cause of death from the inhalation of pharyngeal fluids followed by infection.
3. Varying statistics that need reconciling, and an attempt to do that from numbers printed concerning the epidemic's seriously ill Copenhagen patients. This involves defining the dates of certain events, mortality among patients and some other relevant factors.

The Blegdam Hospital concept of life-threatening poliomyelitis (hereafter, polio**+**) will be revisited, along with epidemiologist Dr Lassen's division of serious polio illness in 345 patients into six anatomico-clinical categories.[227[p7]] Attention is drawn to the severity of assorted cerebral lesions demonstrated in 114 of the 115 autopsies conducted out of the 144 fatal cases. Despite an overall mortality rate of 42% among the epidemic's sickest patients in Copenhagen's Blegdam Hospital, the lowest mortality rate of 11% has been identified for the 18 similar patients treated during the last months (they were hospitalised within the period 20 December 1952 to 2 March 1953).[227[p150]] Various *sources* for the minor discrepancies and conflicting numbers are noted.

The valuable retrospective book on the epidemic, edited by Dr Lassen in 1956,[227] has been referred to freely throughout. Some folklore concerning aspects of patient management during the epidemic will be mentioned. In the light of research by Dr Preben Berthelsen, anaesthetist and medical historian of Copenhagen, an essential correction has been ensured for dating 'Bjørn Ibsen's Day'[368] (correcting my acceptance and previous publication, on the basis of multiple other assertions of the date of 26 August 1952. Instead, the correct day is 27 August.[258[p38]] Thus, initially, with a choice of two dates available, I had 'backed the wrong horse'[368]).

As in the preceding chapter, this commentary has been based principally on accounts in medical papers and books about the Danish polio epidemic. My understanding of events has been reinforced by personal communications received from helpful Danish correspondents, especially Drs Preben Berthelsen and Louise Reisner-Sélénar. Their advice and publications have proved enlightening.

8.2 The role of Dr Bjørn Ibsen

8.2.1 Prof. Ibsen's diagnosis

Bjørn Ibsen recognised that treating physicians held a misunderstanding about carbon dioxide levels.[257[p23],365] In reference to Ibsen's 'anaesthetist's viewpoint'[257[p21]] on the treatment of respiratory complications of poliomyelitis during the epidemic in Copenhagen in 1952, authors Drs Berthelsen and Cronqvist had stated in 2004:

> When Ibsen was consulted in August 1952... he almost immediately realized that polio patients died from respiratory insufficiency with carbon dioxide retention and not from an overwhelming virus infection of the brain, as was generally believed by the epidemiologists.[282[p1193]]

But some deaths continued despite compensation being provided for inadequate breathing. Overwhelming viral infection would prove unable to be dismissed as a cause for some deaths.[227[p7]]

8.2.2 What was the basis for Bjørn Ibsen's viewpoint?

Multiple factors were operative. In their earlier studies together on hypoventilation, anaesthetist Ibsen and thoracic surgeon Engell had already elicited the clinical signs of carbon dioxide accumulation, which by today's understanding appears poorly appreciated in Copenhagen at that time.[257,291,365] Further, Ibsen explained that he was aware of papers by Bower and Ray Bennett, which he had discovered while medical advisor to the Copenhagen library whereby he had access to 'the largest medical library in Denmark'.[257[p22]] He wrote to Bower who sent him reprints. These papers had described improved survival in the polio epidemic at Los Angeles in 1948–1949, with treatment employing AV boosted by additional positive pressure (as described in Chapter 5). Ibsen brought the papers to a meeting of relevant doctors at Blegdam Hospital, called for Monday, 25 August 1952; however, the chief epidemiologist Prof. Lassen and his colleagues were dubious about the success shown in these American papers and considered that many patients had been ventilated even before they needed such treatment.[257[p22]]

After the meeting with the Blegdam doctors, Dr Ibsen made some investigations of his own at the Hospital.[257,291] Ibsen studied the autopsied lungs of four recently dead polio patients. To him, the lungs of one boy he studied did not appear to be sufficiently atelectatic to make adequate ventilation impossible.[369] Further:

1. Ibsen also studied some of Lassen's patients with respiratory paralysis (bulbar and/or spinal), who were receiving AV, six from a cuirass (all that were available), and one other patient from the hospital's single Emerson tank respirator (both kinds of machines supplied INPV),[227[pxi],258[p37],267[p158]] together with studying their clinical records. He found signs he recognised from his studies with Dr Engell as resulting from carbon dioxide accumulation despite the AV provided.

2. He appreciated that an elevated carbon dioxide content, which had been documented for some patients, was indicative of its retention, and not, as the epidemiologists considered, of metabolic alkalosis.

8.2.3 Manual intermittent positive pressure ventilation

The concluding 'Folklore' section of this chapter (Section 8.10) notes that it is medical legend that on 27 August, a now-desperate Lassen allowed ('condescended to ask'[282[p1193]]) Bjørn Ibsen to demonstrate his proposed intervention to try to save a new patient without using machinery.[257,291] After Ibsen's impressive initial success, the application of the methods that he introduced to other critically ill patients at the hospital resulted in the dawn of a new era in the care of polio 'respiratory' patients.[227,257,258,282,291,365,368,369]

8.2.4 Would the same success have been achieved with any other sick patient?

The exploration of this question is helped by considering, first, the severity of the test patient's condition, and then, the severity of the disease in fatal cases. Evidence for the latter comes from autopsy data, giving information for the most likely, ultimate 'final authority'.[227[p151–73]]

8.2.5 Severity of Vivi Ebert's condition

As it happened, the extremely sick girl chosen by Dr Lassen, a new patient by then hospitalised for one day, proved suitable, if difficult, for a critical demonstration. Lassen had classified those at Blegdam Hospital with 'life-threatening poliomyelitis' (again, polio**+**) into six groups: A to F (as per Table 8.1[227[p7]]). Ranking these groups in descending severity of their polio**+** (as per group fatality rates) gave an order of F > E > D > A > C > B. What has been written regarding patient Vivi's ventilatory inadequacy and 4-limb paralysis[257] would appear to place her in Group C ('paralysis of respiratory muscles without encephalitis, cerebralia or pharyngeal paralysis'). But she had already experienced marked clinical deterioration in her breathing, and hence presented a stern test for Dr Ibsen.[282]

8.2.6 Causes of mortality determined from autopsies of polio+ patients

Autopsy findings and the tables of mortality in Lassen's definitive book[227[p7,88,152]] appear to confirm the following:

1. The survival of patients appeared dependent on how the clinico-anatomical features they presented with best fitted the characteristics of one of Prof. Lassen's six groupings. These clinical groupings are depicted in Table 8.1.[227[p7]]
2. Despite overall improved outcomes following the changes in treatment introduced on 27 August, some of the groups still had a high mortality rate. Among Groups A, B, C, D, E and F, the mortality percentage was 39, 25, 32, 46, 63 and 85, respectively (Table 8.1[227[p7]]).
3. Destructive changes in some of the brains seen at autopsy were 'particularly severe' (Table 8.2,[227[p152]]). Thus, Cases 1–4 among the nine case studies detailed in a selective survey[227[p151–73]] had medullary and/or hypothalamic necrosis and destruction, (presumably) drastic enough to

account for death or to contribute largely to it—usually within a few days after admission. Lassen had described 'severe encephalitis', but mostly as affecting children.[227[p1],258[p40]]

4. When the brains of more than 100 of the Blegdam's 115 autopsied patients were examined for the incidence of inflammatory and degenerative processes in various [cerebral] locations, the incidence of changes, as displayed in this chapter's Table 8.2,[227[p152]] was with the highest in

   pons-medulla > hypothalamus > mesencephalon > cerebral cortex.

   However, the order for 'particularly severe' changes was

   pons-medulla > mesencephalon > hypothalamus > cerebral cortex.

   Note from the numbers in the same Table 8.2 that the changes in the pons-medulla were particularly severe for 114 of the 115 patients.

5. Further, Drs Pedersen and Bjerre-Christensen's own 'Table X'[227[p80]] recorded that some deaths were closely associated with, or resulted from, various complications or failures in organ systems, totalling 342 individual manifestations. The writers listed the range of causes with corresponding mortality percentages (see this chapter's Table 8.3[227[p80]]). Surprisingly perhaps, the only respiratory cause of mortality identified on the list was 'pulmonary oedema', in 29(=8%) of the 345 patients, with mortality of 26 (=93%) among those 29 owing to that cause. Therefore, by these tables, after 26/27 August, no deaths would seem to be caused by purely ventilatory failure.

6. Polio, a deadly disease for some patients, was lethal from causes *originating* from other than 'unrelieved respiratory paralysis'. This is clearly documented in Table 8.3.[227[p80]]

Table 8.1: Main classification of the series—distribution and mortality

Group	Clinical group	Principal site of anatomical lesion	Distribution no.	%	Deaths no.	Fatality rate %
A	Polio-encephalitis	Encephalo-bulbar	75	22	29	39
B	Pharyngeal and/or laryngeal paralysis without encephalitis, cerebralia or spinal paralysis	Bulbar	12	4	3	25
C	Paralysis of respiratory muscles without encephalitis, cerebralia or pharyngeal Paralysis	Spinal	157	45	50	32
D	Paralysis of respiratory muscles and pharynx or larynx without encephalitis or cerebralia	Spino-bulbar	28	8	13	46
E	Paralysis of respiratory muscles combined with cerebralia without pharyngeal paralysis	Spino-(bulbar-) Cerebral	60	17	38	63
F	Paralysis of respiratory muscles and pharynx or larynx combined with cerebralia	Spino-bulbar-Cerebral	13	4	11	85
Total			345	100	144	42

Reproduced with permission from Pedersen, Bjørneboe, Johnsen and colleagues (Lassen[227[p7]]).

Table 8.2: Localisation and severity of inflammatory and degenerative processes

No. of cases examined	Localisation	No. of cases with inflammatory and degenerative processes	Particularly severe lesions
105	Cerebral cortex	31	8
106	Hypothalamus	79	16
100	Mesencephalon	75	35
115	Pons, medulla oblongata	114	114
115	Spinal cord	115	115
42	Spinal ganglia	14	–

Reproduced with permission from Vimtrup, Christensen and Schourup (Lassen[227[p152]])

Table 8.3: Complications, incidence and relation to mortality (cases = 345 patients, mortality = 42%)

Complications	Incidence		Mortality	
	No. of cases	%	No. of cases	%
Shock	134	39	90	67
Hyperpyrexia	66	19	60	91
Uraemia*	36	22	28	78
Pulmonary oedema	29	8	26	93
Paralytic ileus	112	32	49	44
Hypertension	65	19	27	42
Total	442		280	

* Only patients aged 15 years and older.
Reproduced with permission from Pedersen and Bjerre-Christensen (Lassen[227[p80]])

8.2.7 Inference

One is left to conclude that it is fortunate Vivi's life-threatening problem appears to have been essentially ventilatory and not cerebral. Because the epidemiologists had not recognised that carbon dioxide was continuously accumulating despite AV by INPV, they had attributed many deaths to the overwhelmingly destructive effects of the polio virus on the brain. Autopsy findings indicate that the epidemiologists were certainly correct on occasion. (How many of the 115 patients of Table 8.1/Table XXXI[227[p152]] could have a cerebral cause of death, perhaps even all of them?) Although Ibsen's new treatment essentially prevented death from ventilatory failure (although 8% of 'respiratory' deaths attributable to other non-ventilatory causes were listed as 'pulmonary'; perhaps, these were due to infections), the overall mortality for 345 patients was declared at **42.7%,** as will be examined.

