Effect of Intermittent Positive Pressure Ventilation on Cardiac Systolic Time Intervals


"It is well known that increased intrathoracic pressure affects the circulation...especially when applied in such clinical conditions as shock and cardiac decompensation...It would thus be of value if myocardial performance could be followed continuously in such patients during intermittent positive pressure ventilation (IPPV). Measuring the heart's systolic time intervals (STI) is a useful non-invasive method for the bedside assessment of changes in left ventricular contractility and stroke volume...."

"It has not, however, been used in patients undergoing ventilator treatment, and there is no information on the extent to which STI are thereby altered. The purpose of this study was to measure the effect of such ventilation on STI in subjects free from cardiac and pulmonary disease. The results have been analysed with reference to changes both in intrathoracic pressure and in arterial carbon dioxide tension...."

"Ten patients, aged between 28 and 63 years (four men and six women), were investigated before and during anaesthesia prior to surgery....The subjects were premedicated with 10 mg diazepam, administered intramuscularly 1 h prior to the commencement of the study. Anaesthesia was induced by means of a sleep dose of thiopentone, after which the lungs were ventilated with oxygen....75 mg of succinylcholine was given intravenously after the administration of 0.5 mg pancuronium. The vocal cords were sprayed with a 4% solution of lidocaine, and a wide-bore, cuffed Portex endotracheal tube was inserted.

"The patient was then ventilated with a mixture of halothane in low concentration and air-oxygen until regular spontaneous respiration was restored. After measurements under spontaneous breathing, the patient was ventilated mechanically by means of a volume-cycled ventilator....The concentration of halothane delivered to the patient was adjusted during artificial ventilation so that the expired halothane concentration remained constant and less than 0.8%, irrespective of minute ventilation.

"The systolic time intervals were measured using an electrocardiogram and a phonocardiogram, and by registering the pulse wave over the carotid artery. Variations in intrathoracic pressure were measured using an oesophageal balloon, 10 cm long and with a diameter of 2.5 cm, attached to a polyethylene catheter...."

"Oxygen and carbon dioxide tensions in arterial blood. P{\text{A}}O_2 and P{\text{A}}CO_2 were determined in samples taken from a teflon catheter (1.2 mm outer diameter) previously inserted into the femoral artery...."

"The cardiac output was measured
using a dye-dilution technique. Statistical analysis of the results was performed using Student's $t$-test for paired values.

"Ventilation and arterial blood gases." The respiratory frequency measured during spontaneous breathing before anaesthesia was $16 \pm 4$ breaths/min and oesophageal mean pressure $-4.2 \pm 1.8$ cm H$_2$O. A moderate tachypnoea was recorded following anaesthesia during spontaneous breathing. The oesophageal pressure was slightly higher than before the induction of anaesthesia. Pao$_2$ was higher in all cases. Paco$_2$ was moderately increased. IPPV with a respiratory minute volume similar to that recorded with spontaneous breathing under anaesthesia resulted in a raised oesophageal pressure.

"Central circulation." The heart rate recorded before anaesthesia was within normal limits. There was no significant change in the heart rate or in the cardiac output following the induction of anaesthesia. IPPV resulted in a reduction in heart rate, cardiac output and stroke volume, and increasing the respiratory minute volume resulted in a further reduction in cardiac output.

The object of this study has been to find out if, and to what extent, artificial ventilation alters STI. Since it is known that anaesthesia alters the circulatory homeostasis, care has been taken to ensure that this effect is both minimal and constant. Thus halothane has been used in the lowest possible concentration, a concentration which we have shown did not alter STI. It is known that altering respiratory minute volume will alter the uptake of a volatile anaesthetic agent, and hence the anaesthetic depth.

The lengthening in PEP with IPPV may be explained by a reduction in preload, a reduced myocardial inotropy, or by an increase in afterload. In this study, the institution of artificial ventilation resulted in an increase in mean oesophageal pressure and a further increase with artificial hyperventilation. Similarly, IPPV results in an increase of approximately 2 mmHg in both the right ventricular end-diastolic pressure, and in the pulmonary arterial wedge pressure. This suggests that the transmural pressures in both the right and left ventricles are lower with IPPV than with spontaneous breathing.

"The lengthening of PEP with IPPV may also be caused by a reduction in myocardial inotropy, due to the concomitant reduction in Paco$_2$. A correlation has been demonstrated between LVET and ETI and the relative magnitude of the stroke volume. In this study, IPPV caused a moderate reduction of the stroke volume. The individual values measured for LVET and ETI under such conditions were found to be lower, although the changes were not significant for the patient group as a whole.

