

From the University Children's Hospital Zurich

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Work performed with the direction of Professor H. Zellweger

The Effectiveness of Iron Lung Ventilation in Poliomyelitis

Inaugural Dissertation

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It is axiomatic that, with rare exceptions, the only poliomyelitis cases that die are those that develop pathologic respiration. (Bower)

The introduction of the Iron Lung in the treatment of poliomyelitis had originally awakened high expectations among both medical practitioners and the lay public, which unfortunately were not all fulfilled. The work undertaken here seeks to draw together our many years of experience with the use of the Iron Lung. A brief analysis of the relevant circumstances that the use the Iron Lung demands follows, including the various mechanisms of artificial ventilation, the therapeutic results and finally, a summary of our current treatments.

An immediately life-threatening situation arises in the progression of Poliomyelitis whenever respiratory compromise is involved, resulting in decreased oxygenation leading to hypoxaemia, hypercapnia and inevitable respiratory acidosis. This is the case when the following centres are affected:

Spinal Respiratory Neuromuscular Impairment

1. Anterior horn of C3-4, and T1-12.

Bulbar Progression

2. Nucleus of the Glossopharyngeal, Vagus and Hypoglossal N.
3. Ventrolateral parts of the substantia reticularis in the medulla oblongata (the Respiratory centre)
4. Ventromedial parts of the substantia reticularis in the medulla oblongata (the Circulation centre)

The numbers of cases with hypoxia varies considerably between the specific epidemics. In the Childrens’ Hospital Zurich the numbers requiring Iron Lung ventilation from 1946 are as follows:

Table 1.

	Total Cases	Cases Requiring IL Ventilation	Percentage
1946	136	5*	3.7%
1947	110	6	5.5%
1948	65	7	10.8%
1949	72	5	6.9%

*We obtained the Iron Lung at the end of 1946, so that from the 5 patients with respiratory failure, only 1 received IL treatment.

Numbers for comparison with other Hospitals are difficult to obtain, however we suspect there would be clear differences if the frequency of bulbar progression in the various institutions were compared with each other. Certainly not all cases with bulbar involvement led to hypoxia, unless there was also spinal respiratory neuromuscular involvement. In the Childrens’ Hospital Zurich in recent years cases with bulbar involvement have accounted for about 10% of all Poliomyelitis cases. In epidemiology from Minnesota during 1946 23% of cases under the age of 16 y.o. and 36% of adult cases had bulbar involvement. Contrasting these numbers, however, has only relative meaning, as it is those cases which also have Respiratory neuromuscular involvement which make up the largest contingent of those with life-threatening hypoxia. We made the following observations (Table 2.)

Table 2.

Frequency of specific forms of progression requiring Iron Lung ventilation				
Progression Form	Number of Cases	Survivors	Died within 1m	Died later
Spinal Neuromuscular	9	6	1	2
Weakness	1	1		
Swallowing impairment	2	2		
Spinal + Swallowing impairment	4	1	3	
Spinal + Respiratory centre + Circulatorion centre	3	0	3	0
Total	19	10	4	2

Spinal neuromuscular paralysis is due to damage of Anterior Horn nerve cells which innervate the diaphragm and intercostal muscles. Isolated diaphragmatic impairment results in paradoxical respiration, as the diaphragm is sucked upward by the movement of the thorax in inspiration and is drawn downwards in expiration. The diagnosis can be clearly confirmed by use of dynamic radiology. Due to the diaphragmatic impairment, those parts of the lung contiguous with the diaphragm suffer from limited ventilation and become atelectatic. Conversely, when the intercostal muscles are impaired and the diaphragm remains intact, the lack of thoracic movement but deeper diaphragmatic excursion results in the upper and paravertebral parts of the lung suffering from hypoventilation. Both cases cause so-called “swinging” or paradoxical ventilation.

If both intercostal muscles and diaphragm are impaired, then no, or at best very limited, respiratory excursion is visible. Before these visible manifestations are recognised however, the patient suffers severe shortness of breath. An absolutely certain early symptom is the inability of the patient to hold their breath for any length of time. One can also ask the patient to count in one breath, and note the number that is reached before a new breath is needed. Children with respiratory muscle impairment can only reach very few numbers. With even greater injury children are no longer able to call out with a loud voice. Within a short space of time obvious cyanosis develops, there is flaring of the nostrils and mouth-breathing as well as use of the accessory muscles of respiration of the throat and neck. The child becomes more cyanotic, distressed and restless, or

remains extremely quiet and, with a terrified expression, avoids even the smallest movement, in order to maintain their oxygen level. Finally lethal asphyxia results. The deadly outcome is hastened when pulmonary complications such as pneumonia, atelectasis or bronchitis appear. The child is then unable to cough up mucus or bronchial secretions, and has little or no respiratory reserve to use. Occasionally a severe weakness can result in a sudden respiratory collapse, without these intermediate stages. (see Case 10).

2. Cranial Nerve Impairment: It is important to distinguish between anterior and posterior cranial nerve impairment. Involvement of the Oculomotor, Trochlear, Abducens and Facial nerves result in no life threatening consequences. The result of CN 9-12 impairment, however, in particular of the Glossopharyngeus and Vagus nerves, is severe. These nerves were affected less frequently in our observations than in the studies carried out by Baker. Symptoms include soft palate impairment, problems with swallowing and vocal cord paralysis (due to vocal cord abductor involvement), as well as tongue function injury. The result of this latter means that the tongue can fall backwards and obstruct the pharynx; vocal cord abductor paralysis causes closure of the glottis. American authors note in particular this vocal cord paralysis which can lead to complete closure of the glottis, and though this has been observed far more rarely by us we can confirm cases of laryngospasm occurring during the progress of Poliomyelitis. Swallowing difficulties first manifest as a nasal speech, due to progressive soft palate paralysis, and eventually becomes unclear, without articulation, and gurgly. On examination the patient drools and the mouth and pharynx is full of saliva that cannot be swallowed. Likewise the salivary glands can produce almost twice as much saliva per day and become larger, ultimately contributing to the obstruction of the airway. The patient is able to take only small breaths which results in worsening hypoxia. Every breath exacerbates the risk of aspiration which can trigger severe coughing and worsen the hypoxia suffered by the patient.

3. Respiratory Centre paralysis results in the loss of regular breathing patterns. Long periods of apnoea are triggered between breaths of varying depth and regulation. Occasionally Cheyne-Stokes Breathing occurs, and the children are in

a short time rendered cyanotic or livid. The course is sometimes foudroyant (Fr: stormy, like lightning) and one observes fluctuations during the illness.

4. Circulation Centre paralysis sometimes results in sudden complete collapse. The pulse becomes irregular, is almost impalpable, the extremities are cold and feel like marble, the skin is cyanotic or livid; often profuse diaphoresis is observed.

Observations on the Pathophysiology of Hypoxia and Hypercapnia

Each of the described disease processes result in Hypo- or Anoxia, to a rise in CO₂ and respiratory acidosis which of itself produces CNS injury from which a *circulus vitiosus* (L: a vicious circle) ensues. Anoxia of 60 seconds produces morphological changes in the brain, after 3-6 minutes necrosis, neural breakdown and death. This brain anoxia first manifests itself in a headache followed by a short-term euphoria and psychological excitement and eventually disorientation and confusion. The patients become lethargic and ultimately comatose. Rises in CO₂ also lead to confusion, distress and again coma. Blood pressure rises initially but later falls again to below normal levels, with death following shortly after due to loss of vasomotor neural activity. Often it is not easy to distinguish between the hypoxic and hypercapnic brain injury caused by Poliomyelitis and that from Encephalitis. Drinker et al. describe how hypoxia and hypercapnia lead to increased capillary permeability. This explains the frequently found pulmonary oedema in these patients. Pulmonary oedema and in Poliomyelitis the occasionally found blood leakage in lung parenchyma and alveoli are not the result of Anoxia and Hypercapnia; they emerge primarily due to long-standing negative intrathoracic pressure applied to alveolar capillaries. This is particularly the case when the airways are partially obstructed (Swallowing impairment, Mucus plugging in the bronchial tree). Here the Americans speak of a cupping action (Minneapolis Polio Research Commission 1947).

A further mechanism leading to pulmonary oedema are changes in blood chemistry confirmed by Bower. This author found that Poliomyelitis sufferers in the first days of their illness became hypoproteinemic, a condition which reached its zenith on the seventh day of the illness. All plasma proteins and albumin are reduced, except for the gamma globulins. The drop in serum albumin is greater the worse the poliomyelitis is, and in particularly bad cases can drop to a level of 2%. Eaton in animal studies showed that a sudden decrease in albumin produced marked pulmonary oedema. It is also likely that the drop in plasma protein and albumin levels also lead to brain oedema, through which the already damaged CNS is affected further.

