POSTURE AND ITS INFLUENCE ON ANAESTHESIA DURING UPPER URINARY TRACT SURGERY

S. V. POTGIETER, M.B., CH.B. (PRET.), *Department of Anaesthesia, Karl Bremer Hospital, Bellville, and University of Stellenbosch*

It is a truism that the proficiency of the anaesthetist is more important than the technique he uses. Nevertheless in describing a technique of general anaesthesia for surgery of the upper urinary tract one is led to suggest that the importance of the technique may be predominant in view of the special circumstances which pertain to the patient's posture in certain operations of this branch of surgery. These circumstances are mainly the cardiovascular and respiratory derangements that occur during operation, and the post-operative atelectasis of the contralateral lung.

The nephrectomy or lateral jack-knife position imposes a severe limitation on ventilation. The table is divided in the middle and both halves can be lowered to form an apex or 'break'. A patient placed on such an adjusted table will have the upper portion of his trunk and its extremities in a steep Trendelenburg position, while the lower portion of the trunk with its extremities are in the reverse Trendelenburg position. Whilst the surgical approach is facilitated by the increase in space between the lower ribs and the iliac crest, the venous return from the lower half of the body to the heart is impeded by gravity. The great vessels (vena cava inferior and aorta) are probably distorted, which must further have a deleterious effect upon the cardiovascular haemodynamics.

The lower ribs on the side in contact with the table have virtually to bear the full weight of the patient in the lateral nephrectomy position. Thus the lower thoracic cage is compressed and expansion of the lower hemithorax severely limited. The movements of the lower hemidiaphragm are also encroached upon, if not completely restricted. Especially in the right lateral position, the larger lobe of the liver is forced higher into the thorax. With the movement of the mediastinum under the influence of gravity also contributing, the lower hemithorax undergoes a severe reduction in volume.

In the ordinary lateral position the lower lung is responsible for the larger moiety of pulmonary gas exchange. This is ascribed to its increased pulmonary blood-flow and improved oxygen uptake[26] and its more efficient hemidiaphragmatic movement.[29] In the lateral nephrectomy position it is restricted in this function. Even under controlled ventilation there is a remarkable reduction in the total volume of this lung as well as a decrease in its compliance.[19]

The upper lung has an increased volume, for the upper hemithorax assumes a position resembling inflation. The ribs are spread, the upper shoulder raised, and the upper hemidiaphragm caudally displaced since the abdominal viscera fall away from it. The effect of these mechanical factors is that neither lung can be effectively ventilated by the spontaneously breathing patient.

Oxygen Cost of Breathing

The work of breathing, i.e. the oxygen cost of breathing,[21] may be multiplied during general anaesthesia[5] and especially during surgery of the upper urinary tract. Factors which interfere with the movements of the chest wall, lung or diaphragm will all cause an increase in the 'oxygen cost'. As the lungs become increasingly more difficult to distend, more oxygen is utilized and carbon dioxide produced. It is, therefore, theoretically possible for this increase in carbon-dioxide production to be greater than can be accommodated by an increase in the alveolar ventilation. With increased oxygen consumption and decreased effective ventilation the danger of hypoxia clearly arises. That it may be insidious has been shown by the finding that competent observers cannot infallibly detect cyanosis until the oxygen saturation of the blood has dropped from the normal 97% to ± 70%. In the anaemic patient one may wait in vain for the development of this sign and in the plethoric its occurrence may likewise be misleading while with high oxygen tensions in the lungs during the maintenance of anaesthesia, even fairly gross abnormalities of ventilation and distribution may go undetected if reliance is placed upon 'colour changes' only.

Of paramount importance is the insidious carbon-dioxide build-up which occurs with hypoventilation long before any observable hypoxia. The oxygen lack may be corrected with a few deep inhalations of oxygen, but a respiratory acidosis cannot be as speedily corrected.

Even if the tidal volumes utilized during anaesthesia are equivalent to those which the patient would exhibit during conscious respiration, they may not be adequate when postural changes alter the ventilation-distribution ratio. An extreme example of this is the hypothetical case where all the inspired gases are distributed to one lung, and the whole of the pulmonary blood-flow diverted to the other.