No denigration at all of Prof. Ibsen (and his colleagues) is implied. From August 27, his method of intervention for hypoventilation was markedly life-saving for many patients, beyond any dispute (the overall improvement in mortality appears to have been 87–42 = 45% in round figures, as will be elaborated further). However, Vivi never advanced out of a residual lesser disability. Wackers[291] has pondered this sobering possibility: 'What if the 12 year-old girl had died, despite the anaesthetist's efforts? Would he [Ibsen] have been given a second chance?[291[p430]]

Polio, a deadly disease for some patients, could be lethal from causes originating from other than unrelieved respiratory paralysis. This is clearly documented in Table 8.3 herein (= 'Table X'[227[p80]])

8.3 Features of the epidemic

Danish/Copenhagen population numbers in Lassen's Table 1[227[p1],267]

Copenhagen's population in 1952 was 1.2 million, or 27.9% of Denmark's total, which was 4.3 million.[227[p1]] The studies for 1952–1953 by Hamtoft, the official statistician, revised the whole country's total official number of polio victims up to 1 May 1953.[396] In 1954, Andersen and Ibsen[371] gave the 1952 epidemic totals for the whole of Denmark as 5,722 cases notified, 3,722 of which passed through Blegdam Hospital,[371] for which Lassen, in his 1956 paper, could verify poliomyelitis for 2,241.[227[p1]] Of these,1,250 had 'paralysis', and **345** required special treatment for the whole epidemic (here, taken to be until 2 March 2).[227[p1]] Lassen's 1956 book, based on Hamtoft's[396] studies also, reduced Andersen and Ibsen's 5,772 all-of-Denmark polio patients to 5,676, with 2,450 of them being 'paralyzed' (=43.16%).[227[p1]] Patients treated outside Copenhagen would have included a proportion of polio**+** patients.

The death rate from polio in Copenhagen in 1952 was approximately 60% of the rate in the rest of the country [at 8% v. 14%][227[p2]]—also deduced from Lassen's 'Table III'(in line with Hamtoft),[227[p2]] one which is less readily apparent. The death percentage rates are defined in that table per 100 paralytic cases. I also found the figures for paralytic polio and mortality (in Lassen's Tables II and III,[227[p2]] both based on the statistician's) less readily apparent. The mortality rate of four age groups is given per 100 paralytic cases (thereby providing a 'fatality rate per cent').

8.3.2 Serious poliomyelitis

Lassen, who defined 'serious' poliomyelitis as manifesting 'insufficiency of respiration or impairment of swallowing or both',[227[p1]] became forced to recognise that those afflicted needed what came to be called 'new methods of treatment',[227[p150]] that is, 'tracheotomy, artificial respiration, postural drainage, or combinations of these'. Throughout the 1956 book,[227] the total of such Blegdam polio**+** patients for the whole epidemic (which many took as being until 2 March 1953) is usually given as or very close to 345, such as on p4 (comparably, Neukirch and Søttrup[227[p150]] listed 318 similar patients for the period 26 August to 2 March, who, together with 30 or 31 patients for the 7 July to 25 August period, made their total 349). 'Special therapeutic measures', as above, were required for 345 of the paralytic patients.[227[p3]]

Lassen writes of the whole epidemic being between 24 July and (confusingly, what he always seemed to refer to as) 'at the end of the year' (i.e. end of 1952), for which he verified a diagnosis of poliomyelitis in 2,241 patients. Of these, 1,235 (55.1%) experienced paralysis.[227[p3]] A confirmed diagnosis of polio+[227[p3]] among 2,241 would amount to **15.4%.**

In early 1953, Lassen had reported polio**+** patients numbering 316 of the 866 paralytic cases (=35.7%),[258[p37]] 'between July 24 and Dec 3', 1952 (elsewhere in the same paper, the 'point of reference' was 'Dec.6'[258[p40]]). This represented 'an enormous load'. The caption to the *Lancet* 'Figure-2'[258[p38]] confirmed 316.

It is not clear whether Lassen's 31 'serious' patients of 'July24-Aug.25' by his 'Mortality-Rates' Table 'III'[258[p40]] (of whom 70% died within 3 days) were regarded as within the group for this treatment or not, as for those dates, such treatment was then unknown to Lassen (although he was applying his own fifth treatment mode of 'postural drainage and stomach-tube', etc.). Were the 31 included in his 316 patients? They can hardly be added in without making the total polio**+** number 348 patients by 6 December 1952, when that is the approximate total for the entire epidemic (or at least until 2 March 1953).

A calculation can be attempted for serious patients, quoting this Chapter's Table 5,

From 26 August to 6 November, Lassen gives cases	250[226[p40,TableIII]]
From 7 November to 19 December	501[226[p150,Table XXIX]]
From 20 December to 2 March	18[226[p150,Table XXIX]]
Total	=318
From 24 July to 25 August	31[226[p40,TableIII]]
Total for the epidemic	=349

8.3.3 Patients requiring 'special treatment'

1. Prof. Lassen's total of '316 patients', for 26 August to 3 December, has 250 from his Preliminary Report Table III's five periods (II–VI)[258[p38,Fig2]] up to 6 November, and hence needs 66 more to make that total by 3 December. But Neukirch and Søttrup's Table XXIX[227[p150]] shows from 7 November, 68 such patients not until 2 March.
2. In his 1955 WHO text, Lassen numbers 349 patients for the epidemic 'till the end of the year',[267[p158]] but 345 in that article's tables.[267[164,167]]
3. Andersen supplies a number of 349 also, but for the whole period, 7 July 1952 to 2 March 1953.
4. The various writers for the definitive 1956 book[258] have differing numbers for such patients needing 'special treatment', even though Lassen was its editor.
    a. Lassen, referring to 'Between July 24 and the end of the year', quotes 345.[227[p.ix]]
    b. Pedersen and colleagues quote the same number, 345, 'During … July 7, 1952, to March 2, 1953'.[227[p5]]
    c. Pedersen and Bierre-Christensen state 42% mortality for '345 cases'.[227[p80]]
    d. Neukirch and Søttrup record 318 patients between 26 August and 2 March (as at 1 January 1956).[227[p150]]
    e. Bjørneboe and colleagues have the same number[227[p19]] of '318 patients treated according to the new therapeutic principles', implying 349 patients for the total period (again, once the earlier 31 patients of 24 July to 25 August, as per Lassen's original *Lancet* Table III,[258[p40]] are added).

In summary, consistency is not exact, only close.

8.4 The time span for the epidemic

As regards the time span of the epidemic:

1. Lassen has starting dates, supplying numbers from the beginning of August[227] but also from 24 July.[258] He never refers to the ending of the epidemic, other than to state 'the end of the year' (which was 1952).
2. For a number of treated patients identical to Lassen's total of 345 in the 1956 book[227] (which Lassen stated to be only for a period from the beginning of August to the end of the year), an April 1954 paper by Andersen and Ibsen[371] described the treatment of patients as extending over a period starting earlier on 7 July and finishing later on 2 March 1953.
3. For the same end date of 2 March, the last two papers in **item 4.d** and **e** in Section 8.3.3 are concerned only with a period starting with the employment of (any) special measures, that is, 27 August.
4. Hamtoft[396] of the Danish National Health Service has the epidemic starting with a few cases in June 1952 and ending by 1 May 1953.
5. Neukirch and Søttrup in their 'Results' chapter's Table XXIX[227[p150]] state the 'old' treatment ended on 25 August, with cases then numbering 30 and deaths 26, and the 'new' method started on 26 August and ended on 2 March.

In summary, again, there is variability in the starting date.

8.5 Varying severity of illness among these polio+ patients:

This topic, now expanded from my earlier writing, was reflected in

1. non-survival versus survival from the illness, for individual patients of groups A–F, as can be seen in the percentages of mortalities listed; see Table 8.1 herein, and also item no. 5 in Section 8.2.6 and Table 8.3 for the high mortality from 'Complications and Special Conditions, Chapter X'.[227[p80]]
2. need for ventilatory assistance ± tracheostomy, versus the absence of need. In the latter instance, less interventional measures alone (e.g. postural drainage, continuous gastric tube suction, and supportive measures, such as nutrition) apparently provided adequate treatment.

In 333 patients, 'one or more of the four criteria set up for respiratory failure' were met; 12 others had '[impaired] deglutition without respiratory insufficiency'.[227[p5]] 'Within the first month a total of 277' received AV, with 97 of these dying within the first week.[227[p13]] Lassen reported that from 26 August to 6 December, the 'great majority' of the Blegdam polio**+** patients were ventilated.[258[p40]] In a subgroup wherein all were treated by tracheotomy and bag ventilation, 70 patients or 46.7% died. But regarding the whole epidemic, Lassen elsewhere says 'about 300 patients [were] subjected to manual bag ventilation'.[227[p60],371[p184]] (After 3 years, 25 were still needing ventilatory support completely or for part of a day).

8.6 Tracheotomy

The number of polio**+** patients with a tracheotomy was 267, of whom 112 or 41.9% died.[267[p167]] Not every AV patient received a tracheotomy (26 did not), while not every tracheotomy patient was ventilated (31 were not). Søttrup set down carefully the indications used when tracheotomy was (or was not) considered required.[368[p27]]

Other complications have already been mentioned, as in this chapter's Table 8.1.

8.7 Mortality during the epidemic

(See Table 8.4 and summarising Table 8.5.)

Berthelsen and Cronqvist also stated: 'In the following months [of the epidemic] mortality markedly decreased to approximately 25%'.[282[p1193]] Polio patients to whom this mortality rate of approximately 25% applied came from the group of patients at the Blegdam specified as having life-threatening poliomyelitis, that is, polio**+.**

Table 8.4: Mortality rates—new methods of treatment introduced on 26 August 1952 (date of reference 1 January 1956)

Period of admission	No. of cases	Died	%
7 July – 25 August*	30	26*	87
26 August – 7 September	50	25	50
7 September – 23 September	50	23	46
23 September – 5 October	50	22	44
6 October – 20 October	50	15	30
21 October – 6 November	50	18	36
7 November – 19 December	50	13	26
20 December – 2 March	18	2	11
Total: 26 August – 2 March	318	118	37

(Perhaps 'from the 26th August' is meant to mean starting on the 27th. Otherwise, why it is the 26th is not obvious.)
Reproduced with permission from Neukirch and Søttrup (Lassen[227[p150]]).
*Note: Usually 25 August is 26 August, and 26 died is 27.

