An investigation of the development of intensive care of adults in England and Wales

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Background

This investigation was undertaken for an MD in the University of Liverpool, England. I was a medical clinician. It may be useful to explain why I applied to enrol as a post-graduate student and to present my research as a Thesis for the degree of Doctor of Medicine. I am a retired Consultant in Anaesthesia and Intensive Care Medicine, and the MD would not in any way enhance my career. So three questions arise: Why an MD (or PhD), why now, and why me?

Why an MD? Although I was interested in the history of my specialty and was experienced in research, I knew I was not competent to conduct research into medical history, or indeed any other sort of history. Although a doctorate is not a taught degree, my supervisors guided me towards some degree of expertise in historical research. My experience as a post-graduate student has convinced me that those who write medical history should endeavour to learn to research and write in a way that will be respected by other historians.

Why now? It was obvious that the history of intensive care in England and Wales needed to be conducted now because many of the pioneers in the specialty were of somewhat advanced age and it was important that their experience should be recorded.

Why me? I had, through two lectures and two papers, tried to persuade others to undertake the task, but apart from one paper and one excellent and informative chapter in a book on the influence of anaesthesia on medicine, none had done so. No definitive work on the subject had emerged. I realised that if a history needed to be written now, I would have to write it or the opportunity to record the testimony of the pioneers would be lost.

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Acceptance of the research project was not straightforward. After submitting my proposal for research I was asked to justify the acceptance of a historical investigation as research appropriate for the degree of Doctor of Medicine, which hitherto had always been awarded for research related to clinical medicine. I argued that knowledge of the history of medicine was a necessary component of the education of a clinician. I used the example of the history of the decline and resurgence of tuberculosis. Contemporary practitioners of respiratory medicine would be poorly equipped to participate in the world-wide control of this disease if they were unaware of the difficulties with which their predecessors had had to contend. My argument was accepted but supervisors had to be found for my study. They needed to be competent historians so that they could direct my research but they also needed to know what was demanded by the Faculty (now School) of Medicine before they could award the Faculty’s highest degree. It was fortunate that Dr Sally Sheard held the post of Senior Lecturer in the History of Medicine and that Professor Geoffrey Gill, in the School of Tropical Medicine, had been awarded his MD for a study of the long-term health effects of Far East imprisonment in World War II.

At the start of what were to become our monthly meetings I was directed by my supervisors to books about how to research history. Oral history was to be an important part of my research. I had to learn how to interview witnesses and to record and transcribe the interviews. I gained instruction in interviewing technique at an Oral History Society course in Sheffield and at the BBC North West Sound Archive at Clitheroe Castle.

The other main constituent of my research was to research written sources. People often say that that must be easy because now we have the internet. The internet is of course enormously useful: one can find databases, newspapers, Hansard, Government Bills etc. But the internet has severe limitations. Not all the material needed is to be found in medical databases (such as MEDLINE or PubMed) or in commercial search engines. Moreover searching the databases to retrieve the material that it contains requires a certain expertise if the search is to find all the relevant articles (sensitivity) and reject irrelevant ones (specificity). Searches of Medline have been shown to suffer from surprisingly large deficiencies in both sensitivity and specificity. Searches by less experienced trainees and medical staff at a university medical centre retrieved only 55% of the articles retrieved by reference librarians (so they were not sensitive) and retrieved 55% more irrelevant articles (so they were not specific). Searches conducted by skilled librarians detected only half the eligible citations. Reasons for these apparent deficiencies lie in the fact that many relevant articles would have been published before the databases existed.

Retrograde addition of published material is labour intensive and expensive but is still progressing. When this study started in 2008 it was not possible to find any articles about intensive care which had been published before 1966. It is now possible to search back to the 1940s. Another source of failure to find relevant articles is the relatively recent use of what would now be obvious search terms, such as intensive therapy or intensive care. Where such terms were used their meaning was not what would attach to them in modern usage. Intensive therapy of tuberculosis for example was used to denote simply very high dosage of anti-tubercular drugs.