Pulmonary Circulation

The pulmonary circulation is a low-resistance system in which the arteries are readily distensible, and blood-flow can be greatly augmented without entailing a rise in pressure.[14] The opening or closing of capillaries depends upon several factors. Thus Burton[6] and Nichol *et al.*[18] have suggested a mechanism whereby a critical 'opening pressure' determines whether or not a given capillary allows blood to flow through it. It has also been shown that local blood-flow to a lung segment depends upon the quality of its gaseous content.[10] A raised carbon-dioxide tension or lowered oxygen tension results in vasoconstriction which shunts blood to better aerated portions of the same lung. The mechanism whereby this vasoconstriction is probably activated is based on the liberation of lactic acid, which effects a change in the local blood pH.[17]

Stroud and Rahn[24] have suggested that maximal pulmonary vasodilatation occurs when 30% oxygen is breathed, while Rahn and Bahnson[20] in their work on dogs suggested that the mechanism for an independant regulation of blood-flow through each lung depends upon the local alveolar arterial oxygen tensions and the resistance to flow of the contralateral lung. However, blood-flow is not dependent only upon local alveolar gas content, but also on the state

of aeration and inflation of the alveoli. Björk,[2] in work on humans, has shown that the pulmonary blood-flow, after an hour of acutely induced atelectasis, was not diminished in the atelectatic lung, and thus caused a large venous-admixture effect. The undoubtedly low oxygen tensions in the lung, when atelectatic, did not cause a reduction in its blood-flow.

When the pulmonary artery to a lung was occluded the broncho-spirometric tracings showed normal ventilation for that lung, but no oxygen uptake, while the unoccluded lung showed double its previous oxygen uptake.[7] Thus, while some of the adjustment of pulmonary blood-flow may be brought about passively as the result of an alteration in posture, there is also evidence that the smaller branches of the pulmonary arteries are capable of constricting or dilating under the influence of a wide variety of stimuli. Probably as soon as a steady state is attained the flow of blood is regulated according to the efficiency of ventilation. The blood-flow is impaired where ventilation is poor and it is increased in the better ventilated areas.

It is not clear how general anaesthesia affects these various controlling mechanisms, but they are probably less affected by the method of anaesthesia to be described in this article than by other techniques which incorporate larger quantities of respiratory and cardiovascular depressant drugs.[13] It seems possible that in the lateral nephrectomy position the larger moiety of pulmonary blood flows to the under lung while the larger moiety of the ventilating gases passes into the upper lung.

Choice of Anaesthetic

In view of the severe limitations and disturbances of respiration produced by the lateral nephrectomy position, controlled ventilation is obligatory, and this fact would determine the anaesthetic agents to be used. Spinal (subarachnoid) or epidural block cannot usually be employed without added respiratory assistance and amnesia, for the lateral nephrectomy position is more than most conscious patients can tolerate. For those who may be tempted to use a 'local' technique in order to avoid possible pulmonary complications, Brock's words[4] may well be heeded: 'To imagine that the simple replacement of inhalation anaesthesia by local or spinal . . . will avoid such a complication shows a childlike faith born of inexperience or insufficient observation.' This fact is well illustrated in a series described by Faulconer *et al.*[11] in which 5 of the 8 patients who developed post-operative atelectasis had had spinal anaesthesia.

Many studies on conscious and unconscious spontaneously breathing patients have been undertaken,[5,22,23] which proved that severe limitations are imposed on certain aspects of ventilation by the lateral nephrectomy position. Nevertheless, Dripps and Severinghaus,[9] consider that 'these data do not apply to anaesthetized subjects, who require for tidal volume only about one-third of the inspiratory reserve volume'. However, in the studies where observations on ventilation and cardiovascular dynamics were made,[1,15] it was concluded that the lateral nephrectomy position seriously impairs both.

It must be borne in mind that almost all general anaesthetic agents are central depressants, and their administration may lead to depression of respiration, especially if muscular relaxation is to be obtained.

All the commonly used volatile anaesthetic agents have been shown[28] to exert a peripheral effect also. They affect the activity of the pulmonary stretch receptors, and thus exert a profound behavioural influence upon the respiratory rate and pattern.

Controlled and Assisted Respiration

With controlled respiration there is no respiratory muscle activity and the oxygen requirement is thus diminished. As the tidal volume and rate of ventilation are under the control of the anaesthetist a greater alveolar ventilation can be achieved than is possible with spontaneous respiration, and a better state of oxygenation and carbon-dioxide elimination, together with adequate muscular relaxation can be obtained. But even controlled respiration cannot perfectly surmount the obstacles imposed by the lateral nephrectomy position. As indicated above, owing to the diminished compliance of the lower lung[19] the upper lung receives the larger moiety of the ventilating volume, so much so that absorption atelectasis of the alveoli of the lower lung may well occur.