Bower also found in severe cases of Poliomyelitis that there was an increase in potassium excretion and an inhibition of sodium excretion in the urine. However in some cases the potassium level remains normal and in others, where renal function is disturbed, it can be raised to a level 50% higher than normal. It is therefore not surprising that in such cases heart dysrhythmias or even cardiac arrest can occur.

A further, extremely rare mechanism leading to hypoxia has been described by Smith, Rosenblatt and Limauro. There have been instances where even without respiratory muscle paralysis or bulbar symptoms a lethal cyanosis has occurred despite iron lung ventilation. The authors attribute this to spasm of the pulmonary arterial system that is so severe that gas exchange is essentially prevented. Thieffrey reported a similar observation.

Overview of the Treatment of Respiratory Paralysis

The earliest mechanical ventilator used was the Biomotor. This consisted of an airtight rubber suction cap that was placed on the abdomen and by changing the pressure within the suction cap produced diaphragmatic movement. It is however both an extremely noisy apparatus which the patients found inordinately disturbing, and only produced ventilation in those lung segments adjacent to the diaphragm. Effective in mild cases of diaphragm paralysis only, the Biomotor proved disappointing in severe cases of respiratory paralysis and those with bulbar impairment. In the cases we observed, those who received Biomotor ventilation developed atelectasis in the upper lobes and the paravertebral parts of the lung. Furthermore the Biomotor can only be used for a maximum of 14 days, as use longer than this results in the development of lung perfusion/ventilation mismatches and aspiration of bronchial secretions. Bolt and Valentin, who carried out spiographic measurements of poliomyelitic respiratory paralysis, found that the Biomotor in cases of prolonged paralysis could not prevent the emergence of respiratory failure.

Children with diaphragmatic paralysis may also occasionally be treated with the Danish “Wippe” apparatus, known in the USA as the “Rocking Bed”. The patient is fastened by the feet and shoulders, and the desired rhythm and frequency is established, moving the body in a see-saw fashion. This can be maintained for several hours at a time, and provides sufficient ventilation by passive diaphragmatic movement. It is only viable in mild cases, however, and cannot be used for long periods.

Much discussed, if little used in actual practice, is the electric stimulation of the Phrenic nerve, as presented by Sarnoff, Witzensberger and Harburg. The Phrenic nerves on each side of the diaphragm are stimulated with electric current in a rhythmic fashion, producing diaphragmatic contraction. Sarnoff showed in animal models that a completely adequate ventilation could be produced by this method, presenting his findings at the 6th International Paediatric Congress. In the majority of cases the Phrenic N. can be stimulated percutaneously, more

rarely the nerve must be exposed. The advantage of this method is that the patient is free to move around, relieving much of the care required.

Until today, however, the most important mechanism of artificial ventilation has been with the Iron Lung. It was first built by the Americans Drinker, Wilson et al in 1928. The Drinker-type and the later Emmerson-type are torpedo-shaped hollow tubes, about 2 metres long, in which the patient is placed up to the neck, so that the head remains outside. The neck is enclosed by a foam rubber and nylon collar which is airtight. At the foot end of the machine is a removable leather housing, through which an electrically-driven pump produces a regular rise and fall in pressure within the chamber and which in turn causes a regular compression and relaxation of the abdomen and thorax. The usual pressure gradient is between minus 10 to minus 15 mmHg and plus 2 to 5 mmHg. If the electricity supply is lost, the machine can be easily operated by hand. Different openings in the side of the machine allow further care of the patient. Injections may be given, urine and faeces may be emptied even while the Lung is in use. Because the whole body with the exception of the head is enclosed, the patient is entirely dependent on the care of nursing staff. Hence smaller IL types have been constructed, in which ostensibly the thorax and abdomen are enclosed, but the arms and legs, as well as the neck and head are free. Different versions of this type exist, including the Mulliken Type, the Monaghan Lung, the Chest-Respirator etc.,. These devices have gained a reputation as providing completely adequate mechanical ventilation. We in Zurich have up until now no experience with these smaller machines. A definite advantage of them is that they allow not only treatment of a respiratory-compromised patient, but also allow that patient to be transported, as the entire apparatus may be placed in an automobile. A further disadvantage of the large Emmerson-type machines is that since the patient is completely enclosed, necessary physiotherapy is not possible. Patients who spend months ventilated in these machines emerge with completely withered limbs which may also have developed severe contractures. Ritchie Russel has recently also observed and noted the dangers of hyperventilation. Healthy "Controls" who were placed within an Iron Lung developed severe Hyperventilation Syndrome, including tetany and ultimately loss of consciousness, after being ventilated for a period of 20 minutes at a respiratory

rate of 12-15 breaths per minute. However as most of the patients with Poliomyelitis who are placed in an Iron Lung have a respiratory acidosis, we consider the risk of developing a Hyperventilation Syndrome to be low. At least in our experience we have not yet seen this syndrome develop in our patients. Great difficulties ensue when babies or very young children require placement in an Iron Lung. Due to the current models of IL being unable to provide the higher respiratory rate required by babies, it is only very rarely that they are ventilated in the Lung. Smaller versions of the IL have been constructed for babies, but these machines are currently only available in the largest Poliomyelitis treatment centres.

A very important question to be considered is: when is the optimal time point at which the patient should undergo IL treatment? Wilson considers that early treatment in the Lung provides the best possible prognosis in avoiding damage to respiratory muscles and preventing further damage from occurring. His statistics show that patients who were treated early in the course of the disease had a better prognosis than those treated late. An exact time point remains very difficult to judge, as the course of the illness, and the emergence of respiratory failure, varies so widely among patients. Of the 9 children in our hospital who died despite IL ventilation, 4 were ventilated from the 5th day of illness, 3 on the 6th day, and 1 on the 8th day. Of the 10 children who survived due to IL ventilation, 4 were ventilated from the 5th day, 2 earlier and 3 later. 1 was ventilated from the 8th day of illness, and 1 from the 12th day. Therefore a wide variation in response exists even within our patient cohort. On the other hand, we concur completely with Wilson's conclusion and would argue strongly for the early ventilation of patients within the Iron Lung. As soon as the first signs of respiratory compromise appear, the patient should be placed in the IL. For the earliest possible measurement of an oxygen deficit, the use of the Oximeter developed by Millikon and Smalles can be used. This may be placed on the earlobe and allows measurement of Oxyhaemoglobin in flowing blood by a photoelectric cell. Bolt and Valentin confirm spirographically the maximal possible ventilation per minute with oxygen and in room air. As soon as maximal respiration with room air less than with oxygen occurs, respiratory failure is present and mechanical ventilation is indicated.

Very often the opinion is expressed that patients shouldn't be left in the Lung too long: at the earliest time possible, at least for a short time, the Lung should be turned off and the patient forced to breathe for themselves. This is an attempt to prevent muscular atrophy that otherwise occurs, as well as avoid a psychological dependence that sometimes develops, where the patient doesn't want to leave the security of the Lung. The idea that psychological security plays a large role in patients not wanting to leave the Lung comes from the oximetry measurements carried out by Schwartz. Schwartz measured the CO₂ and O₂ levels in the blood of respiratory failure patients within the Lung, then turned off the ventilation mechanism by opening one of the windows in the side of the Lung without actually turning the device off. The blood O₂ level sank immediately, but to a much greater degree in those patients who were aware mechanical ventilation had ceased. These experiments showed how strong the psychological influence, anxiety etc, was on blood oxygen concentrations.

If in the acute inflammatory phase of the illness the Iron Lung is turned off too frequently, the damaged nerve cells may be further damaged. As well, the hypoxia that results can lead to greater CNS injury. We have good grounds to presume, that for example the patient in Case 8, through frequent and forced removal from the Iron Lung, developed severe hypoxic brain injury. This case shows that the Lung should only be turned off if very small risk of hypoxia and hypercapnia exists.

The care of patients in the Iron Lung requires great expertise and diligence from dedicated nursing staff to be a success. The nursing staff must be experienced and practiced in Iron Lung care, and must be able to instantly respond to patients who have severe respiratory paralysis. That those treated in an Iron Lung require far more care than that demanded of usual nursing practice is obvious. There must be objection-free emptying of urine and faeces, combined with constant measuring of physiological parameters, however these details will not be pursued here. We will, however, discuss the special therapeutic and nursing problems inherent in the use of Iron Lung ventilation by means of the case presentations.

SEVERAL CASES

1. Respiratory Muscle Paralysis due to Spinal Cord Involvement

Case 1. J. No. 6972/48 - Sch. Paul, born 22.10.1939, 8 10/12 years old.

