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ANAESTHESIA FOR CARDIAC SURGERY (*)

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The recent advances in the surgery of the heart and great vessels have been permitted by the progress of anaesthesia. The first condition for success in operations on the heart is the prevention of deterioration in the patient's condition during the period of the open pneumothorax. Thoracic surgery was sadly hampered until the technique of controlled respiration by intermittent positive pressure was introduced. Before this the only elective procedure in cardidac surgery to gain an established position was the decompression of the heart in constrictive pericarditis — even this was limited to an anterior approach to avoid opening the pleural cavity. Injury to the pleura was then a serious — occurrence leading to operative and post-operative complications.

A further obstacle to the development of heart surgery in the past was the difficulty of induction of anaesthesia in cases with severe circulatory embarrassment. When the art of anaesthesia consisted of the gentle administration of irritant vapours through the respiratory tract, the second stage of anaesthesia, that of heightened reflexes was always a time of hazard. The apprehensive patient was liable to struggle and throw an unwarrantable burden on a disordered circulation. Laryngeal spasm causing sub-oxygenation was troublesome in the healthy patient, but it was terrifying when the myocardium was so little removed from failure. A prolonged circulation time caused the concentration of the anaesthetic in the brain to lag behind that in the air-passages:

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hence laryngeal spasm was more frequent and induction more difficult. Finally there was the fear of reflex disturbance of cardiac rhythm from vagal stimulation during intubation. The methods of anaesthesia that I am going to discuss have been developed against a background of these past difficulties.

A wide variety of conditions are now presented to the anaesthetist for operation with a defect of the heart or great vessels. Operations for the relief of mitral stenosis, for the closure of a patent ductus arteriosus, to improve the pulmonary circulation in Fallot's tetralogy, to decompress the heart in constrictive pericarditis, to excise a structure in coarctation of the aorta or an aneurysm as well as the operations on the open heart provide a variety of problems as difficult to rationalise as to anaesthetise.

First a brief mention of patent ductus arteriosus. This is usually a straighforward thoracic operation on a healthy child, for although the murmur is loud and the patient may be incapacitated for athletic games yet the direction of the shunt from left to right ensures full oxygenation of the circulating blood and easy control of inhalation anaesthesia. It is only when the condition has been neglected until right heart failure has developed, or infective endocardits has complicated the lesion that anaesthetic difficulty has to be feared. The child should have fairly heavy premedication. I use pentobarbitone (Nembutal) ½ gr. per stone body weight which is equivalent to 4 mgm. per kilo, an hour and a half before operation followed by morphia and atropine in proportion to age and weight., half an hour before operation. This should ensure that the child arrives for operation either asleep or so sleepy as to be indifferent. Anaesthesia is with nitrous oxide oxygen and curare. It may be necessary to give about 100 mgm. of thiopentone at the outset of the anaesthetic if the child is not deeply asleep. Controlled respiration is used from the start. The other requirement is a reliable intravenous transfusion. Although normally the blood loss is trivial, if the wall of the ductus is degenerate it may tear and cause sudden severe haemorrhage requiring immediate replacement. When the chest has been opened the upper lobe can be easily packed away to expose the ductus whilst the rhythmical inflation of the chest is continued. When the ductus has been tied two changes are apparent. The action of the heart becomes much quieter. The beat which was laboured and thrusting becomes smooth and efficient. This corresponds to a change in the blood pressure. The diastolic pressure is raised whilst the systolic is little changed. When the ligation is completed attention is directed to reinflation of the lobe which has been retracted. First the inspiration is prolonged for a few breaths, if this is not sufficient the inspiratory pressure is raised and if still some areas of lung are blue and airless then the surgeon gently handles the lung in these areas which

seems to release some local bronchospasm and permit full inflation. The chest is now closed; if no air is trapped in the pleura these children do very well without drainage of the pleura. If some air is left in the pleural cavity either because the lung is not kept fully inflated as the final closure is made or because the patient is allowed to make an active inspiratory movement before the chest is airtight, they are liable to develop a pleural effusion which will need aspiration and may even collapse a lobe. At the end of the operation the return must be made to adequate spontaneous respiration as has already been fully described. The only modification is in the dose of Neostigmin which must be varied according to the size of the child. Roughly 0,5 mgm. for a two year old, 1 mgm. for a five year old and 1.5 mgm. for a ten year old child. If the air passages are clear and there is no residual paralysis or respiratory depression there should be no need for oxygen therapy postoperatively. If one of these children is not well oxygenated when returned to bed I would rather find the reason than prescribe an oxygen mask. I sit them up in bed as soon as they are sufficiently conscious and treat pain with suitable doses of pethidine.

I want to take as my main theme anaesthesia for the operation for the relief of mitral stenosis. It is I suppose the most generally useful of the cardiac operations. It restores to full activity an adult and useful citizen. Often a mother is enabled to undertake afresh the care of her family. In Great Britain it was recently estimated that there were 240,000 sufferers from Rheumatic heart disease of whom 2/3 had predominately a mitral lesion. To deal with those suitable for surgery would require 3,000 valvotomies a year, so the problem is worth while.

Our first consideration must be the preparation of the patient. The selection of the case is not our problem. If the cardiac physician thinks that the patient will benefit from surgery then I think we can find a suitable anaesthetic technique. Of these cases none is so well that it is safe to operate without a preliminary stay in hospital, none so severe that they cannot be anaesthetised. I think the patient must be admitted to hospital for at least a week. A few will need absolute bed rest, but for the majority a relief from the tension and bustle of life at home and at work will give the margin of cardiac reserve we require for the induction and maintenance of anaesthesia. They need to be digitalised and this period can be used to establish them on a suitable dosage. We do this both for those with auricular fibrillation as well as those with normal rhythm. We do not now give quinidine preoperatively. If there is fluid retention they are put on a salt restricted diet and probably a mercurial diuretic is used. In this period they are placed in the care of

a physiotherapist. Instruction is given in breathing exercises, particularly the expansion of diferent segments of the chest against manual pressure. This teaches them to eliminate sputum more readily and to breathe more effectively both of importance after thoracotomy when pain and stifness cause reduced chest movement and sputum retention. In those cases where a high pulmonary vascular resistance is suspected there is an added risk of venous thrombosis in the legs and physiotherapy can be usefully directed to reducing stasis in these vessels.

The assessment of the optimum time for operation may be difficult and is properly left to the cardiologist. To the anaesthetist the cheering signs will be the relief of breathlessness, the steadying of the pulse, reduction of oedema and on X-ray the clearing of the lung fields and the reduction of the heart size. Once the decision is made that the patient is suitable and ready for operation they must be reassured. This is not only kind to the patient but benefits the anaesthetist. The frightened patient is more difficult to anaesthetise and so requires more of the anaesthetic agent which may cause postoperative depression and he may struggle in the induction thus throwing an unwelcome burden on the heart. The patient with a known cardiac lesion presenting for a minor operation e. g. a dental extraction is often more frightened of the effect of the anaesthetic on their heart than of the operation, possibly justifiably. Fortunately when seeking relief for their cardiac disability the feeling of trust inspired by the surgeon extends even to the anaesthetist. This trust can be reinforced by premedication. Premedication normally serves three purposes: to allay anxiety, to dry secretions and to reduce reflex activity. Sedation is most important for it reduces the metabolic demands of the body and so eases the burden on the heart. It serves to some extent as a basal anaesthetic and smaller doses of other drugs are required in the operation. Secretions are dried by scopolamina as ordinarily atropine is liable to produce an undersirable tachycardia — inconvenient to the surgeon and dangerous because the shortening of the diastole and incomplete filling of the ventricle may cause circulatory embarrassment. On the night before operation I give pentobarbital 200 mgm. reinforced if there is any bronchospasm by promethazine 25-50 mgm. On the day of operation Omnopon 20 mgm. and Scopolamine 0.4 mgm. one and a quarter hours before the anaesthetic is due to commence.

