The 1952 Polio Epidemic and the Birth of the Modern ICU and Mechanical Ventilation

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INTRODUCTION: **The modern hospital ICU is a more recent development than most current practitioners realize. As with most progress in patient care, the development of the specialized ICU and the care of the critically ill, has been incremental. One success builds for the next. Improved knowledge of physiology, insight into disease processes, and development of specialized equipment and medical disciplines have created many ICU stories. Here we present one of the earliest. Presented with a huge public health crisis, physicians rethought respiratory physiology, and devised new clinical models that saved many lives. This abstract is written to honor their gift to medical care, one that has continued to evolve, and that continues to save lives every day.**

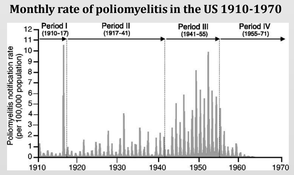
ABSTRACT: **Infectious paralytic poliomyelitis has likely been a scourge for thousands of years. Infection is limited to humans.**

This Egyptian drawing from
The 14th Century BC shows
a priest with an atrophied right leg.
It suggests polio as a cause.

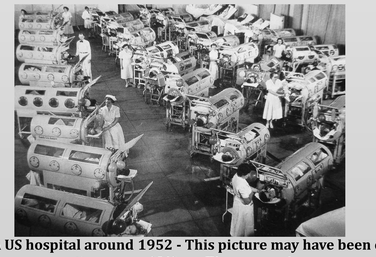
Picture from Wikipedia

Through the, 19th century widespread small outbreaks of *infantile paralysis* occurred in the US. The twentieth century brought the beginning of large epidemics of polio that continued until vaccination started in 1955.

Monthly rate of poliomyelitis in the US 1910-1970



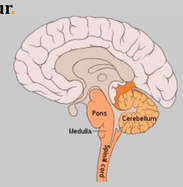
Trevelyan, Ann Assoc Am Geography, 2006



A US hospital around 1952 - This picture may have been on the cover of Life or Time magazine

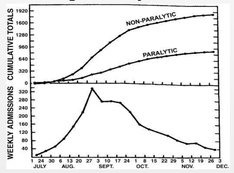
Poliomyelitis is highly contagious and caused by a virus of the Picornaviridae family. The clinical features range from mild constitutional symptoms of respiratory illness, gastroenteritis, fever, and malaise to severe forms of paralysis. Spinal polio is the most common form of paralytic polio. It occurs with spinal cord invasion of the anterior horn motor neuron cells. Bulbar polio occurs with involvement of the upper cervical cord and bulbar region of the brain stem. Loss of the IX, X, and XI cranial nerves interferes with neck movement, swallowing, and clearing of mucus from the pharynx and lungs. Collection of mucus and fluids in the upper airway can lead to suffocation. Involvement of the V and VII cranial nerves can cause loss of upper airway sensation and facial muscle paralysis. Dysfunction of the respiratory centers in the medulla and pons can alter depth and regularity of the respiratory pattern. Respiratory arrest, pulmonary edema and shock can occur.

Location and anatomy of the bulbar region



In August of 1952 an epidemic of poliomyelitis began in Copenhagen,
Denmark. In the next three months over 3000 cases would be seen at Blegdam Hospital (Blegdamshospitalet), the city's infectious disease center. Most of the patients would be children. Of the total cases about 1,250 involved paralytic symptoms, and 345 were bulbar involvement cases—affecting the respiratory and swallowing muscles.

The polio epidemic rapidly peaked in August



Graph from Blegdam Hospital, Copenhagen

1952

Patients were presenting with varying degrees of spinal cord and bulbar paralysis, fever, encephalitis with altered consciousness, increased airway secretions, and irregular respiratory patterns. This myriad of signs complicated the recognition of the life-threatening situation of bulbar polio involvement. During the first weeks of the epidemic 27 of 31 patients with clear bulbar involvement died. Many deaths wee occurring at home before transport to the hospital were, and likely most had bulbar polio. Bulbar patients were presenting with shallow breathing, choking on their own secretions, inability to swallow, and often in shock. The Hospital's chief physician, Henry Alexander Lassen, realized the obvious; the proper management of these respiratory problems was not known. The Hospital had one Emerson tank ventilator (an "iron lung") and six cuirass ventilators. The cuirass ventilators were jackets that fitted around the chest. By alternating positive and negative pressure they inflated and passively deflated the chest—the same mechanism as the Emerson. These devices did provide ventilatory support, but both were inadequate for the complete respiratory failure of the bulbar polio victims. All the seriously ill patients treated with this negative pressure ventilatory support were dying. The only available lab test we would recognize in a present-day acid-base evaluation was total carbon dioxide concentration of blood, closely equivalent the bicarbonate level. This value was markedly elevated and initially interpreted as an alkalosis of unknown cause. Only much later when a pH meter became available could the pH and total CO2 be used on a nomogram to extrapolate the pCO2—which was recorded as high as 150 in some cases.