In conclusion, systolic time intervals are affected by IPPV as a consequence of reduced preload, the reduction in inotropy and increase in afterload probably being subsidiary factors. The results of their measurement should therefore be interpreted with reference to the actual intrathoracic pressure.

Pressure and Volume Changes in Tracheal Tube Cuffs during Anaesthesia


"For more than 20 years it has been known that a closed gas pocket in the body will expand if it contains an insoluble gas. The effect will be similar if the gas present in the pocket is not quite insoluble in blood, but less soluble than the inspired gas. Nitrous oxide is 34 times more soluble in blood than nitrogen. Only recently has it been shown that gas diffusion is also important for tracheal tube cuff pressure and volume during anaesthesia.

"Excessive cuff-to-tracheal-wall pressure (C-T pressure) may cause ischae-
mic necrosis of the tracheal wall, often with subsequent tracheal stenosis and/or malacia. These complications have become an increasing clinical problem during the last 15 years. The present clinical study was performed in order to:

1. Study the pressure increase with time and volume changes within the large-resting-diameter, large-residual-volume cuff and the small-resting-diameter, small-residual-volume cuff during nitrous oxide-oxygen anaesthesia (70%/30%).

2. Show how the intracuff pressure reacts if the cuff is filled with nitrous oxide-oxygen (70%/30%) instead of air during nitrous oxide-oxygen anaesthesia (70%/30%).

3. Register the intracuff pressure of the air-filled cuff during halothane-oxygen anaesthesia, and the saline-filled cuff during nitrous oxide-oxygen anaesthesia.

"The intracuff pressure and volume of the tracheal tube cuff were registered in connection with anaesthesia during general surgery. The pressure was periodically measured with an ordinary sphygmomanometer, which was directly connected to the cuff inflating tube via a three-way stopcock and the shortest possible connecting tube. Volume was measured during pressure controls at the start and at the end of anaesthesia using a precision, all-glass syringe without silicone. The cuff volume was read to the nearest half ml and given at atmospheric pressure and room temperature. The t-test for paired data was used for statistical analyses.

"In groups... where the cuff was filled with air during nitrous oxide-oxygen anaesthesia, the pressure rose continuously with time. The pressure increases in the large volume and small volume cuff were of the same magnitude. When nitrous oxide-oxygen in the large volume cuff was coupled with nitrous oxide-oxygen ventilation, the change in intracuff pressure was insignificant. Under the same circumstances, the small volume cuff showed a minor pressure drop. In the group... [when] the small volume cuff was filled with saline, and the patient was ventilated with nitrous oxide-oxygen; almost no pressure changes were noted. In the group... [where] the small volume cuff was filled with air during halothane-oxygen anaesthesia... a slight pressure drop was registered.

"A major increase in volume was noted during nitrous oxide-oxygen anaesthesia if air was initially present in the cuff... but with nitrous oxide-oxygen in the cuff the volume remained unchanged. Cuff volumes were also unchanged with saline and during halothane-oxygen anaesthesia. It can be seen that high pressures are rapidly produced in the small volume cuff. With a large volume cuff no pressure is needed to keep the cuff expanded if the volume is less than 20 ml (8 mm ID).

"In 1955, Fink described a simple and illustrative experiment showing the importance of solubility for the pressure increase in a closed chamber. Nitrogen and nitrous oxide have about the same solubility in water as in blood.

"Discomfort in the throat is common after tracheal intubation, even when the utmost care is taken during manipulation in the throat. Minor damage to the tracheal mucosa seems to occur as a rule and major damage often leads to tracheal stenosis. It is apparent that diffusion of nitrous oxide into the cuff may occur to such an extent as to present some element of risk. The present observations of the magnitude and time of the pressure increase are in agreement with earlier reports.

"Thus, the volume may be doubled within a few hours. Even the large volume cuff cannot accommodate this increase in volume without a commensurately large rise in pressure. Our data clearly show that the increase in volume and pressure is strongly correlated to time.

"When the cuff is filled with nitrous oxide-oxygen, no increase in intracuff pressure occurs. The large-resting-diameter, large-residual-volume cuff (filled with the anaesthetic gas mixture...
being used) probably provides the simplest and most secure system for maintaining a constant, low, sealing pressure in the trachea during nitrous oxide-oxygen anaesthesia. Measurement of the intracuff pressure is important. . . . Cuff pressure in the large volume cuff is equal to the pressure against the tracheal wall.”

