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PROBLEMS OF ANAESTHESIA IN CHEST SURGERY

Lecture by
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In anaesthesia it is our aim to maintain as far as possible conditions favourable to the continuance of the normal metabolic processes of the body. This most concerns us in the proper ventilation of the circulating blood stream. When the lungs or portions of them are diseased they are less efficient in this vital function of conveying oxygen from the inspired air to the blood and removing carbon dioxide from it. The causes of this inefficiency may be classified under four heads.

1) Reduced breathing capacity.
2) Increased barrier between the air and the blood.
3) Uneven distribution of the inspired air.
4) Pulmonary vascular shunts.

1. The reduction in breathing capacity may be due to a slowing of the rate at which air is drawn into and expelled from the lungs, or to a reduction in the vital capacity. Three important factors may slow the passage of air in and out of the lungs. 1) Pain from injury or from inflamation will limit the muscular force exerted. 2) Obstruction in the air-passages. If the obstruction is in the main air passages it slows both inspiratior and expiration, but in the smaller tubes, as in the bronchospasm of asthma the slowing affects chiefly the expiration. The slight increase in the diameter of the tubes during inspiration permits air to enter the alveoli more easily than it can be expelled. 3) In emphysema the reduced elasticity of the lung slows the emptying of the lung and if active

expiratory efforts are made some of the small bronchi may collapse and add an obstructive element to the slow expiration. The reduction the vital capacity may be due to limitation of movement of the thoracic cage from pain, deformity, or paralysis, or to space occupying lesions. These may be tumours of the lung or mediastinum, large areas of lung rendered functionless by disease, pleural effusions whether of air or fluid, and the accumulation of blood in the lungs in heart failure. Lesions below the diaphragm, driving it up into the chest, act in a similar way.

2. Oxygen in passing from the alveolus to the red blood corpuscle has to pass through five barriers. The alveolar membrane, the interstitial fluid, the capillary wall, the plasma, and the cell membrane of the corpuscle. Pulmonary fibrosis as in Beryllium poisoning, Boerk's sarcoid, asbestosis, etc., or pulmonary vascular changes from prolonged pulmonary hypertension can increase the barrier at the alveolar or capillary membrane, but the practical problems are presented by an increase in the fluid barrier either from pulmonary oedema in the interstitial layer or sputum in the alveolus. Those factors which initiate an outpouring of oedema fluid into the lungs must be carefully avoided. This is more a problem of anaesthesia in cardiac cases and will be dealt with later. The interference with the diffusing capacity of the lung is only one of the problems presented by the presence of sputum in the lungs and a consideration of the steps necessary for the control of sputum will form a complete section of this paper.

3. Uneven distribution of the inspired air may occur from partial obstruction in the respiratory tract, from pleural adhesions, from paradoxical movement, from posture, and from the use of endobronchial blockers and tubes. The effect of uneven distribution is an increase in the physiological dead space and therefore the amplitude of each respiration must be increased to ventilate the blood.

4. If some portion of the pulmonary circulation passes to the left auricle without flowing through ventilated alveoli, the blood in the left heart will be a mixture. Part will be arterial with an oxygen tension of 100 mm. and a carbon dioxide tension of 40 mm. and part will be venous with an oxygen tension of 70 mm. and a carbon dioxide tension of 46 mm. When this mixed blood reaches the respiratory center the raised carbon dioxide tension stimulates

deeper breathing. This washes more carbon dioxide from the ventilated blood, but cannot change its oxygen saturation, the unventilated blood is of course unchanged. The final result is that the mixed blood has normal carbon dioxide levels but is low in oxygen. These shunts in the pulmonary circulation occur

1) Congenital heart disease
2) Haemangioma of lung
3) Disease of Lung: a) Consolidation, b) Collapse.
4) One Lung anaesthesia.

The overall picture is that diseased lung is inefficient. We can by preoperative treatment reduce this inefficiency. A pleural effusion can be removed. Heart failure can be treated. Anaesthetic techniques involving the use of low oxygen tensions or excessive rebreathing must not be used. The patient breathing spontaneously is liable to suffer from anoxia and will be relatively slow in his response to restorative measures.

The second special problem is the need to control secretions. The sudden release of sputum from a lung abcess, bronchiectasis, or through a broncho-pleural fistula may overwhelm the patient and cause sudden death from anoxia. More insidious spread of secretions will form a barrier to ventilation. The hypoxia produces shock and the associated carbon dioxide retention causes a cardiac irritability which may be disastrous if the dissection involves openening the pericardium. The spread of secretions can contribute to post operative morbidity by causing segmental collapse and bronchopneumonia. This superadded dysfunction may cause death from respiratory insufficiency in a patient who has undergone an extensive resection. Finally the spread of secretions during the operation may carry disease to previously uninfected areas of lung.

The first weapon in combating these dangers is in the preparation of the patient. A period of rest under good hygienic conditions whilst the necessary investigations are carried out will greatly reduce the amount of sputum. This general approach can be reinforced by postural drainage, physiotherapy, and antibiotic treatment when the lesion has been accurately localised by X-ray, bronchoscopy, and bronchography, and the sensitivity of the organisms determined. If possible, operation is postponed until no further improvement is occuring. Postural drainage, in a position

that has been found to be most effective, is used immediately before operation. A careful toilet of the bronchial tree is carried out by suction through a bronchoscope. At this stage a decision must be made as to the method of control to be used.

1) It may be justifiable, if the preoperative treatment has been successful and the toilet thorough, to rely on intermittent bronchial suction to protect the patient.

2) An attempt may be made to retain the secretions in the diseased area of lung until the bronchus has been isolated and clamped. This may be attempted by posture, by tamponade, or by endobronchial anaesthesia. Overholt describes a prone position for patients with upper lobe cavities. As the upper lobe is anterior, in the prone position the cavities will be lower than the bronchus and the sputum retained in them until the bronchus has been dissected. In lower lobe bronchiectasis a similar protection can be obtained in the lateral position of a head-up tilt of 15° is used. Whilst the sputum is retained in the diseased area it causes little interference with the respiratory exchange. As a final example, an empyema with a broncho-pleural fistula can be drained with the patient sitting up. One of the first methods of control was by packing the bronchus of the diseased lobe with ribbon gauze through a bronchoscope, retaining the pack in position with a malleable wire. A simpler method is to inflate a small balloon in the appropriate bronchus, fig. 1 and 2. Finally an endobronchial tube can be introduced into the stem bronchus on the other side and a one lung anaesthetic administered, fig. 3.

3) The secretions may be drained away, whilst preventing contamination of the healthy lung. The prone position with the head and shoulders dependent gives a downhill inclination of the trachea of nearly 30° so that any secretions squeezed out of the lung will gravitate towards the mouth rather than contaminate the other lung. The use of a Trendelenburg tilt in the lateral position is misleading as there is always a danger of spill into the under lung. Bronchus blockers with a lumen may be inserted into the bronchus leading to the diseased lobe or lung. Secretions can be continuously removed by aspiration through the

lumen. On the left side an endobronchial anaesthetic tube can be inserted so that its cuff is entirely contained in the left main bronchus. In this way the anaesthetic can