Table 8.5: Mortality rates at different periods during the epidemic and their derivation

1952–1953 Dates	Thesis ref.no.	Polio+ numbers	AV numbers	Deaths number	% of deaths
Total numbers					
24 July – 6 December	258	312[ii] below	?	134	42.9
		[=31 + 250 + 31]		[=27 + 100 + 7]	
	258	v. 316 (p37, 40)	?	134	42.4
7 July – 2 March	267	345 (p164, 167)	262 (p167)	142 (p167)	41.2
	227	345 (p4, 11)	277 (p13)	144 (p7, 150)	41.7
	227	348[iii] below (p150)	?	144 (p150)	41.3
	227				
	227	[= 30 + 318)		[= 26 + 118]	
Fractional numbers					
24 July – 25 August	258	31	31	27	87.1
		(v. 31 + 4)	31	27	(v. 77.1)
	227	30 (p150)	30	26 (p150)	86.7
26 August – 6 November	258	250	'great majority'	100	40
		[= 50 × 5]			
7 November – 6 December	258	31	?	7	22.6
				[from 23% of 31]	
7 December – 'end'	227,258	33 v. 29	?	10	30.3
	227,258	[= 345 – 312 v. 316]		[= 144 – 27 – 100 – 7]	
7 November – 2 March [i] below	227	68 (p150)	?	15 (p150)	22.1
		[= 50 + 18]		[=13 + 2]	
7 December – 2 March [if] below	227,258	37	?	8	21.6
		[= 19 + 18]		[= 6 + 2]	

i. From Neukirch and Søttrup's Table XXIX; ii. from Ch.8's ref. 227 at p.150; and iii. Lassen's Preliminary Report[258]

	Ref.	Patients	Deaths	%
7 November – 19 December	1	50	13	26.0
7 November – 6 December	3	31	7	22.6
7 December – 19 December	3,1	∴ 19[= 50 − 31]	∴ 6[= 13 − 7]	31.6
20 December – 2 March	1	18	2	11.1
7 December – 2 March	1	∴ 37[= 19 + 18]	∴ 8[= 6 + 2]	21.6

ii). In his Preliminary Report,[258] Lassen repeatedly (= 5 X) wrote of 316 patients requiring special forms of treatment until 6 December 1952, even though the numbers he supplied added up to only 312 patients: possibly, short of the four polio+ patients from before 27 August who survived but perhaps did not receive AV?
iii). With a reference date of 2 January 1956.

8.7.1 Overall mortality rate among the whole of the epidemic's 345 (or 348) polio+ patients

Mortality became 41.7% (sometimes written as 42%) by the death of 144 (see Table 8.4 herein),[227[p7]] while those before '26 August' make the percentage considerably weighted against success by the death of 27 (or 26) of 31 (or 30) patients, that is, 87% mortality.[258[p40]] (With the changing [for some unrecognised reason] of the 27 August day to 26, as seen in this chapter's Table 8.4 from Neukirch and Søttrup,[227[p150]] deaths then become 41%).

Following the introduction of Prof. Ibsen's new methods and their heroic employment, the earlier drastic mortality rate more than halved. Lassen provided an *interim* statistic from (after) '26th August' until the next milestone date of 6 November, for all those treated by bag ventilation/tracheotomy (first starting with patient Vivi on August 27).[258] Again, it needs noting that these percentages refer only to those Blegdam patients worst affected (42% of the 100 deaths occurred within 3 days[258[p40]]) and not to all 2,241 polio patients, for whom overall mortality, again, is calculable at 6.4%.

8.7.2 Mortality among successive cohorts of 50 admissions with polio+:

Neukirch and Søttrup, in their 'Results' chapter in Lassen's definitive publication on the Copenhagen epidemic,[227[p147–50]] updated the Lancet 'Preliminary' original *percentage* figures[258[p40]] for the mortality percentage rate of Lassen's five cohorts of successive 50 patients (a total of 250 patients, of whom 100 died). Their figures are reproduced in this chapter in Table 8.4. These changed from the percentages for the five cohorts between 26 August and 6 November from 52, 48, 38, 26 and 36[258[p40]] to 50, 46, 44, 30 and 36 (see Table 8.4; the dates of the cases in the two tables do not match exactly). A minor change in the total mortality up to 6 November also followed, reaching 103 (41%) compared with Lassen's total of 100 among the 250 patients (40%) in Table III in his 1953 *Lancet* article.[258[p40]] But the overall trend was downwards, and for the next month, 6 November to 6 December, Lassen reported the mortality rate among the following 31 polio**+** patients as being further reduced to a striking 23%,[258[p40]] which must represent seven deaths.

By including up to the date of 19 December for their last (sixth) cohort of 50, Neukirch and Søttrup (see Table 8.4) completed their documentation of this group with a mortality rate of 26%. Hence, with mortality eventually approaching the 'approximately 25%', this marked reduction entirely justifies the assertion quoted from Drs Berthelsen and Cronqvist[282] of an end-mortality rate of 25%. That holds, as long as it is remembered that the figure is taken as that finally achieved with the total from *after* Prof. Ibsen's methods were implemented, and not as the mortality rate of polio+ patients from the start of the epidemic.

8.7.3 Mortality in the last patients of the epidemic

Neukirch and Søttrup's[227[p150]] total of polio**+** patients from 26 August to 2 March was 318 with 118 deaths, resulting in a 37% mortality within those dates. For their last cohort of 50 (from 7 November to 19 December), 13 died, that is, mortality was 26%. With their reference date of 1 January 1956, Neukirch and Søttrup[227[p147–50]] listed only 18 more polio**+** patients after 20 December 1952, until 2 March 1953—their end-date for the

epidemic—with the death of two of these bringing that mortality down to a remarkably low end-rate of 11%. The absolute severity of disease is not documented; it possibly could have been milder by this time, but Lassen always spoke against such a suggestion. For instance, he had declared the 'terrible weeks' of the epidemic like 'a state of war',[258[p50]] 'by far the worst ever recorded in Europe',[267[p158]] worse than Minnesota's in 1946 or New York's in 1934, 1931 and 1916 and, as he could later add, Sweden's in 1953. Along with the accumulated experience gained by the clinicians and nurses by that time, the death of only two polio**+** patients certainly was an achievement to be celebrated, and it warrants recognition. However, the number involved (18 patients) was relatively small. (Neukirch and Søttrup had also arrived at that 11% mortality figure for the 20 December to 2 March period, in their Table XXIX [227[p150]]).

Even if all the new admissions are calculable at 37 patients, by counting backwards from 2 March 1953 to (say) 6 December 1952, the final date of Lassen's Preliminary Report[258] (see the last lines of this chapter's Table 8.5), then, with eight dying, the mortality is 22%, which is still nearer to 20% than 25%. The Danish achievement was truly remarkable.

8.8 Difficulties related to Lassen's figures from 7 December 1952 until the 'end of the epidemic'

As already implied, there are problems in trying to derive some figures for this period from Lassen's writings. His famous, groundbreaking *Lancet* 1953[258] report (which, written for a 3 January issue, must have been compiled under very trying circumstances with the epidemic still ongoing, even if diminishing in terms of the number of patients) details either 312 or 316 of such patients being admitted up to 6 December 1952,[258] while the epidemic's total with polio**+** in the1956 book he edited is usually 345.[228[p4,5]] Hence, his number of polio**+** patients after 6 December until the end had to be either 33 (=345 − 312) or 29 (=345 − 316). As the total number of deaths during the epidemic was 144,[227[p7]] as indicated in the data Lassen supplies,[258] the total after 6 December can be deduced as being 10 (= [144 − 27 − 100 − 7] as per Table 8.5 herein). This delivered a mortality rate among 33 or 29 patients for this period from 7 December as being either 30% or 35%, respectively, which is at least 9% higher than the 22% just quoted.

8.9 Bravery of the Blegdam staff

The epidemic was powerful, and one expects that many hospital staff (with some unaware of possible immunity acquired naturally) were placing themselves at serious risk of becoming infected, particularly with the close contact in bag-ventilating of patients or in supplying their nursing needs. To my enquiry of Dr Hans Jørgen Clementsen, my friend, who is now a retired anaesthetist of Hillerød but was a resident doctor during the epidemic, whose wife was a Blegdam nurse during the epidemic, he told me (personal communication) that none of the 1,400–1,500 students or doctors contracted the disease. In his *Lancet* paper,[258[p40]] Lassen made what may have been a 1952 guestimate for a number of about 1,000 for 'baggers' ventilating the polio patients. Dr Clementsen referred to Maag A., Polio-Smitteproblemer, Ugeskrift for Læger 1953;115(32):1212–6, which is without an English summary. The title would translate to 'Problems with the contagion of polio'.

8.10 Some folklore

In discussing the Danish poliomyelitis epidemic of 1952–1953, one can occasionally detect a kind of folklore among latter-day intensivists in Australasia. Some would seem to have a belief along the following lines: during a massive polio epidemic in Denmark in the early 1950s, there were limited numbers of the customary tank respirators available to supply (negative pressure) AV for respiratory inadequacy. Therefore, anaesthetists replaced these by battalions of medical students supplying manual (positive pressure) AV. Thus, the simple ventilatory problem was solved, and thereafter, mortality from the disease was virtually abolished.

Worse, a 2003 published text declares: 'There is an *apocryphal* story [emphasis added] about Scandinavian medical students taking turns to provide assisted ventilation to polio sufferers during an epidemic in Denmark in the 1950s.'[397[p3]] So much then for documented facts. According to Lassen: 'During several weeks we had 40-70 patients in our hospital requiring continuous or intermittent bag ventilation. To do this we employed about 200 medical students daily. Their pay has been about 30s (shillings) for eight hours.'[258[p39]] Hardly apocryphal.

The true story has been clearly set out in journals by participants,[227,257,258,267,295,369,371] as well as by reliable others[282,291,365,398] and in a definitive book.[227] In addition, some of the statistics in this commentary highlight the naive scepticism of the above viewpoints.

