THE USE OF NERVE BLOCKS FOR STUDYING CARDIOPULMONARY PHYSIOLOGY IN MAN

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Anaesthetists employ nerve blocks for purposes other than providing surgical anaesthesia; that is, for diagnosis, therapy, and clinical investigation. In this regard the anaesthetist is in a unique position to make physiological observations in normal as well as abnormal subjects during nerve block procedures. Areas of specific importance to both physiologists and clinicians concern the innervation of the heart and the lungs. Cardiac and pulmonary sympathetic blockade is frequently produced in the course of spinal and peridural anaesthesia. Less frequently, block of the vagus or phrenic has been performed for special situations. Certainly it is feasible to block selectively the sympathetic, para-sympathetic, and somatic nerves to the heart and lungs or the entire thorax. It is the purpose of this paper to bring together information obtained during nerve block procedures involving the heart and lungs and to suggest questions that could be answered by a concerted approach of anaesthetists.

PULMONARY PHYSIOLOGY

Spinal and peridural block.

High thoracic peridural anaesthesia blocks the thoracic sympathetic nerves—often up to T1. Such anaesthesia usually blocks the sensory innervation of the chest wall, the pleurae and, to a varying extent, the intercostal motor neurons. Many studies have shown that such blocks are well tolerated in normal conscious man. Moir (1963) demonstrated a small reduction in expiratory flow rate during high thoracic peridural block, but no impairment of ventilation or vital capacity. Similar effects have been noted during high spinal anaesthesia with the only change being reduction in the ability to cough due to paralysis of abdominal muscles (Egbert, Tammersoy and Deas, 1961). In another study with electromyographic evidence of complete motor and sensory blockade up to T1 using spinal anaesthesia, there was no change in ventilation as indicated by the arterial Pco₂ despite reductions in inspiratory capacity ranging from 42 to 80% (fig. 1) (Eisele et al., 1968). In this study four healthy subjects were able to detect added respiratory loads and demonstrated a normal ventilatory response to carbon dioxide rebreathing.

In evaluating the respiratory effects of high peridural or spinal block in patients with lung disease, there are very few available data. Bromage (1954) indicated that patients with severe emphysema and bronchoconstriction may improve during thoracic epidural block; however, there are no supporting data. Paskin, Rodman and Smith (1969) demonstrated only a slight impairment of forced expiration and a moderate increase in physiological deadspace in nine emphysematous patients during spinal anaesthesia which reached an average level of T7. They concluded that spinal anaesthesia is as well tolerated by patients with chronic obstructive pulmonary disease as by normal subjects. Moir and Mone (1964) evaluated blood gas changes in eight chronic bronchitic patients during epidural anaesthesia up to the T3 level and noted only a small rise in Pco₂ (+2.5 mm Hg) compared to a slight fall in Pco₂ in twelve normal

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USE OF NERVE BLOCK FOR STUDYING PHYSIOLOGY

One problem in evaluating studies on peridural or spinal anaesthesia is that all the subjects have not received the same nerve block, i.e. the same number of dermatomes. Bonica and associates (1966) have demonstrated that there is little difference between high spinal and epidural anaesthesia in terms of cardiovascular effects but respiratory influences were not studied.

The effects of high thoracic motor and sensory block on ventilation in patients with restrictive lung disease as opposed to obstructive lung disorders are not known. These studies raise an important question concerning the influence of thoracic block on airway mechanics. It seems probable that in the presence of an irritant or bronchoconstricting drug, blockade of the sympathetics would do more harm than benefit. It might have the same deleterious effect on airway resistance as the use of adrenergic blocking drugs when given to patients with obstructive airway disease. If airway resistance is increased and the ability to cough is impaired during high peridural anaesthesia, then this technique may be harmful for the patient with chronic obstructive airways disease and cough. Indeed, this aspect of airways resistance needs to be investigated during thoracic regional anaesthesia.

Another equally challenging aspect for investigation is the effect of thoracic sympathetic block on pulmonary vascular resistance. It would be particularly important to study patients with pulmonary hypertension. One haemodynamic study during high spinal anaesthesia (T1, T2) (Lynn et al., 1952) indicates an average reduction in mean pulmonary artery pressure of 28% as the cardiac output also fell by 28%. The transpulmonary arterial pressure (pulmonary artery minus left atrium), however, only fell 18%, which suggests that there was little or no vasodilatation of the smaller pulmonary vessels. In this study one of the five patients had chronic pulmonary emphysema and high spinal anaesthesia produced the smallest change in mean pulmonary arterial pressure (18.7 to 17.5 mm Hg) and essentially no change in transpulmonary arterial pressure.

In a study on newborn dogs Bauman and Fletcher (1967) suggested that high sympathetic block (1% lignocaine peridural anaesthesia) reduced hypoxia-induced pulmonary artery constriction. Most of the studies on this subject, however, indicate that hypoxia has a direct effect on the pulmonary vasculature. Bilateral vagal block has been shown in man not to influence the rise in pulmonary artery pressure when exposed to hypoxia (fig. 2) (Eisele and Jain, 1971). There is little or no information on sympathetic block in subjects with pulmonary hypertension and this should be an important area to study. Certainly it would be ideal to produce a selective sympathetic thoracic block using a dilute concentration of local anaesthetic. In practice this has been difficult to achieve; nevertheless, it should be pursued.