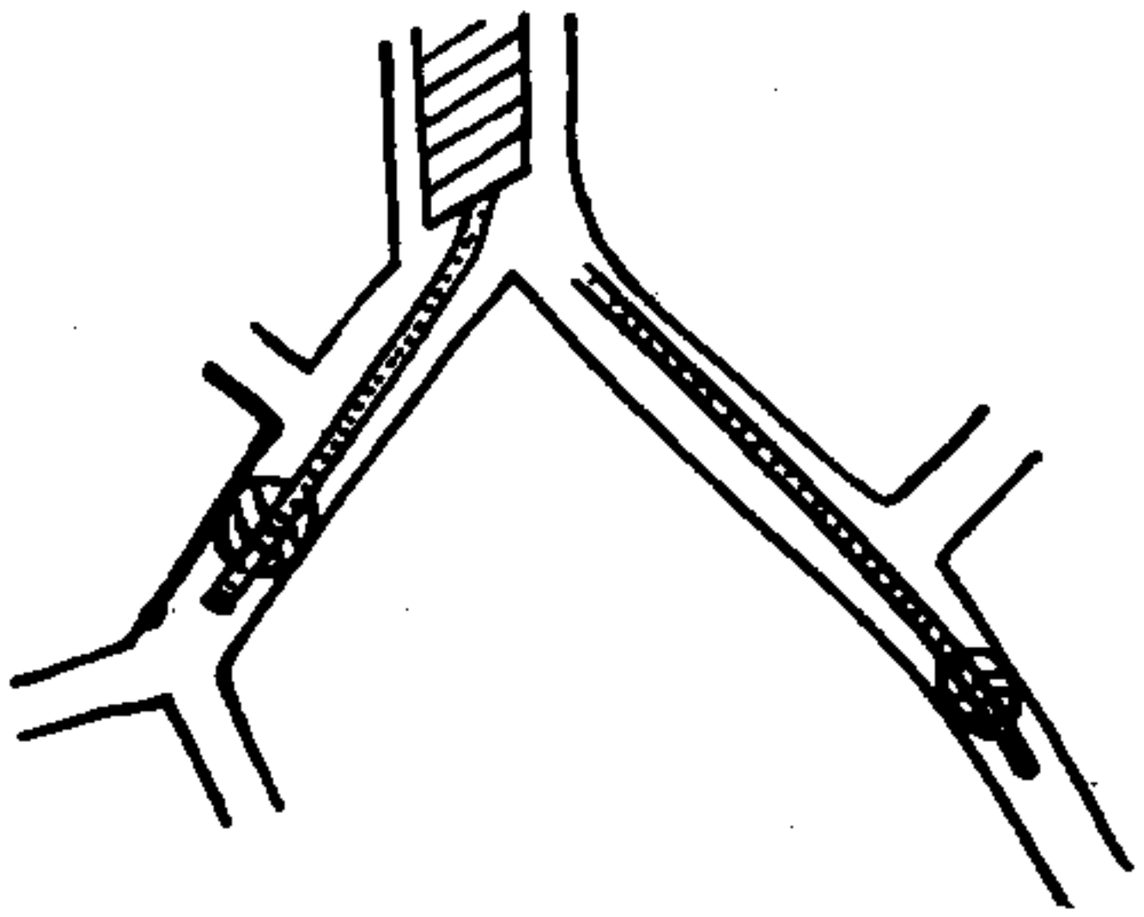


Fig. 1

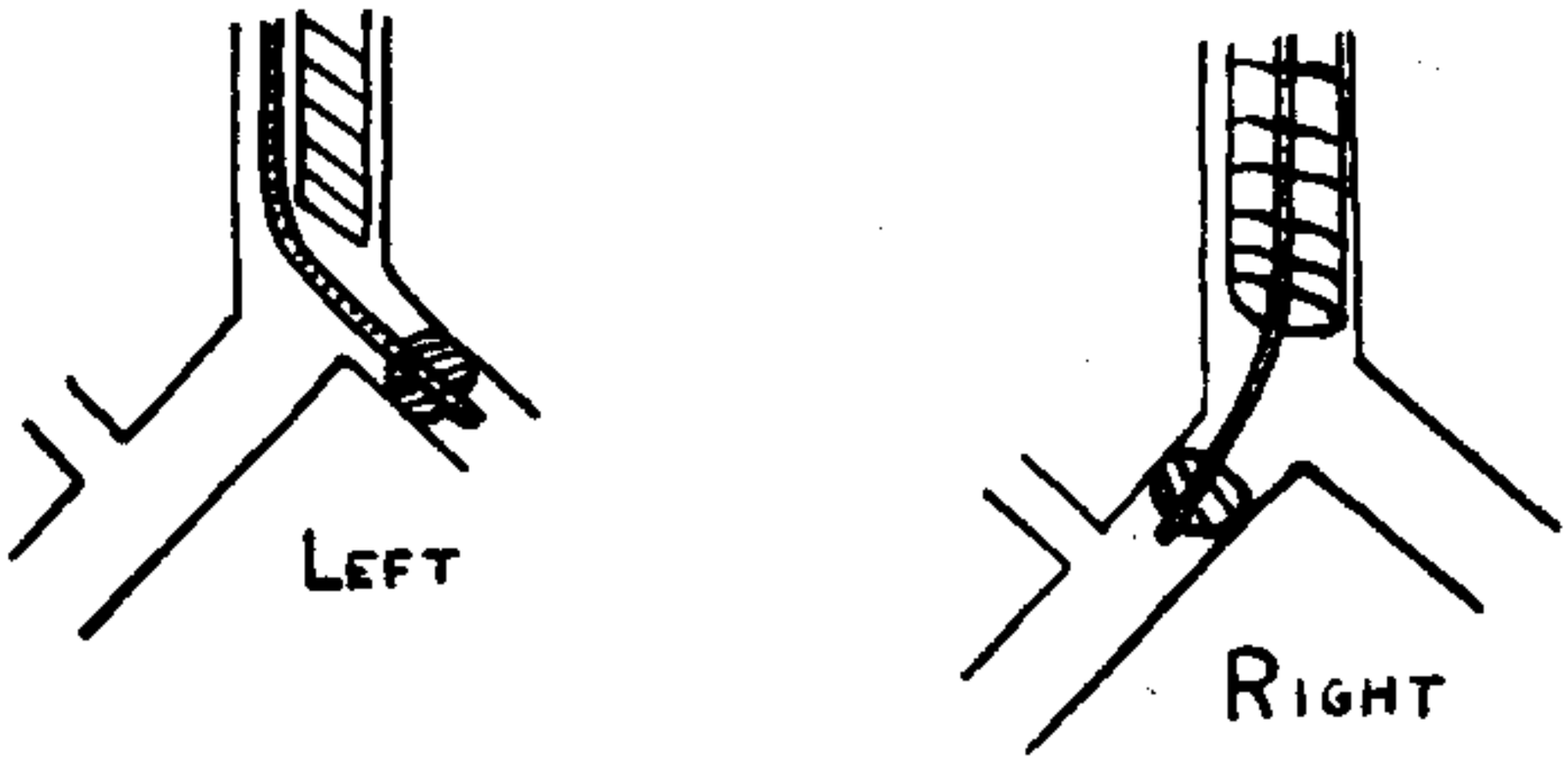


Fig. 2

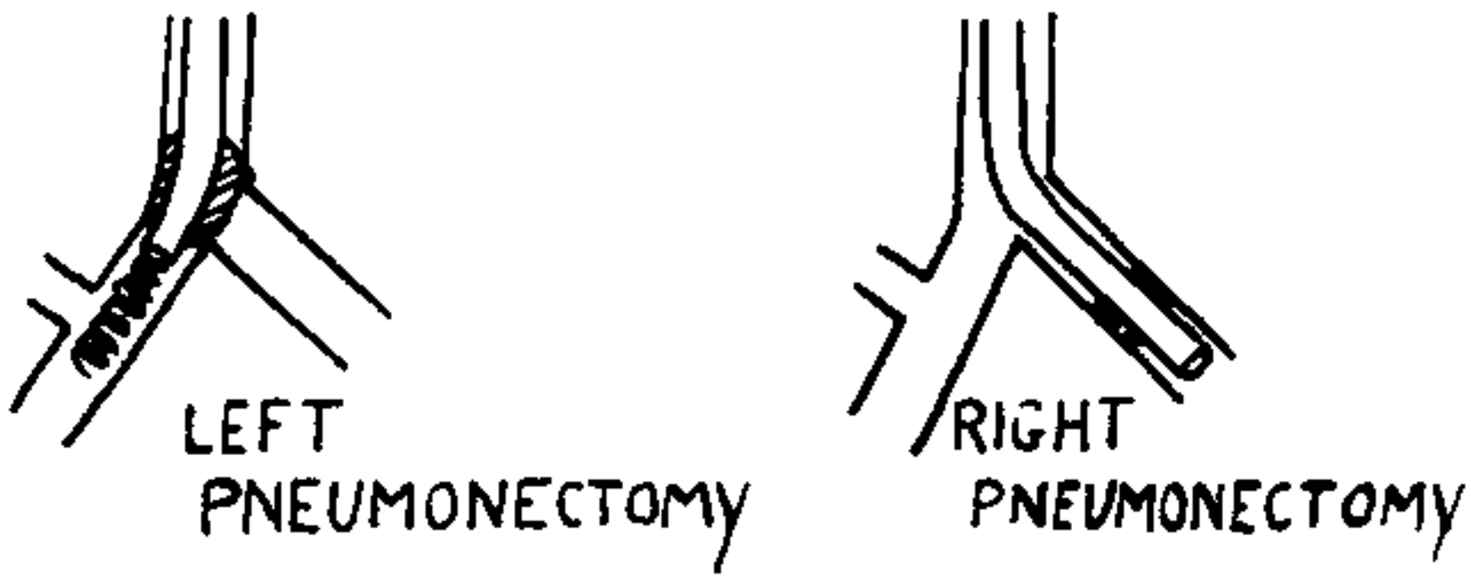


Fig. 3

be given into the left lung while secretions from the right, have free exit to the mouth outside the tube. This is not so readily achieved on the right but there is a tube with a side hole in the cuff to allow inflation of the right upper

lobe and a rubber hook to engage the carina so ensuring its insertion to the proper depth. Alternatively a double lumen tube, such as Carlen's, will effectively separate the two sides and so allow ventilation of the right and free drainage of the left. I will leave the choice until we have discussed the effect of respiration or the open chest.

The third of the general problems presented by chest surgery is that of maintaining the respiratory exchange whilst the chest is open. The first observation is that air is drawn into the chest through the wound as well as through the trachea. The ratio of the air drawn into the wound to that normally inspired is a measure of the waste of inspiratory effort caused by this defect. There are three considerations.

1) In the open hemithorax the lung collapses because of its own elasticity, unless there are pleural adhesions, or the lung has lost its elasticity from age or emphysema. The expansion of the hemithorax cannot be transmitted to the collapsed lung and the effort is wasted in driving air in and out of the wound.
2) The mediastinum is a mobile estructure and moves in response to the forces acting on it. During inspiration the pleural pressure on the normal side becomes more negative, and during expiration less negative or even positive. On the open side the pressure remains atmospheric in both phases. Under these forces the mediastinum moves into the normal side during inspiration and to the open side during expiration, fig. 4, increasing the volume of air drawn through the wound and reducing that passing to the lungs. When the patient is in the lateral position there is added a displacement due to the weight of the mediastinal structures and the volume of the working lung may be critically reduced, fig. 5.
3) If the respiration becomes forced or particularly if there is any obstruction to breathing, part of the air expelled from the working lung will inflate the exposed lung instead of passing up the trachea. In inspiration this vitiated air will pass back into the working lung. The exposed lung will expand in expiration and collapse in inspiration. It moves paradoxically. The air passing from lung to lung is

known as the "pendulum air" and adds to the dead space as well as carrying infection from one lung to the other, fig. 6.

By an open pneumothorax we lose the function of the exposed lung. Fortunately as the lung collapses the pulmonary circulation through it is reduced so there is no great vascular shunt. The remaining lung is made inefficient by the mediastinal movement and the pendulum air. Finally there is the loss of the assistance normally given to the venous return by the negative pleural pressure in inspiration.

Our final choice of technique must be influenced by all three factors. The inefficient lung requires a high proportion of oxygen and an adequate tidal air. Pre-operative therapy must concentrate on removing factors, such as a pleural effusion or ascites, which mechanically embarass respiration. Breathing exercises can improve the respiratory function and so give the anaesthetist a greater margin of safety. Their greatest value is in the post-operative management, for a patient who is able to co-operate intelligently is less liable to post-operative atelectasis. The treatment of heart failure will be of help by making the breathing easier. In this connection I would advocate the pre-operative digitalization of three groups of cases. a) Those where the pericardium is likely to be opened e.g. carcinoma of the lung, when the vessels will have to be tied within the pericardium. b) Carcinoma of the middle third of the oesophagus, where the oesophagus will have to be dissected from behind the heart. c) Patients with hypertension even though showing no signs of failure. These patients are liable to give rise to anxiety postoperatively from cardiac failure we think that the incidence in our cases has been reduced by this simple precaution. One of the three methods of controlling secretions that has been discussed must be adopted and finally the challenge of the open chest must be met.

I think the advantages of the controlled respiration are overwhelming. It provides an adequate tidal air. It prevents paradoxical movement. It permits a quiet field when necessary and allows periods of inflation of the lung for defining the limits of segments of the lung. None the less its disadvantages should be reviewed. Intermittent positive pressure ventilation is not so efficient as normal in ventilating the blood stream. If a minute volume