However thoroughly they may be premedicated it is important that the orthopnoeic patient should come to the theatre confortably propped up (fig. 1). An attack of pulmonary oedema may be precipitated if such a patient is made to lie flat on a trolley, and even those patients who have protected themselves against the transudation of fluid through the pulmonary capillaries by devel-

oping a high pulmonary vascular resistance will suffer a respiratory distress which will render anaesthesia more difficult.

The cardiac reserve is poor. The output is limited by the stenosis. If vasodilatation occurs in the peripheral vascular bed either from struggling in the induction or as a result of the action of drugs the blood pressure will fall severely. This fall may impair the coronary circulation. The pulmonary function is impaired as a result of congestion, winter bronchitis or raised pulmonary vascular resistance. Any interference with regular respiration quickly results in oxygen lack, whilst the response to the inflation of the

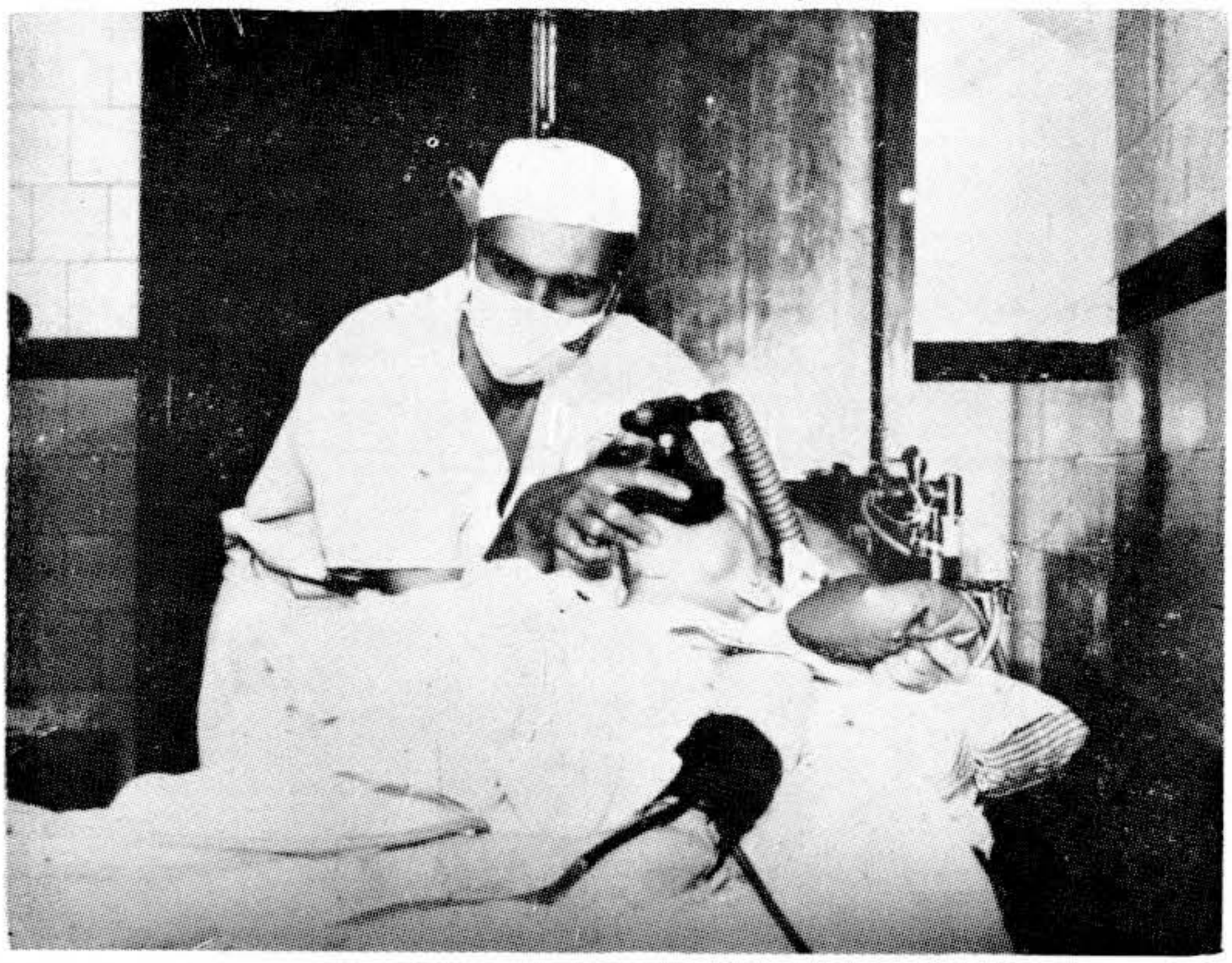

Fig. 1

lungs with oxygen is delayed by poor absorption and by sluggish circulation. If the myocardium is deprived of oxygen by diminished coronary circulation and deficient oxygenation of the blood its action becomes weak and irregular. The poorly oxygenated ventricle may go into fibrillation when the heart is handled. Our aim then must be a completely smooth induction and full oxygenation of the circulating blood.

I think topical analgesia of the respiratory tract is very helpful in these cases. It obviates the risk of laryngeal spasm, and reduces

the chance of reflex disturbance of cardiac rhythm during intubation. I use lignocaine 4 % as an analgesic agent and I give 2 ml. through the cricothyroid membrane. This is easily done if first the thyroid and then the cricoid cartilage is identified. The mid line of the cricoid is recognised by two minute papillae on the upper border of the ventral surface of the cricoid. The cricothyroid memorane is placed immediately above and between these two projections (fig. 2). In any case in which I have special anxiety I anaesthetise the larynx in this way before inducing anaesthesia, but if there are ample reserves I make the injection after the patient is unconscious. It is not quite so safe but it is much nicer.

The first step in anaesthesia is to have the patient breathing quietly and steadily from an oxygen mask. I use the simple Magill's attachment to a Boyle's apparatus with about 6 litres per minute