Other methods of retrieving literature must be used to supplement electronic searching. Ancestry searching is used to describe an iterative process; one consults the sources cited in papers already found. These earlier sources in their turn will cite papers of an earlier generation. This method is particularly useful for finding articles published before, say, 1946 or in obscure journals or archived material. Articles like that are unlikely to be found in a database. Another method of finding material is browsing. One manually searches journals likely to contain papers on the subject of study and proceeds to progressively earlier editions until the first appearance of an article on the subject is found.

Other University facilities such as courses, libraries and access to sources of material which would otherwise be only available on expensive subscription to journals are additional reasons for registering as a post-graduate student.

An aspect of historical writing with which clinicians writing on medical history are probably not familiar is historiography. Historiography largely consists of analysis of what others have written on the subject and comparison with ones own findings. ‘On the subject’ is interpreted very widely: In the context of this study, historiography did not refer exclusively to writing about the development of intensive care in England and Wales. It would equally refer to comparison of this development with the development of intensive care in other countries, or to how the development of the specialty of intensive care differs from the development of other specialties, e.g. orthodontics, sports medicine, or genetics, or to how specialties evolved – practitioners of physic, apothecaries, barber surgeons etc.

Having described the way is which a medical clinician acquired some credibility as an investigator of history, an outline of the results of that investigation will be presented: how did the specialty of adult intensive care in England and Wales evolve?

**Beginnings of modern intensive care**

Writers on the development of modern intensive care have taken the view that intensive care really started in Copenhagen in 1952 when the Danish anaesthetist Dr. Bjørn Ibsen (1915-2007) used a new technique to treat
respiratory failure during an epidemic of poliomyelitis. The use of this new technique became the basis of early modern intensive care. However I discovered that: 1. The story had started much earlier, and: 2. Copenhagen was not as immediately influential as has been assumed.

The importance of the 1952-3 Copenhagen epidemic cannot be understood without knowing something about polio and how it was treated before 1952. A case of what appears to have been polio was described in 1699 but for many years after that time the disease was endemic (there were just a few sporadic cases). However large epidemics of polio started in the USA and Europe in the late 19th century. In 1916 in the outbreak in the City and State of New York over 13,223 persons caught the disease and over 25 percent died. Epidemics did occur in Great Britain. They were, however, minute in comparison to those in the United States and some in mainland Europe.

Polio is a febrile illness affecting mainly children (hence its popular name ‘infantile paralysis’). In later epidemics it occurred increasingly in young adults. A mild attack might, in the absence of an epidemic, be thought to be just influenza. There would be nausea, drowsiness, and a stiff painful back or neck. But in more severe cases, perhaps 15-30% of the total, there will be paralysis: most commonly the paralysis involves nerves supplying limb musculature. This may be transient, improving after a couple of weeks, or lifelong leading to permanent disability. Paralysis of the phrenic and/or intercostal nerves leads to respiratory difficulty or paralysis which, until the invention of mechanical respirators (later called ventilators), was fatal. Paralysis of the nerves to the throat (bulbar palsy) will make swallowing impossible. Paralysis of the vasomotor centre may cause vasomotor collapse.

The first really practical respirator was invented by Philip Drinker (1894-1972), an Assistant Professor of Industrial Hygiene at Harvard, in 1928. Patients whose respiratory muscles were paralysed by polio were put into a wooden or iron cabinet. The head is outside the cabinet on a pillow. The device is called a cabinet respirator, colloquially known as an ‘iron lung’ (Figure 1). A pump sucks air out of the cabinet 10-40 times per minute and this causes the chest of the paralysed patient to expand and air is sucked into the lungs through the mouth, which is outside the cabinet. The air is then allowed back into the cabinet and the chest shrinks again and the air is expelled from the chest. For an adult patient this cycle is repeated perhaps 20 times per minute to mimic normal breathing. Iron lungs kept thousands of people alive (some for more than fifty years) and this was early intensive care.