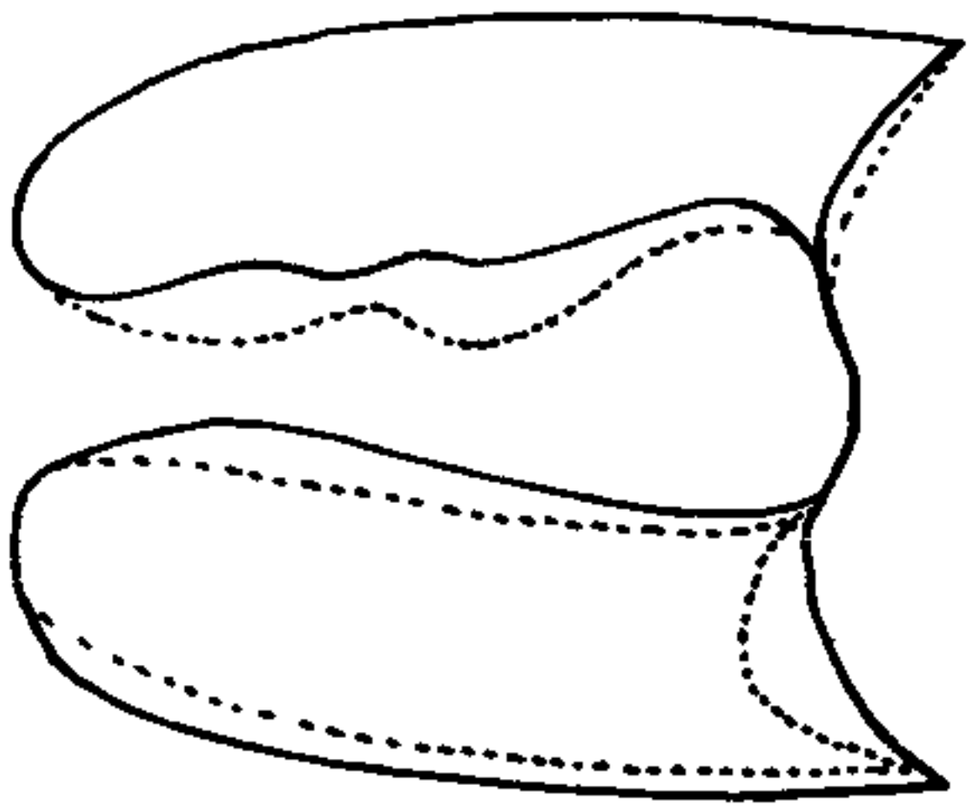


Fig. 4

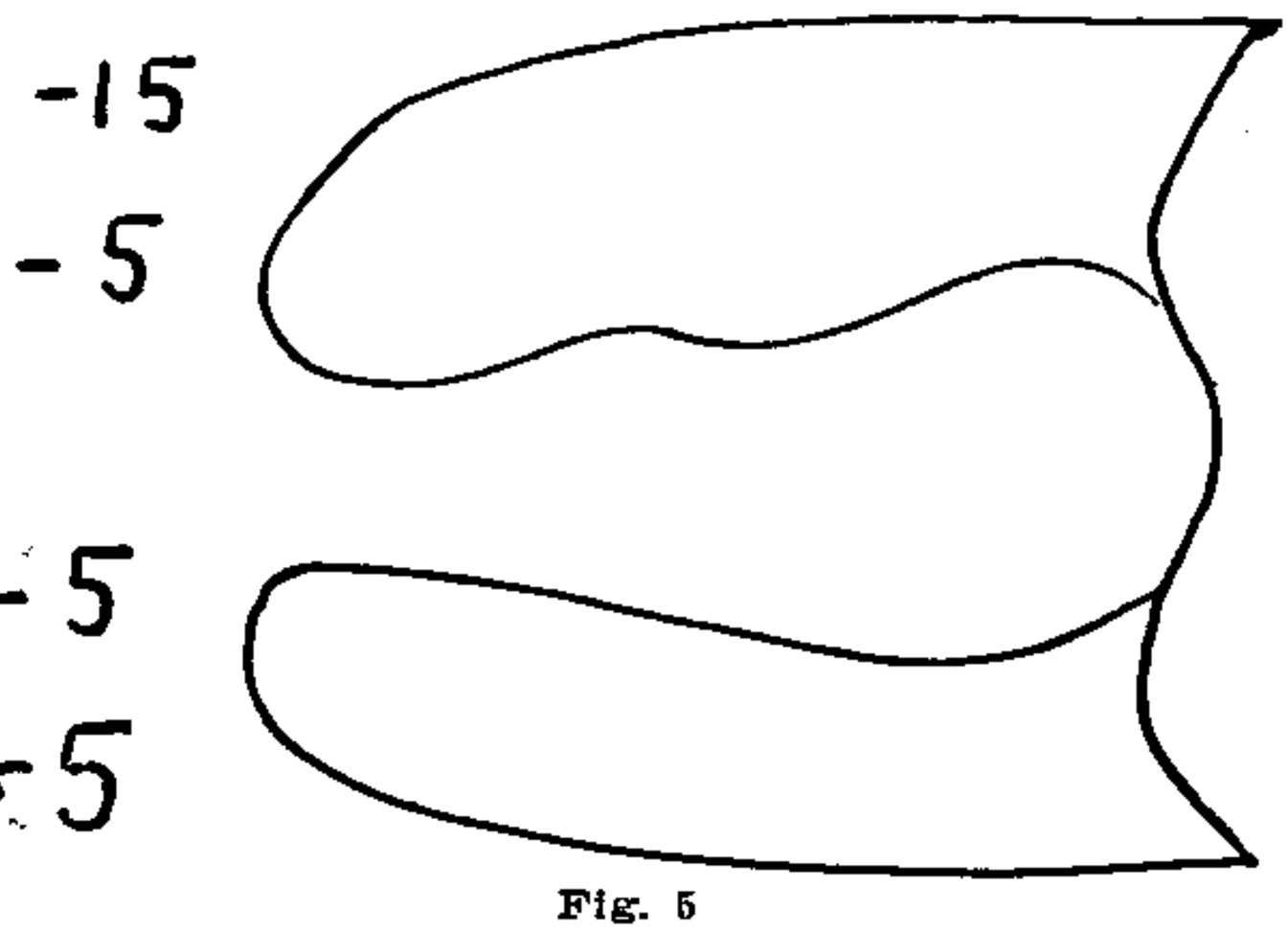


Fig. 5

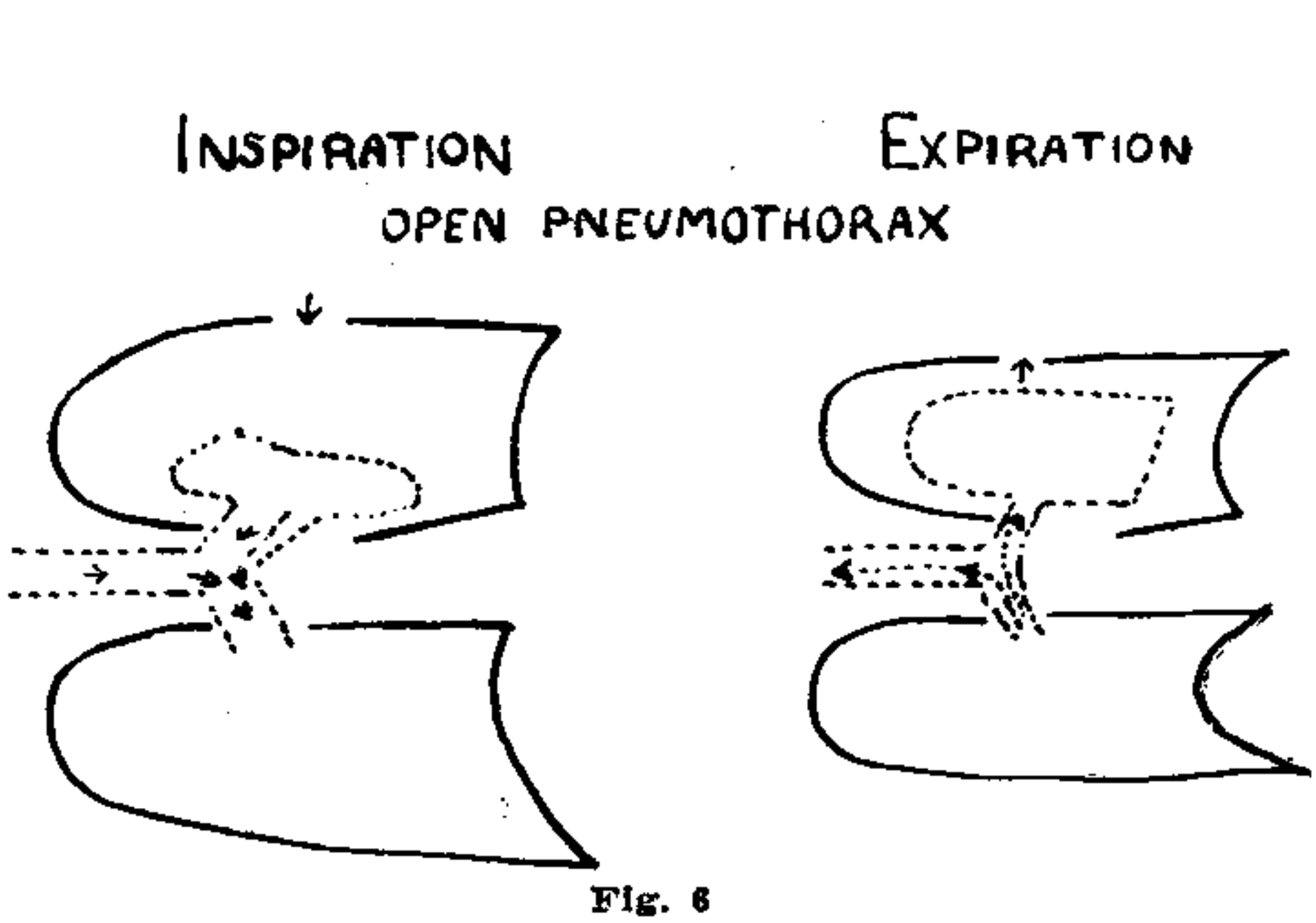


Fig. 6

of 5/6 litres maintains a normal pH this will need to be doubled on positive pressure to maintain it. Controlled respiration is dependent on suppression of the cough reflex, so that unless secretions are well controlled sputum will be spread around the lung the forced inspiration. The change from passive to active respiration occuring at the end of the operation at the same time as the patient is emerging from anaesthesia, is being moved from operating table to bed, and changing from an oxygen rich anaesthetic mixture to normal air makes this transition a little more dangerous. If these points are kept in mind I know of only one condition in which the attempt at controlled respiration is dangerous. This is when air is trapped beyond a valvular opening from the bronchus, an air cyst or a tension pneumothorax. If positive pressure is applied the cyst may distend and collapse functioning areas of lung. During expiration the air is unable to pass back into the bronchi. In such a case spontaneous respiration should be maintained until either the bronchus leading to the effected part of lung has been blocked or the chest has been opened so that the pressure of the trapped air can be released. In the repair of a large bronchopleural fistula embarrassment can be caused because the balloon of the endobronchial tube interferes with the closure of the bronchus and has to be removed. If the patient is completely paralysed it will be difficult to ventilate until the bronchus is closed.

The simplest and a very satisfactory way of maintaining controlled respiration is by manual squeezing of the rebreathing bag in a carbon dioxide absorption circuit. This gives information as to the condition of the patient and the need for drugs from the feel of the bag. Pressure can immediately be varied to suit the surgical requirements at any moment: lung can be inflated to search for a leak or define a segment breathing can be quietened or even suspended in a difficult dissection, the movement of the lung can be timed to assist the surgeon in his stitching. There is a place for mechanical respirators for this task. They can be adjusted to give a steady and measured ventilation which is of particular use in segmental ressection or decortication of the lung. The anaesthetist is freed from the mechanical task of respiration and enabled to record blood pressures, manage the transfusions and prepare his drugs. I think that the control of the machine should be by pressure and the force transmitted pneumatically rather than

mechanical transmission set to deliver a given volume. I have used a Blease pulmoflator with satisfaction for nearly five years. On theoretical grounds there is a place for a respirator with a negative pressure phase in expiration. I remember with interest having such a machine demonstrated to me on a previous visit to South America. I have used such machines I think with advantage to the patient, certainly with gain to the surgeon for the ventilation is achieved with much less positive pressure to there is less movement and less bleeding. Valuable though this negative pressure phase is in the ventilation of curarised patients for general surgery, but when the chest is open and the patient is in the lateral position, it allows the under lung to collapse and the mediastinum becomes so far away as to make surgery difficult. I have used it in the prone position and been delighted with the large tidal air obtained for an inspiratory pressure of 10 cms. of water or less. A ventilation obtained with a quiet field and an unobtrusive lung on the open side. When I have used it in the lateral position I have had to abandon the negative phase after opening the chest.