Assisted respiration is a term which is very difficult to define, since the degree of assistance can vary so greatly. Indifferently applied it is probably a hinderance to respiration, while enthusiastically applied it differs but little from controlled ventilation. In any event, it is a taxing technique for the anaesthetist and less efficient than fully controlled ventilation, and does not warrant separate consideration.

Technique

From the above considerations it is clear that any technique allowing spontaneous respiration cannot be accepted for use when the patient is in the lateral nephrectomy position. That patients do survive when such techniques are employed is irrelevant.

In this hospital a technique employing controlled ventilation is used. The average adult is premedicated with 0·65 mg. of atropine and 50 mg. of promethazine (phenergan) 1 hour before operation. Anaesthesia is induced with a test dose of 5 mg. of d-tubocurarine chloride (dTc) *via* an intravenous infusion, followed by a further 25 mg. of dTc and a sleep dose of about 250 mg. of thiopentone in a 2·5% solution. After the lungs have been inflated for a few breaths with 3 : 1 nitrous oxide and oxygen the patient is intubated with a large-size Magill cuffed endotracheal tube. The cuff is inflated to occlude a gas leak, and the tube anchored to the patient's neck.

Anaesthesia is then maintained with nitrous oxide and oxygen (2 : 1 litres) while ventilation is controlled through a semi-closed circle absorption system. Apnoea is maintained by hyperventilation and further doses of 5 mg. of dTc when necessary. On termination of the operation, after adequate time has been allowed for an intravenous dose of 1·2 mg. of atropine to become effective, the curarization is reversed with neostigmine, $\pm$ 3·75 mg. These doses are not absolute and may be varied.

It is imperative for the patient to be intubated, for by this means only is it possible to provide an airway which will maintain the lowest resistance to breathing that is mechanically possible. Intubation circumvents the normal resistance encountered in the oropharynx (tongue, epiglottis, larynx and salivary secretions). Controlled ventilation with a face mask cannot be effectively achieved without

fear of distending the stomach, and a large portion of the ventilating tidal volume is inevitably wasted in alternate ballooning and collapsing of the oropharyngeal airway. There is also always the possibility of regurgitation or reflux of stomach content, which may find its way down the trachea. Further, should the surgeon inadvertently pierce the parietal pleura of the upper lung—a not too remote possibility—the disruption caused by the ensuing pneumothorax can be effectively controlled.

The tendency to allow the patient to recover consciousness in the lateral position while fixing the wound dressings must be avoided, because the lower lung is not then fully aerated. It is advisable to persevere with controlled ventilation post-operatively in the supine position for at least 10 - 15 minutes, so that the atelectatic area of the lower lung may be fully re-aerated.[19]

Controlled ventilation is not without its disadvantages, and its successful application depends upon a knowledge of the disturbances it produces. Circulation is mainly affected by unskilled intermittent positive-pressure ventilation with sustained high mean airway pressures, since the venous return is impeded, especially if the lower part of the body is in the reverse Trendelenburg position. The circulatory reserves of the patient are of paramount importance, and by the use of small quantities of depressant drugs and a light plane of anaesthesia the vasomotor system is the least depressed.

Post-operative Pulmonary Complications

The term post-operative lung complications—which is by no means strictly defined—is used to denote any event pertaining to the lungs in the post-operative period, and the inclination to imagine these events as being essentially of an anaesthetic origin should be carefully controlled. Atelectasis has always been a post-operative accompaniment especially of upper abdominal surgery, and it can be reaffirmed that neither general anaesthesia as such, nor the various aids to anaesthesia like endotracheal tubes, relaxant drugs, carbon-dioxide absorption systems, etc. constitute the threat to the lungs that is imagined by some physicians. Mushin has stated, 'Grudgingly maybe, the truth is now generally accepted that a post-operative chest is due more to the hand in the abdomen than to ether in the lungs'.

The factors determining the onset of pulmonary sequelae are mainly the site of operation, pre-existing respiratory-tract infection, and increased bronchial secretion. These are followed in the post-operative period by immobility and inability to cough effectively, whether this is the result of pain, of analgesic and central depressant drugs, or of instructions from doctors and nurses.

Atelectasis is nor always recognized, for the physical signs are notoriousy inconsistent and variable. Where routine X-ray examinations have been carried out after abdominal surgery, an incidence of about 50% has been reported.[3,25] If more discriminating methods of detection are used, such as the determination of arterial oxygen saturation during the breathing of air and 100% oxygen,[12] it is found that *most* patients have atelectasis post-operatively. This is probably a minor clinical significance in the majority of cases, but under unfavourable circumstances it may give rise to a complicating pneumonitis.