Family history: Asthma and Hayfever

PMH: Unremarkable

Current Illness: Became progressively unwell over 2 days in the first weeks of August. The temperature was not measured initially. On 12th August had a temperature of 38.3 degrees, with headache. On 13th August the temperature was 38.7 degrees with neck stiffness. Admitted to the Cantonal Hospital Winterthur with paralysis of the left arm and severe breathing difficulties.

On the evening of admission he had a temperature of 38.6 degrees, pulse rate was 136, respiratory rate was 40. Mild meningitis symptoms were present, however also a generalised weakness, areflexia, rapidly developing paralysis of both arms and the intercostal muscles, with purely abdominal breathing and nasal flaring. Lumbar puncture showed 12 white cells, 33 mg% albumin and 89 mg% glucose, the Gold Sol Reaction was normal. On 14th August (the 4th day of illness) cyanosis, restlessness, obvious air hunger had occurred and he was placed in the Iron Lung. Until 20th August the Lung could only be switched off very briefly as the patient became cyanotic immediately. He was now afebrile, but had also developed Facial N. paralysis. Pulse rate was 100, RR remained 40. From the 21st August he showed improvement, and the Lung could be switched off for several hours at a time. Until 11th September the patient underwent intermittent IL ventilation, for a total of 348 hours. Only minimal atelectasis occurred due to this intermittent ventilation. In the second half of September the intercostal paralysis clearly receded and a reduced thoracic ventilation was required. The RR dropped to between 20-30 breaths per minute. At the end of October the patient was sent to the Rehabilitation Centre, where he remained until June 1949. By the time of his discharge from the Rehab Centre he still showed some signs of extra respiratory work, needing to use auxiliary muscles of respiration as intercostal muscle use was still limited, as well as the left side of the diaphragm. There was also continuing paralysis of the shoulder girdle and obvious, if not complete paralysis of both arms.

Summary: Iron Lung ventilation for a total of 348 hours due to a severe spinal nerve paralysis of respiratory muscles. At discharge - 9 months after the illness began - there remained diminished respiratory function with limited reserve, further disability of both upper limbs.

Case 2: J. No. 6847/48 - Ch. Olga, born 16.04.1933, 15 3/12 years old.

Family History: Unremarkable

PMH: Marked debility.

Current Illness: On 27th July 1939 developed headache, 28th July vomiting, pain and a generalised weakness, especially affecting the arms. By 29-30th July was no longer able to sit or stand, or move the arms, and the diagnosis of Poliomyelitis was made.

On Admission: Temperature was 39.9 degrees, HR 120, RR 30. Looked extremely unwell, had a soft voice and clear signs of meningitis, marked weakness and neck stiffness, the upper limbs were paralysed but had extremely limited movement with support. CSF: Albumin 81mg%, cell count 70, primarily white cells. On the next day fever was treated with Pyramidon 6g/day, but severe vomiting continued. There was glycosuria. Paralysis progressed over all 4 limbs, and on 2nd August involved the diaphragm and intercostal muscles, with a RR of 80. During the evening of the 7th day of illness, the patient was placed in the Iron Lung. Until the 13th August the patient required almost constant IL ventilation, as even 15 minutes outside the Lung resulted in marked cyanosis developing rapidly with severe respiratory distress and requesting herself to be placed back in the Lung. Subsequently the patient improved to some extent, with movement in both legs possible, though marked reductions in arm movement remained. The thorax showed regular respiratory movement, though this remained reduced. On 27th September spirometry measured a peak flow of 0.8L, which climbed slowly over the next two months to 1.0L. The patient required paraffin oil to use her bowels. The patient was discharged from our Rehab Centre on 17th March 1949 with continuing paralysis of the intercostal muscles, shoulder girdle and right forearm. Leg paralysis had almost completely disappeared. Follow-up in spring 1950 showed marked atrophy of the paralysed respiratory muscles and the arms.

Summary: Debilitated girl with diffuse paralysis of the legs, arms and respiratory muscles, placed in the Iron Lung on the 7th day of illness, and ventilated for a total of 189 hours. Unfortunately the patient had to be released without adequate physiotherapy. 2 years after the onset of illness she was both mentally and physically disabled to the point of demanding a great burden of care from the family. With adequate initial physiotherapy and training she likely would have been able to work and have a degree of independence, but sadly due to the timing of her illness onset coinciding with the outbreak of war, this was not available.

Case 3: J. No. 6231/48 - Z. Ruthli, born 24.10.1942, 5 7/12 years old.

Family History: Mother - Epilepsy due to traumatic injury; Father - Alcoholic.

Personal History: Adopted by another family, with relatively late speech development, otherwise unremarkable.

Current Illness: From the 7th to 10th May 1948 a cold-like illness with a high fever between 38-39 degrees. On 11th May fever resolved but developed neck pain and stiffness, on 12th May remained fever-free in the morning and appeared to be recovering, but during the afternoon developed severe posterior headache and again was febrile to 39 degrees. over the next 3 days fever persisted and paralysis in the left arm, extending later to the left leg appeared, with associated severe pain, confirming a diagnosis of Poliomyelitis.

On Admission: Temperature 39.5 degrees, HR 144, moderate signs of meningitis, marked lassitude and pain in the back, paralysis of the shoulder girdle and bilateral upper arm musculature, respiration predominantly abdominal, and diffuse pain and paralysis in both legs. Reflexes in the right leg diminished, in the left leg present. CSF: pressure was raised to 42cmH₂O, Albumin 14mg%, WCC 44, Glucose 44, Chloride 464mg%, Gold Sol Reaction was positive.

Ventilation was purely via the diaphragm with Intercostal muscles completely paralysed, clearly recognisable Poliomyelitic Facies, and cold extremities. During the night the patient became restless with a mild cyanosis. On the 18th May she had developed complete tetraplegia, except for small movements of the toes and fingers. Due to neck and back muscle involvement, breathing was entirely diaphragmatic without auxiliary muscle assistance, though maintained at a

regular 40 breaths per minute. On the 19th May, - the 8th day of illness - due to clearly progressing cyanosis the patient was placed in the Iron Lung, whereupon she immediately improved her oxygenation, her restlessness disappeared and she fell asleep. The child was ventilated between 5-10 hours per day in the Lung, up until the 26th May - the 15th day of illness. By this time contractions of the intercostal muscles were observable, the child became afebrile and her overall condition improved. As well, there were increased movements of her extremities. On the 2nd of June, at the request of her parents, after 21/2 months in hospital, the child was discharged home. At this time there was still severe paralysis of all limbs and also the rump. Upper arms and shoulders could not be moved, though all movement was possible in the forearms and apparently there was also movement in both feet.

Summary: A 5 ½ year old girl with severe spinal cord Poliomyelitis, without obvious CNS or Bulbar involvement, required 33 hours of ventilation in the Iron Lung. 11/2 years later she was able to stand independently, walk with support, lift her head weakly. The L. arm could not be raised, and the L. lower leg was completely paralysed. The child made further advances over the next months, and as she has no CNS damage from the disease or hypoxia, it is likely she will be able to maintain a career in an area where she is able to sit and work.

Case 4. J. No. 4040/47 - P. Heinrich, born 29.10.1937 9 9/12 years old.

This patient had already suffered a meningitis form of Polio in 1946. On 27th July 1947 he developed a severe sore throat and general malaise. On 30th July severe vomiting, headache, meningism, whereupon he was admitted to an outlying district hospital. A marked meningitis ensued, with CSF showing 750 white cells, half being polymorphonucleocytes and half mononucleocytes. Meningitis was diagnosed and he was treated with Streptomycin. On 1st August lassitude, areflexia appeared, however the Streptomycin treatment was continued. Towards evening the child developed breathing difficulties, which in the following days worsened, and he was transferred to the Children's Hospital in Zurich.

Evening of admission on 3rd August: Temperature 39 degrees, HR 110, severe cyanosis, complete paralysis of the diaphragm and intercostal muscles,

respiration entirely dependent on auxiliary muscles. Bilateral paralysis of the shoulder girdle, moderate paralysis of the legs. CSF: Albumin 34mg%, WCC 21 primarily mononucleocytes, Glucose 72mg%, Chloride 439mg%, Gold Sol Reaction positive. Immediately placed in the Iron Lung (5th day of illness). During the first days of admission was only able to be removed from the Lung for minutes at a time, however after this initial critical period this was extended to several hours. From the 10th August mechanical ventilation was no longer necessary. In October the patient was sent to the Rehab Centre in Affoltern (a suburb of Zurich), with regular breathing and only the shoulder girdle showing residual weakness. The forearms could be moved, however he was unable to raise his upper arms. He also continued to suffer an asymmetric weakness of abdominal muscles which over the next few months lead to a scoliosis of the spinal column. He was discharged on 22 March 1948 to home. By summer 1949 he had made excellent progress, and by August 1950 he was able to climb two flights of stairs, was attending school and looking forward to working in an appropriate career.

