ANAESTHETIC MANAGEMENT OF A VERY PREMATURE INFANT

Case Report

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SUMMARY

Anaesthesia and surgery for premature infants is becoming more common and presents additional hazards in an already precarious life. Attention to detail is mandatory in the management of these infants whose immature physiological systems have a limited ability to compensate for homeostatic imbalance. Body and skin temperatures, fluid balance, ventilation, inspired oxygen concentration and acid-base status should be controlled. Meticulous monitoring is important before, during and after surgery. A rational choice of anaesthetic agents and techniques can be made from a knowledge of the modified effects and altered metabolism of drugs in premature infants. Anaesthesia for an intrathoracic and an intra-abdominal operation on a premature infant is described. Complicating conditions were recurrent apnoea requiring 3 weeks of intermittent positive pressure ventilation, and a patent ductus arteriosus with severe congestive cardiac failure, and pneumothorax.

The attitude of the physician toward the ultimate useful life of the exceedingly premature infant is rapidly changing from pessimism to optimism. Consequently, these infants are undergoing surgical procedures more frequently, and the anaesthetist should be familiar with the differences and the unique difficulties encountered. This report describes the clinical course of a very premature infant who underwent two major surgical procedures, and illustrates the points of interest and importance concerning preoperative, intraoperative and postoperative management.

CASE REPORT

A female infant was born at 28 weeks gestation after a pregnancy complicated only by the premature delivery. Her birth weight was 702 g. On admission to the intensive care nursery she was placed in an incubator with servo temperature control. Pulse rate, electrocardiogram and respiration were monitored by a Beckman vital signs monitor. An umbilical artery was catheterized. On the 2nd day of life she began to have periods of apnoea. Initially, 50% oxygen in the inspired atmosphere prevented further episodes. Over the next 24 hours, however, the apnoeic periods returned and gradually became prolonged until there was complete apnoea. The trachea was intubated with a 3-mm i.d. clear plastic nasotracheal tube, which was then connected to a Bennett PR-2 mechanical ventilator. Respiration was controlled at 18 b.p.m. with a peak airway pressure of 22 cm H_{2}O and inspired oxygen concentration of 28%, as measured by a paramagnetic oxygen analyser. The ventilator settings were adjusted according to arterial blood pH, P_{a}O_{2} and P_{a}O_{2} values. Throughout the period of continuous mechanical ventilation an attempt was made to keep the pH_{a} within the range 7.40–7.55, P_{a}O_{2} 30–40 mm Hg and P_{a}O_{2} 60–100 mm Hg. On the same day, for treatment of hyperbilirubinaemia she was given a 2-volume exchange transfusion. On the 4th day of life, because the infant still required continuous mechanical ventilation, a gastrostomy was performed so that early feeding could be started. The next day a murmur characteristic of patent ductus arteriosus was heard. The clinical course over the next 9 days was encouraging, and by the end of this period for a substantial part of each day she was breathing without aid from a ventilator. Subsequently, however, her condition deteriorated progressively. This was manifested by increasing intensity of the murmur, the onset of irregular cardiac rhythm, a slight opacification of the lung fields, and an increase to 30 cm H_{2}O in the required peak inspiratory pressure. On the 16th day, she was digitalized with digoxin 0.03 mg. The maintenance dose was 0.01 mg/day. On the 21st day she weighed 500 g, which was her lowest weight. The following day she developed frank congestive cardiac failure, with a gain in weight of 80 g (16%), an enlarged liver, a marked increase in the opacity of the lung fields, and an increase to 40 cm H_{2}O in the required peak airway pressure. Two days later, on the 24th day of life, she developed a small pneumothorax, which was drained successfully using an under water seal chest tube. She developed frank pulmonary oedema the next day in spite of a sixteenfold increase in the dose of intramuscular digoxin (0.08 mg twice a day). After much discussion, surgical ligation of the patent ductus arteriosus was elected as the only alternative to death.