8.11 Later communications with Dr Ibsen

In February 2002, Drs Preben Berthelsen and Cronqvist interviewed the great man, long retired, when his memory of distant events was not quite exact for completeness of information. Therefore, they requested the Municipal Copenhagen Archives for permission to see the appropriate clinical pages of the patient Vivi (who had died at age 31 years, in 1971) but were told the records could not be found (subsequently, it became obvious that the Vivi pages were actually lent out at that time and were being examined for a thesis). Meanwhile, in April 2003, as a foreign stranger, I wrote a few questions for elucidation to Dr Ibsen to which he generously responded with an apology and a few short answers, advising he was not really up to being questioned at that time. Later, in 2006, a further interview gained by Dr Reisner-Sélénar achieved historical value regarding Prof. Ibsen and the foundation of ICM, since he provided an account of the events on 27 August and the next day. Dr Reisner-Sélénar recorded his account and then transcribed it into her thesis, in which she augmented it with information from the original hospital recordings. All this material, now documented, is accessible online, as detailed in Chapter 7.

8.12 A central mystery remains

Did Lassen write 26 August in forgetfulness, instead of the 27 August 1952 date he wrote of originally in 1953[258,379] and later,[267[p158]] or did he become 'revisionist' for some reason? His first such mention of 26 August was in the *Danish Medical Bulletin* of 1954,[379] in which he declared in his paper, 'The introduction of bag ventilation on August 26…' (and not the date of the day he had told the Royal Society of Medicine at London the previous year[399]); and then, similarly in his 1955 WHO monograph[267[p209]]. Further, he himself continued with this date in the 1956 definitive book on the epidemic,[227[pxi]] as did the various contributors to it. Under Lassen's editorship, they at times gave or were allowed to indicate 26 August as the introductory date.

For example, in this book, Neukirch and Søttrup repeatedly supply 26 August 1952 as the date of introduction of 'New Methods of Treatment' (p147, and especially p150), while Bjørneboe and colleagues reported, 'from 26 August 1952 the therapeutic regime was altered' (p19). Thereafter, apart from Berthelsen[282] or Wackers,[291] some other writers, such as active observer Poul Astrup, who, on the morning of 27 August, like Prof. Lassen, was present at Bjørn Ibsen's initial demonstration,[291,365] followed the 26 August lead, until Berthelsen's successful confirmation of the 27 August date from hospital records, which was later confirmed by Dr Resnais-Sélénar, and now, can be seen unequivocally, in a Blegdam Hospital clinical record.

While I am aware this theme may be tiresome to readers, it is valuable for historical accuracy that the question of correct dating has been resolved, thanks to the tenacity of Dr Berthelsen. We owe him thanks.

Addendum 8.1: Commentary on Prof. Ibsen's priority rights

To debate which ICU held prime place may seem a trivial pursuit, but the question has been considered before, perhaps with some equivocation, in a published opinion from Bjørn Ibsen himself, generally considered to be the foremost pioneer of the new specialty. In terms of its historical contribution to the sequence of events culminating in a new medical specialisation, the first successful mass treatment in Europe of respiratory failure due to polio was because of the organisation introduced by Bjørn Ibsen and Lassen at Blegdam Hospital, Copenhagen. This became what Ibsen himself described as a recognised ITU. Writing in 1966, he stated

> The second intensive therapy unit [or ITU, this at the Blegdam Hospital] was now in use in Denmark – the first being the one for barbiturate poisoning cases – but this one had the same limitation: only one type of disease was being treated.[295[p284]]

Since then, Drs Berthelsen and Conqvist have judged 'the first ICU in the world' to be a different one in Copenhagen, another which Ibsen developed at the end of 1953 at the Kommunehospital. As they do, I have some reservations over Prof. Ibsen's contention regarding the earlier centralised units. Ibsen's statement, modest in terms of his own achievements, can be examined concerning these options.

1. A Clemmesen ICU or the ITU at the Bispebjerg Hospital, Copenhagen?

Prof. Ibsen referred first to Carl Clemmesen's dedicated unit at the Bispebjerg Hospital, opened on 1 October 1949 to centralise patients into a single area (a 4-room/9-bed unit[333]) for close observation and management after poisoning from barbiturates and other agents (see Chapter 6). There was also a long-continued attitude among the psychiatrists of reluctance to abandon trying new antagonists, in the hope of possibly effecting a nalorphine-type reversal of other intoxicating agents also, such as barbiturates. Although there can be no denying the dedication and care in Clemmesen's unit, to me, it does not warrant Ibsen's generous description of 'first' ITU, and this unit does not hold such a place legitimately, in my opinion.

Dr Clemmesen, essentially a psychiatrist with long experience in treating barbiturate poisoning, did not seem to reveal adequate recognition and treatment for airway and particularly ventilatory problems he saw after poisonings. Treating psychiatric doctors could call on anaesthetists for intubating, or on otolaryngologists for tracheotomy, as well as physicians and others as deemed necessary. The incidence of orotracheal intubation at the Bispebjerg unit is indeterminate. While aware of tracheotomy as an option for airway insufficiency, Clemmesen and colleagues did not favour it because of its previous failure at the Bispebjerg to improve outcomes after barbiturate intoxication.

For some never-explained reason for such selectivity, Clemmesen saw IPPV as eminently applicable to respiratory depression from opiates but seldom to that from barbiturates. When IPPV was tried too late for the latter, he found it unhelpful. Dr Aage Kirkegaard, had already introduced in 1945 intravenous blood volume repletion for shock from poisoning by intoxicating agents, such as barbiturates.

The ability to upgrade Bispebjerg psychiatric treatment for poisoning by visiting Eric Nilsson, an anaesthesiologist successfully treating intoxications at Lund, seemed limited. Thus, Carl Clemmesen's Intoxication Unit should not be regarded as the first ITU/ICU.

2. A Lassen ICU or ITU at the Blegdam Hospital, Copenhagen?

Ibsen's nomination of the second ITU/ICU was the Blegdam area, in which three floors were dedicated to treating polio**+** patients. Ibsen remarked that the Blegdam 'unit' had the same activity shortfall comparable to Clemmesen's, in that only one specific type of illness was being treated, presumably referring here to the respiratory depression of polio**+** (for which Ibsen had been the prime introducer of effective treatment). Examination of the 1956 book edited by Lassen[227] shows that with these patients, much more than respiratory depression from the 'one type of disease' was involved**.** Good, if limited, evidence is available from certain chapters in Lassen's book and remarkable enlightenment from the autopsies conducted on as many as 115 non-surviving polio**+** patients (when all those dying numbered only three more, as per Table XXIX[227[p150]]) among a total of 318 'seriously ill' patients during 26 August to 2 March[227[p147]]). All of these patients who died presumably needed prior intervention for a degree of airway and ventilatory deficiency. Thus, 20-plus anaesthesiologists were in attendance to help students 'bagging' patients. As Ibsen later phrased it: 'For the first time in Denmark anaesthesiologists' knowledge, technique, and equipment were used on a grand scale for other purposes than just for giving anesthesia.'[295[p283]]

This statement echoes the remark made about Prof. Ibsen, attributed to Sir Robert Macintosh, later reported by Prof. Secher in his brief but lucid 1987 account of Ibsen and the Danish epidemic: 'Bjorn you are the one who brought the anaesthetist out of the operating room.'[398[p432]]

The tables and text in Lassen's 1956 book reveal that systemic problems, often vital, were certainly more than solely ventilatory, and also had non-ventilatory causes affecting cerebral and other body systems (heart, lungs, gut or kidneys, and also blood pressure and temperature, as is all evident from 'Table-X'[sic]).[227[p80]]

The order of incidence of the complications among 345 patients, with their mortality percentage (as from Pedersen and Bjeere-Christensen's Table X[227[p80]]) lists

> Pulmonary oedema 93%, > Hyperpyrexia 91%, > Uraemia 78% (those below 15yrs excluded), >Shock 67%, > paralytic ileus 44%, > Hypertension 42%, ('Table-X' [227[p80]]).

Table-X also lists mortality, revealing the incidence of widespread systemic effects of polio, as does Vimtrup, Christensen and Schourup's Table-XXXI for them, in the brain and spinal cord.[227[p152]]

The complications, which resulted in a substantial mortality rate[227[p80]] among the 345 patients, were of pulmonary oedema, with which *mortality* was 93% among the 29 so affected; hyperpyrexia, with 91% dying among the 66 affected; uraemia, with 78% among 36 dying with it (but again, here excluding those below 15 yrs); shock, with 67% dying among 134 affected; paralytic ileus, with 44% dying among 112; hypertension with 42% dying among 65 patients so affected (see Pedersen and Bjeere-Christensen's 'Table-X'[227[p80]]).

Unfortunately, these 1956 tables deal with mortality in these six groups only by total end-numbers and percentages, and not by distinguishing the causes of death separated out within this 118, or not even by whether they were ventilatory or non-ventilatory.

After 26 August,[227[p80]] complications totalled 280 (single for some patients, multiple for others) among 345 patients.[227[p80]] The effects were serious enough for mortality data to provide confirmation that such effects were life-threatening, and in some, could ultimately be fatal. Thus, a mortality rate written at 67%, occurring among 134 instances of circulatory shock,[227[p80]] was recorded for 39% of the 345 patients counted.[227[p80]] The other figures for Table-X demonstrate that a full range of intensive care management was required for multiple serious disorders. By then, respiratory problems that were entirely ventilatory (and not owing to causes such as secondary bacterial infections), should not have made a large contribution to respiratory deaths, because of the availability of m-IPPV from August 27.

The emphasis is that there was ample non-respiratory serious illness among the patients in Lassen's 'ITU', which required intensive-care-type attention. One would also like to know for how many patients was death due to necrosis in significant brain regions. The autopsies define the location of sites with cerebral necrosis,[227[p152]] but not whether its effect was the cause of the mortality, and in how many of them, among the 115 patients autopsied.

The mortality numbers given by Pederson and Bjerre-Christensen in their Table-X[227[p80]] can indeed indicate the *severity* of complications, that is, the need for intensive care in support. That would appear sufficient to negate Ibsen's description of this very large institution at the Blegdam as being only the second treating ICU. The causes of initial referral and admission to Lassen's 'unit' (I have not found Lassen's own use of that word) were not multidisciplinary as it was in Ibsen's Kommunehospital unit, but the polio patients had a high proportion of intensive care problems.

My conclusion is that serious problems in Lassen's unit were not only medical but of an order warranting the description 'intensive care'.

Autopsies secured after 114 deaths (of a total 118 reported from 26 August to 2 March; see Table XXIX[227[p150]]) showed that for the interval 26 August – 19 December, there was a usually progressive reduction in mortality rates among six successive groups of 50 patients (=300 total), from 50% mortality in the first group of 50, down to 26% in the sixth and last group of 50.

Does this 'unit' of Lassen's fit the description of an ITU, the term that Ibsen assigned it? Lassen's unit does not fall short for severity of illness or breadth of medical complications needing expert attention for treatments that were not ventilatory. Unfortunately, despite nine case histories being carefully detailed[227[pp155–67]] by Vimtrup, Christensen and Schourup, it is still difficult to elucidate the findings of their chapter, 'Autopsy Findings', for the 115 patients, apart from noting that 'Particularly severe lesions' were observed (Table XXXI[227[p152]]) in the pons and medulla for 114 of the 115 autopsies examined (all these patients also had severe lesions in the spinal cord).