Summary: A 10 year old boy, due to severe respiratory paralysis required 85 hours of Iron Lung ventilation. Paralysis eventually affected all limbs. Subsequent good recovery, so that 3 years later he expects to be able to work in a suitable career.

Case 5. J. No. 5357/48 /8044/49 - Sch. Walter, born 9.08.1933 14 5/12 years old. Family History: Father undergoing psychiatric treatment due to a psychoneurotic complaint.

Personal History: Unremarkable

Current Illness: On 17th January 1948 suffered headache and back pain; 18th January Temperature 38.6 degrees and bilateral leg weakness; diagnosed as Poliomyelitis on 20th January.

On Admission: Temperature 37.9 degrees, HR 70, typical poliomyelitis facies, obvious signs of meningism, lower limb reflexes weak or absent, left side abdominal wall pull upwards. Widespread paralysis of arms and markedly so of the legs. CSF: Pressure 29cmH₂O, Gold Sol Reaction positive, TP 70mg%, WCC

97, Glucose 55mg%, Chloride 440mg%. Blood analysis unremarkable. Treated with Pyramide 6g daily for fever, which lowered the temperature to 35.6 degrees. Over the next 2 days progressive weakness was noted. Intercostal muscles affected, with associated nasal flaring and use of accessory muscles of respiration. On 25th January (the 9th day of illness) the boy became livid, was exhausted and subsequently was placed in the Iron Lung, where he showed immediate improvement. He required daily IL ventilation until the 30th January, for a total duration of 20 hours. Outside the Lung the patient was intermittently confused, delirious, and livid rather than cyanotic in appearance. He was completely constipated and only able to empty his bowels by aid of enemas. However over the next 1.5 months he improved dramatically, and was able to be transferred to the Rehabilitation Centre in Affoltern. There the patient was initially very low in spirit, and cooperated minimally with attempts at physiotherapy. After 3 months he regained hope for the future and worked hard to improve over the next 10 months. By April 1949 ventilation was completely adequate, though he continued to suffer bilateral upper arm and shoulder weakness, especially affecting the deltoids and serratus anterior muscles. The left leg had completely recovered, however the right leg remained weak, especially the calf muscles, as did the pelvic girdle musculature. This has resulted in a marked scoliosis developing. The patient is currently a business trainee and is able to perform all his tasks as required.

Summary: Diffuse paralysis of the extremities. Due to respiratory muscle paralysis the patient required 20 hours of IL ventilation. With appropriate physiotherapy the boy was able to recover some activity, despite residual paralysis. He is currently pursuing a career in business.

Case 6: J. No. 3554/47 - B. Franz, 7 5/12 years old.

Family and Personal Medical History unremarkable.

On 8th June 1947 the patient became unwell, with stomach pain and vomiting.

On 9th June he developed fever and anorexia, which by the next day was 38.2 degrees, with marked diaphoresis, headache, and a diagnosis of Poliomyelitis was suspected.

On Admission: Temperature 39.2 degrees, HR 140 and an obvious meningitis due to Polio. CSF showed a TP of 62mg%, Glucose 70mg%, Chloride 434mg% and Gold Sol Reaction was positive, with WCC 113. Over the 11th to 12th June he developed a rapidly progressive spinal cord paralysis, with complete involvement of the intercostal muscles and an atonic hemiplegia. Breathing was purely abdominal and sub-cyanotic, and the child underwent mechanical ventilation by the Biomotor Apparatus for 5 hours. He showed marked improvement initially, but later he again deteriorated, developing severe cyanosis and diaphoresis, and required placement in the Iron Lung. He remained in the Lung until the 28th June (the 14th day of illness). During the initial period of IL ventilation, as soon as the Lung was switched off the patient became deeply cyanotic, with tachycardia and marked diaphoresis. Later he was able to be removed from the Lung for several hours at a time, and from the 29th June he no longer required IL ventilation.

However he continued to suffer an almost complete quadriplegia, and was only able to move his fingers and toes and while in the bath. Ventilation was via the diaphragm, primarily on the right side, and the upper intercostal muscles. On 6th July he was placed in the Rehab Centre at Affoltern, but despite intensive therapy showed almost no improvement of his quadriplegia. During the 5th month of illness he developed bronchitis which again led to a respiratory emergency requiring return to the main Childrens' Hospital and a further 12 hours of mechanical ventilation in the IL. Although there was no pneumonia found radiographically, a severe bronchitis was evident, and the child had great difficulty coughing up phlegm. He required nightly IL ventilation, though during the day he was able to breathe adequately without support. Nevertheless, the inability to cough up phlegm persisted. In April 1948 the Vital Capacity was

measured at 1L. During the summer a vaccine against Polio derived from oral flora was introduced by Professor Aqua, however it was ineffective in the treatment of signs and symptoms already present, and in this case a severe quadriplegia persisted. At the end of September and beginning of October, therefore 15 months after the onset of illness, the patient again developed fever, respiratory distress, tachycardia (HR 180) and confusion. and he was again placed in the Iron Lung. He initially improved, including his conscious level thanks to the ventilation provided by the machine. Unfortunately by morning his cyanosis had again worsened significantly, he became distressed and disorientated, and at 10:30 am he died while still being ventilated in the Iron Lung.

Autopsy Results: Chronic inflammatory infiltrate and ganglion cell degeneration in both spinal cord and brain. Atrophy of the skeletal muscles, severe alveolar hyperaemia in all lobes of the lung, dilated heart with hypertrophy of the right ventricle.

Summary: 7 1/2 year old boy with severe spinal Poliomyelitis. complete quadriplegia and almost complete paralysis of the intercostal muscles, paralysis of the L. hemidiaphragm. During the acute phase of the illness the patient required 16 days ventilation in the IL. In the following 15 months almost no improvement occurred with even mild respiratory inflammation resulting in respiratory distress and the need for IL ventilation. The patient was never able to clear his own bronchial secretions, resulting in two episodes of pneumonia needing mechanical ventilation, and ultimately in the patient's death.

The autopsy showed severe bleeding in both lungs. Thieffrey described haemorrhagic pneumonia occurring in Poliomyelitis, very like that seen during the Spanish Flu epidemic of 1918-1920. In our case no pneumonia was found at autopsy, rather a hyperaemia and bloody exudate, which can be best explained through increased negative pressure during respiration brought about by airway obstruction. This has ultimately caused the death of the child due to his extremely compromised respiratory reserve. Our patient displayed the typical fate of polio sufferers with severe respiratory muscle paralysis, who require optimal conditions to achieve adequate oxygenation. They live completely without reserve, so that even the smallest challenge, such as here with a mild bronchitis, results in a

life-threatening hypoxia. With an Iron Lung available sufficient ventilation can be achieved to compensate for this intrinsic hypoxia, but without it the lethal outcome is unavoidable. Simson recommends that every complication requires a bronchoscopy to be performed, above all in cases of atelectasis, so that obstructing secretions can be removed via the bronchoscope.

Case 7: J. No. 911/49. W. Benno, born 27/09/1936, 13 years old.

Family history: Unremarkable

Personal history: Unremarkable

Current Illness: On the 8th September 1949 suffered abdominal pain and back pain. On 10th September severe diaphoresis, headache, pain in upper legs and back. Diagnosed as Poliomyelitis.

On Admission: Temperature 38.6 degrees, HR 39, BP 150/95, pectus excavatum, clear signs of meningism. CSF: Pressure 10cmH₂O, TP 77mg%, cell count 428 - 249 mononucleocytes, Glucose 60mg%, Chloride 432mg%, Gold Sol Reaction positive. On 12/09 paralysis manifested involving both legs as well as the arms, breathing was difficult and only possible with the aid of accessory muscles. On the 13th September the patient had become exhausted and breathing required such extreme effort that he was placed in the Iron Lung. The right intercostals had minimal function, while the left intercostals and diaphragm were paralysed. ECG showed moderate prolongation of conduction time. The patient required IL ventilation until the 21st October, initially for almost 24 hours/day, but later he was able to spend several hours at a time outside the Lung. In total he received 383 hours of mechanical ventilation. The right intercostals and right hemidiaphragm recovered normal movement, however the left side remained disabled. On 22nd October VC was measured at 620ml, and on 18th November 820ml, while the initially raised BP normalised. The ECG continued to show a prolonged conduction time. The patient was sent to the Affoltern Rehab Centre, however was frequently brought back to the Hospital for treatment as every mild respiratory illness resulted in atelectasis of the left lower lobes. This in turn caused hypoxia, cyanosis and respiratory distress requiring IL ventilation, bronchoscopy and suctioning of obstructing lung secretions. There was incremental improvement in limb paralysis, and by March 1949 the VC was 1.4L.