Surgery was performed on the 26th day of life. The patient weighed 608 g. The infant was transferred to the operating room, with the nasotracheal tube in place. In transit, manual ventilation with oxygen was maintained without aid from a ventilator. Subsequently, how- ever, her condition deteriorated progressively. This was manifested by increasing intensity of the murmur, the onset of irregular cardiac rhythm, a slight opacification of the lungs, and an increase to 30 cm H_{2}O in the required peak inspiratory pressure. On the 16th day, she was digitalized with digoxin 0.03 mg. The maintenance dose was 0.01 mg/day. On the 21st day she weighed 500 g, which was her lowest weight. The following day she developed frank congestive cardiac failure, with a gain in weight of 80 g (16%), an enlarged liver, a marked increase in the opacity of the lung fields, and an increase to 40 cm H_{2}O in the required peak airway pressure. Two days later, on the 24th day of life, she developed a small pneumothorax, which was drained successfully using an under water seal chest tube. She developed frank pulmonary oedema the next day in spite of a sixteenfold increase in the dose of intramuscular digoxin (0.08 mg twice a day). After much discussion, surgical ligation of the patent ductus arteriosus was elected as the only alternative to death.

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using a 1-litre bag and a 3 L/min flow. Electrocardiographic leads were connected, an oesophageal stethoscope and a rectal thermometer were placed, while the patient was lying on a warm water blanket on the operating table under an infra-red lamp. Rectal temperature just before induction was 38.2°C. The haematocrit was 38%. The patient was placed in the right lateral position, and intubation of the trachea was begun with 0.2% halothane in 4 L/min oxygen. A gradual increase to 2% over a period of 20 min was needed to prevent movement. An unmodified Ayre's T-piece was used, with ventilation controlled by intermittent occlusion of the outlet. Care was taken not to cool the child by excessive preparation of the skin. The uncomplicated operation of transthoracic ligation of the wide open ductus arteriosus lasted 55 min. Ventilation was controlled at 30 b.p.m. Heart rate varied from 110 to 130 beats/min. Blood gases determined from a blood sample taken from the umbilical arterial catheter after 40 min of anaesthesia revealed a slight alkalosis: pH 7.53, standard HCO3 m.equiv./l. base excess +6, Pco2 38 mm Hg, Po2 240 mm Hg. During the procedure rectal temperature fell to 36.7°C. Blood loss was estimated by weighing sponges. As blood was lost, settled cells were infused in 2 ml increments, to a total of 6 ml. The only other fluid administered was 3 ml of 5% dextrose in water given via the central venous line pump to maintain the patency of the arterial catheter. A chest tube was placed in the left chest to drain the pleural cavity. Postoperatively the patient improved markedly. No digitalization was necessary. By the 9th postoperative day she was able to breathe spontaneously. At first only 1 minute of spontaneous ventilation was tolerated every hour. Initially 5 cm H2O end-expiratory pressure was used during the intervals off the ventilator. Over the 4 weeks following the beginning of spontaneous ventilation, she showed a satisfactory gain in weight, achieving 1020 g. After 11 weeks she required no mechanical assistance to respiration and her weight was 1620 g.

Six days after mechanical ventilation was stopped, she developed small bowel obstruction. Feeds via the gastrostomy were not retained and no stool was passed. The same day, aged 97 days and weighing 1600 g she underwent division of adhesions and jejunoplasty under general anaesthesia. Awake nasal intubation (Rees, 1960) was performed in the operating room. The anaesthetic and monitoring methods were the same as for the previous operation. A total of 25 ml of isoflurane in 95% oxygen was used during the surgery, which lasted 2 hours. Blood loss did not require replacement. The blood gas values half an hour after surgery were the same as those obtained preoperatively.

Postoperatively, mechanical ventilation was continued until all possible effects of anaesthetic agents had been reversed. Three hours after the end of surgery, the nasotracheal tube was removed. Subsequent progress was satisfactory for 2 weeks except for hypercarbia with a compensatory metabolic alkalosis. Chest radiographs showed diffuse patchy infiltrates and focal areas of emphysema, which is a characteristic picture of the lungs of premature infants mechanically ventilated with oxygen for prolonged periods (Northway, Rosan and Porter, 1967). Unfortunately, despite successful cardiac and abdominal surgery and anaesthesia, there was a woeful end to the story. Aged almost 4 months, 3½ weeks after her second operation, the patient had a fit. At this time also retrolental fibroplasia was diagnosed. Initially, the infrequent mild seizures were controlled by 23–25% oxygen.

The cause was never found, the e.g. being normal, but it was surmised that there was diffuse hypoxic brain damage. The patient died of respiratory insufficiency aged 4½ months, 20 hours after her physicians had decided that further treatment was not in the best interest of the patient and her family.

Postmortem examination showed an essentially normal brain and complete bilateral retinal separation. The lungs were characteristic of those of premature infants treated with oxygen and prolonged positive pressure ventilation, having linear areas of atelectasis and focal areas of hyperexpansion. There was evidence of right and left ventricular hypertrophy, and congestive hepatomegaly and splenomegaly. The kidneys had periglomerular fibrosis and acute and chronic inflammatory infiltrates. In addition, the para-pancreatic lymph nodes were acutely involved, which suggests that the pyelonephritis may have been contributory to the generally poor condition of the patient.