Dr. Lassen called a meeting of the hospital staff on August 25, 1952. Notably present were Dr. Paul Astrup, head of the hospital laboratory, and anesthesiologist Dr. Bjorn Ibsen. Dr. Ibsen had earlier that year been involved in the care of a child with tetanus. Paralysis, tracheotomy, then manual ventilation led to successful recovery. He also recognized that the mysterious alkalosis was probable metabolic compensation for CO2 retention from hypoventilation.

Dr. Ibsen proposed tracheotomy and positive pressure ventilation using manual bag setup. The idea was accepted. There was skepticism, but also desperation.

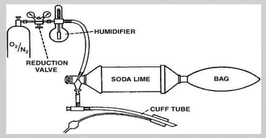
The description of the first case follows:

*

"The patient's name was Vivi. She was 12 years old dying of paralytic polio. All her limbs were paralyzed, she was cyanotic, and gasping for breath. 'Everyone expected her to die.' But Ibsen, an anesthesiologist, thought he could save her. He proposed a radical departure from the standard treatment by suggesting that polio patients could be treated in the same way as he had managed curarized patients during surgery. He said, 'In the OR we intubate and ventilate them and they do OK. We could do exactly the same with polio patients.' A surgical tracheostomy was performed and a cuffed endotracheal tube inserted. But when Ibsen tried to ventilate, he could not push air into her lungs. At that moment, according to legend, all the other doctors left the room. Ibsen persisted, administered thiopentone, and then was able to ventilate without difficulty. Vivi regained consciousness, and her temperature fell."

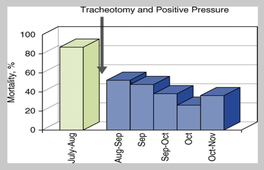
She survived.

(Pincock, Lancet, 2007)



West, J Applied Physiology, 2005

The success of tracheotomy and manual bag ventilation was dramatic. Mortality in this group of patients fell from 90% to 40%. But at that time there were no mechanical ventilators. Continuous bag ventilation by human hands was the only possible option. A trained person had to be at the bedside continuously ventilating the patient. The solution was to recruit and train medical and dental students to work in rotating shifts. A special ward was prepared for the rising number requiring ventilation; it became a *single unit* devoted exclusively to care for polio patients with respiratory compromise. <u>Over the ensuing months about 1500 students provided about 625,000 hours of bedside ventilation.</u> Positive pressure ventilation was now being provided on a large scale. This work continued for the remainder of 1952 until the epidemic abated. Hundreds of lives were saved.



The following year Dr. Ibsen took a job Kommunehospitalet in Copenhagen. He was asked to supervise surgery patients in the recovery room. He did and turned the ward into a unit where all critically ill patients would receive care. He called it an intensive therapy unit; it was the world's first ICU.

Picture from
Berthelsen,
Acta Anaesthesiology Scandinavia,
2003

Bjorn Aage Ibsen
1915-2007

Historical review has learned that the first use of intermittent positive pressure ventilation (IPPV) for polio victims was really at Los Angeles County Hospital/USC in 1948-49. Dr. Albert G. Bower, head of the communicable disease service, had recognized the pattern of respiratory death from bulbar polio. He had made the connection of severe alkalosis being the compensation for severe respiratory acidosis. He has also learned the shortcoming of the Drinker-Collins respirator ("iron-lung) and its inability to adequately ventilate serious respiratory failure. His experience was also a high mortality—79%. In 1948 he began to work with bioengineer Ray Bennett (yes, that of the Bennett Respirator series). Bennett developed a mechanical "attachment" to the Drinker respiratory to provide IPPV. But really it was separate apparatus, thought initially it ran off the electrical motor of the negative pressure "Drinker" respirator. An inspiration could be triggered by pressure changes, a bellows could provide a measured volume, and the inspiratory pressure monitored. Either facemask or tracheotomy could be used. Dr. Bower later commented that tracheotomy should have been used more frequently. Since the Bennett device did not run continuously, the patient was left in the Drinker, so oddly both were working at the same time. Obviously there were synchronization problems. But the mortality dropped to 20%, just as it would a few years later in Copenhagen.

The initial LA County experience was published, but only in a regional journal and was never widely known. Dr. Lassen in Copenhagen may have read the reports, but if so did not realize their importance. It was Dr. Ibsen who designed the push forward.

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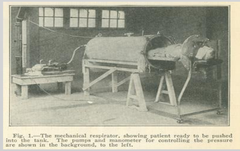

Fig. 1.—The mechanical respirator, showing patient ready to be pushed into the tank. The pump and compressor for controlling the pressures are shown in the background, to the left.

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