The Anæsthetist's Viewpoint on the Treatment of Respiratory Complications in Poliomyelitis During the Epidemic in Copenhagen, 1952

By Dr. Bjørn Ibsen

Consultant Anæsthetist

Blegdam Hospital, Copenhagen, Denmark

On August 25, 1952, I was called in as anæsthetist for consultation by Professor Lassen in the Epidemiological Hospital of Copenhagen.

Within the preceding three weeks there had been 31 patients with bulbar poliomyelitis treated in respirators—tank as well as cuirass. 27 had died.

Four patients were seen in the autopsy room that day. One of them—a 12-year-old boy—had died in a respirator with a blood pressure of 160 and a bicarbonate level in the serum far above the normal level. The lungs did not appear to be sufficiently at electatic to make adequate ventilation impossible.

With enthusiastic encouragement from Professor Lassen I tried to demonstrate on a patient how sufficient ventilation could be administered without the help of a respirator. A patient in a very bad condition was chosen. She was a 12-year-old girl who had paralysis of all four extremities. She had atelectasis of the left lung and was gasping for air and drowning in her own secretions. Her temperature was $40 \cdot 2^{\circ}$ C. She was cyanotic and sweating.

A tracheotomy was done immediately under local anæsthesia, and a cuffed endotracheal tube put in place through the tracheotomy. During this procedure she became unconscious. A to-and-fro absorption system was connected directly to the tube, with good endotracheal suction. Even then it was impossible to inflate the lungs, partly due to secretions and partly due to bronchospasm. In this desperate situation I gave her 100 mg. Pentothal i.v. in the hope that I could stop her struggling. She collapsed, her own respiration stopped and I found that I could now inflate her lungs.

Shortly after this a device for continuous measurement of the carbon-dioxide concentration in the air from one of the main bronchi—a Brinkman Carbovisor—and an Oximeter of the Millikan type, was put to work.

By these means it was shown how under-ventilation gave rise to a CO₂ accumulation—even when full oxygenation of the blood was maintained with pure oxygen.

The usual signs of CO₂ retention could now be demonstrated. There was a rise in blood pressure. The skin became clammy and sweating; and the patient started her own respiration, which soon became gagging and bucking—a well-known sign we had learned to recognize from some of the other patients seen in the respirators. Secretions began to pour out of the mouth and the nose. These symptoms could be relieved within a few minutes, when CO₂ was removed by increasing the ventilation.

Another important point could be seen in this patient. As soon as CO₂ was removed the blood pressure dropped to 80—and the patient appeared to be suffering from shock. Blood was given which improved her condition remarkably. She became warm, dry and pink—a condition which always makes an anæsthetist happy.

X-rays showed that the atelectatic left lung had been inflated completely.

So far, the patient had been improved by measures usually carried out by the anæsthetist during his daily work in the operating theatre: Active securing of free airway, adequate assisted ventilation and intravenous treatment of shock.

The patient was now put in a respirator. All the signs of under-ventilation occurred, and she once again got cyanotic. Administration of oxygen in the same way as to other patients in respirators revealed that her colour could be improved—and nearly full oxygenation ensured—but the Carbovisor showed a continuous rise in CO₂.

When the respiration was assisted in the respirator by intermittent squeezing of a bag connected with the tracheotomy tube, everything went well again. In this patient we had to continue the manually assisted respiration, because it was impossible for the respirator to maintain sufficient ventilation to secure the necessary elimination of CO₂. The reason was thought to be that the patient already had too many lung complications, thus making the respirator inefficient. The aim should be to protect the lungs against secretions, before the patient is put into a respirator. Our experience from a couple of late emergency tracheotomies, done under local anæsthesia, confirmed this.

The operation itself was difficult—performed on the bed or in a respirator. The patients were apprehensive and unco-operative probably due to anoxia. Bleeding into the lungs could not be avoided. During the handling of the trachea, anoxia was further aggravated due to spasm and reflexes. Finally some of the patients suffered from severe vomiting and aspiration during this procedure.

These emergency tracheotomies done late in the disease are almost useless. I do not remember many patients who survived a late procedure more than a few days.

Accordingly, it was felt necessary to have an endotracheal cuffed tube, which had been passed through the mouth, lying in the trachea before the tracheotomy could be performed. To accomplish this a general anæsthetic frequently had to be administered.

A free airway was thus maintained during the operation, and the respiration could be assisted as well, if needed. The surgeon had quiet operational conditions. The bleeding could be under control before the tracheotomy tube was inserted through the tracheotomy opening.

The first patient operated upon in this way, was found the next morning to have been seriously over-ventilated by the respirator during the night.

It was now possible to outline the principles of treatment:

- (1) Any period of anoxia has to be avoided.
- (2) When necessary a tracheotomy should be performed and a cuffed endotracheal tube inserted through the tracheotomy, in order to protect the lungs against secretions from above—allowing effective endotracheal suction—thus securing a free airway.
- (3) The tracheotomy should be preceded by oral intubation. Most often this had to be done under general anæsthesia.
- (4) Inadequate ventilation could be assisted when necessary by connecting the tracheotomy tube with a to-and-fro absorber system, and then manually performing an intermittent positive pressure ventilation. This method was suitable for patients actually in respirators as well as out of them.
- (5) Shock should be treated with intravenous infusions—blood and serum, or vasopressor drugs, after the same principles as in surgical shock.

To carry out this treatment on all patients it was necessary to call upon additional staff. All available anæsthetists and medical students were mobilized and after eight days a big organization was working.