The fact that the pathologists found signs of hyperoxia is disconcerting since every effort was made to keep Pao2 below 100 mm Hg. It may be that infants with this degree of immaturity are more sensitive to normal oxygen levels. On the other hand, the blood was sampled from the aorta and perhaps the oxygen tension in the retinal arteries was considerably higher.

The infant died of respiratory failure at a time when she was having some mild seizures. Other diagnoses present were acute and chronic pyelonephritis, severe toxoaemia with enlarged splenic and para-pancreatic nodes and intrafollicular necrosis; also reticulo-endothelial hyperplasia of the liver and evidence of severe pulmonary hypertension.

**DISCUSSION**

The purpose of this report is to emphasize the adaptations of anaesthetic and monitoring techniques and the possible complications that pertain to very premature infants during anaesthesia. The requirements of anaesthesia for newborn babies have been described already (Rees, 1960; Bachman, 1965; Smith, 1968; Rackow, and Salanitri, 1969; Gilman, 1970). These operations should not be undertaken unless an experienced team can be assembled. We can imagine no such operation that could take 6½ hours (Ward, Crawford and Stevenson, 1970). Finally there must be a reasonable chance of definitive, therapeutic or curative surgery, not palliative.

**Diagnosis.**

The diagnoses in the case described were recurrent apnoea of the newborn, pneumothorax and patent ductus arteriosus. The first two were being treated successfully. The cardiac failure secondary to the patent ductus was life-threatening and could be treated only by surgery.

**Recurrent apnoea of the newborn.** Normal respiration in the premature infant is irregular. The frequency range is 40–100 b.p.m., with no expiratory pause. Many infants develop a rhythmic pattern
of respiration called periodic breathing, consisting of several rapid breaths for 10–15 seconds followed by periods of apnoea of 5–10 seconds. The cause is undetermined, but it appears that the premature brain cannot integrate all the chemical and non-chemical stimuli (Avery, 1968). The infants have a low PaO₂ and a low PaCO₂, and periodic breathing can be prevented by adding oxygen or carbon dioxide to the inspired mixture. The concentration is titrated in each patient. When the period of apnoea is longer than 20 seconds it is called an apnoeic spell. At first these spells can be terminated by external stimulation. If the heart rate slows early during the apnoeic period, it is unlikely that tactile stimulation will start spontaneous respiration. When such episodes occur, many patients develop prolonged or total apnoea. Continuous support by mechanical ventilation is now an accepted treatment. The length of time that mechanical ventilation is necessary can only be determined by trial and error.

**Pneumothorax.** The pneumothorax in this case was probably secondary to the uneven distribution of gas in the congested lungs and the increasing positive pressures used to inflate them. To treat tension pneumothorax a chest tube is inserted into the pleural cavity, and connected to water seal drainage or low suction. This treatment is almost always successful in infants. Chest radiographs should show no air in the pleural cavity and complete expansion of the lungs.

**Patent ductus arteriosus.** Persistent patent ductus is relatively common in premature infants, especially in those who develop respiratory distress or who require prolonged mechanical ventilation (Kitterman et al., 1970). Usually therapy other than digitalization is unnecessary. In our case there was cardiac failure from the 16th day of life and uncontrollable left- and right-sided failure on the 22nd day evidenced by sudden weight gain, decrease in lung compliance and descent of the liver margin.

**Premedication.**

Once surgery is elected, meticulous preparation for monitoring, transfer to the operating room, and anaesthesia begins. Whether or not to give atropine for premedication must be considered (Wilton, 1960). The vagal reflex in the premature is relatively hypoactive (Vallbona, Rudolph and Desmond, 1965) and the dangers of thickening lung secretions in our opinion outweigh the advantages of the use of atropine. Sedatives and narcotics are neither necessary nor safe in the newborn infant.