I want to commend the prone position used by Mr. Holmes Sellors for the majority of his thoracotomies except in heart and great vessell surgery. It serves a double purpose. First it allows the mediastinum to remain more stable. We stop the paradoxical movement of the mediastinum which would occur when the chest was open by controlled respiration, but there remains the movement due to the weight of the structures in it. In the lateral position whilst the chest is closed this movement is limited by a difference in the pleural pressures on the two sides. The upper hemithorax maintaining pressures more negative than the lower in the same phase of respiration. When the chest is open this force is unchecked. Even the use of positive pressure will not of itself prevent this shift of the mediastinum for an attempt to inflate the lower lung also distends the upper and in fact may increase the shift. The air can be directed into the under lung by endobronchial tubes or blockers but such a technique involves difficulties with pulmonary vascular shunts, injury to the bronchial mucosa, and the positive pressure may have to be maintained both in expiration and inspiration with increasing circulatory embarassement. The prone position eliminates this gravitational shift. The other advantage is in the control of secretions. The downward

slope of the trachea ensures that any sputum released from the lung gravitates towards the mouth, fig. 7, 8 and 9. The healthy lung is almost completely protected, which reduces the risk of anoxia in the course of the operation and of atelectasis in the recovery period. I would like briefly to describe the anaesthetic technique for a thoracotomy in the prone position.

The preparation of the patient has already been indicated. A sufficient period of postural drainage is given immediately preoperatively according to the case with which sputum can be brought up. The premedication aims at moderate sedation and drying of secretions. Normally Omnopon 20 mgm. with Scopolamine 0.4 mgm. The patient is given oxygen to breath and anaesthesia induced with intravenous Thiopentone. This is given slowly and a dose twice the sleep dose is given. I then obtain topical anaesthesia of the larynx by injecting 2 ml. Lignocaine 4 % through the cricothyroid membrane into the trachea, fig. 10. I then give Tubarine 18 mgm. When the jaw relaxes I perform a careful bronchial toilet through a bronchoscope. During this period if the dose has been correctly estimated spontaneous respiration will be maintained. If oxygenation is not satisfactory it can be improved by inflation through the bronchoscope. I try to avoid inflation of a patient until I have sucked away at least the grosser amounts of sputum in the respiratory tract. When satisfied the bronchoscope is removed and a large cuffed endotracheal tube is inserted (artificial respiration is started with a mixture of N20 and O2 about 3 litres to 2 litres per minute). The patient can then be rolled into position on the operating table, with rests placed under the chest and pelvis, fig. 11 and 12. This allows the head and neck to hang down giving the important downward slope to the trachea. The abdomen is free from pressure so the diaphragm can move freely when the lungs are inflated, fig. 12. The arm on the side of the thoracotomy hangs over the side of the table and draws the scapula clear of the line of incision, fig. 14 and 15. The other arm lies by the head and is conveniently placed for transfusion, which is set up as soon as the patient has been placed in position. Usually a further dose of Tubarine is given into this drip to allow the control of respiration to be obtained with smaller inflationary pressures. The compliance of the lung to the changes in pressure in the rebreathing bag depends on the resistance of the airway,