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An Australian entrepreneur, Edward (Ted) Both (1908-1987) demonstrated a wooden version of the Iron Lung in London (Figure 2). Lord Nuffield, a millionaire motor manufacturer, offered to supply Both Respirators to any hospital in the British Commonwealth which wanted one. By the beginning of the Second World War there were about 1000 respirators in Britain and in the British Forces.

Cabinet respirators were bulky. A smaller respirator (cuirass) which encased only the thorax (Figure 3) was useful for recovering patients or those who needed to be transported by land or air. The author brought a young army doctor from Singapore back to England using only a cuirass respirator similar to the one illustrated.

Cabinet respirators saved thousands of lives of people in respiratory failure due to poliomyelitis. The majority of such patients recovered sufficient power in their respiratory muscles to be weaned off the respirator in the course of weeks or months. Some however needed the help of the respirator for all or

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part of the day. As recently as 2009, a report of the death of a patient who lived in an iron lung for over 60 years appeared in the *New York Times*.  

**Figure 2: The Both cabinet respirator**

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**Copenhagen 1952-3**

A revolution in the treatment of respiratory failure occurred in 1952. The first the world knew of it was a paper by the Infectious Diseases physician at the Blegdam Infectious Diseases Hospital in Copenhagen, Dr Henry Cai Alexander Lassen. It appeared in the *Lancet* just after New Year in 1953. He first described the first 19 weeks of the epidemic: 24 July-3 December 1952. During that period 2722 patients were admitted with polio. 866 had paralysis (32%) and of these 316 had respiratory or pharyngeal paralysis or both (1:8 of the total) In four months the number of such patients was 3 times as many as in the previous 10 years. At times they had 70 patients needing artificial respiration at once.

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15 The author gives Lassen’s forenames in full because they have been difficult to find and were given wrongly (as Hans Christian Anderson Lassen) in the lecture on which this paper is based. They are given in G. L. Wackers, ‘Modern anaesthesiological principles for bulbar polio: manual IPPR in the 1952 polio-epidemic in Copenhagen’, *Acta Anaesthesiol. Scand.*, 38 (1994), 420-431.

They had one Emerson cabinet respirator and six cuirass respirators. Lassen reported: “We were in a state of war!” In his article Lassen concentrated on the management of respiratory insufficiency. In the first month of the epidemic (14 July-26 August) 31 patients were admitted with respiratory paralysis with or without laryngeal paralysis. 27 of these patients died, 19 within three days of admission. The article continues:

…we consulted our anaesthetist colleague, Dr B Ibsen and on August 27th the first patient was treated with what was to become the method of choice for patients with impairment of swallowing and reduced ventilation - namely tracheotomy just below the larynx with insertion of a rubber cuffed tube into the trachea and manual positive pressure ventilation from a rubber bag.

Anaesthesia had only been recognised as a specialty in Denmark in 1950. It was an emerging but low status discipline. Ibsen was a free-lance anaesthetist in Copenhagen’s University Hospital (Rigshospitalet). Having been chief physician for thirteen years in Denmark’s polio centre and centre of expertise in epidemic diseases (Blegdam Hospitalet), Lassen had to overcome a certain degree of professional pride in soliciting ‘outside’ help. Why did the infectious diseases specialist consult a humble anaesthetist? What did Ibsen know that the physicians did not know?

The physicians thought the patients had renal failure. With the very limited information about acid/base balance available at that time such an interpretation was not as ludicrous as it appears to modern clinicians. The physicians dismissed the possibility of respiratory inadequacy because the
patients were not cyanosed. This was because they were breathing a high concentration of oxygen. Ibsen knew that they were dying because their lungs were damaged and could not be ventilated sufficiently to prevent build up of carbon dioxide and resultant acidosis, certainly not by a cuirass! The lungs were damaged because in patients who could not cough or swallow (those with bulbar paralysis), the secretions from the nose and mouth had trickled into the lungs and damaged them – the respirator could not adequately inflate the stiff lungs.