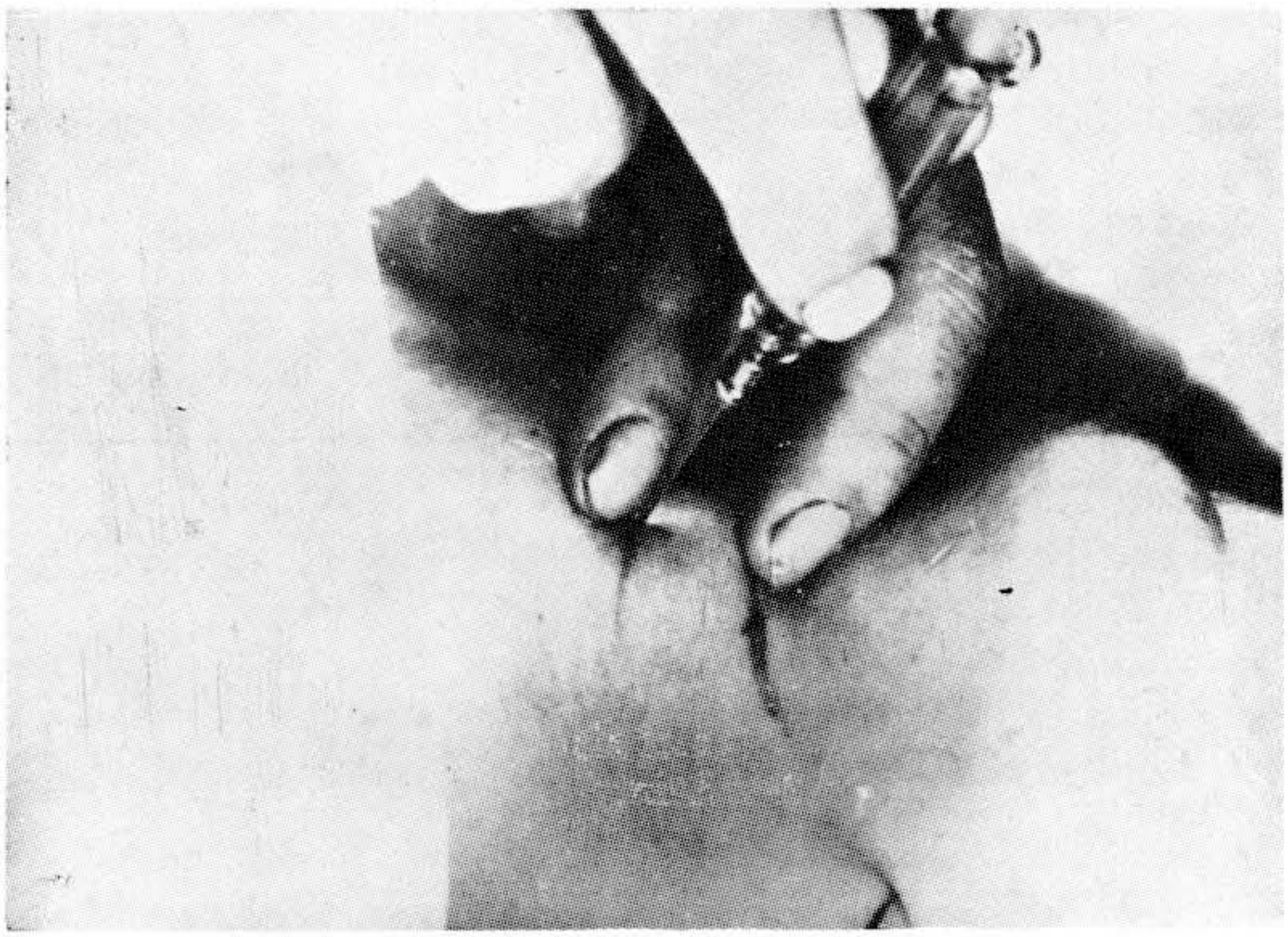

Fig. 2

of oxygen running. When I am sure that they are breathing comfortably on this high oxygen supply I start the intravenous administration of thiopentone. It is essential that it should be given slowly. The prolonged circulation time can cause great delay in the onset of sleep and as a dose of 100-150 mgm. is often sufficent to induce sleep it is easier to gauge the best dose if a dilute solution, say 2.5 % is used. Having ascertained the sleep dose and providing

that it has not caused any obvious deterioration in the patients condition, I give a total of twice the sleep dose. I follow this with 30 mgm. of tubarine. The patient continues to breathe the oxygen and as the curare begins to weaken the respiration the patient's efforts are at first assisted and then the respiration is controlled. Whilst this is occurring the cricothyroid injection of local analgesic is given unless this has been done before starting the anaesthetic, and the blood transfusion is started. The period of some three minutes whilst the full relaxation from the curare is developing gives ample time to arrange this. It is important to avoid overloading the circulation before the stenosis has been relieved yet it is necessary to be prepared to deal with sudden and severe haemorrhage. I find that the most reliable position is the forearm on the right side. It is less subject to interference by the surgeon and his assistant than is the left arm and less liable to venous spasm than a cannula in a leg vein. In severe haemorrhage it may be necessary to put in the blood under positive pressure, and in cardiac failure an intraarterial transfusion may be necessary. Fortunately this can be given directly into the aorta if a sterile outfit and a bottle of blood is kept ready. So a drip through a cannula is set up in the right forearm but the blood is given very slowly until blood loss requires replacement.

When the patient is fully relaxed he is intubated with a large cuffed endotracheal tube. This is connected to a carbon dioxide absorption circuit fed with nitrous oxide 3 litres and oxygen 2 litres per minute. The large flow is used in order to have a reasonable knowledge of the mixture actually breathed by the patient. If small flows are used variations in the metabolic rate and leaks from the circuit make an accurate estimate of the proportion of the gases impossible. Such knowledge is important because the depth of anaesthesia maintained by the intravenous drugs is so shallow that without the nitrous oxide the patient would wake up whilst on the other hand the secret of maintaining a good cardiac action is to prevent any oxygen defficiency. The light level of anaesthesia minimises vasodilatation and aids a rapid postoperative recovery. Suboxygenation occurring with this mixture is very suggestive of under-ventilation. Higher oxygen concentrations can allow a patient to be fully oxygenated but hypercarbic.

The position on the table is lateral with the patient so fixed that lateral tilting can be used (fig. 3). The positioning is carried out slowly and gently. The changes in haemodynamics required by a change in posture are made with difficulty. There is usually a fall in blood pressure which may be severe if the movement is too rapid.

The next step in anaesthetic technique is the inserting of a paravertebral block with 0.5 lignocaine, of segments 2-8. This block for convenience is made by the surgeon. It serves two useful purposes. It reduces the quantity of general analgesic drugs needed to keep the patient free from reaction and it keeps him free from wound pain in the first postoperative hours allowing deep breathing and coughing. We usually have 1/300,000 adrenaline in this local analgesic solution. This is open to criticism in that it increases the irritability of the heart. If I am worried by the conditions of the patient I omit the adrenaline but in the majority of cases the adrenaline does no more than check the fall in blood pressure caused by moving the patient into position.

The blood pressure chart in these cases is interesting (fig. 4). The pressure in the anaesthetic room after premedication is naturally lower than in the ward usually by some 10-15 mmHg. Thio-