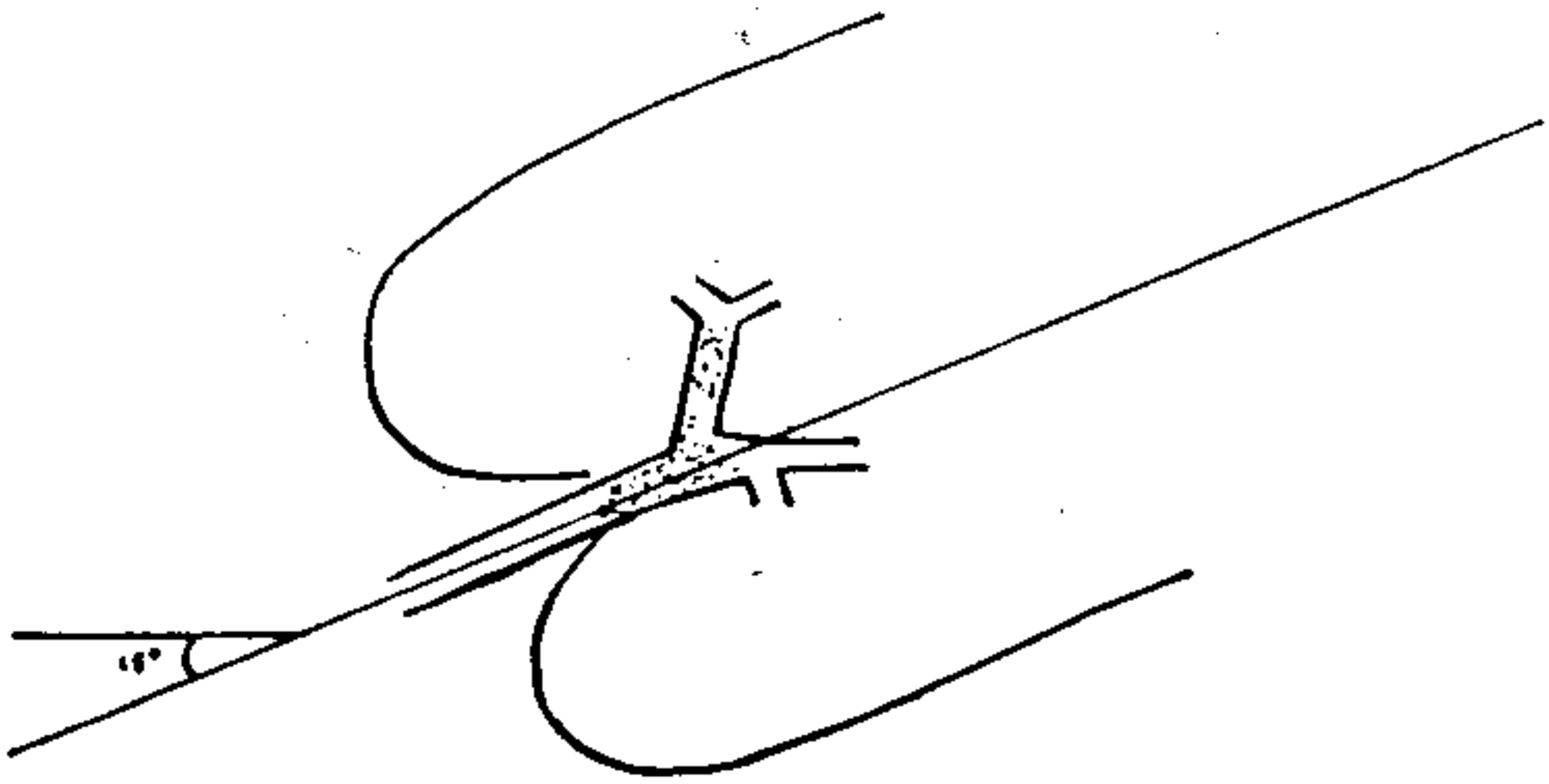


Fig. 7

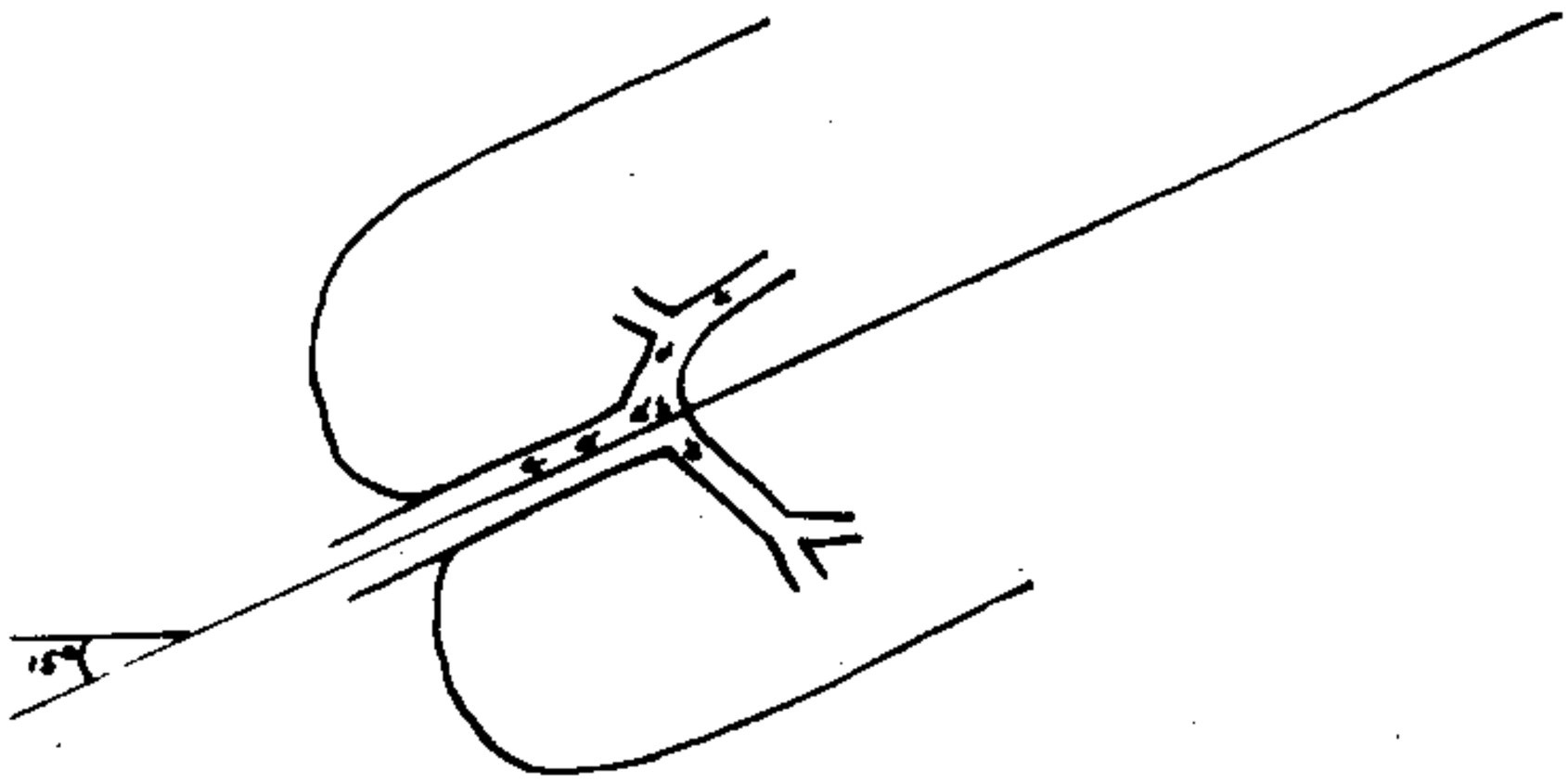


Fig. 8

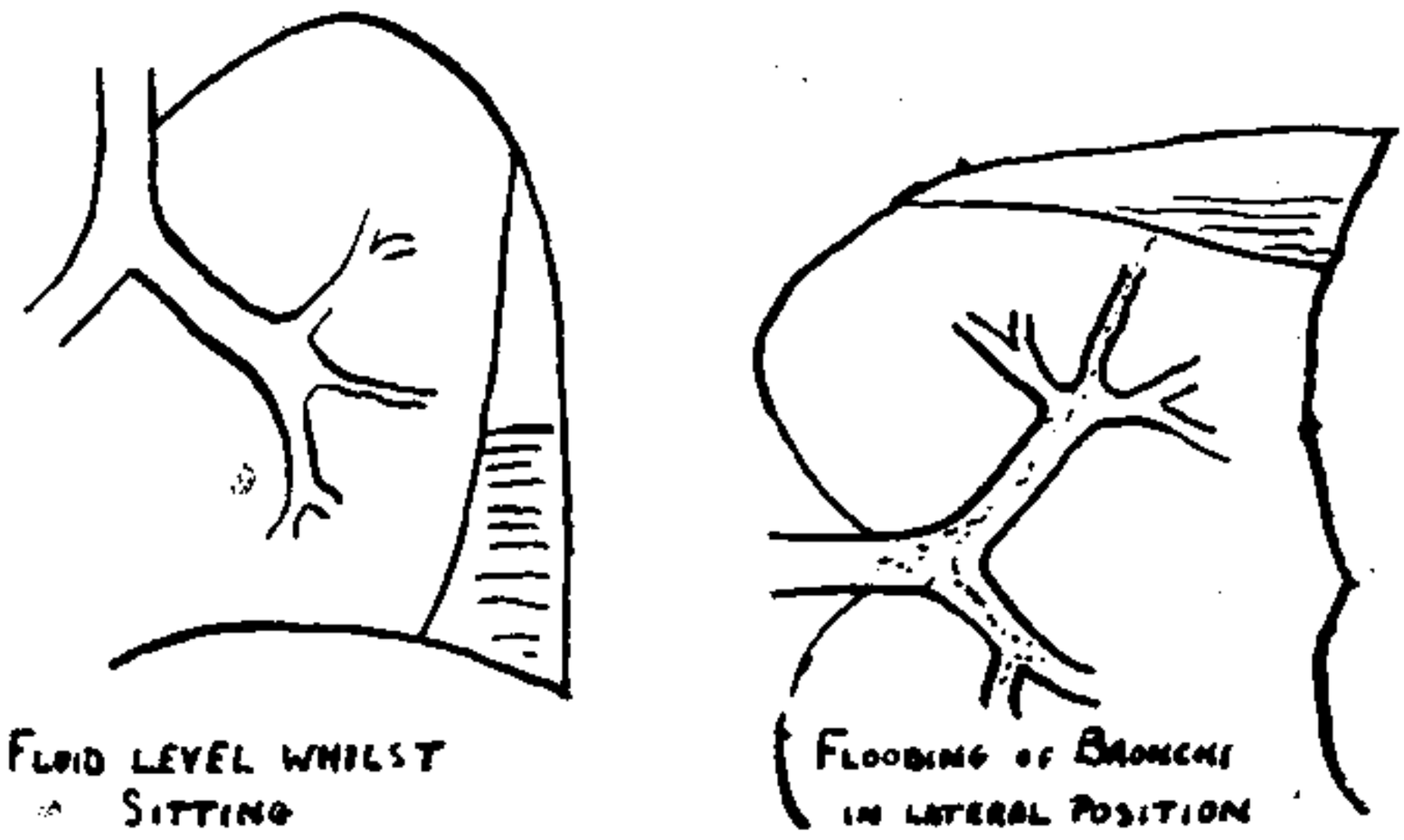


Fig. 9

which we have reduced with a large lumen tube, the elastic recoil of the lung which we cannot influence, and the muscular tone of the chest wall which we reduce with our supplementary dose of curare. The operation can now start. Blood is given in the transfusion to keep pace with any loss. When the chest is opened the pressure of inflation must be modified. If the lung is elastic it

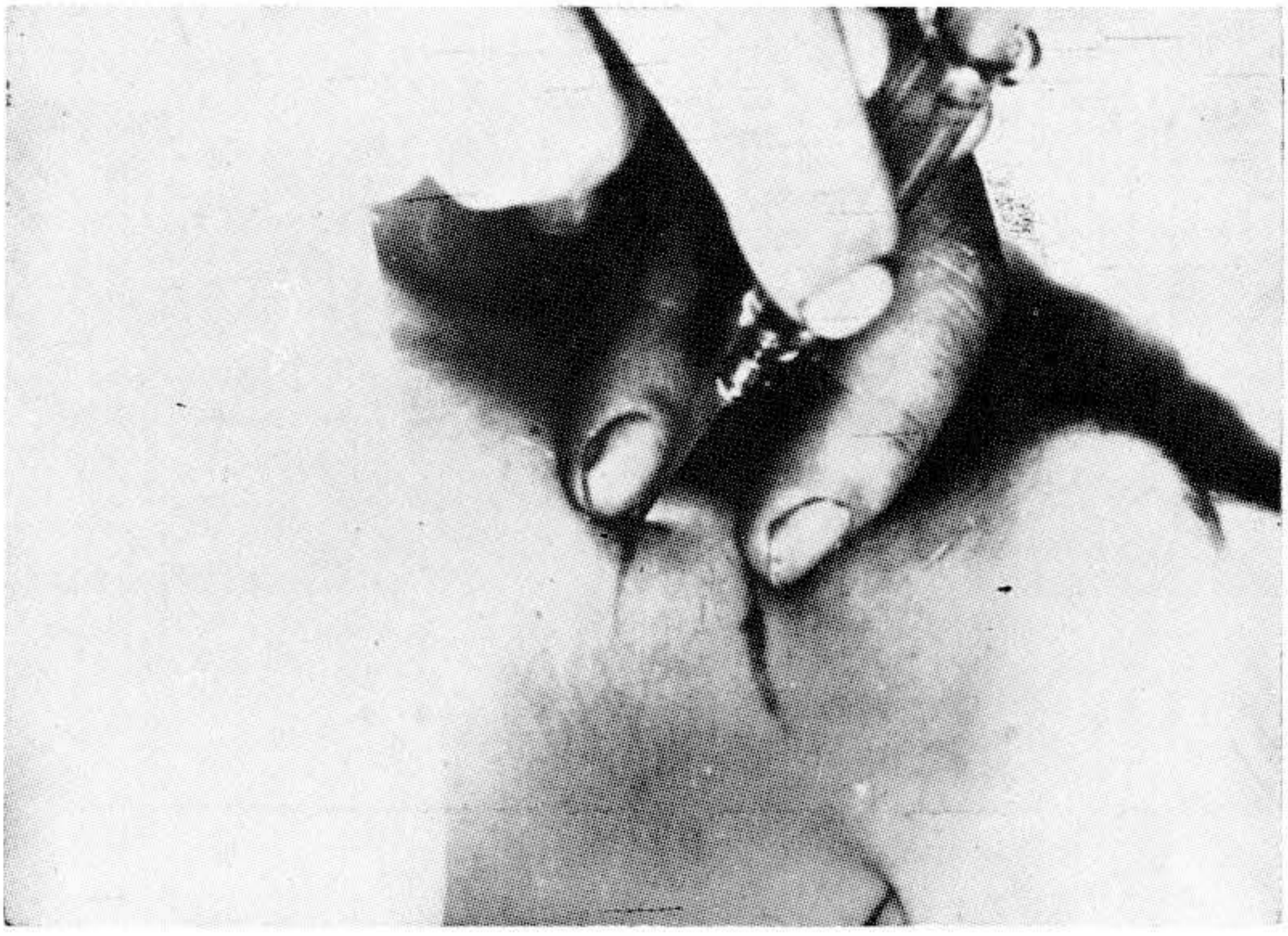


Fig. 10

may be possible to allow it to collapse on the open side, by reducing the pressure. When it is collapsed the pressure can be increased to maintain satisfactory ventilation of the other lung without disturbing the collapsed lung. This manoeuvre gives admirable operating condition but it must not be used at the expense of proper ventilation of the patient. More often the movement of the exposed lung must be controlled by packs and retractors. When the operation is finished assistance having been given as I indicated earlier in discussing controlled respiration, the exposed lung is rexpanded under vision. The chest is closed with a drainage tube connected to a water seal. When it is considered to be airtight an attempt is made to woo the patient back to spontaneous respiration by removing the absorber from the circuit. A blind toilet of the bronchial tree is now done by passing a suction tube along the