**Tracheal intubation.**

Nasotracheal is preferable to orotracheal intubation because the tube is more stable, easier to fix, less likely to kink, does not stimulate oral secretions and may be left in for several days if necessary. Uncuffed nasotracheal tubes of a non-irritant material and internal diameter more than 2.5 mm are recommended. For this 600-g infant we were able to use a 3.0-mm i.d. tube initially and then, as she grew, a 3.5-mm i.d. A Cole tube is an unwise choice since the “shoulder” may damage the larynx and vocal cords if the tube must remain in place for more than 12 hours. Before insertion the tube should be cut to the appropriate length and fitted snugly to a connector of similar internal diameter. The expected length for a patient weighing less than 1000 g would be 8.5–9.0 cm without connector. Fixation should be simple yet prevent repositioning or dislodgement of the tube upon normal movements of the baby (Smith and Daily, 1971). The advantage of prior selection of length and firm fixation is that the tube cannot change position relative to the carina, oesophagus or pharynx. We recommend awake intubation, fixation of the tube, and verification of its position by chest radiography before transfer to the operating room, for any premature or acutely ill newborn baby.

Specifically short laryngoscope blades are made for premature infants. We used a Miller O. The blade is straight and has the light close to the tip, but more important than that, the thickness of the blade between the gums is small (6 mm versus 9–11 mm) and thus manipulative trauma is reduced. We cannot recommend a curved blade (Macintosh) since the greater thickness and its configuration are not suited to the premature infant.

**Monitoring.**

In a newborn intensive care unit the atmosphere of each patient is carefully controlled and adapted. Modern electronic monitoring provides continuous information without disturbing the infant. When surgery is elected, some of the monitoring equipment is replaced by the anaesthetist. Support and monitoring by a team of nurses, paediatricians, technicians and machines must now be entrusted to the anaesthetist who can anticipate sudden changes in physiological parameters, which are liable to occur secondary to the surgical procedure. In the operating room it is advantageous for one anaesthetist to watch the monitor equipment, relay salient information, and keep an anaesthetic record, while
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another cares for the infant beat-to-beat and breath-to-breath (Wilson et al., 1969).

Since the total blood volume of a 600-g infant is only approximately 60 ml, special care must be taken in recording volumes lost and given. Blood lost via suction can be measured in a small graduated cylinder inserted into the suction line. Short small-bore suction tubing makes the measurement more accurate. Swabs placed along the suture line to prevent blood loss among the towels, and dry swabs used must be weighed. It is our policy to replace blood as it is lost and to infuse no more than 9 ml/kg/hr of fluid via the constant infusion pump in addition to this.

The monitoring and maintenance of temperature is a frequently discussed problem. Special care, however, is required in the premature infant. An adult rectal thermistor probe is clearly not acceptable. The gross stretching of the bowel by the thermistor probe in our patient could have easily resulted in perforation (Greenbaum et al., 1969).

Agents and techniques.

Some authorities (Smith, 1965; Mustard, Bedard and Trusler, 1970) feel that paralysis, oxygenation, intubation and general support are all that are necessary for management of an anaesthetic for an infant. Others, including the authors, believe that analgesia and hypnosis are also important (Swafford and Allan, 1968).

Local anaesthesia has been used frequently for abdominal surgery in premature newborn infants. Toxic levels, breakdown rates and long-term effects on developing tissues are not known. It is known, however, that high blood levels in the foetus produce severe bradycardia and that overdosage in the newborn causes seizures and respiratory depression. Unfortunately, during prolonged anaesthesia with local agents, it is sometimes necessary to use a nitrous oxide/oxygen mixture by facepiece in addition, to prevent movement of the infant.

Although nitrous oxide is a good analgesic, it probably should not be used in infants undergoing lung surgery or repair of cardiac defects because high oxygen concentrations may have to be given to prevent hypoxia (Strong, Keats and Cooley, 1966). Nitrous oxide-oxygen with a narcotic, neuroleptic, sedative and/or neuromuscular blocking agent currently is a popular anaesthetic technique. The detoxification, breakdown and elimination of these drugs require mature enzyme systems and renal function. Thus use of such drugs is not advised in premature infants (Eitzman, 1968). Depolarizing and non-depolarizing muscle relaxants should rarely be necessary, since the musculature of the premature infant is underdeveloped. Deepening of the anaesthesia is all that is necessary to abolish active movement. Rarely, when high blood levels of gaseous anaesthetic agents are not tolerated, non-depolarizing agents may be used.

Potent inhalation agents have the advantage that nearly 100% oxygen can be given, and that they are hypnotics, analgesics and muscle relaxants. Their effects on developing human tissue are not known. Unless 100% oxygen is advisable for prevention of hypoxia and acidosis it should be used carefully, for a Pa02 of more than 120 mm Hg appears to be deleterious to developing lung and retina.