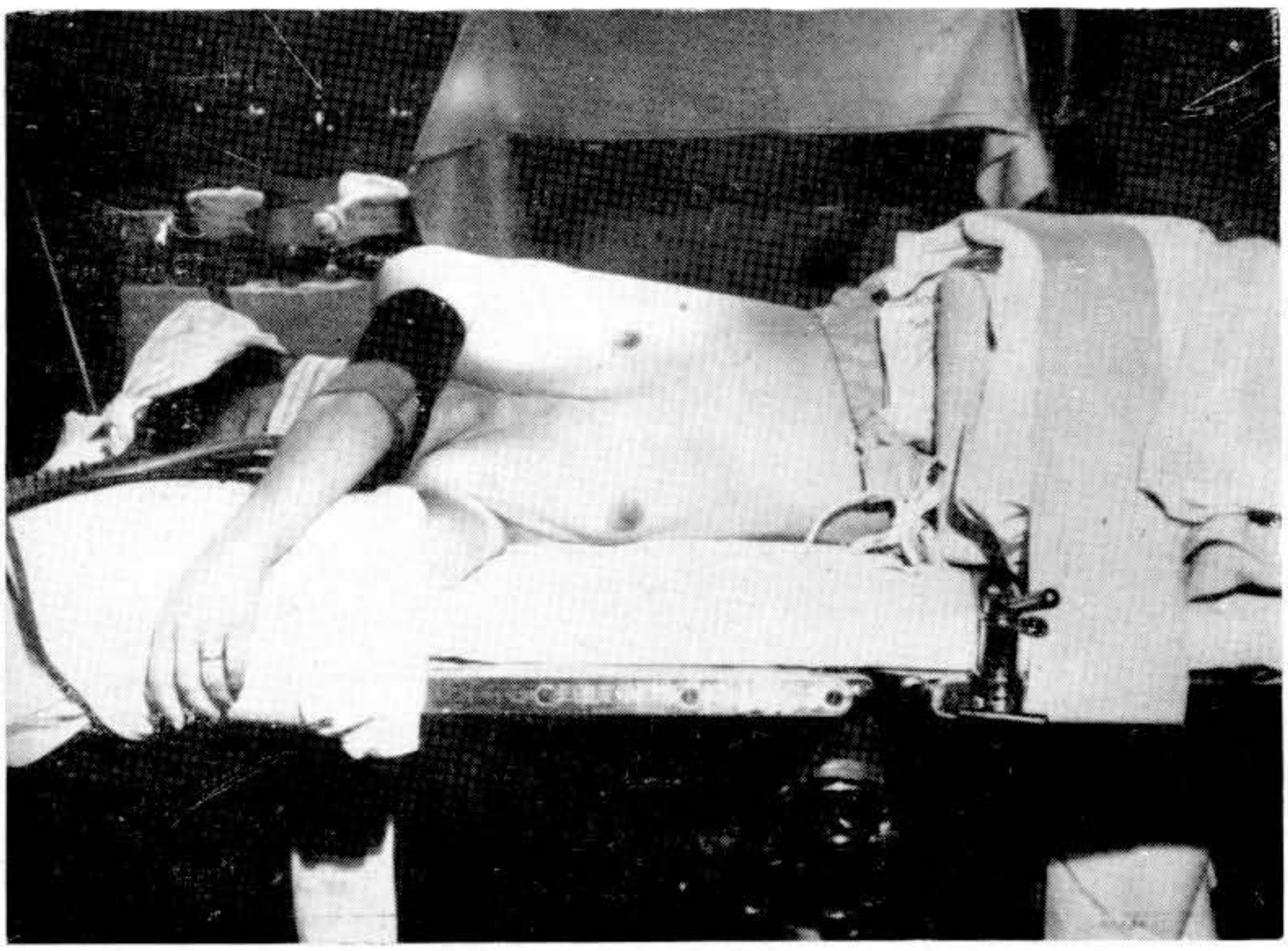

Fig. 3

pentone produces a further fall so that after induction I expect a pressure 90/70. If the thiopentone is carelessly given too quickly the fall may be disastrous. The movement of the patient in placing him in position on the table causes a further fall and I expect a systolic pressure of 70 mmHg. This is usually corrected by the infiltration so that at the start of the operatio I expect a pressure 90/70. When the pericardium is opened there is a further fall

in pressure so that by the time the clamp is on the auricular appendage the systolic pressure is about 60 mmHg. The finger in the auricle does not affect the pressure although the few beats occurring whilst the finger is in the valve are completely inffective. Loss of blood at this stage may cause a critical fall in that the coronary circulation becomes insufficient and the heart fails. I have already stressed the importance of having effective methods of blood transfusion ready. In the normal case the pressure does not begin to rise immediately after cutting or splitting the valve, but a steady rise begins as soon as the pericardium is closed. At the end of the operation a systolic pressure of 100 mmHg. can be expected.

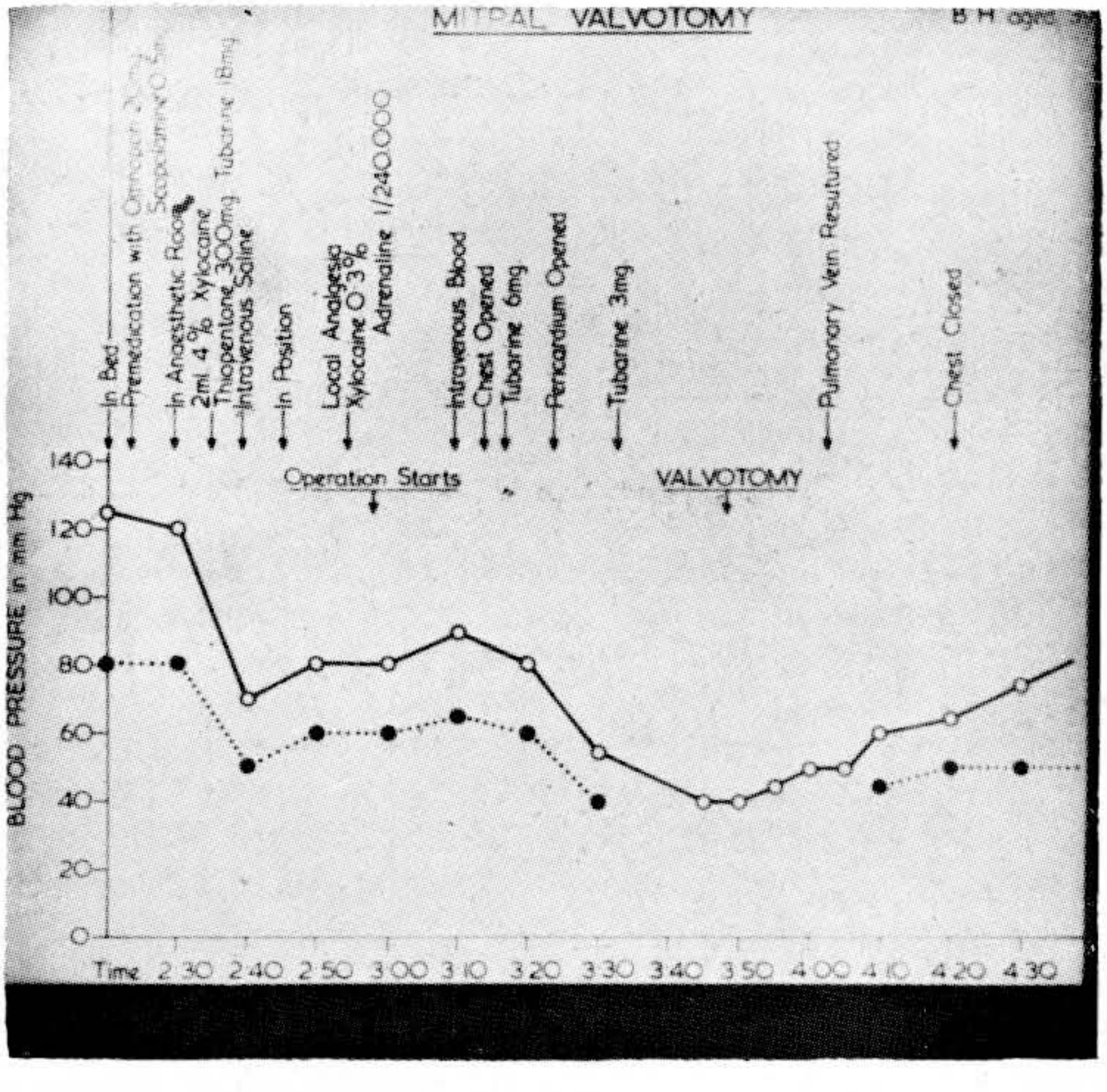

Fig. 4

In order to give a continuous record of the changes in blood pressure we have leapt ahead of our discussion of the general management of the cases. The chest is opened by an antero-lateral incision through the base of the 5th. rib. When this has been done and the side towels and retractors are in position the table is tilted laterally, towards the surgeon. The lung in an established case is

firm and rubbery and does not pack away as readily as normal lung. It is essential that this packing should be done efficiently for unless this is done either the patient will be underventilated in order that the surgeon may have a good field or the lung will billow forward obscuring the heart. We make use of malleable retractors which control the lung very effectively and enable the use of pressures of up to 25 cms. of water for inflation without the lung invading the field. The actual pressure used will vary from case to case and depends on the build of the patient and the elasticity of their tissues but the tidal air volume must be maintained and the mediastinum must not be allowed to fall away too far from the midline. If the mediastinum sags it becomes more difficult for the surgeon to introduce his finger through the auricular appendage into the valve, and the diminution in the capacity of the right hemithorax will progressively limit the ventilation. Is is very characteristic of the return of muscle tone that the tidal air is reduced whilst the ventilating pressure is unchanged. One advantage of a mechanical respirator is that if the ventilating pressure is kept constant the changes in the volume breathed can read of the scale and a warning is given of the need for more drugs.

For many patients no further drugs are administered but in resistant patients and if the procedure is prolonged additional drugs may be needed. The indications for additional relaxant are (1) an increase in the inspiratory pressure to maintain the tidal volume (2) an increased tendency for the lung to billow into the wound owing to decreased expansion of the other lung (3) the return of spontaneous respiratory movements usually heralded by jerky diaphragmatic movement. In those operations where an arterial suture is used it is wise to give an extra dose of relaxant before commencing the suture lest a sudden jerk should tear out the stitches. The end point of the action of curare is indefinite and a further dose of curare can be avoided at the end of the operation by over ventilation, which removes the stimulus of carbon dioxide.