Advantages of Infant Ventilators Over Adapted Adult Ventilators in Pediatrics


“A lung simulator with variable compliance and resistance components was used to evaluate the dynamic compliance of the Bournes, Babybird, and Pediatric Emerson postoperative ventilators. . . . When mechanical ventilators are used for infants and small children, significant volumes of gas may be lost to the patient as airway pressure increases as a result of (1) compression of gas in the internal volume of the ventilator and (2) distention of the flexible tubing that connects ventilator and patient. As airway pressure rises . . . there will be a decrease in delivered volume. These volume losses may have significant effects on the minute volume and hence gas exchange in infants.

“We compared the volume losses from increased airway pressure in an adult ventilator adapted for pediatric use and in 2 ventilators specifically designed for infants. . . . Three infant ventilators were compared: the Emerson postoperative with pediatric piston and modified for IMV, a Babybird, and a Bournes. . . . Ventilator function was tested with a Bournes Infant Lung Simulator. . . . The simulator also allows changes in airway resistance from 50 to 500 cm H2O/L/sec by interchanging small metal filters proximal to the compliance component. In all studies, a 3.5-mm OD endotracheal tube was interposed between the ventilator and the lung simulator. Air was used as the gas in all studies.

“Peak airway pressure and lung pressure were measured with aneroid manometers connected to the simulator. These pressures were secondarily displayed on an oscilloscope that could be marked for calibration. . . . To test the accuracy of this pneumatic integrator, a 3.5-mm OD endotracheal tube was connected to a Fleisch #0 pneumotachograph. . . . The calculated internal compliance of the ventilator using the two differently derived volumes was essentially the same.

“The internal compliance of a ventilator is a measure of the volume lost within a ventilator system following rises in airway pressure during gas flow. The higher the internal compliance of a ventilator system, the greater the amount of gas it can accommodate without transferring this volume to the patient in response to an increase in pressure within the ventilator.

“The experiment was divided into 3 parts. In each part, the ventilators were adjusted to provide ± 100, 60, and 40 ml delivered lung volume at the lowest resistance and highest compliance on the lung simulator at a ventilator rate of 20/min. In part A, compliance was decreased in steps and resistance was increased to give 12 progressive increases in airway pressure. . . . In part B, airway resistance was kept constant while compliance was decreased in 4 steps from 10 to 1 ml/cm H2O, and again delivered volume was calculated. In part C, compliance was maintained constant at 10 ml/cm H2O, while airway resistance was increased in 7 steps from 50 to 500 cm/L/sec. For each volume in all 3 parts, a linear regression equation was derived from the calculated lung volume and airway pressures.

“In part A of this study, where both airway resistance and lung compliance were varied, the Bournes ventilator had the lowest internal compliance, the Babybird had twice this value, and the Emerson had 5 times this value at the several volumes studied. . . . The volume losses to the patient as peak airway pres-
sure rises are significantly greater with the Babybird and Emerson than with the Bournes; volume losses with the Emerson were greater than with the Babybird.

"When the airway resistance is constant, the Babybird and Bournes have comparable volume losses as airway pressure increases. . . . The Emerson has 4 to 5 times the volume loss of the other two. . . . With constant lung compliance and increasing airway resistance at all tidal volumes, both the Babybird and Emerson had significantly higher volume losses than did the Bournes. . . . A lung simulator allows variation in airway resistance and lung compliance separately, approximating the conditions encountered in a patient with pulmonary disease. Such a device provides a method for comparing the performance of various pediatric ventilators. . . .

"Relation between flow, pressure, and resistance in the airway is generally analogous to the relationship between current, electromotive force, and resistance in an electric circuit, as expressed in Ohm's Law. . . .

"No ventilator functions as a true 'volume' ventilator when used to ventilate infants. In part, compression and distention volume losses can be compensated for by increasing the preset tidal volume of the ventilator above the required patient tidal volume. This provides an adequate patient tidal volume when airway pressure remains constant. . . . One of the factors influencing the compression volume loss is the size of the internal volume of the ventilator. . . . There is a definite advantage in using infant ventilators (with small internal volumes) for the mechanical ventilation of infants rather than adapting adult ventilators for this purpose."

The Anaesthetist's Contribution to the Care of Head Injuries


"Head injuries continue to be a major medical and social problem. . . . Many of these accidents take place some distance from a neurosurgical unit, and anaesthetists are becoming increasingly involved in the problems of immediate and intensive care, as well as the anaesthetic management. They should therefore understand the physiological and pathological changes that can take place following head injury. The main problems are the diagnosis and removal of significant intracranial clots, the prevention of hypoxia, and the control of cerebral oedema and increased intracranial pressure (ICP) . . . .