Vagus nerve block.

The entire pulmonary parenchyma including the airways is richly innervated with vagal fibres. Several receptors have been identified which respond to mechanical forces (inflation and deflation), specific chemical agents (phenyl diguanide, lobeline), irritants and inflammatory agents, and increases in vascular pressures (interstitial oedema, microembolism). Mushin (1971, personal communication), using a technique described by Bertola (1940), has blocked the vagus nerves bilaterally at the base of the skull 1-2 cm below the jugular foramen. The glossopharyngeal nerve is also blocked by this technique and, occasionally, the hypoglossal nerve. Studies on normal volunteers with bilateral vagus nerve block indicate no alteration or impairment in respiration (Guz et al., 1966). Lung volumes and mechanics are essentially unchanged, though there is probably an increase in anatomical deadspace. In patients with specific lung disorders, i.e. pulmonary embolism, pulmonary fibrosis, vagal block has produced a substantial reduction of the accompanying...
tachypnoea and dyspnoea (Guz et al., 1970; Berglund et al., 1971) despite the absence of any significant improvement in pulmonary function.

Other studies have examined the effects of vagus nerve block in asthmatics (Eisele and Jain, 1971). The block does not relax airways that are already constricted, but it may reduce the unpleasant sensations associated with it. Most of the vagal block studies have been performed on patients breathing high concentrations of oxygen to eliminate any chemoreceptor influence, thus the results in dyspnoic patients indicate that there is an abnormal degree of vagal afferent impulses presumably bombarding the central nervous system resulting in inappropriate alveolar hyperventilation. As a result of vagal block investigations, several patients have undergone cervical or perihilar vagus nerve section. This area of investigation has tremendous potential for involvement of the anaesthetist.

Phrenic nerve block.

Attempts to block the phrenic nerve bilaterally and evaluate its respiratory effects have recently been made (Eisele et al., 1972), although the precise degree of phrenic motor block could not be determined. Three of eleven healthy volunteer subjects showed marked diaphragmatic weakness (fluoroscopically) following cervical injection of a small volume of 4% lignocaine solution. There was no evidence of other nerve involvement. The breathing pattern changed to slower, deeper breaths with activation of upper intercostal muscles. In the sitting position, the partial phrenic block was well tolerated with no change in Pco₂. The vital capacity fell 25%, which agrees with other estimates of the diaphragm's contribution to the inspiratory capacity (Bergofsky, 1964). Diaphragm weakness has not been a problem during cervical epidural anaesthesia, suggesting that the phrenic nerve is quite difficult to block. It has been noted, however, that partial or complete diaphragmatic paralysis, though well tolerated in the erect position, cannot be tolerated in the supine position (Comroe et al., 1951).

CARDIAC PHYSIOLOGY

Studies on the neural regulation of the heart have, for the most part, involved a combination of sympathetic and parasympathetic blocking drugs. A unique approach of high thoracic spinal anaesthesia (sympathetic blockade) and large doses of atropine has been performed by O'Rourke and Greene (1970). They showed that man's heart denervated in this manner beats at a faster than normal rate. That is, the heart rate increased (64%) following atropine when sympathetic block was present, indicating that in the resting state parasympathetic influence predominates. During beta-adrenergic blockade with propranolol, Robinson and co-workers (1966) noted a much higher rise in heart rate (102%) following atropine, suggesting greater parasympathetic control in the resting state than that observed during spinal sympathetic block. The same investigators demonstrated that during exercise, painful stimulation or sympathomimetic drug therapy, sympathetic influence predominates, and that simultaneously there is a decrease in parasympathetic tone.

Cardiac studies using atropine have the disadvantages of side effects from large doses, and suffer from a lack of knowledge of the direct cardiac effects, if any, of atropine separate from positive chronotropy. Bilateral vagal (parasympathetic) block studies in man without atropine indicate a maximum heart rate of 130 beats/min in two young subjects free of heart disease (fig. 3) (Eisele and Jain, 1971). One subject was excited during the procedure, yet the heart rate did not
exceed that of the other subject who was completely relaxed. This raises the question of why the heart rate did not go higher following vagal blockade in a subject whose sympathetic tone was high as evidenced by an elevated pulse and blood pressure. In the relaxed subject the heart rate increase of 63% corresponds with the increment in heart rate produced by atropine in the sympathetically denervated subjects reported by O'Rourke and Greene (1970). Heart rate control is complex and an understanding of the role of the autonomic nervous system must include consideration of the delicate interplay with baroreceptor reflexes. Glick and Braunwald (1965) point out that the effect of baroreceptor stimulation can be parasympathetic augmentation as well as sympathetic withdrawal. Block of the vagus nerves in the neck also blocks the baroreceptor's central connection (glossopharyngeal nerve) thus making it extremely difficult to draw conclusions regarding vagal control of heart rate. Further, there are metabolic factors that can increase the heart rate in the denervated heart which should be considered (Donald and Shepherd, 1963).