Lassen asserted—despite repeatedly referring to the epidemic lasting to 'the end of the year', thereby presumably omitting many of the last 18 patients—that the epidemic was of great severity throughout its duration.[267[p158]] Only two of the last 18 succumbed, indicating less severe incidence among them of brain lesions in vital regions. Compare the findings of Vimtrup, Christensen and Schourup of 'particularly severe [brain] lesions' in 114 of 318 (=35.8%) patients.

Berthelsen commented about the last 18 patients (personal communication, 2019), that a count among these last 18 admissions of *'2 deaths, I guess, must be* [=have been] *caused by the cerebral lesions – in rounded figures 15%'.* But that does not approach the previous unequivocal mortality rate at 36% of the total. One is left with the conclusion that the 318 patients certainly formed a group of ICU-type patients needing care of vital functions. One can expect this care was supplied to the best of the abilities of the supporting doctors and nurses.

A latter-day ICU would have a Clinical Director, a role that Lassen pursued, but it is perhaps surprising that he could have personally fulfilled the leading clinical role, when he was writing so much 'new territory' material (concepts, analysis, data gathering) contained in his *Lancet* report of 3 January 1953. However, Ibsen could report that 'To secure continuity in the treatment, conferences were held every single day for 2 hours in Professor Lassen's office, where all problems were discussed by all those involved'.[372[p10–1]]

Lassen was someone who, according to Ibsen, 2002 (Berthelsen, personal communication), was 'actually damned intelligent'. Secher stated, 'As chief of epidemiology it was Lassen who took the responsibility for the establishing of the treatment'.[398[p431]] Then, the treatment is listed as including tracheostomy (early), m-IPPV, physiotherapy, etc., but *without* mentioning non-respiratory concerns.

My conclusion is that Lassen's treatment organisation did partially fill a role of 'first ICU'. In terms of later ICU models, Ibsen's Kommunehospital unit did so fully.

3. An Ibsen ICU at the Kommunehospital, Copenhagen?

What was different about Ibsen's regimen for his ICU at the Kommunehospital, compared with Lassen's management at the Blegdam (or in comparison with 'special care units', known of especially in the US after the early 1950s)? Ibsen's training and experience, brought from basic anaesthesiology, increased the range of management he could offer. Initially, it was mainly trained anaesthesiologists who were in charge of newly established ICUs (always with competent nursing support of the medical staff) with their skills in detection and treatment of airway obstruction, ventilatory inadequacy and circulatory insufficiency involving the central pump function of the heart, or the peripheral vasculature, and the oxygenation of vital organs. In addition, renal function adequacy, acid–base balance and nutritional needs were managed. Ibsen's expectation of anaesthetists in ICUs was for them 'to act as responsible physicians'.[295[p289]] (One can note that Ibsen soon appreciated from French medical articles[295[p287–8]] that adrenergic vasoconstrictors were not conducive to peripheral blood flow, and hence, he came to promote the use of vasodilating agents, such as chlorpromazine.[257,295])

As the Kommunehospital unit's clinician in charge, Ibsen was the leader who had the authority to determine admission to 'his' unit, aware of the treatment options he could supply, based on his assessment of needs. These would be managed more specifically than in a conventional medical or post-surgical special care area. One can note the following well-known remarks of Ibsen on admissions to his unit. He requested that the referring doctor enter in the notes that the patient for whom he sought admission was moribund: 'if the patient recovered then that would be due to our treatment, and if he did not recover, our treatment would not be blamed.'[257[p33]] This writer accepts the contention of Berthelsen and Conqvist that Bjørn Ibsen's unit at the Kommunehospital was 'The first intensive care unit in the world, Copenhagen 1953'. I would add for emphasis the word 'true' or else 'general'. Secher had previously stated, *'*at the Kommunehospital ... he established the first intensive care unit in a general hospital'.[398[p432]]

After the single patient at end-1953, a total of 259 patients over the next four years were documented by July 1958,[378] in the first paper in the medical literature coming from a general ITU, it being the founding one. That total comprised 121 patients with respiratory or circulatory insufficiency, 60 trauma patents, 34 with primary disease of heart or lungs, 19 with neurological disease and 25 others, mainly with poisoning.

Much of 'finding the way' with the earliest patients was from employing basic principles and learning from trial and error.

The Danish poliomyelitis epidemic, 1952–1953

Chapter 9: Bjørn Ibsen—commemorating his life, 1915–2007

Chapter 9: Table of Contents

9.1 Introduction

Intensivists, anaesthesiologists and other health professionals have regretted the death on 7 August 2007 of Professor, dr med. Bjørn Aage Ibsen, notable Danish pioneer of modern ICM, close to his 92nd birthday. Ibsen is generally recognised as the father of intensive therapy as we know it today.[400] This claim arises from the combination of:

- his remarkable improvements to the treatment of problems from acute paralytic poliomyelitis in the Copenhagen epidemic in 1952–1953,[227,369] and
- the foundation of the 'first ICU in the world', also in Copenhagen, in 1953.[282]

The first of these achievements took place at the Blegdam Hospital (Blegdamshospital) for communicable diseases, and the second at the Kommunehospital (Municipal Hospital), both in Copenhagen. This chapter will outline Ibsen's life and expand on some of the material already presented in Chapters 7 and 8.

Figure 9.1: Dr Bjørn Aage Ibsen

Prof. Bjørn Ibsen, with thanks to him (date unknown, personal communication, Hans Clementsen, April 2003).

9.2 Ibsen's early years

Ibsen was born on 30 August 1915 in Copenhagen and graduated in medicine in January 1940 from the university there (the Nazis invaded in April that year). He began training in thoracic surgery, but when surgery was starting to seem a doubtful venture for him,[257[p9]] decided to transfer to anaesthesia (thoracic surgery had made him aware of the necessary role of anaesthetists in the surgeons' performances). At that time in Denmark, anaesthesia was not classified as an independent specialty and was considered low in the medical hierarchical order.[291] Then, Ibsen went for a year to Prof. HK (Harry) Beecher's Department of Anaesthesia at the Massachusetts General Hospital in the US as an assistant resident from 1 February 1949.[257] Ibsen's friend, Dr John Zorab, reported that the year-long course at that hospital provided Ibsen with an excellent training.[400]

Ibsen returned to Denmark in 1950, a year in which an enlightened 1950 report from a Danish 'Second Commission' recommended that hospitals establish departments of anaesthesia, whose staff should care for the patients during and post an operation.[282] Ibsen spent three years at Copenhagen's Rigshospital, freelancing as an anaesthetist. During that time, in 1951 he defended his thesis '*Necrosis capitis femoris veri et fracturam pertroch antericam*', which he had written when he still wanted to become a surgeon (Dr Preben G Berthelsen, anaesthetist, of Copenhagen, personal communication, 2009).

During a devastating poliomyelitis epidemic in Denmark, at the suggestion of Dr Bjørneboe, a senior resident in the medical department (see Footnote 9.1[1]), Ibsen was called in to a crisis meeting of doctors at the Blegdam Hospital on Monday, 25 August 1952, organised by head epidemiologist Prof. Lassen. After careful study of patients, records and some relevant autopsies, Ibsen offered his suggestions to Lassen.[257,291,369]

9.3 Ibsen at the Blegdam Hospital, 1952–1953

The story of Lassen's scepticism and Ibsen's success in saving the life of Vivi Ebert has been covered in Chapter 7, with further commentary in Chapter 8.

Lassen was initially sceptical of the ideas and proposals offered by Ibsen, whom many other physicians at the time would have thought of as 'a mere anaesthetist'.[291] Ibsen not only pointed out some erroneous clinical interpretations the physicians had made but also made recommendations that would be unwanted—of tracheotomy (at that time abandoned there for polio) and of AV, which he would provide without machinery. At that time, the few machines available, namely, one Emerson tank respirator and six Kifa or Sahlin cuirass respirators,[227[pxi]] all supplied INPV. Functionally, these had proved inadequate for Lassen.[257]

[1] Footnote 9.1.[291] An anecdote with a certain charm may account for this. Ger Wackers, who consulted Ibsen when writing about the Ibsen therapeutic revolution, related that, in January 1950, Mrs Ibsen (Doris Kirsten Petersen, a trained nurse, and the mother of their four daughters) and physician Mogens Bjørneboe and his wife were together on the same trans-Atlantic ship returning from the US to Denmark, with Mrs Ibsen explaining the type of training her husband had been undertaking. Later, in Copenhagen, in June 1952, Bjørneboe had sought Ibsen's help with a neonate with tetanus, for supplying not only anaesthesia, but curarisation and m-IPPV through a tracheostomy tube. After three weeks, the tiny patient died, but Bjørneboe found Ibsen's alternative method of treatment much more impressive in controlling the tetanus than had ever been the case previously, when he was using sedation. Accordingly, Bjørneboe suggested to Lassen that an anaesthetist could have a solution for the Blegdam's polio crisis. All credit to informed nursing wives. (Ibsen's second wife Ingrid died in 1986.)

Here was a man now who had the vision to propose ventilating patients for their breathing failure somehow without respirators. A sceptical Lassen reluctantly agreed (desperately or prudently?) to allow Ibsen a trial to show his capabilities with a moribund 12-year-old, Vivi Ebert,[291,368] admitted the previous day. The treatment succeeded in relieving her respiratory distress, and she lived on until 1971, although she was an invalid after eventual discharge to home care for her shortened life.

Ibsen himself wrote of receiving enthusiastic encouragement from Lassen to begin.[369] After tracheotomy, followed by some initial alarms, Ibsen's successful management of the girl's condition by m-IPPV, with an anaesthetist's inflating bag and a Waters to-and-fro canister (packed with carbon dioxide-absorbing soda lime), spoke for itself, and Lassen became an enthusiastic supporter of the method and collaborator. They solved the workforce problem in four days[369,371] by recruiting in sufficient numbers Copenhagen's anaesthetists, medical students and some other anaesthetists attending Copenhagen, to sustain the large team needed. Thus, a large organisation was working after eight days.[369] The Blegdam was overwhelmed with patients (as per Addendum 9.1), and it was demanding work for everyone concerned, as Ibsen has described,[369] but he was fortunate in having Lassen's support and organisational skills for the immense effort needed over the next five months. The whole story was well detailed later by Wackers in 1994[291] and then more recently in 2003 from Prof. Ibsen directly, by Dr Resnais-Sénélar.[367]

Some positive consequences had flowed from the establishment of an international training centre in anaesthesiology in Copenhagen by the WHO only two years earlier. Prestigious visiting instructors (whom Berthelsen & Conqvist listed[282[p1192]]) from the US, the UK and elsewhere provided the courses.[257[p18]] Thus, when the polio epidemic struck, there were 20 to 30 trainees[369] to help with the 1,400–1,500 students and the nurses (and also to gain experience), as well as local Danish anaesthetists, including some who had retired.[371] Immunisation against poliomyelitis was unknown then and was introduced only late that decade (inactivated virus 1955, oral vaccine 1961), before which contagion was still a real risk for these brave helpers.