The patient recovered movement in both arms, however the legs and back remained paralysed to the extent that he was unable to raise himself vertically, though he could move horizontally and in water. He was discharged to continue treatment on an out-patient basis, but on 13th May 1950 he again developed atelectasis and pneumonia of the left lower lobes, requiring rehospitalization. He was discharged on 10th June 1950, Unfortunately his paralysis was unchanged but the patient showed remarkable resilience and ability despite his incapacity. He was counselled to build on this resilience so that he could possibly be employable in an appropriate career.

Case 8: J. No. 9776/49. N. Ursula, born 14/04/1939 10 5/12 years old.

Family History: Mother suffers migraines

Personal History: Unremarkable, tall girl for her age at 149cm.

Current Illness: On 22nd August 1949 fell on to her back, with persistent back pain. On 25th August developed fever 38.7 degrees, which by 28th August was 39 degrees with associated headache, and a developing weakness initially affecting the right arm, which prompted a diagnosis of Poliomyelitis.

On Admission: 28th August fever 39.9 degrees, HR 120, RR 32, BP 126/96, ECG showed right axis deviation in Lead II. Clear signs of meningism, typical Facies Poliomyelitica, atony of neck muscles, paralysis of the R. deltoid. Lumbar puncture showed a pressure of 30cmH₂O, TP 32mg%, Cell count 110 - 90 mononucleocytes, Glucose 76mg%, Chloride 433mg%, and Gold Sol positive.

During the night of 29-30/08 the paralysis progressed so that the arms were completely affected and only extremely weak movement of the hands was possible. The legs could no longer be raised. The child became rapidly cyanotic and ventilation was only possible via the accessory muscles, so that placement in the Iron Lung became a matter of urgency. On 31st August the child was afebrile but the BP was 165/110. As soon as the Lung was turned off, the child became profoundly cyanotic with marked air hunger. Thoracic and abdominal ventilation was completely absent, and breathing was only possible with neck muscles.

Within the IL cyanosis rapidly disappeared and the child was completely settled. Initially she also suffered from constipation and inability to empty her bladder, but this recovered by the 3rd September. By the 22nd September the child was

able to spend 2 hours at a time outside the Lung, though the diaphragm was still completely paralysed. The intercostals were capable of limited excursion. The limbs were also still affected, but not as severely as at the onset of illness. Physiotherapy and Water Therapy was commenced. The child suffered sudden intermittent relapses again requiring mechanical ventilation as she became profoundly cyanotic, confused or even lost consciousness. On 22nd October the VC was 240ml, the child was a pale grey colour, she was exhausted and was placed once more in the Lung, however after a week of mechanical ventilation this became difficult and ultimately no longer possible. At 10pm she asked to eat something, but at midnight she died suddenly while ventilation was still being attempted.

Autopsy: Poliomyelitis affecting the anterior horn cells, an eccentric hypertrophy of the right heart, chronic extensive emphysema and atelectasis, alveolar hyperaemia. Diffuse fatty liver and fatty deposition in the myocardium.

Summary: This is also a case of pure spinal cord Poliomyelitis with severe consequences. The child was ventilated intermittently in the Lung for a total of 548 hours, frequently for prolonged periods. She was able to be removed from the Lung for several days at a time, but redeveloped hypoxia and respiratory acidosis, which doubtless also over time resulted in a hypoxic brain injury. The child developed over time obvious psychological and behavioural changes, and became extremely institutionalised. Without the close examination of the brain it is difficult to decide how much of these behavioural and cognitive changes were due to brain injury itself and how much to a severe depression as the result of her overwhelming illness. Nevertheless as a result of these observations may we conclude by stating that children with such badly-affected respiratory muscles be treated aggressively, so that hypoxic brain injury may be avoided.

Case 9: J. No. 9803/49 E. Walter-Bruno, 5 2/12 years old.

Personal History: Very lively, if nervous child, otherwise unremarkable.

Current Illness: A short prodrome of nausea and vomiting began on 19th August 1949. On 29th August accompanying abdominal pain and a fever to 38 degrees manifested. Became constipated, suffered back stiffness, restless sleep, and on the

30th August was no longer able to walk or sit up, leading to the diagnosis of Poliomyelitis.

On Admission: Temperature 39 degrees, HR 140, BP 120/75, meningism, severe quadriplegia with only limited hand and foot movement possible. Respiration was predominantly abdominal with assistance from the accessory muscles. CSF had a pressure of 30cmH₂O, TP 49mg%, Cell count 93 - primarily monocytes.

On 31st August continuing high fever, BP 130/100, severe cyanosis developing requiring immediate IL ventilation (4th day of illness). ECG showed mild ST depression and Lead III had become biphasic, indicating myocardial injury.

The patient remained in the IL until 13th September (the 17th day of illness), and could only tolerate a few minutes at a time free of mechanical ventilation before he again became deeply cyanotic. The total duration of IL ventilation was 295 hours. On the 13th September, due to the IL being urgently needed for another patient, the patient was placed in the Biomotor Ventilator. After one hour ventilation with this apparatus he became profoundly hypoxic, was unable to be adequately ventilated and died due to anoxia.

Autopsy: Severe Poliomyelitis of the anterior horn cells, partial atelectasis in both lungs, emphysema, pulmonary oedema, dilated right heart.

Summary: This case is also one of pure spinal cord Poliomyelitis, which reached as high as the brain stem, as indicated by the high blood pressure measurements recorded. Doubtless the child would have survived for longer if Iron Lung ventilation had been continued, however the severity of his paralysis was such that any significant improvement was unlikely.

In rare cases the weakness developing in the pre-paralytic phase is so severe that hypoxia results and IL ventilation is necessary. In such cases early ventilation to control the hypoxia is more promising, as here after abatement of the acute inflammation there can be a restitution of reasonably normal function.

Unfortunately this patient did not receive early ventilation, leaving him with an illness that was not survivable.

Case 10: J. No. 3668/47 M. Vreneli, born 13/10/1940, 6 ½ years old.

Family History: Unremarkable

Personal History: Birth weight 2600g despite normal gestation, normal development.

Current Illness: On 22nd June 1947 developed headache, fever to 39 degrees. On 23rd June was extremely tired and slept for long periods, but also developed abdominal pain. On 24th June the fever continued and pollakisuria manifested. Poliomyelitis was subsequently diagnosed.

On Admission: A very small girl, weighing only 20kg, Temperature 38.7 degrees, HR 120, Facies poliomyelitica, mild weakness, obvious meningism. LP: pressure 19cmH₂O, Cell count 182, primarily monocytes. Pandy test was positive.

On 26th June the temperature was 40.2 degrees, the patient was apathetic and displayed a severe weakness with areflexia and migration of the navel indicating a marked involvement of abdominal muscles. There was inability to raise the head. On 27th June the fever abated but a sudden, rapidly-developing respiratory distress, cyanosis, weak and rapid pulse became evident at 10am. Diaphragmatic movement was not noticeably affected but intercostal function was limited. The child was immediately placed in the Iron Lung, and recovered well after 2 hours of mechanical ventilation. She required no further IL treatment, and continued to recover good function. On 15th August she was discharged with mild paralysis of the quadriceps mild weakness of the truncal musculature.

Summary: In this case there was no or only mild respiratory muscle involvement, but rather a high grade generalised weakness which required a brief period of IL ventilation.

2. Swallowing Impairment

Case 11: J. No. 7199/48 K. Heidi, 12 11/12 years old.

Family History: Unremarkable

Personal History: Mediocre student, bright child difficult to control. Menarche at 12 ½ years old.

Current Illness: Since 5th September swinging temperatures, frequent headaches, constipation. On 10th September high fever, meningism, unequal pupils and nasal speech. Referred to hospital.

On Admission: Temperature 38.8 degrees, HR 104, atonic facial expression, very nasal speech (rhinolalia aperta), left pupil more dilated than the right. Mouth and pharynx full of yellowish saliva, which the patient was unable to spit out or swallow. Marked coarse tremor, meningism, good power and reflexes in the extremities. Lumbar puncture showed a pressure of 20cmH₂O, TP 81mg%, Cell count 175 - 50% were lymphocytes, 50% monocytes. Glucose 57mg%, Chloride 448mg%, Gold sol Reaction clearly positive. On 11th September there was clear inability to swallow and saliva had to be regularly suctioned. On the 12th September (8th day of illness) the patient became cyanotic and had to be placed in the Iron Lung. She received nutrition via a nasogastric tube. On 13th September severe cyanosis persisted and the patient continued to need regular suctioning due to build-up of saliva. The IL had to be intermittently halted, but even with the Lung the patient could only breathe with great difficulty. The main focus was maintaining the integrity of the upper airway. On 14th September the patient suffered a severe collapse with loss of consciousness for several hours. Use of the medication Lobelin (a nicotinic alkaloid) and the Lung were able to maintain ventilation, however whenever the Lung was ceased a marked cyanosis immediately occurred. Regular suctioning was essential.