An analgesic is indicated by (1) movements of the fine muscles of the face and hand (2) increase in the pulse and fall in blood pressure not explained by the surgical manipulations at the time (3) dilatation of the pupil (4) secretion of tears (5) vasoconstriction evidenced by pallor, drop in skin temperature and delayed capillary refil (6) sweating (7) the onset of bronchospasm, a slight increase in the resistance to inspiration and a prolongation of expiration. When an analgesic is necessary I use pethidine 20-25 mgm. and repeated if indicated.

Opening the pericardium almost always lowers the blood pressure and often produces a severe disturbance of cardiac rhythm. Whilst surgical manipulation is the principal cause of irregularity,

anoxia and hypercarbia are important contributary factors. We used to reduce this irregularity with intravenous procaine or procaine amide, but if the condition is ignored and good ventilation is maintained it usually settles as soon as stimulation ceases. We have abandoned its use because a dose sufficient for this purpose causes vasodilatation and the associated myocardial depression results in severe falls in blood pressure.

In a straigntforward case the clamp is now placed around the base of the appendage. An incision is made large enough to admit the index finger. The finger is introduced and the clamp removed, this allows the finger to advance into the auricle. The size of the orifice is palpated. The size of regurgitant stream determined if there is any associated incompetence.

The adherent cusps are separated by forcing the finger through the orifice or the finger is withdrawn and reinserted with a small ring knife to start the separation. This is a matter of a few moment and normally without any adverse effects on the patient. It is with the abnormal that we must concern ourselves.

If the appendage is too small to admit the finger, an incision is made in the wall of the auricle. This involves the loss of between 50-100 ml. of blood each time the finger is inserted and withdrawn. This loss must be anticipated and replaced by transfusion.

The presence of clot in the auricle or the appendage is more troublesome. If the clot is in the appendage and the clamp can be placed between the clot and the atrial chamber the procedure is fairly simple. When the incision is made and before the finger is inserted, the clamp is temporarily released and blood allowed to flush out any clot which may be loose in the chamber. The blood loss is made good and the operation proceeds. If the clot extends further the risk of embolism is much greater and may demand a reassessment of what the patient has to gain as opposed to the risk of cerebral embolism. To reduce this risk the carotids may be compressed for short periods either manually in the neck or by clamps or tourniquets in the chest. Naturally this compression can only be applied for short periods. We apply the pressure for not more than 30 seconds at a time at the critical times when we anticipate that the surgical maneouvres may detach clot. The occurrence of cerebral emboli can frequently be diagnosed by the anaestetist. Sweating, a sharp fall in blood pressure which does not return to its former level, a deterioration in colour despite adequate ventilation, and deep unconsciousness at the end of the operation when most patients are awake are suggestive. Peripheral emboli may show themselves by the loss of pulse in a limb, but this is not usually found until the routine examination at the end of the operation. The rapid return of consciousness at the end of the ope-

ration allows simple instructions to be carried out by the patient in order to establish whether an embolus has occurred. The anaesthetist may be able to assist by an appropriate sympathetic block.

The emergencies of cardiac arrest and ventricular fibrillation require special attention. This is one of the greatest responsibilities of the anaesthetist. The success of restorative measures depends on the promptitude with which they are instituted. Paradoxically enough the attention of the surgeon may be concentrated on some detail and although he is actually operating on the heart he may not notice the cessation of an effective circulation. I think that the anaesthetist should feel the pulse and so far as possible observe the heart throughout the critical periods of the operation. To facilitate this we use a mirror suspended from the overhead light which allows the anaesthetist to see whilst seated at the top of the table (fig. 5). The necessity for continous observation as

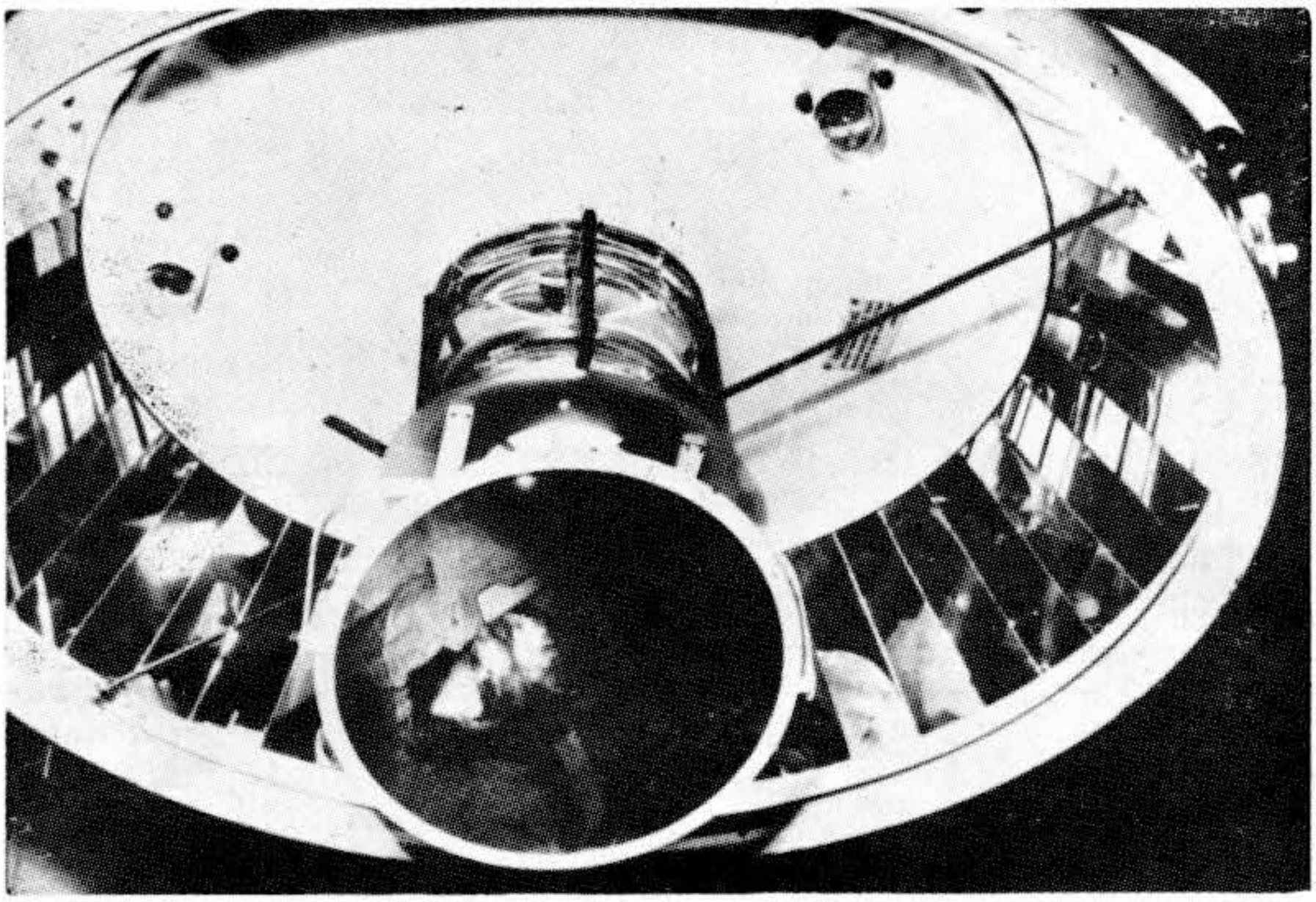

Fig. 5

well as the duty of maintaining ventilation means that the anaesthetist is not free to keep a record of the pulse and blood pressure. This must be deputed to an assistante. If an arrest of the circulation occurs two duties immediately devolve on the anaesthetist. The exact time of the stoppage must be noted and the sugeon informed. Artificial respiration with pure oxygen must be maintained throughout the emergency. When the survival of cerebral cells is in the balance the presence of nitrous oxide might interfer

with their oxygen uptake so the change to 100 % oxygen is made. Surgically the stenosis or other cardiac lesion should be relieved if possible. The restoration of normal action and even the maintenance of an artificial circulation by cardiac massage is far more possible if the lesion is corrected. Except for the proviso that an attempt will be made to correct the lesion the survival of the patient depends on the promptness and effectiveness of the cardiac massage. This can be quite effective with a clamp closing the incision in the auricle but the heart must be watched for leaks. The squeezing of the ventricle should be at a rate of 60-70 per minute and at a pressure sufficient to produce a palpable carotid pulse. The effectiveness of the artificial circulation may be gauged by the maintenance of a small pupil, the colour of the lips, and the nature of the capillary refil. If these are not satisfactorp or are deteriorating more drastic steps are necessary for although cardiac action may be restored in a patient whose pupils have become large and irregular, whose skin is mottled and lips remain bluish white there is little hope of recovery from the accompanying cerebral damage. It is worth emphasizing that once an artificial circulation has been established, haste is no longer necessary. There is time for a considered approach. Oxygen to the cerebral cortex is essential and is so far as possible being provided. Oxygen for the heart muscle is the other great requirement.