"Following a severe head injury there is widespread neuronal damage and diffuse ischaemia . . . leading to tissue acidosis from increased formation of lactic acid. . . . It is essential to prevent further widespread cerebral damage that can occur as a result of a vicious circle of secondary factors which reduce the oxygen supply to the brain. . . . The primary lesion is aggravated by respiratory insufficiency, hypoxia, hypotension and anaemia, . . . hypercapnia, . . . volatile anaesthetic agents, . . . and careless anaesthesia, all of which contribute to neurological deterioration and a poor prognosis. . . .

"The establishment and maintenance of a clear airway and adequate oxygenation, from the time that the patient is first seen until there is sustained neurological improvement, is of fundamental importance. . . . The next priorities are to ensure an adequate circulation and to control haemorrhage so that cerebral perfusion pressure is maintained. . . . Once a clear airway is secured and an adequate circulation established, there is then time to examine the patient and establish and record base-lines of conscious level, vital functions and focal neurological signs. . . .

"The patient must be examined fully and carefully. Thirty per cent of cases with head injuries will have other injuries, 8% of them serious; these should be suspected in the presence of hypotension, which is rare in cases of closed head injury alone. . . . If there are fractured ribs, the anaesthetist must be
aware of the risk of a tension pneumothorax developing should controlled ventilation be instituted. Prophylactic insertion of chest drains is not advised because of the risk of direct lung trauma. . . . Before a patient is moved from the accident room, to either another department or another hospital, it is essential to ensure that there is a secure and clear airway, an empty stomach and an adequate circulating blood volume. . . .

“General anesthesia may be required for angiography, exploratory burr-holes, and craniotomy for the removal of intracranial haematomata. The same precautions and care as for any neurosurgical procedure will be required. . . . The patient should be examined carefully before anaesthesia so that any changes resulting from acute cerebral compression or concealed haemorrhage can be observed at once. If the stomach has not been emptied, full precautions against regurgitation should be taken, and a nasogastric tube passed when a cuffed endotracheal tube is in place.

“The type of induction of anaesthesia will depend on the level of consciousness, on the general condition of the patient and whether intubation has already been performed. . . . Brain swelling may not be checked adequately by controlled ventilation and hypocapnia because, although ICP may be decreased, the position on the pressure/volume curve may not be altered. . . . Patients with an apparently minor head injury may require anaesthesia for an extracranial procedure such as orthopaedic fixation or laparotomy; they present a particular hazard. A halothane anaesthetic, with spontaneous or controlled ventilation, may cause hypotension with severe increase of ICP resulting in cerebral ischaemia and herniation, and these cases should receive the same meticulous care that a neurosurgical procedure requires. . . .

“In most cases, provision of a clear airway with humidification and maintenance of \( \text{PaO}_2 \) greater than 10.6kPa (80 mmHg) is satisfactory. The blood-gases should be estimated at least every 24 h. Prolonged airway care will be necessary in those patients where lengthy coma is anticipated. . . . In some cases it may be decided that controlled ventilation should be continued for some time after operation. . . . Many neurosurgical units now record the ICP continuously as a guide to the effect of therapy and prognosis. . . .

“When the obvious causes such as hypostatic, infectious or aspiration pneumonia have been corrected, persistent hypoxaemia may remain a problem. . . . Hyperthermia increases cerebral metabolism and oedema and the body temperature must be controlled. . . . Controlled hyperventilation is now widely used as a method of reducing mortality and morbidity in severe head injuries. . . . Controlled hyperventilation should be considered when the ICP is greater than 4kPa (30 mmHg) with no adequate response to other therapeutic measures. . . .

“To be effective, controlled ventilation should be instituted early, but it is of no value in patients with severe head injuries who have had fixed dilated pupils for more than 24 h and who are flaccid, areflexic or have become apnoeic and have failed to establish satisfactory spontaneous ventilation in spite of energetic treatment. . . .

“Coughing, straining and suction all increase ICP, and breathing against the ventilator should be avoided. . . . Ventilation should be continued until there is maintained neurological improvement with spontaneous and purposeful movements, a sustained decrease in ICP, adequate spontaneous respiration and until arterial blood-gases are normal. There may be an abrupt increase in \( \text{Paco}_2 \) following discontinuation of prolonged hyperventilation. . . .

“Anaesthetists, with their particular interest in intensive care and the respiratory and cardiovascular systems, can contribute a great deal as members of a team concerned with the management of a severe head injury.”