In their publications during and after this devastating epidemic, Ibsen[257,369,371] and Lassen[227,267,371] demonstrated to the world the feasibility of successfully treating ventilatory failure in large numbers of polio patients by supplying m-IPPV, whether required for days, weeks or months, while at the same time reducing dramatically the mortality rate from its previously appalling level (see Addendum 9.1). Later, a few locally designed machines were constructed,[227[p68]] (e.g. the Bang ventilator) and Dr Carl-Gunnar Engström made his single prototype available at the Blegdam from autumn 1952.[374] The effectiveness and reliability of IPPV, and the supremacy of the Swedish Engström ventilator,[374] were established. Ibsen's (and Lassen's) well-publicised lessons were taken up in other parts of Europe that were fearful of polio epidemics. The wider production of IPPV machines began, and polio patients with breathing problems were gathered into early respiration units.[68] Ibsen's achievements during the polio epidemic are summarised in Box 9.1.

Box 9.1: Ibsen and Copenhagen's 1952–1953 polio epidemic

- Bjørn Ibsen, although acting out of accord with general understanding at the time, recognised the signs of carbon dioxide accumulation (learned earlier from studies using a Carbovisor),[291,371] resulting from underventilation (the high total CO_2 content represented respiratory acidosis, and not, as the physicians considered, metabolic alkalosis), and distinguished them from what were thought to be the infectious consequences of poliomyelitis.
- He demonstrated the benefits of tracheotomy and m-IPPV to effect a substantial decrease in the mortality rate. He and his colleagues successfully developed a large-scale, organised system of treatment without mechanical ventilators, by utilising medical students to provide m-IPPV.
- He demonstrated the superiority of large-scale, prolonged m-IPPV over INPV, which it now replaced.
- By utilising pCO_2 estimations, derived by using Poul Astrup's new methods of determination, Ibsen obtained an effective guide to optimal ventilatory control.[374]
- He recognised and emphasised the importance of ventilatory intervention before complications such as shock or pulmonary oedema became established and significant.
- He recognised and emphasised the need for stabilisation of a patient before planned, secure inter-hospital transport by a retrieval team, in ambulance or plane, together with the benefit of medical staff going out from a central to a peripheral location for a sick patient: 'Help should come to the patient, and not the patient to the help.'[257[p25]]

9.4 Further documentation

The Copenhagen polio story can be read in its ample documentation by the principals, Ibsen and Lassen, while others, especially Wackers and Severinghaus and colleagues,[365] then further, Prof. Ibsen himself again[257] and Dr Berthelsen[366] have shone more light on details, while on the 50th anniversary of the end of the epidemic and the foundation of our specialty, an Australasian journal acknowledged Ibsen.[298] In recent years, 'Bjørn Ibsen's Day' (27 August 1952, the day he first applied IPPV to a patient with polio; see Footnote 9.2[2])[266,367] has been revisited in the Scandinavian anaesthesiological literature.[368] The professor also received fine tributes in English medical writing, from Atkinson in 1997,[401] and Ibsen's friend John Zorab, 2003, in the regular series 'The Resuscitation Greats', featured in the journal *Resuscitation* in 2003.[400] In this century, significant revelations have been contributed from their studies by Dr Reisner-Sénélar[367] from her interview with Prof. Ibsen, reported with clinical data in her 2009 thesis, and then, in 2014 by Dr Berthelsen,[366] Copenhagen anaesthesiologist, from his retrospective look at the 1952 epidemic for setting the record straight.

[2] Footnote 9.2. Following Lassen's mixed offerings of dates, I had set 'Bjørn Ibsen's Day' to a day earlier than the true 27 August 1952.[368] Dr Berthelsen kindly wrote after he could confirm from excavating original hospital records, that this day was not that 26th date. Hence, I added a correction to the 27th in my next article, succeeding the one, 'Bjørn Ibsen's Day'. This error is now clearly resolved.[266, 367]

9.5 An intensive care unit and intensive care medicine

In April 1953, Ibsen was appointed senior resident for anaesthesia to the Department of Surgery at the Kommunehospital and undertook to determine the best postoperative fluid replacement therapy. This enabled him to take charge of the recovery room.[282] His vast experience gained in polio work[295] treating respiratory insufficiency had given him the notion of an ICU for treating multiple critical conditions.[257] He converted an existing observation and recovery unit to establish his own truly multidisciplinary ICU ('multidisciplinary' here, in the sense of the conditions treated, now medical as well as post-surgical recovery and others, and not as referring to doctors from multiple specialties: the ICU was 'his' unit). Ibsen's 1966 article[295] describes its evolution from a previous recovery room to a true 'ICU', what he called his intensive therapy unit or ITU (see Footnote 9.3 for the sequence[3]).

Confirmation that Ibsen's 'own' ICU at Kommunehospital was the world's first comes from fine sleuthing through original records and the medical literature by Drs Berthelsen and Cronqvist[282] (see Chapter 8). The unit's first patient on 21 December 1953 was a non-surgical one, transferred from a medical ward. Ibsen thereby started and established the practice of modern ICM[295[p285],375[p268]]—although he was not using that term—starting 'little by little', with one patient at the end of 1953, followed by 13 in 1954 and 120 by 1957.[282[p1192]]

The firm line he took over admissions can be seen in the conditions he established for entry to his ICU. He required a written statement from the referring doctor in the patient's records that the patient was moribund before allowing admission: 'I wanted to make sure that if the patient recovered, it would be recognised to be due to our treatment, and that if he did not recover, our treatment would not be blamed.'[257[p33]]

By 1957, Ibsen was able to report[295[p285–6]] on his first four years of ICU experience, and presented the notion, 'let us use the cooperation of anaesthetists who can form a pool of trained personnel and the respirator centers for treatment of any respiratory insufficiency' from 'all conditions', 'to stimulate further the development of ITUs in general medicine and surgery'. Then, a year later, when his department of anaesthesiology attained beds of its own, he published details, in his epoch-making[255] article (written in Norwegian with anaesthetist, Tone Dahl Kvittengen [1911–2001]),[378] of the treatment of 258 patients, of whom 165 were survivors. Its title translates from the Danish as 'The work in an anaesthesiologic observation unit'—which was at the Kommunehospital. (Berthelsen and Cronqvist provided a summary in English.[282]) Eight years later, Ibsen listed, this time in English, the wide spectrum of conditions and their associated problems managed in his ITU.[295] In that study, he also described various ICU issues,[295] such as aspects of leadership and anaesthetists' direct responsibility for patient care in ICUs; the control of bacteriological problems; air-conditioning and humidification; heat regulation and fluid therapy; and record-

[3] Footnote 9.3. Ibsen described [295[p284–5]] a transition of ideas from:

- the hospital's polio-specialised unit; to
- a rostered pool of anaesthetists for clinical crises in Copenhagen, available on standby, with equipment; to
- (after successful management of two tetanus patients with sedation–curarisation–IPPV at different sites in the hospital) the installation of a tetanus room, with four successive patients similarly treated, 1953; to
- the first ITU, one 'combined with the service from a conventional recovery room', inaugurated at the Kommunehospital in Copenhagen on 1 August 1953; to
- the first non-surgical but 'physical medicine' patient treated there from 21 December 1953.[282]

keeping. Reflecting on his unit's 'intensive therapy' in a 2002 interview, he said, 'What we did was just to use the principles and techniques, which served us so well in the operating theatre, also on patients with medical diseases'.[282[p1190]]

Ibsen enlarged on his intensive therapy experience in the 1975 supplement to the *Acta Anaesthesiologica Scandinavica* for his 60th birthday; the article was entitled 'From anaesthesia to anaesthesiology. Personal experiences in Copenhagen during the past 25 years'.[257] That work provides an engaging retrospective. At the first International Symposium on the History of Anaesthesia in 1982,[402] he was still discussing the vasodilating 'lytic cocktail' for shock: he first used chlorpromazine in patients with shock in 1955.[295] (His book *Intensiv Shockterapi* was published in 1969, Berthelsen advises.) Zorab described how, with organisational changes impending at the Kommunehospital around 1975, Ibsen moved to exploring the field of chronic pain, to develop and make pain clinics the major interest in his final years of clinical work.[400] His achievements in the foundation of intensive care are summarised in Box 9.2.

9.6 Ibsen's perspective

In relation to his designation as the father of intensive care,[400] Ibsen wrote generously of the contributions of others. He acknowledged repeatedly that it was in the library that he had first read an account (from Los Angeles, by Bower, Ray Bennett and colleagues[268,269]) of using PPV for polio. Not many others could have taken up that option, as the methods of Bower and colleagues were evidently not adopted elsewhere in the US at the time, whereas news of the merits of m-IPPV by Ibsen and Lassen produced immediate imitation. Ibsen wrote to the Americans asking for a copy of their article, and when the epidemic in Copenhagen was reaching crisis point, he tried to interest Lassen in the Bower and Bennett approach of supplying IPPV for the ventilatory problem.

Box 9.2: Ibsen and the foundation of intensive care medicine

- Ibsen revolutionised the management of respiratory insufficiency in acute poliomyelitis.
- He applied basic anaesthetic principles of care, to maintain the vital functions of critically ill patients.
- He suggested the notion of the ICU for the critically ill, which originated from established respiration units, and then, in 1953 established Denmark's first ICU at Copenhagen's Kommunehospital, where he was in charge of care of his own patients.
- He established working rules for safe practice during procedures, such as:
    - avoiding any period of anoxia;
    - preceding tracheotomy with endotracheal intubation;
    - assisting inadequate ventilation or performing IPPV;
    - treating shock with intravenous fluids or initially;
    - vasopressors, then vasodilators.[369]
- In establishing the legitimacy of anaesthetists working in such sites ('the anaesthetist came out of the operating room'[403[p419]]), he promoted the concept of the anaesthesiologist–intensivist. This led to the concept of the dedicated intensivist.
- He established the concept of intervention at the right time for patients whose condition is deteriorating.
- He developed the concept of organising safe transport to the ICU from outside and from distant hospitals, as well as intra-hospital.
- He established retrieval teams.