Until it receives an adequate blood supply normal rhythm is unlikely to occur. Two further steps can be taken to promote that circulation. While the surgeon continues the cardiac massage the assistant turns the lung forward and applies a light clamp to the descending aorta. This clamp improves the cerebral and coronary circulations at the expense of organs whose tolerance of oxygen lack is much greater. The second step is to start a transfusion under pressure into the aorta.

Drugs have their place once these measures have been instituted. In the case of cardiac standstill adrenaline is most valuable 5 mk. of 1/10,000 injected into the cavity of the left ventricle and massaged into the circulation may cause the heart to spring into action. It may even be useful in ventricular fibrillation if the heart is distended and the fibrillation weak, for the electrical defibrillator will work more effectively on muscle with good tone. If a defibrillator is not available, procaine 5-10 ml. of a 1 % solution may prolong the refractory period sufficiently to stop the uncoordinated waves of contraction and it may be hoped that when the arrested ventricle restarts it will have a normal contraction. Neostigmin 0.25 mgm. may stop a fibrillation. I have never had any success with any other drug.

In the absence of these excitements the incision in the heart is sutured with or without the amputation of the auricular appen-

dage. The pericardium is loosely closed. The packs and retractors are removed and the lung reexpanded as already described. These cases are usually drained with the tube atached to a nonreturn water seal. When the chest has been closed the soda lime cannister is removed from the circuit. The over ventilation associated with the reexpansion of the lung washes out an excessive amount of carbon dioxide. Until the chest is closed, this is advantageous for it ensures that there will be no spontaneous respiration which could draw air into the chest. Once the chest is closed the normal stimulus to breathing should be restored as soon as possible. I always use neostigmin to counteract any persisting action of the curare. It is preceded by atropine 0.6 mgm. and I use 1 mgm. repeated if necessary.

The routine of examination for peripheral emboli has been described. The patient is returned to bed and is given analgesics to control pain and restlessness. Oxygen is given if there is the slightest indication. I have reverted to an old fashioned method of supplementing the oxygen by using the Tudor Edwards spectacles. These were condemned as inefficient as compared with masks such as the B. L. B., but if the two catheters are directed along the floor of the nose and not as is supposed by the makers up to the frontal sinuses a considerable enrichment of the inspired air can occur without any carbon dioxide retention.

Whilst this has outlined the general management of cardiac cases there are particular points in some other lesions. In dealing with Fallot's tetralogy some special points arise. The premedication and induction is similar to that used for a patent ductus but the absorption of nitrous oxide is not so good and not more than 50 % can be given so that a supplement of thiopentone is more likely to be necessary. In judging the condition of the patient pulse and blood pressure must be the main guide but in spite of the cyanosis colour can still be a useful indication. In a case going well there is a distinct pinkness underlying the cyanosis in the nailbeds. In spite of the high haemoglobin these cases need transfusion to replace any blood lost for if allowed to react to shock by haemoconcentration thrombosis may occur. If Blalock's procedure is contemplated it is unwise to place the blood pressure cuff on the arm whose subclavian artery is to be anastomosed to the pulmonary artery. For when the artery is cut the blood pressure readings will be lost at a time when they are most required in the assessment of the patients condition. Before the surgeon is finally committed to the operation it is wise to test that the patient can survive with a clamp on the pulmonary atery that is going to be anastomosed. A clamp is placed temporalily and the pulse and general reaction noted. Another point is that even though a clamp is on the pulmo-

nary artery on the open side the lung should still be inflated for in these cases the bronchial circulation can be usefully ventilated.

Patients with constrictive pericarditis sometimes tolerate thiopentone badly. It is suggested that cardiac tamponade may be set up by the loss of pentone of the heart muscle in a rigid pericardium. In these cases in order to avoid this danger we use an induction with nitrous oxide oxygen and ether after premedication with Omnopon and scopolamine and topical analgesia of the larynx. As soon as the stage of surgical anaesthesia is reached intravenous tubarine is given. The respiration is assisted until the relaxant has worked, when intubation under direct vision is easy. In nearly every case we find that we can abandon the ether as soon as the patient is intubated, anaesthesia being maintained on nitrous oxide oxygen and curare supplemented, if necessary, by pethidine, thus permitting the use of diathermy in the operation. These patients require special care with transfusion for they have a very raised venous pressure and although a canulla must be inserted to deal with emergencies minimal fluid must be given until the heart is decompressed. Postoperatively they benefit from a slow transfusion of blood.

Hypotension and Hypothermia

I want to discuss two techniques developed for other branches of surgery and found to have special uses in cardiac surgery. The first successful use of hypotension was in cerebral surgery and was obtained by arteriotomy. The rapid withdrawal of blood from the circulation can lower the blood pressure. If the blood is stored, it can be retransfused when the difficult part of the operation has been completed and so aid the recovery. This technique probably only justifiable in dealing with vascular cerebral tumours had a short period of use but it has the inherent disadvantage that it invokes the body's responses to stress. The next development was to make use of the hypotension associated with high spinal analgesia. Much good work has been done by this technique and in some centres it is still the favoured method when a dry field is essential as in fenestration of the lateral semicircular canal or is helpful as in pharyngectomies. Spinal analgesia is in great disfavour with us because of neurological sequelae and so such a technique has great difficulty in gaining wide acceptance. The more elegant technique of epidural analgesia is free from this risk and has its advocates as a routine method of hypotension. But the method which I want briefly to describe and which I have used in cardiac surgery is ganglion blockade.

I think it right first to discuss its use in general surgery as its use in cardiac surgery is modified by particular conditions.

First as to choice of agent which lies between the short acting "Arfonad" and the long acting methonium compounds. Continuously administered intravenous "Arfonad" gives a control of blood pressure which can be varied from moment to moment by changing the rate of drip. But I find in cardiac surgery that the continuous block produced by the methoniums is more helpful. I have found little difference between the various methonium drugs but I have tended to use hexamethonium iodide, remaining faithful to one drug for simplicity of dosage. The hypotensive agent is given intravenously after a normal anaesthetic induction. The dose is calculated on an age basis. 100 - Age in miligrams. In the case of children the dose so calculated, is further modified to allow for the weight of the patient, by multiplining by the weight in kilos, divided by 60. This dose will produce a fall in blood pressure usually to a level of 80-90 mmHg. systolic. The mechanism being a reduction in the peripheral resistance whilst the cardiac output remains unchanged. The final control of the level of the blood pressure is by posturing the patient. Lowering the foot of the operating table some 10^{o} - 15^{o} will allow blood to pool in the dependent limbs. The slight diminution in the amount of blood returning to the heart establishes a new level of maintenance of the pressure. Supplementary doses of hexamethonium can be given in a dosage about one quarter that originally given. Procaine amide, 100 mgm., can be given in resistant cases to assist the vasodilatation.