How did Ibsen know that the lungs were damaged and the ventilation inadequate? He knew the lungs were damaged because he had gone to the post mortems of those who died and he had seen that they had stiff, solid lungs. Ibsen also had evidence that the lungs were not being adequately ventilated: he had a prototype of an instrument called a carbovisor which measure the expired carbon dioxide concentration and was therefore able to demonstrate adequacy or inadequacy of lung function. When Ibsen used the carbovisor during anaesthesia he had noticed that when the carbovisor indicated that the anaesthetised patients were not adequately ventilated they sweated and the blood pressure rose. Ibsen observed that the dying polio patients were sweaty with a high blood pressure, which he recognised as signs of inadequate ventilation of their lungs. He concluded that he needed: 1. a better way to inflate the stiff lungs and: 2. a way to protect the lungs from damage by the secretions trickling down into the lungs from the nose and throat.

Figure 4: Anaesthetic circuit used by Ibsen to apply intermittent positive pressure respiration to paralysed polio patients.
The circuit Ibsen devised (Figure 4) was simply a system for delivering anaesthetic gas and of course oxygen for a patient to breath. It was not designed for ventilating the lungs, but it had a rubber reservoir bag from which and into which the patient breathed. Anaesthetists had in the 1930s and 1940s realised that anaesthetic agents in use at that time, particularly cyclopropane, depressed the patient’s respiration. They aided respiration by occasionally squeezing the bag, thus forcing gas and oxygen into the lungs. With increased understanding of the physiology of so-called intermittent positive pressure respiration (IPPR), later renamed intermittent positive pressure ventilation (IPPV), it was realised that IPPR could be applied safely and with advantage whenever cyclopropane was being used, so the more advanced anaesthetists were familiar with using their circuit for maintaining adequate ventilation when necessary.

Ibsen asked a surgeon to perform a tracheotomy high in the trachea and insert a cuffed tracheotomy tube and to connect the circuit to it. Ibsen was then able to inflate the patients lungs rhythmically by squeezing and releasing the bag. The cuff was inflated when the tube was in position in the trachea. It made the tube an airtight (or gas-tight) fit in the trachea thus preventing gas escaping round the tube when the bag was squeezed and ensuring that the pressure on the bag inflated the lungs, even though they might be stiff and uncompliant due to the damage caused by inhaled secretions. The cuff also prevented any further contamination of the lungs by upper airway secretions. This simple apparatus was therefore able to inflate the lungs more efficiently than a cabinet respirator, the equipment was readily available, and dozens of patients could be treated at once – all that was needed was a considerable number of bag squeezers. These were recruited from medical students and dental students who had to squeeze the bag continuously in 8 hour shifts for which they were paid 30s (now £60).

Between 26 August and 6 November 1952 Ibsen, his anaesthetic colleagues and his student assistants, treated 172 cases by tracheotomy and bag ventilation and 77 died. The mortality rate of 42% in this group of patients had, before Ibsen’s innovatory treatment, been 85-90%. By the last month of the epidemic the mortality rate was down to 11%.

It has to be said, but it is not often said, that American physicians still using ‘iron lungs’ achieved results as good as Ibsen’s but they had plenty of more advanced cabinet respirators with positive pressure attachments and which could be used with tracheotomies. Ibsen only had one cabinet respirator and sometimes needed to treat seventy patients at a time.

After Copenhagen

Ibsen had shown that anaesthetic techniques could efficiently save patients in respiratory failure, and later commentators have sometimes assumed that anaesthetists immediately followed his example, but at least in England and Wales very few anaesthetists were ready or able to undertake Intensive Care.
Respiratory failure continued to be treated by the people who had always treated it – Infectious Disease Physicians.