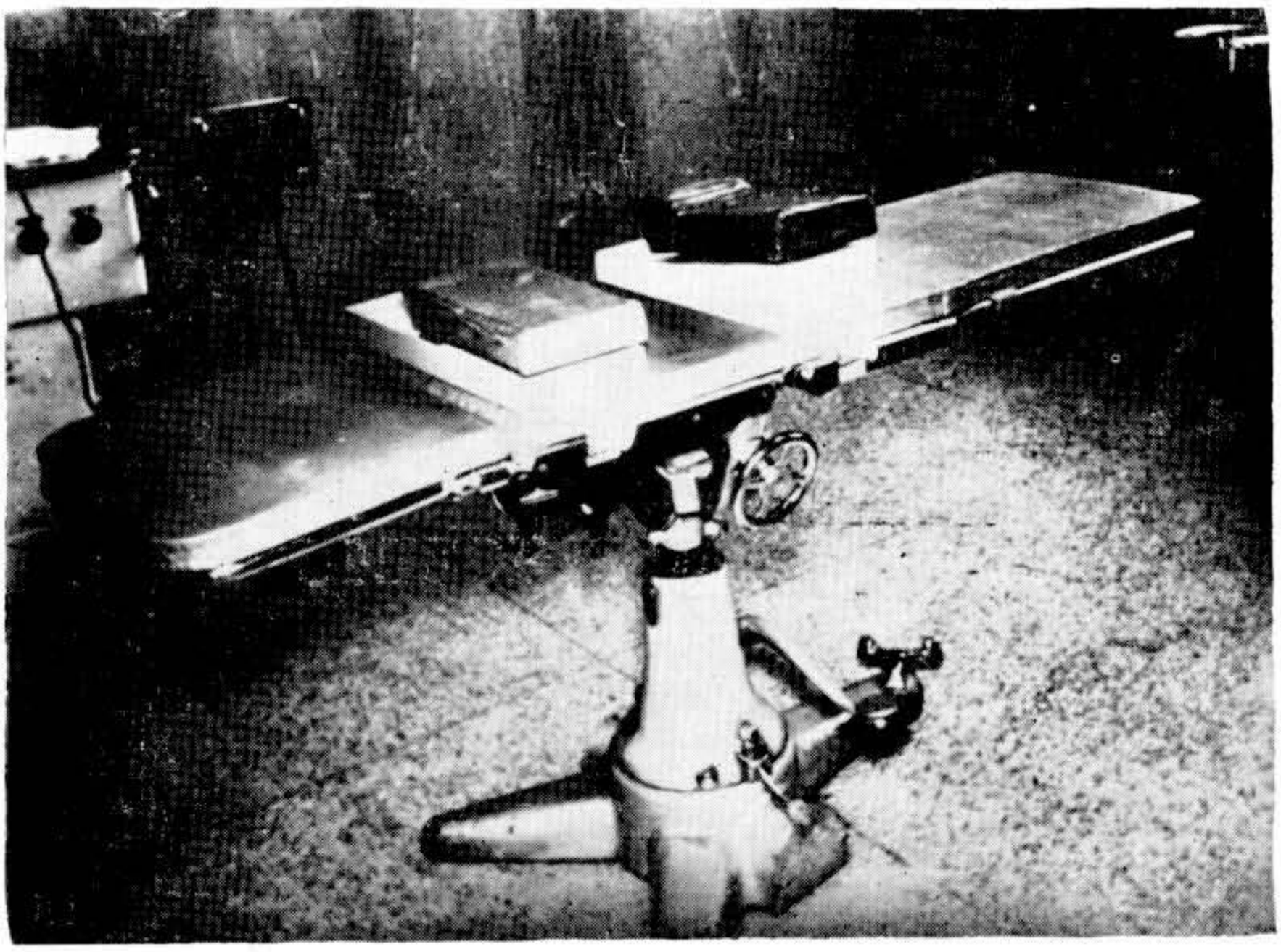


Fig. 11

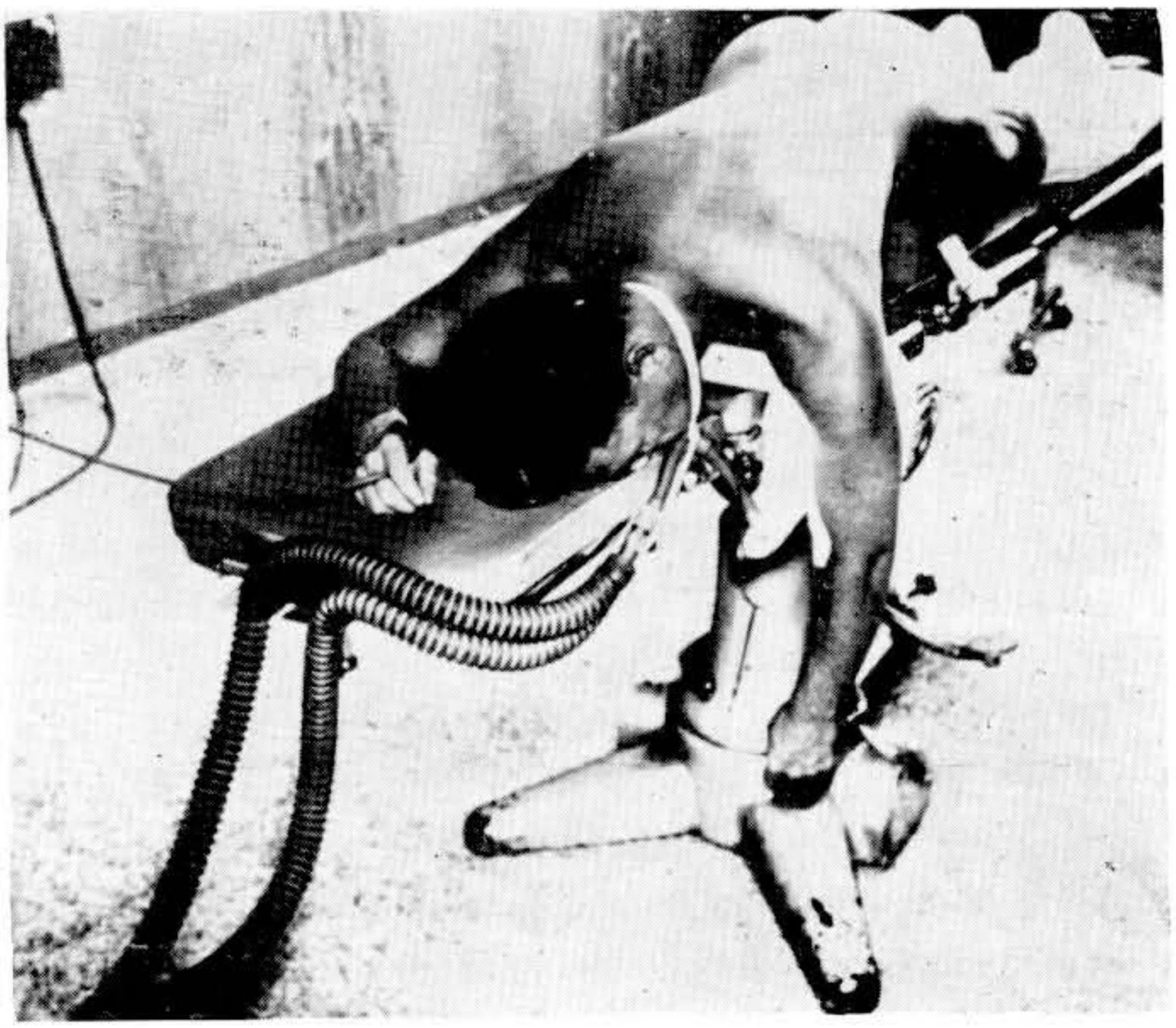


Fig. 12

endotracheal tube as has been done in the course of the operation if moist sounds are heard in the tube. When the dressings are in place Atropine 0.6 mgm. followed by Neostigmin 2.5 mgm. will help restore full respiration. I think that Neostigmin should always be used at the end of these cases where curare or a similar competitive blocking agent has been used to aid the control of respiration. Death from vagal inhibition of the heart has been reported but if atropine is given first and the patient has not been

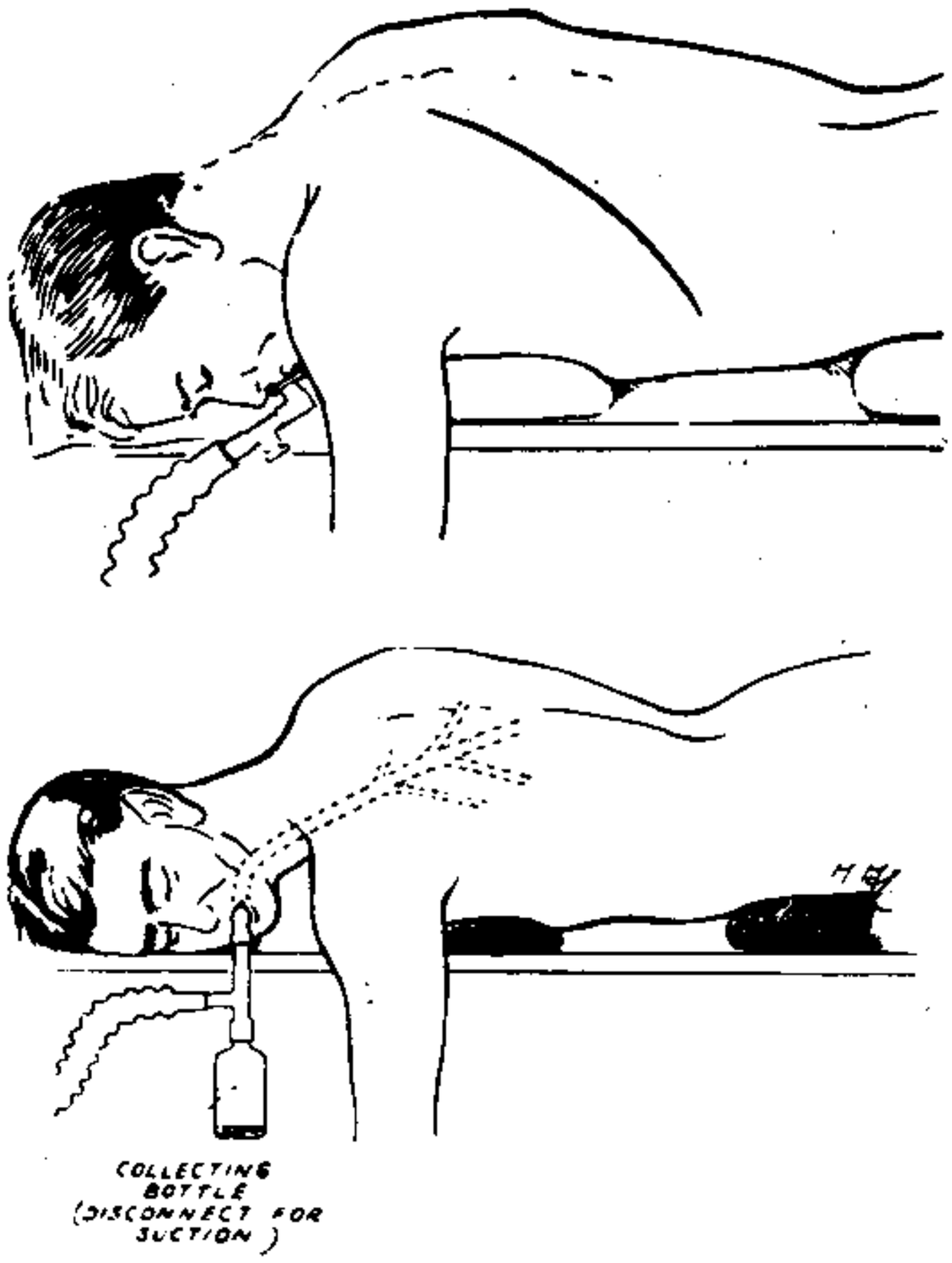


Fig. 13

allowed to develop an oxygen lack in the attempts to restore normal respiration I think it is safe. Ideally Neostigmin should not be given until there is some evidence that the curare block is passing off e.g. the first signs of returning spontaneous respiration or of voluntary movement. If Neostigmin is given whilst the curare block is complete the acetylcholine which accumulates from the inhibition of the cholinesterase cannot be effective and may produce a prolonged paralysis by depolarisation. If a patient will not breath at the end of the operation and neostigmin has been given some other cause should be sought. Overventilation and lack of carbon