An anaesthetic system should prevent accumulation of carbon dioxide and any increase in work of breathing by resistance to inspiration or expiration during spontaneous ventilation. An Ayre’s unmodified T-piece satisfies these requirements. Ventilation should be controlled for all premature infants, regardless of the system chosen. Manual intermittent positive pressure ventilation can be achieved by occluding the open end of the T-piece. When the lungs are visible through an incision this technique is safe and in our case it was an advantage to have manual control and not mechanical, since the upper lung was almost completely compressed by retractor.

The inspiratory time and degree of lung inflation could be adjusted breath-to-breath to accommodate surgeon and patient. The main disadvantage of the open system for prematures is the cooling and drying effect of high flows of dry gases. A to-and-fro system is preferred from this point of view but it is unwieldy, and the paediatric soda-lime canisters are too large for premature infants. The Jackson Rees modification of the Ayre’s system (Rees, 1960) and the Columbia paediatric circle valve (Rackow and Salanitre, 1968) are highly acceptable. Unfortunately, both have bulky, heavy hoses and connectors, which, being placed between anaesthetist and patient, tend to divert the concentration of the anaesthetist from the baby to the equipment and monitors.

Postoperative care.

Postoperatively the anaesthetist must return the baby directly to the incubator, which should have remained in the operating room with its warming system on. Ventilation should be controlled on the way to the intensive care nursery and maintained
mechanically until all possible effects of the anaesthetic agents have gone, and rectal temperature and acid-base balance are back to normal. In the nursery the monitoring of vital signs, electrocardiogram, temperature and respiration should be continued. Pain relief is not necessary. Extubation should be performed after consultation with and under the supervision of the anaesthetist.

REFERENCES


CONDUITE ANESTHESIQUE CHEZ UN ENFANT TRES PREMATRE
RAPPOR D'UN CAS

SOMMAIRE

L'anesthésie et la chirurgie des prématurés deviennent plus fréquentes et constituent un risque supplémentaire pour un état de vie déjà précaire. L'attention à tous les détails est indispensable dans la conduite à suivre chez ces enfants, dont les systèmes physiologiques immatures ne sont qu'incomplètement capables de compenser le déséquilibre homéostatique. La température corporelle et cutanée, l'équilibre des liquides, la ventilation, la concentration d'oxygène inspiré et l'état acide-base doivent être contrôlés. Le monitoring mécanique est important avant, pendant et après la chirurgie. Un choix rationnel des agents et techniques anesthésiques peut se faire sur base de la connaissance des effets modifiés et du métabolisme altéré des médicaments chez le prématuré. Les auteurs décrittent l'anesthésie pour une intervention intrathoracique et intraabdominale chez des prématurés. Les complications étaient une apnée récidivante, nécessitant 3 semaines de ventilation à pression positive intermittente, et un ductus arteriosus avec insuffisance cardiaque congestive grave et pneumothorax.

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ZUSAMMENFASSUNG

Nevertheless, one must admit that some of the reasons for employing surgery in a few of the patients mentioned with an odd contribution from America and Turkey, mostly from Germany, Austria, Switzerland, to Japan, the up-to-date information from so many centres, ranging neurosurgeons, neurophysiologists, psychiatrists and psychologists will value the opportunity to have available all the summary of the Conference which is the devout purpose of this book. There is no doubt that many neurosurgeons, neurophysiologists, psychiatrists and psychologists will value the opportunity to have available all the up-to-date information from so many centres, ranging mostly from Germany, Austria, Switzerland, to Japan, with an odd contribution from America and Turkey. Nevertheless, one must admit that some of the reasons for employing surgery in a few of the patients mentioned in this book would not be tolerated by any worker in this country, certainly not to the best of the knowledge of the reviewer. This volume may be of interest to anaesthetists concerned with neurosurgery.

C. B. Sedzimir


Comment on a book which has achieved its fifth edition since its first publication in 1964 is really superfluous. The fact that the volume can achieve such success is itself an indication of its excellence.

The authors have taken the opportunity of the preparation of a new edition to benefit from constructive criticism which has been offered to them from many sources and in their preface acknowledge this. Indeed such minor revisions constitute the main changes which have been made in this volume as compared with its predecessor.

There is, however, one very small point which the reviewer would feel impelled to comment on. The authors mention the use of paraldehyde for rectal basal narcosis and in so doing accept the widely held belief that paraldehyde is not a dangerous drug for this purpose. Indeed experience of its intravenous use for the treatment of status epilepticus, a procedure which does not come within the purview of the present book suggests that it is just as dangerous as any other sedative drugs.

A R. Hunter