The physicians often used improved cabinet respirators, or they sometimes used IPPR with or without an anaesthetist. They got good results – they improved their ventilators so that they could use a tracheostomy with a cuffed tube to protect the lungs from secretions trickling down in patients who could not swallow.

After a slow take-up of the injected Salk killed vaccine for polio in 1958, a publicity campaign and the acceptance by the Ministry of Health in 1961 that the Sabin live vaccine, which could be administered on a sugar lump, was safe led to a rush for immunisation and the incidence of the disease dropped sharply. Although occasional cases continued to occur in Britain, the effect of polio on the development of intensive care in England and Wales was negligible after 1963 (Figure 5).

![Figure 5: Notifications of polio between 1956 and 1970](http://www.hpa.org.uk/Topics/InfectiousDiseases/InfectionsAZ/Polio/EpidemiologicalData/polioAccutePoliomyelitisAnnualNotifDeathsEW/)

So the infectious diseases physicians and their respiratory units gradually left the Intensive Care scene and anaesthetists gradually took up the challenge.

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Why had they not done so immediately after 1952? Ibsen was an anaesthetist and he had shown the way. One could go further - equipment almost identical to Ibsen’s had been invented in 1913. Why had the anaesthetists not used it to treat respiratory inadequacy then? And what had happened to make anaesthetists willing and able to undertake intensive care by the mid-1960s?

What had happened was the professionalisation of anaesthesia. In the 19th century there were only a handful of experts in the country. Anaesthetics were given by anyone who might be available; students, porters, and in a case widely reported in the 1960s, a Blackpool dentist’s grandmother! Unfortunately no reference can be found.

After the 1st World War, expertise became recognised. A large number of operations were needed for soldiers needing thoracic surgery for chest injuries and the anaesthetist had to deal with the problem of pneumothorax when the chest was opened. Anaesthesia for surgery for facial injuries also presented a problem because masks could not be applied to the face during the operation and the anaesthetic needed to be given through an endotracheal tube which few anaesthetists were capable of inserting. A few experts emerged who developed the necessary skills and they wrote papers describing their methods, but most general anaesthetics were still given by general practitioners. Anaesthesia was a ‘Cinderella specialty’.

A seminal event was the founding in 1908 by the Royal Society of Medicine of its Section of Anaesthesia. Learned articles on anaesthesia appeared regularly in the Society’s Proceedings and internationally recognised experts and teachers emerged. For example in November 1913 H.E.G. Boyle and G. E. Gask demonstrated to the Section an apparatus for the insufflation of ether into the trachea similar to the one described by Charles Elsberg in New York the same year.

The Association of Anaesthetists of Great Britain and Ireland (AAGBI) was founded in 1932 because by reason of the terms of the Royal Society of Medicine’s Royal Charter the Section of Anaesthesia was not permitted to concern itself with organisation of the nascent specialty or payment or conditions of service. The Association’s first and most far-reaching achievement was the establishment of the Diploma in Anaesthetics (DA). The first examination was held on 8 November 1935. It was a two-part examination.

Then in 1948 Lord Webb Johnson, President of the Royal College of Surgeons, told the AAGBI that to be Consultants in the new NHS anaesthetists would need an exam comparable to the FRCS or MRCP. On his advice a Faculty of Anaesthetists was founded in the Royal College of Surgeons, and the exam for Fellowship of the Faculty was introduced.\(^\text{22, 23}\) Anaesthetists were gradually recognised as consultants of rank equal to that of surgeons and physicians. Anaesthetists could then look after their own patients in ICUs. However in the years following the establishment of the National Health Service there was a general shortage of consultants and this was particularly acute in the newer specialties such as anaesthetics. Following a report in 1961 by a Committee chaired by Sir Harry Platt the number of consultant anaesthetists increased, and GP anaesthetists became uncommon in major hospitals.\(^\text{24}\)

Several further developments had qualified this new generation of consultant anaesthetists to participate in intensive care:

- Modern anaesthesia in the 1950s demanded artificial ventilation of the lungs;
- Cyclopropane was introduced in 1934.\(^\text{25}\) It was a good anaesthetic but a severe respiratory depressant. In 1940 Guedel wrote that IPPR was essential when cyclopropane was being used. Moreover he said that, contrary to earlier opinion, anaesthetists should be competent to pass endotracheal tubes and to apply IPPR (which he called ‘passive respiration’).\(^\text{26}\)
- In 1952 TC Gray wrote that IPPR was mandatory with curare.\(^\text{27}\)
- Anaesthetists had to treat post-operative respiratory failure. Curare occasionally could not be reversed and the patient could not breathe after the operation so ventilation was necessary until the curare wore off.
- Automatic lung ventilators became more commonly available. Before they had ventilators in the operating theatre, anaesthetists had to ventilate the patient by squeezing the anaesthetic reservoir bag for hours and hours.
- Cardiac surgical patients needed post-op intensive care including IPPV and individual nursing.\(^\text{28}\)
- Treatment of barbiturate poisoning changed from respiratory stimulants to IPPV.\(^\text{29}\)

\(^{23}\) Anon., ‘Association News’, *Anaesthesia*, 3 (1948), 129.
\(^{26}\) A.E. Guedel, ‘Cyclopropane anesthesia’, *Anesthesiology*, 1 (1940), 13-25.
\(^{29}\) F. Plum, A.G. Swanson, ‘Barbiturate poisoning treated by physiological methods; with observations of effects of beta-beta-methylethyl-glutarimide and electrical stimulation’, *JAMA*, 163 (1957), 827-35.
• IPPV was gradually found to be successful in treating all recoverable forms of respiratory failure, such as asthma, trauma, and poisoning.30

Hence, there was an increasing involvement of anaesthetists, there were more ICUs, intensive care medicine was treating an ever widening range of illnesses. But in 1995 a boy of 10 died for want of an ICU bed. There was media uproar and an investigation by the Audit Commission.31 The Commission came to the conclusion that ‘the development of intensive care has been unplanned and haphazard and has largely relied on the local interest of local clinicians to develop it.’ £142.4 million was made available for the enhancement of adult critical care services. This was the first planned investment in intensive care since its inception.

A document called Comprehensive Critical Care published by the Department of Health in 2000 contained 29 recommendations for the modernisation of critical care services.32 Further development of the specialty of Intensive Care medicine hinged on the development of specialist training and accreditation. Over a period of almost 50 years what had been an additional interest of a small minority of anaesthetists became an accredited specialty in England and Wales and in the EU. Time prevents a fuller description of this process, but organisation of intensive care into a specialty was stimulated when Dr John Nunn, as he came to the end of his term as Dean of the Faculty of Anaesthetists of the Royal College of Surgeons on 17 March 1982, said that the Faculty had been concerned with the matter of training in intensive therapy. The fact that this branch of medicine is multidisciplinary contributes to its strength but it also means that intensive therapy units tend to lie outside the competence of any single Faculty or College acting on its own to inspect and recognise units for training or, indeed, to formulate criteria for recognition for training. The Faculty had recently instigated a debate on this problem at the Conference of Medical Royal Colleges and their Faculties. A working party had been formed and it comprised representatives of the Faculty of Anaesthetists, the Surgical Colleges, the Colleges of Physicians and the Intensive Care Society. The first meeting would take place that spring.33

The first report of the group (by then called the Liaison Group) was accepted by the UK Conference of Royal Colleges and Faculties in January 1985. They formed a Joint Accreditation Committee for Training in Intensive Therapy in 1988. After lengthy negotiation the unprecedented collaboration of seven Royal Colleges resulted in the formation of The Faculty of Intensive Care

Medicine in the Royal College of Anaesthetists on 7 May 2010. It can surely be said that with the formation of the Faculty, intensive care has come of age.