Placement of the nasogastric tube was particularly difficult due to the strong gag reflex present. The tongue needed to be sewed forward to prevent it falling backwards and obstructing the airway.

The patient remained in the Lung until 16th September, a total of 97 hours, and on 17th September she was able to be removed from it altogether. From then there was a slow improvement in her swallowing and she no longer needed to be suctioned as frequently. From the 24th September there was a rapid improvement and the patient was again able to swallow. From the 4th October there was a mild soft palate impairment, but otherwise no other symptoms. On 11th October, with only very mild soft palate impairment the patient was discharged home.

Summary: Severe Poliomyelitis involving the posterior cranial nerves, which led to marked impairment of swallowing and the soft palate, but without affecting the

Respiratory Centre or muscles of respiration. Saliva needed to be rigorously suctioned for many days, but despite this severe cyanosis developed requiring Iron Lung ventilation for a total of 97 hours.

Case 12: J. No. 3836/47 B. Emil, born 23/03/1942 5 3/12 years old.

Family History: Unremarkable

Personal History: Premature birth, enuresis ceased at 5 years, otherwise normal development. Tonsillectomy 3 months prior.

Current Illness: On 9th and 10th July low-grade fever, neck pain and swallowing difficulties, frequent vomiting. On 11th July paralysis of facial muscles. Sent to hospital.

On Admission: Small, underweight boy, 14.7kg. Temperature 39 degrees, HR 140. Obvious signs of meningism, mild weakness without hyporeflexia.

Right-sided facial paralysis, impairment of swallowing, the Glossopharyngeus and the soft palate. Breathing was normal and regular.

Lumbar puncture showed a pressure of 29cmH₂O, TP 20mg%, Cell count 31 - mostly lymphocytes.

On 12th July: very restless, mild respiratory distress, mild cyanosis, mouth and pharynx continuously filling with saliva which he was unable to swallow.

Frequent mouth rinsing and suctioning was necessary.

On 14th July suffered a sudden, severe cyanosis and was no longer able to breathe. Required immediate placement in the Iron Lung for mechanical ventilation and to have his mouth continuously suctioned. After 1 ½ hours the boy much improved, but towards evening developed fever and a rapid respiratory rate. Coarse, wet crackles could be heard in both lungs and a diagnosis of aspiration pneumonia was made. The patient slowly improved over the next few days, but required continuous suctioning and could only be fed via a nasogastric tube.

By the 30th July the patient could again swallow without choking, but by the date of his release on 18th August the right-sided facial paralysis and soft-palate impairment were still obviously present.

Summary: This is also a pure paralysis involving the posterior cranial nerves, which for a period required IL ventilation.

3. Swallowing Impairment in Combination with other Forms of Paralysis Leading to Hypoxia

Case 13: J. No. 1108/46 Z. Sonja, 13 years old.

Family History: Unremarkable

Personal History: Mild intellectual impairment, School difficulties, Tonsillectomy at 9 years old.

Current Illness: 1st September 1946 developed fever, abdominal pain. Underwent appendicectomy on 2nd September, low-grade fever and frequent vomiting. On 3rd September had speech articulation impairment, and diagnosed as Poliomyelitis that evening. Temperature was 39.5 degrees, HR 120, vigorously tried to fend off staff with terror-filled eyes. Increasingly gurgly speech and sallow face, with saliva dribbling from the corners of her mouth. The mouth and pharynx were full of saliva which she was unable to swallow. The tongue was immediately pulled forward to prevent airway occlusion. There was normal respiratory excursion of the thorax but high-grade weakness of both arm and leg musculature. Saliva had to be continuously suctioned.

On 5th September breathing became difficult, with jerky movements of the mouth floor and drawing in of the epigastric area during inspiration, and with cyanosis and listlessness developing despite the application of oxygen, stimulation and continuous suctioning. Underwent ventilation with the Biomotor apparatus. BP 155/120. Nutrition given intravenously.

On the 7th September thoracic ventilation on the left side was better than the right, however there was only minimal diaphragmatic excursion. The alkali

reserve was 70. Listlessness slowly improved and that evening at 11pm she was placed in the Iron Lung (7th day of illness). Cyanosis immediately disappeared. The child became calmer and her respiratory distress abated. On the following day she was afebrile but continued to require mechanical ventilation over the next several days. Her BP remained high at 155/120 initially, but slowly normalised to 105/75 subsequently. Her swallowing impairment continued however, and she continued to need regular suctioning and parenteral nutrition until 18th September. From that point the child was able to swallow or spit out her own saliva and her speech became clearer as her facial musculature regained its innervation. On 12th October she was discharged.

Summary: This is a case where although swallowing impairment was the most significant injury, breathing difficulties were also present. In particular the normal respiratory excursion was limited due to severe generalised weakness. The swallowing impairment causing a build-up of saliva in the mouth and pharynx had an additive effect in combination with these respiratory limitations to cause hypoxia and respiratory acidosis, as indicated by the high alkali reserve of 70.

Case 14: J. No. 7166/48 L. Ernst, 7 4/12 years old.

Family History: Unremarkable

From the Personal History the only note-worthy fact is the child suffered from enuresis until he was 5 years old. 7 days before the onset of Poliomyelitis he received the Smallpox vaccination.

Current Illness: Over 2nd-3rd September 1948 suffered a prodromal illness with fevers between 38-39 degrees. Anorexia and abdominal pain. There was a fever-free period from the 4th September, but on 6th September fever again manifested along with headache. The family doctor recognised signs of developing meningism and diagnosed Poliomyelitis.

On Admission: Temperature 38.8 degrees, mild cyanotic complexion. Neck stiffness and mild generalised weakness, obvious meningism. Oppenheim Reflex was positive, and there was hyperreflexia. Lumbar puncture: Pressure 28cmH₂O, TP 115mg%, Globulins 17mg%, Cell count 347 - approx. 50% monocytes. Glucose 180mg%, Chloride 430mg%, Gold Sol test strongly positive.

The following night the patient became delirious, was extremely restless, though the generalised weakness increased. There was nasal flaring and breathing was only possible via the accessory muscles, as the intercostals could barely move. The patient's voice was very soft, and there was diffuse paralysis in all the extremities. On 8th September the patient was placed in the Iron Lung, whereupon he became calmer and was able to sleep. The IL was only able to be turned off very briefly, as the patient became deeply cyanotic within a matter of minutes. He was by this time suffering an almost complete quadriplegia, with only small movements of the right hand and foot possible.

By the 13-14th September the Lung was able to be switched off for several minutes at a time, but only if supplemental Oxygen was provided to the patient. On the 14th September fever also abated and he seemed to be improving. On the afternoon of 15th September he again became restless and distressed with a livid appearance. At 10pm the report was better, but at midnight he was no longer able to speak, his tongue fell backwards obstructing the airway and he died 40 minutes later.

Summary: Severe spinal cord Poliomyelitis with Quadriplegia and Respiratory Muscle paralysis, requiring several days continuous ventilation in the IL. Initially there were no Bulbar signs, but on the 9th day of illness fever recurred. There appeared a definite Facies Poliomyelitica, he had glycosuria, obvious developing paralysis of speech and tongue muscles and subsequently died as a result of this further paralysis.

Whether in this case, despite IL ventilation, the combination of swallowing and respiratory muscle paralysis led to a lethal outcome, which may have been hastened by the addition of bronchiolitis, or whether there was a contribution from a vasomotor paralysis as well, is difficult to ascertain. There was no histological examination of the medulla oblongata carried out. Certainly an assumption that the first mechanism was responsible indicated an urgent tracheostomy was doubtless required.

Case 15: J. No. 4072/47 H. Hilda, born 5/02/1943 4 ½ years old.

Family History: Unremarkable

Personal History: Still suffering enuresis, poor appetite. A delicate, underweight girl.

Current Illness: On 4th August severe irritation, 5th August headache and low-grade fever. 6th August fever climbing to 39 degrees and no longer able to sit up. Diagnosed as meningitis.

On Admission: Temperature 38.5 degrees, HR 120, typical Facise Poliomyelitica, meningism, clear generalised weakness. Lumbar puncture: Pressure 28cmH₂O, TP 48mg%, Cell count 69 - primarily lymphocytes.