One area in which cervical vagal block can provide useful information, however, is in vagal influence on the cardiac conduction system. In the author's reported study it is of interest that the right vagus contributed approximately one-half the total vagal tone since the rate increase was only half that produced by bilateral vagal block. This is surprising because the right vagal fibres supposedly go to the S-A node, while the left vagus has been thought to influence the conduction system between the auricle and ventricle (Cullis and Tribe, 1913). Thus, it is not clear how left vagal release can raise the heart rate when a sinus rhythm prevails. In normal man the effect of right and left vagus nerves on conduction have not been evaluated by separate and combined blocks. This could be performed in anaesthetized man during neck surgery when the vagus nerves are exposed. Perhaps the greatest value could be derived from recordings from the bundle of His while the vagus nerves are blocked.

A related question is that of vagal influence upon ventricular performance. Depression of contractility by vagal stimulation has been shown in the dog (DeGeest et al., 1965), but little is known about this in man. Cervical vagal block investigations could contribute important information in this area and would offer an excellent opportunity for collaboration with cardiologists. Only the surface has been scratched in resolving the interesting problems of neural control of circulation and respiration in man. It is, indeed, apparent that the area is worthy of the collaboration of the physiologists and the anaesthetists.

REFERENCES


CORRESPONDENCE

NEUROLOGICAL SEQUELAE OF SPINAL AND extradural ANALGESIA

Sir,—In recent years there has been an increase in the use of spinal and extradural (lumbar and sacral) analgesia, in surgery, obstetrics and physical medicine. In an effort to determine to what extent these techniques may be associated with neurological sequelae, the Council of the Association of Anaesthetists of Great Britain and Ireland has appointed a small committee to collect details of any such complications.

May I appeal through your columns to all workers in whose practices such events occur, and for which records are available, to write to the Honorary Secretary of the Association for a "Neurological Sequelae" form which should be completed as fully as possible and returned. (The address is: Room 126, Tavistock House North, Tavistock Square, London WC1H 9HR.)

It is important that all cases are reported, no matter how minor the neurological deficit that has occurred and irrespective of whether this deficit is thought to be actually caused by the procedure or just associated with it.

The information about individual cases will be regarded as strictly confidential by the committee. If this project results in the collection of interesting and significant facts, the results will be published.

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PEEP

Sir,—It is generally accepted that lung function is impaired after surgery under general anaesthesia. "Sighing" had its brief heyday. Is it now the turn of positive end expiratory pressure (PEEP), although at one time any respiratory obstruction was frowned on? Should we use this manoeuvre throughout general anaesthesia, or at least towards its termination until its value in this context has been elucidated? Increasing the tension on the expiratory valve, during both controlled and spontaneous ventilation, would be one method. Perhaps, as usual, someone has already tried it!

ALAN GILSTON
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SIMPLE AND RELIABLE METHOD OF INSERTING A NASOGASTRIC TUBE DURING ANAESTHESIA

Sir,—Anaesthetists have difficulty occasionally in inserting a nasogastric tube during anaesthesia. Several methods have been proposed (Steen, 1964; Ogawa, 1970; Tahir and Adriani, 1971) but these are complicated and some-what traumatic. The method described here is simple and reliable and needs no expensive equipment.

A guitar string (E) is well lubricated and is introduced into the lumen of a nasogastric tube. The tubing must be cut off so that it is shorter than the string by 10 cm. The well-lubricated nasogastric tube so prepared is introduced through a nostril gently and advanced smoothly through the oesophagus to the stomach. Correct location of the tube is confirmed with aspiration of gastric contents or with manual palpation by the surgeon if the abdomen is opened. The guitar string is then taken out slowly while the nasogastric tubing is held tightly with the other hand. It takes about a minute from the insertion to the withdrawal of the guitar string. This method is very simple and there have been few instances of nasal bleeding and other complications which are often observed with the other methods. This method can be adapted for children when a smaller gastric tube and guitar string are used.

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REFERENCES


A POTENTIAL DANGER

Sir,—I think it right that I should draw the attention of your readers to a problem encountered with Vickers/ Puritan flowmeters.

Owing to a design fault in the upper end of the flowmeter, it is possible for a spur, which prevents the flowmeter bobbin from blocking the exit orifice, to break off and thus allow the bobbin to impinge upon the outlet orifice. This, of course, cuts off the flow of oxygen through the device.

The harder you turn on the tap the more certain it is that no flow will occur, although the pressure of oxygen on the proximal side of the bobbin holds it up, maintaining the unsatisfactory state of affairs while giving the appearance that a very high flow rate is occurring.

The attention of the representative of the firm producing this flowmeter has been drawn to the problem and I am sure that they are taking measures to deal with it, but it is important that those using this flowmeter should be aware of the potential hazards some of the older models may present.

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