In 1966, Ibsen referred back to Lassen's polio unit thus: 'The second intensive therapy unit was now [1952] in use in Denmark – the first being the one for barbiturate poisoning cases – but this one had the same limitation: only one type of disease was being treated'[295[p284]] (see Footnote 9.4[4]). He put his own multidisciplinary unit into perspective a page later: 'The first intensive therapy unit not concerned with polio or barbiturate poisoning was inaugurated at the Kommunehospital in Copenhagen August 1, 1953, combined with service from a conventional recovery room'.[295[p285]] (Note that this ITU's first patient was admitted on 21 December 1953.[282])

[4] Footnote 9.4. Yes, but the type of polio disease required more than 'limited' management of hypercarbia and hypoxia by effective respiratory support. Other features of the disease included shock, 'cerebralia'[267[p164]] and devastating cerebral destruction,[227,266] gastric atonia, paralytic ileus, pulmonary oedema, azotaemia and hyperthermia.[227,267]

9.7 Honours received by Ibsen

Ibsen received due honours in recognition of his outstanding contributions to medical practice. He was an honorary member of the Faculty of Anaesthetists of the Royal College of Surgeons (Dublin), the European Association for Intensive Care, the European Association for Resuscitation, the Danish Association of Anaesthetists, and the Scandinavian Society of Anaesthesia and Intensive Care (Dr Preben G Berthelsen, personal communication).

From 1 April 1954, Ibsen was Chief of the Department of Anaesthesiology at Kommunehospital and, from 1971, Professor of Anaesthesiology, University of Copenhagen.[282]

9.8 Conclusion

As the ICM history grows longer, it catches up with the life spans of the pioneers. To name just two, Australasia's Matt Spence died in 1992, and American Peter Safar in 2003, but now, the original pioneer of modern ICM has finally joined them. Scandinavia must be proud of Ibsen's lifetime achievements (Box 9.3); many people owe their lives to the changes he introduced.

Box 9.3: Ibsen's general achievements

- founded the world's first truly multidisciplinary intensive care unit;
- laid the foundation of intensive care medicine ('intensive therapy');
- established the superiority of positive pressure mechanical ventilation, initially for acute poliomyelitis;
- advocated early, preventive intervention in patients whose condition was deteriorating;
- established the principles for safe transportation of the critically ill;
- initiated vasodilator instead of vasopressor therapy in shock;
- produced the first article (in Danish) on intensive therapy, in 1958; and
- increased the reputation of anaesthesiologists.[281]

Addendum 9.1: Summary of features of critically ill patients at Blegdam Hospital during the Danish poliomyelitis epidemic, 1952–1953[227]

The epidemic initially[227,258]

- It was Europe's worst polio epidemic, with more than 3,000 polio patients treated at the Blegdam Hospital, and was termed a state of war.[258]
- 4 July – 3 December 1952: 2,722 polio admissions,[227,258] 866 with paralysis and 1,856 without.[258]
- 7 July – 2 March 1953: 3722 (by then, many adults included) admitted with polio.[371]
- Regarding the epidemic being devastating, Else Tønnesen referred to it in retrospect in 2000 in the Danish medical journal *Ugeskrift for Læger* with her viewpoint (in translation) that from a global point of view it was a negligible affair.[404] But locally, the epidemic was drastic, while the innovative treatment mode eventually became universalised.
- Lassen started treating ventilatory failure with one Emerson INPV tank and six cuirass respirators.[227]
- From the epidemic's beginning to 26 August: 31 patients needed special treatment.[258[Table 3]] (compare 349 total for the whole epidemic[371]).
- After approximately the first six weeks of the epidemic, 27 of 31 NPV patients, or 87%, died (19, or 70%, within 3 days of arrival[227,258]).
- 25 August 1952: Ibsen attended a crisis meeting.[369]
- 27 August 1952: Ibsen conducted the first intervention.[266]

Staff organisation

- Three hospital floors, each with 35 patients (mostly in single rooms),[369[p73]] directed by a minimum of 40 assistant doctors trained in anaesthesia, from Copenhagen's four largest hospitals, who provided special care for 24 hours each day.[371,379]
- A senior anaesthetist gave the anaesthetic for tracheotomy and supervised the assistants who were guiding medical students for the entire period.[371]
- Eventually around 1,400 (or 1,500[291]) students were mobilised to provide m-IPPV, for a total of 165,000 hours.[282,291]
- In all, 600 trained nurses were needed.
- The students worked in 6-hour shifts (at times 8), with four shifts daily, until sufficient 'mechanical students' (i.e. ventilatory machines) became available to replace their services.[291[p427–8]]
- This effort required the close cooperation of all physicians (working 12–16 hours daily, for months), 2–hour daily meetings and numerous discussions.
- Over 4 months, there were 1,500 emergency calls to otolaryngologists during the hours 19:00–07:00 alone.[371]
- During the epidemic, 34 physiotherapists attended.[371[p788]]

Patients and treatment

- 28 August – 3 September: Total 335 admissions (around 50 per day),[227] with the epidemic peaking around 1 September.[258[p37–8],291[p427]]
- 26 August – 7 September: 50 needed 'special treatment' (25 died).[227[p150–TableXXIX]]
- During several weeks, 40–70 patients needed bag ventilation from about 200 medical students, daily.[258]
- On one single day after the first 900 admissions, 75 patients were receiving m-IPPV,[371] which required:
    - 250 medical students, 260 bedside nurses from outside[371] and 27 workers, as required to change
    - 250 cylinders with 10 gallons (45.5 L) oxygen[371] for IPPV, with oxygen and nitrogen in a 50:50 ratio.[371]
- 30 shillings equivalent was paid to a medical student for 8 hours; or for a day's mixed gases; or for a day's soda lime.[258]
- Of total patients: critical, 349[267,371]; respiratory failure, 333; tracheotomy, about 267 and ventilated, about 277.[266,368]
- More than 75 patients were brought in from localities outside the Blegdam's usual area.[258]
- Some patients received manual bag ventilation for more than 3 months.

Mortality

- A great proportion of the patients treated by tracheotomy and bag ventilation were in a very bad state on admission.[258]
- 26 August – 6 November: 250 needed tracheotomy and m-IPPV (100 died, including 5 admitted moribund, who died forthwith).[258[p40]]
- With the associated treatment, this reduction in mortality from 87% to 40% represented 100 lives being saved.[258[p40, Table III]]
- 26 August – 6 November: 47% mortality rate for 150 consecutive patients treated by tracheotomy and m-IPPV.[258]
- 6 November – 6 December: 31 admissions; death rate of 23%.[258[p40]]
- Overall, mortality decreased from 87% to 42%, but was down to 11% for the epidemic's last 18 patients,[227[Table XXIX]] despite the 'constant severity of the cases throughout the whole epidemic period'.[267[p158]]
- As at 1 January 1956 (more than 3 years later), 25 patients still required ventilatory assistance every day and 13 of them required it for most of the 24 hrs.[227]

Other participants

In addition to Lassen, Ibsen particularly acknowledged Poul Astrup, Mogens Bjørneboe, Erik Wainø Andersen and Frits Neukirch.

*Variations in the numbers of polio patients are discussed in references 266, 298 and 368.

Summary and Conclusion

The focus of this monograph was on the foundation of my own medical specialty of intensive/critical care medicine. It contains two streams of particular interest to me, the first being a small, late-19th-century group of people who, I feel strongly, warrant better recognition for their pioneering achievements in the history of that specialty. I have detailed their contributions as originally documented and have corrected misconceptions as accurately as possible. These practitioners were followed in the first half of the 20th century by others who also warrant reassessment, to bring recognition of their contributions to the beginnings of ICM/CCM. Primary sources have been used wherever feasible. I have been aided tremendously by generous and valuable support from my personal helpers at the University of Auckland's Philson and General Libraries in locating papers and books, some of which pose their own difficulties from being printed long ago or, on occasion, not written in English. The second stream of interest concerns the treatment within, and the outcome of, the Danish epidemic of poliomyelitis in 1952–1953 and the subsequent 'first ICU in the world', and the emergence thereby, at the end of 1953, of a new specialty.

In the monograph, with the aid of accounts from principal participants, is a retelling of the story from Denmark, especially as regards 27 August 1952, which is a day that can be described in this context as a landmark. It was the start of a new line of treatment for ventilatory failure in poliomyelitis, which had its origin in the anaesthetic world and in anaesthetists who, principally, were the first practitioners of ICM/CCM. The prime mover was Dr Bjørn Ibsen whose personal account of events was recorded just over 50 years later in an interview with him conducted by Dr Louise Resnais-Sénélar, who generously made her PhD thesis, based on discussion with Dr Ibsen in 2006, available for me to see (discussed in Chapters 7 and 8).

Regarding the Introduction

The aforementioned main themes were preceded by a short Introduction that scans some of the resuscitation events before Copenhagen's 1952 crisis. Information from before the 18th century is sparse, perhaps non-existent apart from brief mention of mouth-to-mouth resuscitation for breathing difficulties, chiefly for newborns. From a few vague hints encountered, one is left wondering whether there could have been occasional resuscitation activity going on in the 17th century, especially in Central Europe. This was likely to have been after apparent drowning and more often than reported, or rarely documented, apart from by those such as Pastor Sebastian Weiss, 1620. The Introduction referred to a few such episodes told and retold, with those of Petri Borelli and Georg Grübel being the best known from the 17th century.

A reflection on pioneers, 19th century and pre-1950

Certain pioneers are noted, all from the US (Joseph O'Dwyer, George Fell, William Northrup and even Charles Trueheart), with the intention of honouring their extensive achievements. The courage displayed by physician **Joseph O'Dwyer**, in trying to achieve a satisfactory endo(-laryngo-)tracheal intubation system for children with respiratory diphtheria, was profoundly impressive. How daunting and depressing for everybody involved (parents, nursing attendants and intubating doctor) must have been those few years when every patient died (one can imagine the difficult conversations/consultations with parents). Perhaps

as O'Dwyer expressed regarding death occurring during an operation, there was a little relief from 'allowing the little sufferers to die easier. It was a justifiable form of euthanasia'.[100[p10].] After success was eventually achieved, in 1885–1886 still only 24% (of a group of 50) survived treatment. But following the arrival of antitoxin and later, active immunisation, O'Dwyer's methods became largely redundant for diphtheria. But he had re-established the credibility of intubation of the airway (which Charles Kite and others had secured in the later 1780s), virtually abandoned for its preservation of vital functions.

George Fell, in the US, a physician seemingly readily offended but always available when called on for assistance, worked tirelessly for more than a dozen years at resuscitating victims. He strove to convince his fellow doctors that many lives could be saved if they would only try his methods, as he had shown with his IPPV system. His demonstrations over more than a dozen years, even when he was disheartened and in impaired health, should have been convincing. His rescues were well documented, as were those of O'Dwyer. The time Fell spent attending some patients (even up to >80 consecutive hours) is indicative of his convictions, determination, courage and dedication.