On 8th August temperature was 39 degrees, with intercostal muscle paralysis, diaphragmatic breathing obviously increased. Paralysis of both upper arms and the shoulder girdle, with quadriceps involvement in both legs. At this stage no cranial nerve impairment, but on 9th August severe cyanosis, respiratory distress, mouth and pharynx full of saliva requiring immediate placement in the Iron Lung. During the evening intermittently confused.

On 10th August afebrile and condition stable, but still needing mechanical ventilation. During the night of 11th-12 August she died suddenly within the Iron Lung.

Autopsy: Poliomyeloencephalitis; Hyperaemia of the brain, brain oedema; atelectasis of the right upper lobe due to mucus and saliva; meningitis; dilated heart, especially the right heart; organ congestion; large fatty droplets in the liver.

Summary: This case also concerns a combined bulbar and spinal involvement, complicated by atelectasis. The contribution also of Respiratory or Circulation Centre disease cannot be definitely excluded.

Case 16: J. No. 356/49 G. Jean-Pierre, born 2/03/1936 13 7/12 years old.

Family History: Mother suffers from Graves' Disease (Basedow's D. in Central Europe)

Personal History: Late to cease enuresis, otherwise unremarkable.

Current Illness: Began a prodromal illness on 29th October, with abdominal pain, headache, fever to 39 degrees. A period of latency occurred between 30th-31st October, but on 1st November severe headache, abdominal pain and fever recurred, and on 3rd November had developed paralysis of the legs. Sent to hospital.

On Admission: Temperature 39.1 degrees, HR 120, RR 32, BP 120/75. ECG showed ST depression especially in Lead III, indicating possible myocardial damage. Facies Poliomyelitica, mild cyanosis, paralysis of both legs and the right arm, and weakness of the left arm, paralysis of the abdominal wall and back musculature, marked signs of meningism.

LP: TP 68mg%, Globulins 4%, Cell Count 266 - almost all monocytes.

During the course of the afternoon rapid progression of paralysis, by 8pm paradoxical respiration had developed with intercostal muscle involvement.

On the morning of 4th November the patient was deeply cyanotic, the arms and legs were completely paralysed, and only minimal ventilation was possible via the accessory muscles. The patient was then placed in the Iron Lung (6th day of illness). Despite generalized weakness he was still able to swallow, but by the following day this had also become impaired, with associated speech difficulties. The patient became deeply cyanotic within seconds of the Lung being turned off.

On the 7th November both intestinal and bladder paralysis had also manifested, with a low-grade fever and a BP 155/115.

By the 12th November the swallowing impairment had begun to improve, with clearer speech, and clear conscious state, despite continued paralysis of all four limbs, and intestinal and bladder function absent. Over the following days the patient's condition remained stable, but he continued to suffer profound hypoxia, to the point of loss of consciousness within two minutes, if the Lung was turned off. The cranial nerves had recovered fully, but from C4 down almost complete paralysis persisted. Only small movements of the left thumb were possible.

Nursing care was difficult and compromised, as any removal from the Lung resulted in severe hypoxia and profound distress to the patient due to the awful feeling of asphyxiation he experienced. On 26th November he died suddenly during one of these very brief periods when the Lung was turned off to allow nursing care to be carried.

Autopsy: Poliomyelitis of the anterior horn, dilated ventricles of the heart bilaterally, bronchopneumonia bilaterally, tracheobronchitis. Histologically fragmentation of otherwise normal looking myocardial fascicles.

Summary: This case also presents combined respiratory muscle and swallowing paralysis. The later was limited and not life-threatening. The spinal involvement was of extraordinary high grade, and after a month of illness showed not the slightest degree of improvement. It is likely that even if the patient had eventually been capable of independent ventilation, he would have suffered from severe disability and morbidity. The atrocious feeling of asphyxiation he suffered after even a very short time without mechanical ventilation prevented us from using a positive pressure face mask (Bennet's positive pressure respirator attachment. A form of CPAP).

Case 17: J No. 3611/47 I. Paul born 13/04/1934 13 ½ years old.

Family History: Psychiatric illness on the father's side.

Personal History: Delayed development, speech at 8 years old, enuresis until 9 years old.

Current Illness: On 14th-15th June developed severe headache, but patient still mobile. On 18th June became febrile with a temperature of 38.2 degrees, headache and vomiting.

On 19th June could no longer sit up, the temperature was 38.3 degrees, and the diagnosis of Poliomyelitis was made. Temperature rose to 38.7 degrees and clear Facies Poliomyelitica was recognised. The patient was markedly diaphoretic, had developed a red-coloured dermatographica and signs of meningism with high-grade neck stiffness and differential reflexes. There was mild facial paralysis with soft palate involvement particularly on the left.

LP: Opening pressure 18cmH₂O, Cell Count 120 - half being monocytes. The Pandy Test was clearly positive.

On 19th June there was progression of paralysis bilaterally, involving the soft palate, orbital muscles and muscles of facial expression, as well as generalized weakness and areflexia. By that evening the right intercostals and diaphragm were paralysed, with the patient intermittently confused and restless.

On the 20th June the cranial nerve paralysis was even more marked, with complete loss of swallowing requiring a subsequent gastric tube and constant suctioning. At 2:15pm the pulse became suddenly faster and weaker, the patient developed profound cyanosis and rapidly lost consciousness and continence.

Although he received immediate mechanical ventilation in the Iron Lung there

was no improvement in his condition. He was extremely diaphoretic, had unequal pupils and severe cardiovascular instability. The right hand was much cooler than the left, and by that evening the patient was completely comatose, with a weak tachycardia. He died from cardiovascular shock in the early hours of 21st June.

Autopsy: Poliomyelitis of the anterior horns, cerebral oedema, dilated right heart, congested organs generally, lung and liver petechiae and in subpleural and subcapsular areas frank blood. Pleurisy, mucopurulent bronchopneumonia, areas of atelectasis.

Summary: This is also a case of combined respiratory muscle and swallowing paralysis, but with involvement of the circulation centre as well. The child subsequently died as a result of circulatory collapse due to this central nervous system involvement.

Case 18: J. No. 9364/49 O. Horst born 10/08/1941 7 ½ years old. Family and Personal History unknown. The child was from Linz, Austria, on holiday in Switzerland. On 27th June, the day he arrived, he had a fever of 38.4 degrees, and developed a headache and vomiting the next day. The local doctor recognised the signs of meningism and differential reflexes as being likely poliomyelitis, and sent him directly to hospital.

On Admission: 28th June Temperature 38.5 degrees, HR 112, RR 48, complaining of headache. Clear signs of meningism, neck signs. LP: Pressure 26cmH₂O, TP 46mg%, Cell count 357 - 125 Monocytes, Chloride 432mg%. On 29th June at 9am the fever was 40 degrees, respiratory distress, mild cyanosis, fearful expression. The upper two-thirds of the thorax musculature was completely paralysed. At 11am cyanosis had worsened, disability had increased. By 1pm cyanosis was severe, ventilation now almost entirely through accessory muscles only. Placed immediately in the Iron Lung, whereupon the cyanosis disappeared and the previously fearful expression took on a more peaceful mien. BP 138/110.

At 3:30pm the patient was briefly removed from the Lung, but within minutes became profoundly cyanosed, with a terrified expression and minimal

autonomous ventilation. Temperature 40.2 degrees, HR 80 and barely palpable, circulation diminished. Although placed immediately back in the IL, cyanosis remained, the patient developed a glassy-eyed expression and vomited frequently. However he remained fully conscious.

On 30th June there was a mild worsening of cyanosis and confusion, and the patient could only be removed from the IL for a matter of seconds before he lost consciousness. On 4th July he was afebrile but immediately became profoundly cyanotic once the Lung was removed. On 9th July the electricity supply was lost for a period of about 15 minutes, and although the Lung was manually operated after 5-7 minutes, during this time and despite bag-mask ventilation, the patient became severely cyanotic and lost consciousness. He rapidly improved once the Lung was once again able to be used.

As well as the respiratory muscle paralysis there was also extensive paralysis of the arms and legs. The patient was visibly weaker. On 12th July the pulse became irregular, even within the Lung the patient was profoundly cyanotic and by the 13th July completely comatose, with absent corneal reflexes. He died while still being ventilated within the Iron Lung on the 20th July, having never recovered from this coma.

Autopsy: Poliomyelitis of the anterior horn, cerebral oedema, follicular injury of the spleen, dilatation of the right heart, diffuse fatty deposits in the myocardium, general organ congestion, petechiae within the accessory muscles, pericardial bleeding.