The good management of these cases depends on the maintenance of proper oxygenation of the circulating blood. There have been a number of disasters due to cerebral anoxia, but I have had to use hypotension for the extensive resection of malignant ulcers of the head and neck after the failure of previously used X-ray therapy. These have often been patients who except for their dire condition were quite unsuited by age and vascular state for a period of hypotension. Yet although some had periods of unrecordably low blood pressure by maintaining a high level of oxygenation they have made good recoveries.

The first guide to their condition is of course the blood pressure. If it remains steady at about 60 mm. systolic there is little cause for anxiety. Although even in this case there is no margin for suboxygenation, whether due to obstruction of the airway, poor design of apparatus leading to increased dead space, or errors such as the failure of an oxygen cylinder. Factors which might be trivial under normal circulatory conditions can under hypotension lead to disaster. If the blood pressure becomes unrecordable either for technical reasons or because it really is low, I have two guides to the continued wellbeing of the patient. 1. The maintenance of the normal respiratory pattern. The respiratory centre requires a supply of well oxygenated blood for the maintenance of its proper

function. If deprived of this it is unable to respond in its normal way to the stimulus of carbon dioxide. At first the respiration will become periodic. Waxing and waning in depth. A period of deep respiration passing into one of apnoea, and then deepening again. This is followed by gasping respiration and finally by respiratory failure.

I think this so useful a sign that so far as possible maintain hypotensive patients on spontaneous respiration so that I have this warning of failing cerebral circulation. If it occurs the first easy remedial measure is to make the table level thus restoring to the circulation the volume of blood that has been pooled in the legs. This is most effective for raising the legs will frequently raise the systolic pressure by 20 mmHg. If this is not sufficient then a sympathetico-mimetic drug such as methyl amphetamine 5-10 mgm. intravenously will raise the pressure for it acts distally to the ganglion block. I must reiterate the importance of avoiding anoxia and should the disorder of rhythm produce under ventilation then the respiration must be assisted. This assistance should be accompanied by some step directed to raising the blood pressure for the institution of controlled respiration tends to lower the pressure. 2. The other guide of which I make use is the warmth of the hand and the colour and circulation in the capillaries of the nail bed. In most patients readings of the pressure can be obtained throughout. A level chart is the best evidence of the continued wellbeing of the patient. After about 45 minute the pressure begins to rise. Further doses of hypotensive drugs are given if a longer period is required or the pressure is allowed slowly to rise so that by the time that the dressings are applied the pressure is about 100 mmHg. systolic and the patient can safely be returned to bed.

Amongst the operations on the heart and great vessels that which benefit most from hypotensive techniques is the operation for coarctation of the aorta. Three particular hazards present themselves in this operation:

(1) Blood loss during the thoracotomy. The circulation below the stricture is maintained by collaterals running from the subclavians and upper intercostals to anastomose with the lower intercostals and phrenic vessels. These dilated vessels must cross the line of incision.

(2) The hypertrophied upper intercostals subjected to the hypertension above the stricture of the aorta soon become aneurysmal and degenerate. These may easily tear during the dissection of the aorta which must precede the application of clamps.

(3) When the clamps are applied the upper one may so occlude the left subclavian artery as to force the cardiac output through

a still smaller field, with the result that the B. P. rises to unprecedented levels.

Controlled hypotension would help in all three of these problems lessening the blood loss in the incision, reducing the strain on the aneurysmal intercostal vessels, and by controlling the rise in pressure after the application of the clamps it would reduce the risk of cerebral haemorrhage. Unfortunately its application is not easy. The peripheral arterial vasodilatation produced by ganglion blockade cannot increase the flow through the aortic stricture and can have little effect on the flow through the collaterals which are already dilated. Its effect is limited to the head, neck, and upper limbs, which are supplied by branches from the aorta above the stricture. The fall in B. P. that can be expected is small. I quote from an example. A girl of 15 years whose resting blood pressure was 160 mmHg. and whose highest known systolic pressure was 180 mmHg. under quiet general anaesthesia had a blood pressure 150/100. She was given 80 mgm. hexamethonium iodide. The pressure fell to 120/90, which I would regard as an excellent response. This fall is adequate to reduce the risk of tearing aneurysmal vessels and for that reason I strongly recommend its use. It is not going to reduce the blood loss whilst the chest is opened. When a ganglion blockade has been established the body has lost its ability to cope with a change in the circulating blood volume. Any blood lost must be quickly and adequately replaced or circulatory ~~failure~~ will ~~ensue~~. ~~This must~~ be ~~remembered~~ whilst the chest is being opened for the loss may be considerable.

The next point that arises is after the application of the aortic clamps when the pressure may begin to rise. There is a natural temptation to use supplementary doses of hexamethonium in an attempt to stop this rise. Usually this must be resisted. Although the vascular bed over the whole body has responded to the methonium, only those vessels supplied by arteries proximal to the stricture can contribute to the fall in pressure and the response will be even more restricted if the clamp impairs the flow into one subclavian artery. It is not, however, because it is illogical to expect good results that I warn against pushing the hypotensive drug. In a very short while ~~the~~ stricture will be excised, the aorta sutured and the clamps removed. If this flow of blood into the descending aorta is going into a field recently and completely subjected to ganglion blockade, there will be a disastrous fall in blood pressure. On the first occasion that I used hypotension for a coarctation I had not reasoned this out. The level of the B. P. remained quite satisfactory until the clamps were in place. It then started to rise in spite of two supplementary doses of hexamethonium. When the clamps were removed it fell catastrophically. In spite of intra-

venous vasopressors it was some hours before the blood pressure was maintained at a satisfactory level.

This period of hypertension can be very worrying. The pressure is rising steadily to levels above the highest recorded preoperatively. Cerebral haemorrhage seems imminent. My colleague Sellick has made use of a simple and ingenious method of lessening the pressure. He has had inserted into the aorta above and below the stricture two wide bore needles connected with tubing previously lined with silicone to prevent clotting. This effectively checks the rising pressure. I personally have not had to fall back on this safety valve, but it is a simple device which it is worth having in reserve. I do not advise its routine use because of the risk of haematoma formation at the site of punctura.

The general anaesthetic management of these cases is as for any other cardiac patient. A peaceful and unhurried induction of anaesthesia in a well premedicated patient is followed by controlled respiration using nitrous oxide, oxygen and curare. The need for controlled respiration to maintain ventilation with the open chest far outweighs the value of the sign of adequate cerebral circulation given by spontaneous respiration, particularly as the pressure does not fall to levels that give rise to any doubts as to its sufficiency.

The other special aid to which I want to draw attention is hypothermia. There are in principle three methods of producing hypothermia. First pharmacological by the action of drugs we deprive the body of its temperature control mechanism. If heat loss exceeds that produced by metabolism, body temperature will fall. Secondly by surface cooling with cold baths, ice packs, refrigerated blankets and electric fans, heat loss can be greatly accelerated. Thirdly direct cooling of the blood by exteriorising part of the venous circulation cooling the blood and retransfusing it. Finally combinations of these methods can be used.

In considering which method or combination of methods will best suit the demands of cardiac surgery it is as well to be clear in our minds as to what benefit we hope to confer on the patient by hypothermia. By far the most important is a prolongation of the period of survival of the cerebral cortex when deprived of an adequate blood supply. It is I think misleading to suppose that we are confering any protection to the heart by lowering the general metabolic demands of the body. The cardiac muscle requires oxygen for its own metabolism, and a further larger supply for the external work it must do in driving the circulation forward against the peripheral resistance. As the patient is cooied the metabolic demands of the heart are lowered as are those of the whole body, but the external work although reduced by the slower pulse

and lowered blood pressure continues. As the body cools a temperature is reached at which the heart muscle working at its lowered metabolic rate is unable to perform the external work required of it. This failure is expressed as fibrillation of the muscle. First the auricle fibrillates. The change from sinus rhythm is common occuring as the temperature falls below 31°C. Sometimes the change in rhythm follows an external stimulus, a change of position, the sudden access to the heart of colder blood, or the start of the operation, but essentially it is related to temperature and the diminished rate at which the muscle can extract oxygen from the blood and metabolise it. If the temperature is lowered still further ventricular fibrillation may supervene.