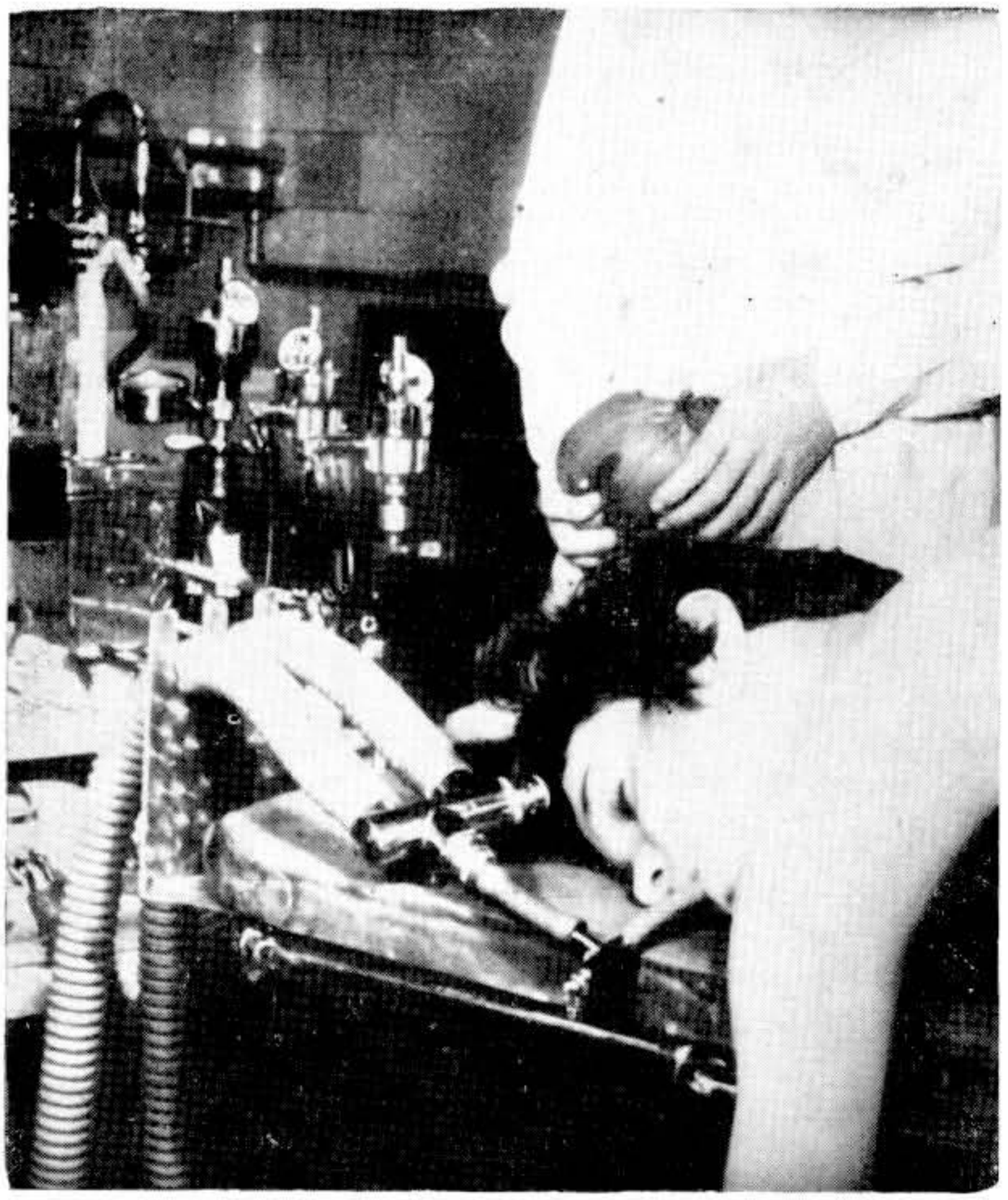


Fig. 14

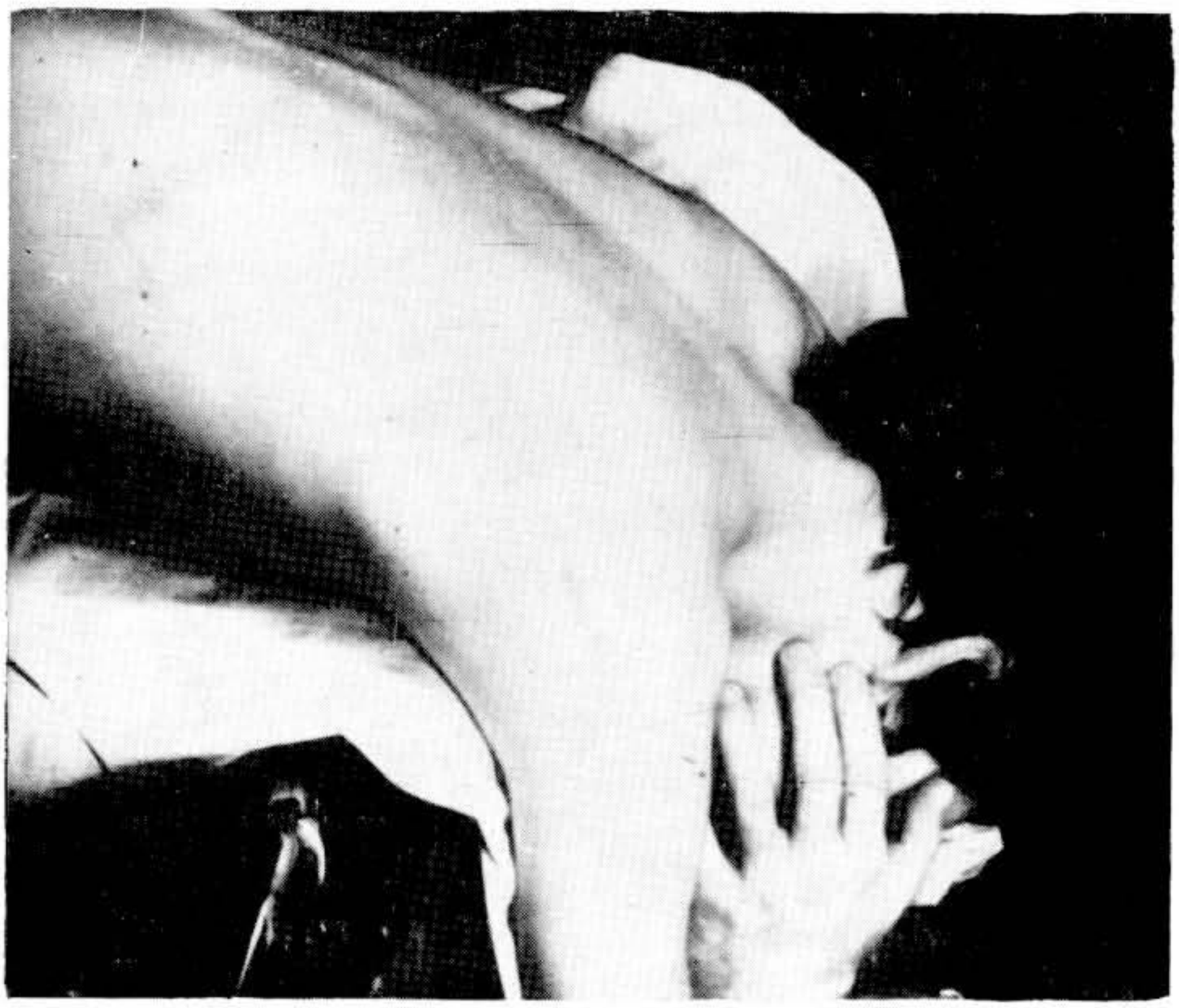


Fig. 15

dioxide is common but if the closure of the wound has been made with the absorber out of circuit this is unlikely. Anoxia of the respiratory centre may be the fault either as a result of poor ventilation or of circulatory failure. Inflation of the lung with oxygen will cure the one and analeptics can be tried for the other. The inhibition may be reflex if the presence of the endotracheal tube is resented at a light level of anaesthesia. The bold removal of the tube restores respiration. This is not so heroic as it sounds for if the diagnosis is wrong reintubation is easy. The centre may be depressed by morphia or pethidine given as a supplementary analgesic during the operation. If there are good grounds for suspecting this N. allyl morphine 10 mgm. is useful. Management on these lines will be sucessful unless there has been some cerebral catastrophe or there is a myasthenic state. Thymectomy for myasthenia gravis presents an interesting problem but briefly the patient is adjusted to intramuscular Neostigmin at 4hrly intervals. The operation starts 3hrs. after a routine dose. After an induction with thiopentone and nitrous oxide and oxygen it will be found that the myasthenia has caused sufficient relaxation to allow intubation and that overventilation will control the respiration without the use of curare. At the end intravenous neostigmin will relieve the muscle weakness and restore normal breathing. The trouble comes if the condition has not been diagnosed preoperatively or in those rare cases of carcinoma of the lung complicated by a neuropathy in whom a normal dose of tubarine will produce a paralysis lasting many hours. The sure way of avoiding this possibility is to use a test dose of curare before starting the anaesthetic, but if this has not been done and I should admit that it is not my practice, continued artificial respiration until the curare has been excreted is necessary.

Assuming that these difficulties have been met the patient is returned to bed. Given supplementary oxygen as required and general aftercare.

Although purely anaesthetic considerations would dictate the use of the prone position, surgical convenience must decide the approach used. In a dry case the lateral position can be managed with exactly the same anaesthetic technique, but displacement of the mediastinum may cause difficulty. This can be corrected by placing packs to restrain the distension of the exposed lung and

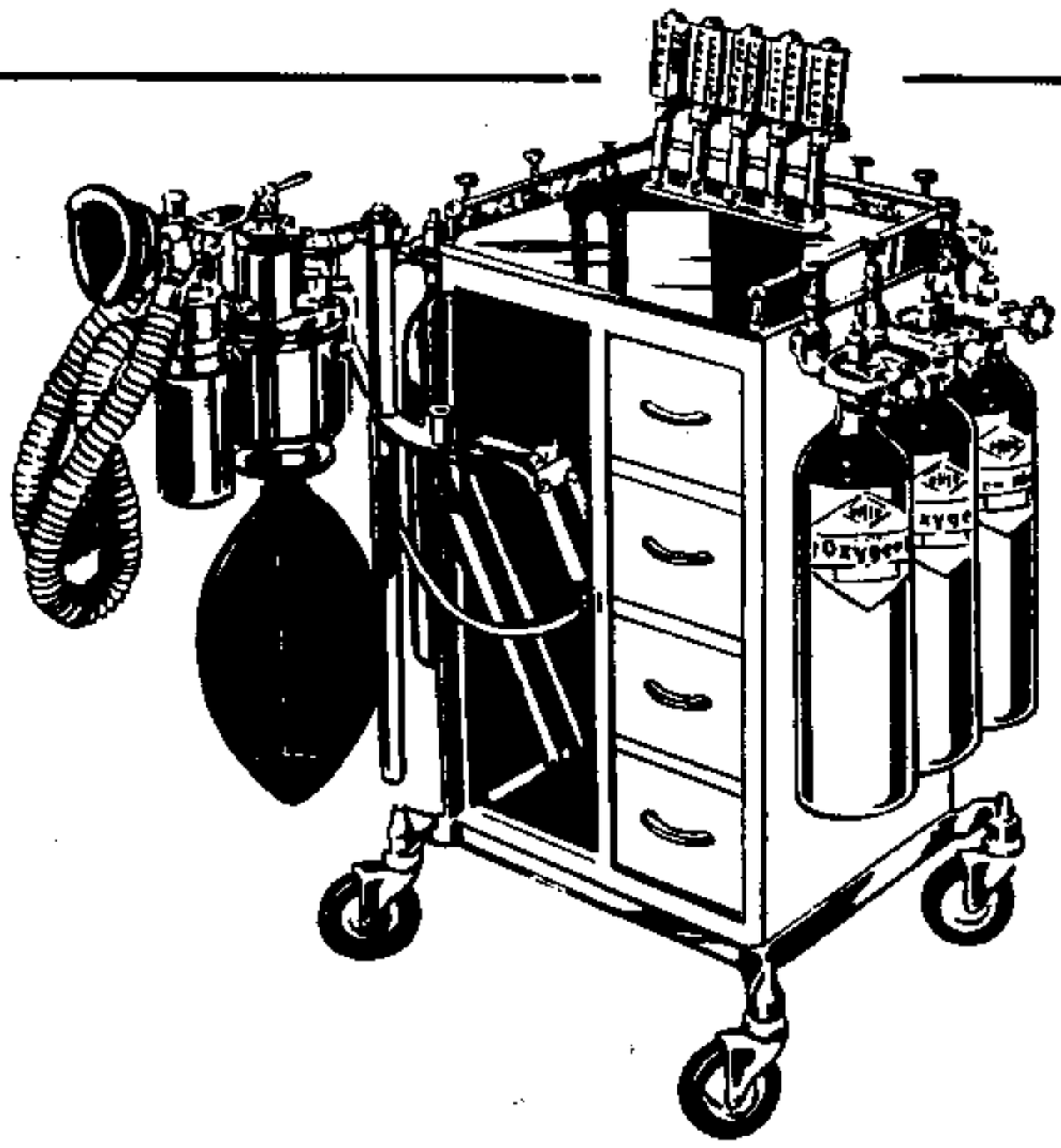
then inflating the under lung by using an increased positive pressure for the artificial respiration. If the patient is wet I think that secretions should be controlled by endobronchial anaesthesia or by the use of a bronchus blocker. I use a Thompson blocker.