All patients with respiratory problems were collected in a special department, where they were under constant observation by a team, consisting of the epidemiologist, the ear, nose and throat surgeon, and the anæsthetist, all working with help from an excellent and capable laboratory. Later on radiologists and physiotherapists also helped.

There were in the department three floors, each with 35 patients, most of whom had their own room.

In order to secure continuity in treatment, conferences were held every day at which all problems were discussed. Specialists were invited to attend these conferences—physiologists, cardiologists, neurologists, &c.

Patients from the general wards—there were 500 beds, and 50 to 60 new cases coming every day—were transferred to the special department when they showed any of the following signs of respirator impairment: Difficulty in swallowing. Accumulation of secretions in the airway. A weak and insufficient cough, when the intercostal muscles or especially the diaphragm became paralysed. Paresis of the upper extremities. Progressive paresis moving upwards. Marked encephalitic signs.

A sudden vomit with aspiration could alter the situation in a few seconds, and make the use of the equipment necessary. A spare set of this equipment was kept ready on each floor, in case of failure of a patient's own set. This precaution has more than once proved to be life-saving.

Experience shows that the equipment to perform an artificial emergency respiration, and suction too, should be available within a reasonable time to every patient in respiratory trouble—even in his own home. During the polio epidemic, it became the principle, that this help should come to the patient, and not the patient to the help. Thus we went out by ambulance or by plane into the country, and rendered many patients capable of being transported.

In the observation department the patient's condition, and the indications for the different types of treatment, were evaluated. Patients were divided into wet and dry cases, according to the amount of secretion in the airway. They were also divided into a group, where the respiratory muscles seemed to be able to perform an adequate exchange when free airway was present, and a group who were thought not to be able to do so. The dry cases with sufficient ventilation, very few in number, were merely kept under observation. The dry cases with insufficient ventilation were most often put in a tank or cuirass respirator, in order to give them a chance to get through without a

tracheotomy. Only too often this was not possible. I remember only two patients who were put dry in a respirator, and remained dry. The wet cases who seemed to have sufficient muscle power to maintain an adequate exchange, were put in postural drainage. We tried to keep the airway free, but very often a tracheotomy had to be done, in order to maintain a free airway. This was especially the case in children, where there was little co-operation for suction and the drainage position was difficult to maintain.

The wet cases with insufficient ventilation had tracheotomy immediately, and were put on artificial respiration, usually administered through the tracheotomy tube.

As a rule we did not dare to put wet cases in respirators without a preceding tracheotomy. The few times we tried to do so we always regretted it.

In many cases where we were in doubt whether the impaired respiration was mainly due to secretions, or to decreased ventilatory muscle power, we did a tracheotomy. When the airway was made as free as possible, it was then decided whether the patient could be left in postural drainage, or had to have artificial respiration. Most patients had to have the latter.

The choice of anæsthesia for the tracheotomies was a difficult one. Pentothal and Scoline were used in the beginning, but were given up after one emergency, with cessation of the heartbeat of a patient in a tank respirator. I am not sure this was fair to the method. We got it started by heart massage and the patient improved but died the next night due to blocking of the tubes by secretions from a many days old complete atelectasis of the lung.

Later on cyclopropane was used. No premedication was given. Very often the patient stopped spontaneous breathing as soon as oxygen was given and had to be anæsthetized by squeezing the cyclopropane down into them.

A high tracheotomy was performed in order to stabilize the tube as much as possible in the trachea. A special device to maintain the tube in place was invented by one of the E.N.T. surgeons. The tube was changed when it was felt necessary. For practical reasons it was not possible to arrange a regular change. To our surprise, however, these rubber tubes gave rise to very little local irritation in the trachea.

The first hour after the tracheotomy was always very difficult. The reflexes might become exaggerated with the onset of pulmonary cedema. We were rather afraid of giving intravenous fluids during this time, before the tube was firmly in place.

The manual artificial respiration was done with a to-and-fro absorber system with a flow of 5 litres per minute and a mixture of 50% nitrogen and 50% oxygen. Since we had to use medical students, it was felt that this system had some buffer effect:

CO₂ excess could not develop if the absorber did not work.

Oxygen poisoning was not likely to occur.

Oxygen surplus was given to help in shock and pulmonary complications.

The high flow secured against low oxygen due to re-breathing.

There was no trouble with valves.

Drying out of the mucous membranes was probably not a problem as long as condensed water was present in the breathing bag.

Most of the patients had a stomach tube passed through the nose just after admission to the observation ward. The tube was left in place in patients who could not swallow, and used for giving food. Paralytic ileus was a common complication, which made the stomach tube a most valuable therapeutic measure in order to release the pressure on a weak diaphragm.

The cuffed rubber tube in the tracheotomy was replaced with a silver cannula as soon as possible. The E.N.T. surgeon decided when, by following the patient's capacity for swallowing. This we learned to respect. In some patients who were doing well on their own spontaneous respiration the chance was taken too early. The patients got atelectasis due to aspiration, and had to fall back into artificial respiration for weeks.

It was amazing, however, to see how the result could be improved by a very simple technique familiar to all anæsthetists. As soon as a patient is in trouble in a respirator he should—if the trouble cannot be corrected—have help by some other means. In Copenhagen it was artificial respiration through a cuffed tube inserted through a tracheotomy. The technique of performing this artificial respiration manually or mechanically can always be discussed and improved upon. But by the very simple technique used in Copenhagen, it was made possible, during the grave epidemic, to provide artificial respiration for every patient who needed it.