The Fell--O'Dwyer apparatus may have been somewhat cumbersome, but it did work as required and enabled the first successful open-chest thoracic surgery in the US, in 1900, because of the foresight and tenacity of **Rudolph Matas** (after a different initiative had been successful in France in 1898). **William Northrup** was a tireless promoter trying to open new fields of application for IPPV with the apparatus. But it could not make headway in the early 20th century in the US against the popularity of the insufflation method with anaesthetists. The apparatus gave the first hints of anaesthetic intervention also being helpful for some intracerebral disasters, and results might have been more productive had Sir Victor Horsley in Britain been drawn in more closely and better supported. The key point regarding IPPV with the Fell–O'Dwyer apparatus being used for resuscitative as well as anaesthesiological purposes is that the innovators did lead the way in developing viable ICM modes, even if eventually, the method chosen later was not the pioneers' actual practice.

Charles Trueheart described his novel apparatus in 1870, but thereafter, it seems his new IPPV device and practice for neonates was known and employed only in middle Europe. He is acknowledged here only in a Supplement to Chapter 3 since, apparently, he never brought his apparatus with him back to his homeland's Galveston, Texas. However, the trail to run Charles 'Truehead' to ground to determine his true identity proved a fascinating if protracted chase, impossible other than with librarians' help received here in Auckland and also from Galveston.

Intermittent negative pressure respirators

The expensive INPV Drinker respirators, available since 1928–1929 from the US, were life-saving not only for respiratory depression in poliomyelitis but also when tried for a variety of life-threatening conditions (as set out in Table 4.2 in Chapter 4). The alternatives to them included the **Both** brothers' effective INPV machine, with outer wooden (not steel) construction, which was hence cheaper than the Drinker. The Both was many times life-saving with its ready availability during a late-1930s polio outbreak in Australasia. Lord Nuffield's generous donations made the Both respirator available in the British Commonwealth as needed, worldwide: 1,755 in all. These included 33 to New Zealand, and the Both becoming established in suitable substitution for the Drinker respirator. Neither of these INPV machines, while fulfilling its function satisfactorily, allowed ready medical and nursing access to the subject's whole body, nor provided reasonable bodily comfort for

its subject. Both respirators inarguably delayed the development of mechanical IPPV, which, from the early 1930s, was being provided manually for thoracic surgery as needed, in the Western world (and was provided by some Scandinavian machines, which still failed to establish the IPPV mode further afield). Prof. Macintosh and Prof. Mushin at Oxford, UK, tried unsuccessfully to extend the range of INPV use postoperatively. **John Forbes** at Fairfield, Melbourne, is an unsung hero, who was committed full-time, day or night and lived onsite for years; he is perhaps overlooked now but is an Australian hero and is lauded in Chapter 4.

Albert Bower's INPV Drinker respirators at the LACH substantially reduced the rate of mortality with severe poliomyelitis after their modification by **V Ray Bennett** (the total mortality rate at Los Angeles in 1949 was even better than achieved at the Blegdam). Unfortunately, the publication of good results from Los Angeles in a medical journal (which proved too modest to be high profile), especially with the use made of the positive pressure supplementation, did not seem to have encouraged and recruited followers in other polio units (apart from Dr Bjørn Ibsen's discovery of the documentation of the Los Angeles successes). This was still cumbersome machinery compared with the later, outside-the-body IPPV machines developed quickly in Europe after the lessons from the Copenhagen crisis. But Ray Bennett's great technical expertise and multiple respiratory inventions contributed to the American success and certainly helped reduce mortality where used. Bennett was markedly inventive and his quality respiratory and other products were widespread in the US, not only in medicine but also in aviation.

Carl Clemmesen, with a longstanding dedication to improving outcomes, showed foresight in housing together victims of certain poisonings or intoxications, usually barbiturate, for supervision and standardised treatment in 1949. Despite his commitment, in the face of what proved to be an overlong dependence on stimulating agents, poorer outcomes ensued with barbiturate poisoning than with morphine poisoning, which was treated with supportive m-IPPV if necessary for apnoea along with the adequate employment of nalorphine antidote.

A brief consideration of other Scandinavian units at that time, apart from the achievements of anaesthesiologist **Eric Nilsson** at Lund, show that the Copenhagen treatment experience with intoxications was not comparable with Nilsson's successes with the same problem. One is still left admiring Carl Clemmesen's valiant efforts. He may be remembered, but unfortunately, his colleagues **Aage Kirkegaard** and Eric Nilsson have been too overlooked for their positive successes in this field.

The Copenhagen poliomyelitis epidemic, 1952–1953

Chapters 7 and 8 relate to the severe epidemic of poliomyelitis at Copenhagen in 1952–1953 and the subsequent emergence of a dedicated specialty, while Chapter 9, deriving from my obituary of Ibsen, is a repetitive summary that honours his work.

This period has proved a fertile field for investigation because of personalities, misconceptions, errors and contradictions, which have taken years of unravelling in Chapter 8. The main challenge has been that the formal book on the epidemic under Lassen's editorship has contributions from multiple authors, and it has not been ensured that dates and numbers in successive chapters match. There is disparity revealed for vital aspects such as the start and end date of the epidemic; the number of patients who were admitted,

died, received a tracheotomy and received IPPV; the relationship of complications listed separately to the cause of death; the number dying from central nervous system destruction and from solely respiratory deficiency. Nevertheless, statistical reporting has been available to quote from and compare when needed.

Denmark's 1952–1953 epidemic was widespread, powerful and devastating for a small country. About equal proportions of victims were in Copenhagen and in the rest of the country. Those with the disease who were 'seriously ill' with life-threatening complications were gathered into a localised area of the Blegdam Hospital for close attention. The devastation is characterised by 27 of this group dying during 7 July – 25 August (at other times, 24 July – 27 August is quoted for the same figures). The essential feature for this monograph is that a new method of ventilatory treatment was provided for affected patients at the suggestion of Dr Bjørn Ibsen (fostered by Bjørneboe's recommendation of Ibsen to Lassen) contrary to beliefs of the hospital's clinicians. There was an overall reduction in the mortality rate from 87% (for 30 patients up to 25 August 1952) to 37% (after that date until 2 March 1953).

Ibsen's transformation of treatment, and the increased survival therefrom, represent a major medical breakthrough. Credit for the method of treatment, known already in the anaesthetic world (and acquired by Ibsen during his year at HK Beecher's department at the Massachusetts General Hospital, Boston, in 1949) but not otherwise suggested in Copenhagen by any other anaesthetist, must go to the perspicacity of both Ibsen and Bjørneboe. Ibsen worked at the Blegdam during the epidemic from 27 August, but the details of his continuing working time are largely unknown although he was a known recipient of municipal earnings.

The initial demonstration day was compared with the day of William TG Morton's demonstration of anaesthesia for surgery at Boston on 16 October 1846. That launched anaesthesia as a (hospital) specialty, and Ibsen set the wheels in motion for the foundation of ICM. The medical system at the Blegdam was hierarchical, as well defined later by Preben Berthelsen, the Copenhagen anaesthetist. Later, in 1953, Lassen refused to support Ibsen for an academic posting at the Rigshospital, and Ibsen stayed in Copenhagen (his good fortune, as it happened) but went to the Kommunehospital from 1 April 1953, where he was able to convert his anaesthesia/surgery recovery room (of 10 beds) into the first of the modern style *general* ICUs, with himself as independent chief. This inspired the formation of ICUs in other countries, which were initially alarmed at the prospect of a polio epidemic akin to that of Denmark.

Ibsen's intensive care credentials became further apparent from his report on his new ICU (historically, the first paper from a formal ICU) in 1958. Thus, he earned the appellation (as in John Zorab's splendidly detailed short biography of Ibsen in *The Resuscitation Greats*), of 'the father of intensive therapy as we know it today'. Intensive care activity started with units founded worldwide (including at Auckland in 1959), following Ibsen's lead.

A viewpoint from this century

ICM/CCM practice and units have progressively flourished worldwide since the 1950s but differ today from those founded in the 1950s. Initially, treatment for preserving vital functions was based on basic principles and attempts to distinguish between effective and ineffective treatments through trial and error. The personal experience of ICU directors and their dedicated staff contributed to general pooled knowledge. Following the formal establishment of colleges offering dedicated training and certification in ICM (thus, for instance, a six-year postgraduate course in Australasia), clinicians fully trained in the specialty provide treatment, and their research and knowledge is now recorded in dedicated journals.

Nowadays, research into the validity of treatment processes is conducted with more general participation by units in carefully planned, randomised, multi-centred, numerically large, clinical trials (with thousands of participants enrolled in accordance with strict criteria); the results are statistically evaluated and correct answers are sought for treatments when current practices are uncertain or legitimately questioned.

We now stand on the shoulders of our pioneers and value and honour the contributions they made in having started treatments with results unknown or unproven. The work of self-sacrificing pioneers, such as Ibsen, O'Dwyer and Fell, among many, warrants being held in the highest esteem. However, **the development of this speciality is far from over.** Even today, there are many areas where patients still die through a lack of knowledge, effective treatments and appropriate technology. By examining the past, this monograph aims to emphasise such a viewpoint.

Cautionary note

Caution is still advisable. All the sequential events described herein are written with due diligence (as far as that can be established), quoting dates that are taken to be accurate. However, some noteworthy principals and commentators who were deeply involved and are quoted have written about significant happenings or facts on occasions for which their date is a day or more incorrect (this especially applies to Lassen and his 1956 book). Readers are expected to trust that I have noted and compensated for such inaccuracies, wherever detected. My apologies for any residual unrecognised errors of historical fact that remain undetected.

Ron Trubuhovich

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Notes:

- References with an asterisk, thus**:***, are not accessible through PubMed when listed on it; those with a double asterisk, thus,**, also do not have an English summary;
- Secondary titles of papers in brackets [..] are the English translation of European languages.
- The LA Reynolds & EM Tansey 2011 conference booklet covering the *History of British intensive care, c.1950-c.20O0* (in 156 pages), does provide fair coverage for that era in Britain, while fortunately for readers of texts who can read them only if in English, some significant European papers may also be found translated into the English language online, within the (US) National Library of Medicine's PubMed. 'Foreign' authors are thereby generally made more accessible to such readers. One example is the paper originally offered by T Kucmin in the Polish language, the title of which is translated to English (see the next note, for English-only readers). PubMed also identifies by name V Gajic, whose 2011 article is in the Serbian language; but that paper's name becomes recognisably named to English-only readers at PubMed. Apart from these two papers, other European writers here on ICM are recognisable (i.e. the names of the two aforementioned authors, Kucmin and Gajic, do not in any way complete the list possible). PubMed is of immense help.
- See online for Kucmin T, Plowaś-Goral M, Nogalski A., translated from Polish, into English, together with the online title, translated as 'A brief history of resuscitation – the influence of previous experience on modern techniques and methods', available from PubMed.