The technique with a bronchus blocker is similar until the bronchial toilet at the start of the anaesthetic. This must be performed through an adult (11 mm.) bronchoscope. The Thompson blocker will not pass through a smaller bronchoscope. The site of election for the balloon is then visualised through the bronchoscope, which is then withdrawn 1 cm. The blocker is then passed until the balloon is clear of the end of the instrument. The balloon is inflated to fix it in position and the bronchoscope withdrawn. The anaesthesic is given through a cuffed endotracheal tube inserted beside the blocker. The management is similar until the time comes to clamp the bronchus before which the blocker must be removed. The drill for the removal of a blocker is straight forward. Suction is applied to the lumen of the blocker to remove secretions. The balloon of the blocker is deflated. This allows air to drawn past the balloon clearing any sputum that may be trapped there. The cuff of the endotracheal tubes deflated and the blocker can be withdrawn. The bronchus is clamped and the controlled respiration restarted into the remaining lung. Subsequent management is similar to that described for the prone position. The blocker can be placed in the left lower lobe, in the left main bronchus, on the right side below the middle lobe orifice, blocking the right basal branches, between the middle and upper lobe orifices, blocking middle and lower lobes, and in such a position that the whole right lung is isolated. For upper lobe resections the whole of one lung is isolated so that the contralateral lung is protected. The lower lobe on the diseased side is carefully aspirated postoperatively. Some anaesthetists favour endobronchial anaesthesia especially left endobronchial anaesthesia for right pneumonectomy. I do not favour it as I have found that the tube is liable to slip into the trachea when the hilum is pulled in the process of dissection, fig. 2 and 3. I think double lumen tubes offer too great a resistance to respiration to justify their use.

A study of the problems of the open chest is very rewarding to the anaesthetist for if anoxia is avoided and blood loss made good patients tolerate the most formidable operations.

SEGURANÇA **EFICIENCIA**

HEIDBRINK KINET-O-METERS



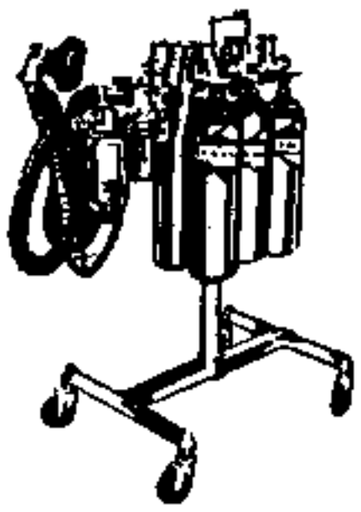
KINET-O-METER
Modelo 550 - Tipo
Armário - 5 Gases

Os KINET-O-METERS, ainda que desenhados especialmente para a utilização do método de grande economia da absorção do CO_2, também permitem o uso do método da reinalação parcial. Os debímetros são construídos e calibrados de maneira apropriada, indicando e medindo com a maior precisão os fluxos necessários ao sucesso de ambas as técnicas. Há sempre um KINET-O-METER que se enquadra a qualquer exigência técnica.

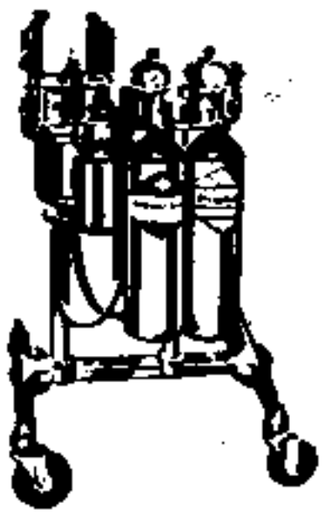
Modelos: Stand, Carro, Gabinete, Carro-Gabinete e Midget.

Peça o novo Catálogo Heidbrink com informações completas sôbre Aparelhos de Anestesia.

KINET-O-METER
Mod. 212A - Stand - Cilindros pequenos - 2 Gases: N2O-O2



KINET-O-METER
Mod. 650 - Midget portátil ou em stand.



KINET-O-METER
Mod. 403A - 4 Gases: N2O e O2 em cilindros grandes - C3H6 e CO2 em cilindros pequenos.

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(1-nor-epinefrina)

HORMÔNIO PRESSOR

LEVOFED é a 1-nor-epinefrina, sob forma de bitartrato, de indicação precípua no tratamento dos estados de hipotensão aguda: choque operatório, hemorragias graves, depressão vaso-motora central, infarto do miocárdio.

LEVOFED, mediador simpático da vaso-constrição geral eleva a pressão sanguínea sem maiores alterações do volume-minuto cardíaco e sem constrição dos vasos coronários.

Apresentado em solução a 1:1000 (base), em ampôlas de 4 cm^3, o LEVOFED é de aplicação *estritamente intravenosa*, em gôta-a-gôta, após prévia diluição em 1000 cm^3 de solução glicosada a 5 %.

Amostras e literatura à disposição da classe médica

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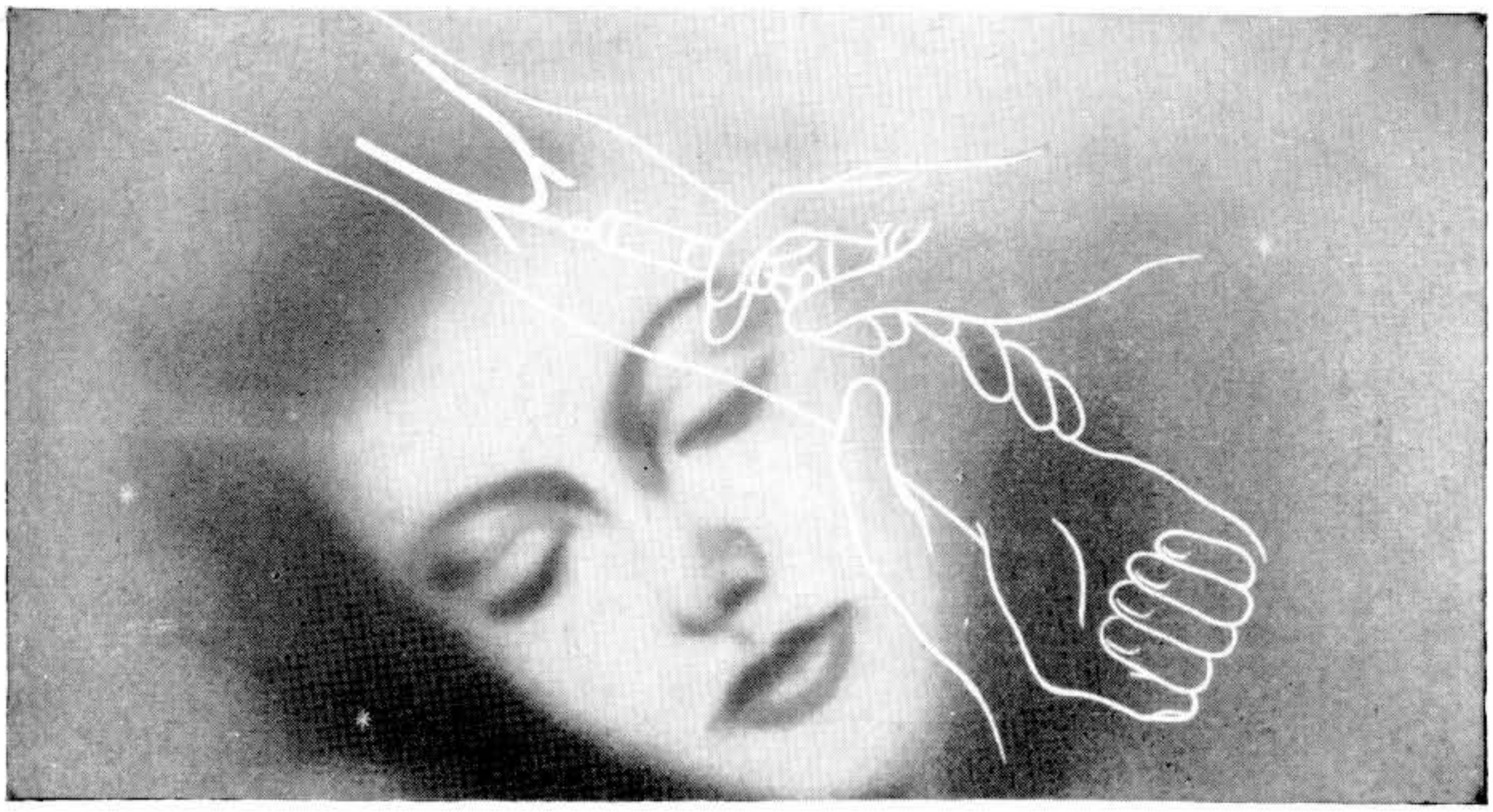

ANESTESIA ENDOVENOSA

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KEMITHAL

(Tialbarbitona Sódica)

- indução suave e rápida.
- complicações, tais como espirros, tosse, laringospasmo, excitação e tremores, são raras.
- o despertar é rápido e não se observam inconvenientes post-anestésicos.

(*Lancet*, 1946, 1, 768)

Ampolas de 1 g e 2 g, acompanhadas de ampolas de água bidestilada.

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