There is a tendency for atelectasis to develop in the lower topographical area of the under lung,[11,16,19] and the upper lung may be affected by a pneumothorax in the more conventional surgical position. Therefore an anterior approach to the upper urinary tract has recently been advocated[27] to circumvent some of the pulmonary and cardiovascular problems which might develop. However, these derangements may effectively be controlled during surgery in the lateral jack-knife position by using the method of anaesthesia described in this paper.

SUMMARY

The lateral nephrectomy position imposes severe limitations upon the cardiopulmonary systems and it is within these boundaries that an anaesthetic has to be administered. The lower topographical area of the contralateral lung is predisposed to post-operative atelectasis and this and other sequelae can best be dealt with by a controlled ventilation anaesthetic technique.

OPSOMMING

Vir chirurgie aan die boonste deel van die urine-weë word die pasiënt gewoonlik in 'n posisie geplaas wat meebring dat daar aansienlike beperkings op die kardiovaskulêre en respiratoriese stelsels geplaas word. As gevolg hiervan word 'n narkose-tegniek waarby gekontroleerde asemhaling ingeskakel word, as die voordeligste beskou. Oor die ontstaan van na-operatiewe longkomplikasies is daar dikwels buitensporige gissings, en die metode wat hier aanbeveel word sal daartoe bydra om veral atelektase te voorkom.

I wish to acknowledge the advice and criticism of Drs. P. A. Foster and J. W. Mostert in the preparation of this paper.

REFERENCES

1. Attinger, E. O., Monroe, R. G. and Segal, M. S. (1956): J. Clin. Invest., **35**, 904.
2. Björk, V. O. (1953): J. Thorac. Surg., **26**, 533.
3. Brattström, S. (1954): Acta. chir. scand., suppl. 195.
4. Brock, R. C. (1936): Guy's Hosp. Rep., **86**, 191.
5. Brownlee, W. E. and Allbritten, F. F. (1957): A.M.A. Arch. Surg., **74**, 846.
6. Burton, A. C. (1951): Amer. J. Physiol., **164**, 319.
7. Carlens, E., Hansen, H. E. and Nordenström, B. (1951): J. Thorac. Surg., **22**, 527.
8. Case, E. H. and Stiles, J. A. (1946): Anesthesiology, **7**, 29.
9. Dripps, R. D. and Severinghaus, J. W. (1955): J. Phys. Reviews, **35**, 741.
10. Duke, H. N. (1952): *Visceral Circulation*. Ciba Foundation Symposium. London: Churchill.
11. Faulconer, A., Gaines, T. R. and Grove, J. S. (1946): Anesthesiology, **7**, 635.
12. Gordh, T., Linderholm, H. and Norlander, O. (1958): Acta anaesth. scand., **2**, 15.
13. Gray, T. C. (1957): Lancet, **1**, 383.
14. Harvey, R. M. and Ferrer, M. I. (1954): Dis. Chest., **25**, 247.
15. Henschel, A. B., Wyant, G. M., Dobkin, A. B. and Henschel, E. O. (1957): Anesth. and Analg., **36**, 69.
16. Lee, J. A. (1953): *A Synopsis of Anaesthesia*, 3rd ed., p. 337. Bristol: John Wright and Sons.
17. Liljestrand, G. (1958): Acta physiol. scand., **44**, 216.
18. Nichol, J., Girling, F., Jerrard, W., Claxton, E. B. and Burton, A. C. (1951): Amer. J. Physiol., **164**, 330.
19. Potgieter, S. V. (1960): Brit. J.Anaesth. In the press.
20. Rahn, H. and Bahnson, H. T. (1953): J. Appl. Physiol., **6**, 108.
21. Riley, R. L. (1954): Ann. Intern. Med., **41**, 172.
22. Slocum, H. C., Hoeflich, E. A. and Allen, C. R. (1947): Surg. Gynec. Obstet., **84**, 1051.
23. Sokalchuk, A., Ellis, D., Hickcox, C. and Greisheimer, E. M. (1949): Anesthesiology, **10**, 577.
24. Stroud, R. C. and Rahn, H. (1953): Amer. J. Physiol., **172**, 211.
25. Stringer, P. (1947): Lancet, **1**, 289.
26. Svanberg, L. (1957): Scand. J. Clin. Lab. Invest., suppl. 25, vol. 9.
27. Tauber, A. S. and Maluf, N. S. (1958): J. Urol. (Baltimore), **80**, 13.
28. Whitteridge, D. L. and Bülbring, E. (1946): Brit. Med. Bull., **4**, 85.
29. Zoltan, M. (1958): Dis. Chest, **33**, 550.