Summary: This patient also initially suffered a purely spinal cord paralysis affecting respiratory muscles. On the 13th day of illness however the temperature increased and bulbar paralysis also occurred. The child died subsequently died of a cardiovascular collapse.

Case 19: J. No. 6304/48 G. Vreneli, born 25/10/1941 6 ½ years old.

Family and Personal History unremarkable.

Current Illness: Prodromal illness from 20th-21st May, with vomiting, sore throat, temperature between 37.5 and 38 degrees. A latency period over the 22nd-23rd May. On 24th May temperature 38 degrees, pain in both legs, on 25th May no longer able to sit up and recognised as Poliomyelitis.

On Admission: Delicate, small girl of only 16kg weight, temperature 38 degrees, HR 130, RR 28. Very obvious signs of meningitis, mild weakness of both legs, respiration normal, reflexes normal on abdomen and upper limbs.

LP: Pressure 23cmH₂O, TP 106mg%, Cell count 213 - half monocytes, Glucose 54mg%, Chloride 434mg%, Gold Sol test clearly positive.

Treatment with Pyrimidon (after Deuretsbacher) in 5% Glucose solution commenced, at 4g per day over 4 days. Progressive worsening of lower limb paralysis during the course of the 4th day of treatment, and the i.v. Pyrimidon was changed to PR administration.

On 27th May the temperature was 39 degrees during the evening, although during the morning had been normal. There was increasing somnolence, respiration had become laborious and the RR was 42, with increasing signs of respiratory muscle involvement, requiring Iron Lung ventilation at 3.00 am, the 4th day of illness.

On 28th May the child had a temperature of 40 degrees and was completely unconsciousness, with irregular respiration, diaphoresis and marked use of the accessory muscles despite IL ventilation, pupil dilation was marked. The child had frequent drooling, indicating swallowing impairment. From the 29th-31st May the temperature showed a low-grade fever, but was otherwise unchanged. The child was profoundly cyanotic despite continuous IL ventilation, with irregular and gasping breaths. She died at 21:00 on 1st June of respiratory failure.

Autopsy: Poliomyelitis of the anterior horn with accompanying severe injury to the Medulla Oblongata and Pons. Bleeding in the lung serosa, aspiration pneumonia bilaterally, mucopurulent tracheobronchitis, dilated right heart, pulmonary oedema, follicular injury to the spleen, fine fatty deposits in the liver.

Summary: Initially this young girl was afflicted with respiratory muscle paralysis, however subsequently swallowing impairment occurred, although this was at this stage still survivable. Unfortunately there was, later, also central respiratory centre involvement, which meant a lethal outcome was unavoidable.

Summary

If we consider the successes we achieved in the preceding cases by use of the Iron Lung as a whole, they can be described under the following groupings:

1. Spinal respiratory muscle paralysis
2. Swallowing impairment
3. Swallowing impairment in combination with other types of paralysis leading to hypoxia
4. Circulation and Respiratory Center involvement.

The outcomes reached within these groupings are:

1. Spinal respiratory muscle paralysis -

Life saved with complete recovery	1 Case
Life saved with minimal remaining impairment	1 Case
Life saved with severe remaining impairment	4 Cases*
Life saved but current status unknown	1 Case
Died during the acute inflammatory stage	1 Case
Died after the acute inflammatory stage had abated	2 Cases

*In hindsight Case 2 was in a therapeutic sense badly neglected, so that severe disability remained, and which may have been lessened significantly with appropriate treatment.

2. Swallowing Impairment -

Pure swallowing involvement is rare. We could only confirm 10% of the cases had swallowing difficulties. The prognosis of isolated swallowing involvement is good, as the affected cranial nerves only very rarely remain impaired. Our two patients made very good recoveries, and were discharged almost completely well.

However despite frequent suctioning aspiration pneumonia could not be avoided in Case 12. Aspiration and its consequences, pneumonia, atelectasis, pulmonary oedema, are the most common complications of upper airway muscle paralysis, and cannot be avoided by mechanical ventilation in the Iron Lung. Indeed some authors believe that ventilation of these patients in the IL is contraindicated, and suggest rather that the aspiration risk should be avoided by use of a tracheostomy instead of almost continuous suctioning. Some American authors recommend that a tracheostomy be carried out at the first sign of the posterior cranial nerves becoming affected. Yet even larger Centres, with greater experience, for example that run by Stimson, remain opposed to use of the tracheostomy and never employ it. The greatest supporter of the tracheostomy is Galloway, who in 1943 rescued two children with severe swallowing paralysis by its use. By 1949 he reported using a tracheostomy in 15 cases of swallowing impairment, all of whom recovered. He compared his statistics with those of other American hospitals, where the tracheostomy was not employed, and who had a lethal outcome in 30-40% of cases where swallowing was paralysed. Galloway suggests the following indications for use of a tracheostomy:

1. Progressive hypoxia through secretions in the upper airway
2. Loss of consciousness or particularly severe restlessness in the patient which does not respond after a short time to other treatment.
3. Pronounced restlessness or stupor of patients who are mechanically ventilated within the Iron Lung with pure spinal paralysis
4. In patients where there is no way of removing upper airway secretions
5. Bilateral paralysis of the vocal cords
6. Rapid progression of bulbar syndromes
7. Severe signs of vasomotor failure
8. Insufficient nursing staff or equipment, or poor cooperation on the part of the patient.

In questionable cases a tracheostomy should be performed rather than a wait-and-see approach taken.

Patients with swallowing paralysis require constant observation and detailed care, almost continuous suctioning of secretions from the mouth and pharynx to avoid aspiration, eventually through means of a tracheostomy, careful nourishment

which may be intravenous or rectally during the acute phases, antibiotic treatment to avoid lung infection. Should all these aspects of care be fulfilled, then the prognosis is very good.

3. Swallowing Impairment in Combination with other types of Paralysis Leading to Hypoxia -

In these cases the prognosis is poor. The majority of our cases died due to pulmonary complications, brought about by the double insult of respiratory muscle paralysis and swallowing impairment. Our conclusion is that in these cases lung complications should be avoided through use of a tracheostomy and prophylactic Penicillin injections.

4. Circulation and Respiratory Center Involvement -

These cases are currently untreatable with contemporary methods. The prognosis is universally grim. Sarnoff (pers. comm.) suggests that using electronic stimulation of the Phrenic N. may produce a good outcome in cases where the Respiratory Center is only temporarily affected. We have intermittently attempted this in our patients, sometimes at high voltages, in an effort to prolong the life of the patient, even for a few hours hoping for an abatement in symptoms.

Unfortunately to date we have had no success.

Patients with Respiratory and/or Circulation Center involvement are frequently not in a condition to tolerate the rhythmic ventilation of the Iron Lung. Bower recommends the use of Curare at a dose of 0.9-1.5E intercostally, 2-3 times a day, to offset the irregular breathing impulses from the damaged Nerve Center.

Of our 19 Cases treated in the Iron Lung, 9 died. Of the 10 surviving patients, 5 either made a complete recovery or were minimally affected by the disease long-term, and are currently able to work in any field. Five other Cases remained disabled to varying degrees, but with significant impairment nevertheless. This follows the observations of Laruelles, that half the patients treated in an Iron Lung do not survive, and of the half that do, half of those will continue to suffer permanent disability to some extent.

We therefore have to reduce our initially high expectations of Iron Lung treatment, if only a quarter of those treated by mechanical ventilation achieve a full, or near full recovery. Despite those outcomes, the Iron Lung remains an advance on the earlier means of mechanical ventilation such as the Biomotor, where, as reported by Fanconi, Zellweger and Botsztejn, there remained a 60% mortality rate.

To conclude, I would like to give my warmest thanks to Professor G. Fanconi for the use of material.

Warm thanks also to Dr. H. Zellweger for initiating this study, his valuable counsel and advice, and his always kind and generous support.

Glossary

Pyramidon: Phenacetin, developed 1883. An effective treatment for mild to moderate pain, and fever. Unfortunately a renal toxin

Gold Sol Test: A test of the ability of globulins in the CSF to precipitate gold from colloidal solution. A positive result indicates infection, initially developed to diagnose syphilis.

Pandy Test: A test to detect elevated levels of globulins in the CSF. Named after Pandy Kalman, the Hungarian neurologist. A positive result indicates only that a pathological process in the CNS is occurring.

Pollakisuria: Frequent urination, but in very small amounts.

Curriculum Vitae

I was born in Budapest on 30th August, 1914, and attended Primary and Secondary school there, obtaining my Matura. Due to the war I was unable to attend University until 1945, when I enrolled in the Medical Faculty at Budapest University, completing 7 semesters. In 1949 I left Hungary, continuing and completing my studies at the University of Zurich.