This serious complication does not usually occur until the temperature has fallen below 28°C. and again it may be associated with some external stimulus but it is clear that the cold is not protecting the heart and indeed temperatures below 28°C. impede its normal action.

Ther are two further benefits of hypothermia which might be considered. It has been fairly conclusively shown that the cooled animal is protected in some degree against shock. This was discovered fortuitously when some animals which had been subjected to trauma in an experiment were accidentally chilled owing to an electrical failure. The chilled animals shewed a much higher survival rate than those kept in what had previously been considered ideal conditions for recovery. After this clue it was not difficult to demonstrate the reduced response to traumatic stimuli. Shock may develop from shivering in mismanaged cooling or in rewarming for if rewarming of the surface opens blood vessels more quickly than the heart is able to increase its output a state of shock develops. Whilst it is cool the body is spared those efforts at maintaining vital activity which although lifesaving in the hunted animal are inappropriate in the operating theatre. Traumatic shock is not a feature of heart surgery. The uncomplicated operation produce singulariy little shock; blood loss must be immediately replaced. Shock will certainly develop from tissue anoxia if the lungs are not ventilated or the action of the heart is weakened by anasthetic technique or surgical interference. Here indeed cooling may give some further protection by reducing the demands of the tissues. The other advantage is a reduction in the intracranial pressure by an actual reduction in brain volume as well as by a reduction in the C. S. F. pressure. Whilst this is of the greatest value to the neurosurgeon in dealing with concussion and post operative reactions it may be of great value in minimising the damage should an embolus be dislodged in the operation.

Simple pharmacological cooling or hibernation will not produce the fall in temperature required to permit the occlusion of the

circulation, but many of the techniques make use of chloropromazine to facilitate the cooling by other means and to prevent a stress reaction to the cold. Some worckers prefer extracorporeal circulatory cooling or pervascular cooling. This was first tried by an arteriovenous shunt. Blood being led from a cannula in the femoral artery through silicone lined polythene tubing to the jugular vein. In its passage the blood is cooled by immersing a coil of the tube in a cold solution, or by covering the tube with a wider tube through which the cooling solution is pumped in the opposite direction. It was soon felt that apart from the risk of complications arising from the cannulisation of the femoral artery the arteriovenous shunt imposed an unwarrantable burden on the heart. The technique has been abandoned in favour of veno-venous cooling using a pump in the circuit to drive the blood through the cooling coil. Brock, who advocates this method, claims that by its use he is enabled to make a preliminary examination of the heart at normal body temperatures. If cooling is wanted polythene catheters are introduced through the right auricle into the superior and inferior venae cavae for the abstraction and return of blood. Cooling takes place steadily and its rate is controlled by the speed of pumping. A cooling rate of 1°C. in five minutes is aimed at so that suitable conditions can be expected in about one hour. Rewarming can be commenced as soon as the need for occlusion of the circulation is passed by substituting warm for cold water around the coil. In this way rewarming can be confortably started before the cannulae are withdrawn to close the chest.

We have preferred to use surface cooling and our experience is divided between the use of refrigerated blankets and immersion in a cold bath (fig. 6 and 7). The refrigerated blankets contain coils through which a solution of ethylene glycol is pumped by the machine. The machine contains a refrigerating unit which cools the circulating fluid. It can be switched to heat the coils for rewarming. This machine cools the patient with a minimum of disturbance and without the mess which is apt to be associated with immersion in a bath. It is slow; it takes two to three hours to achieve the necessary temperatures. We are finding it more suitable to the neurosurgical interventions requiring cooling where the temperatures need not be so low but need to be maintained over a longer period than for cardiac surgery so I shall describe the details of cooling using the cold bath.

Two special points arise in considering premedication. Drugs are broken down and excreted much more slowly at low temperatures. Liver perfusion experiments have shown that the half life of morphia is prolonged ten times at 30°C. The action of depressant drugs is enhanced when the body temperature is reduced. I think that this demands early premedication and careful dosage. The other

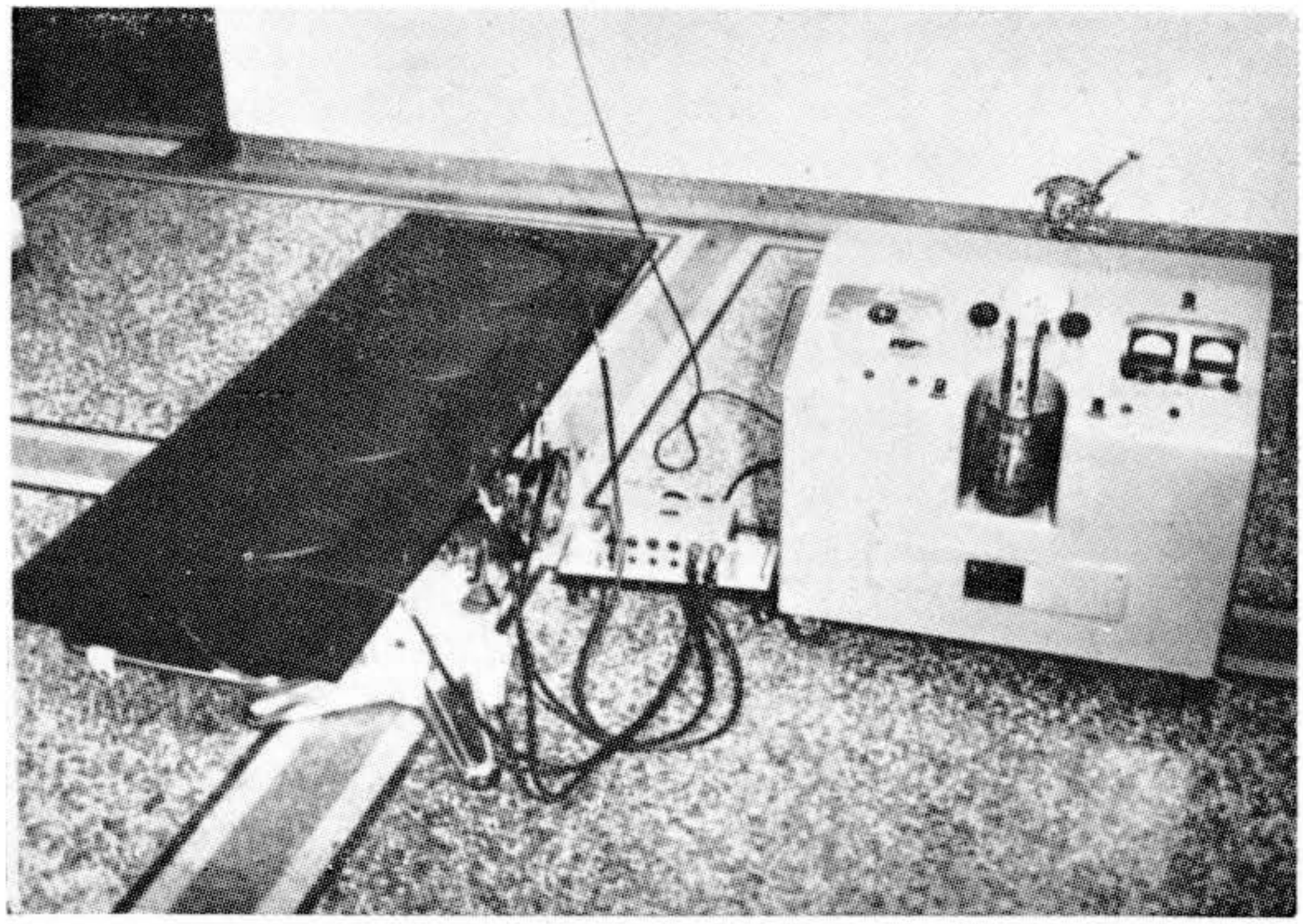

Fig. 6

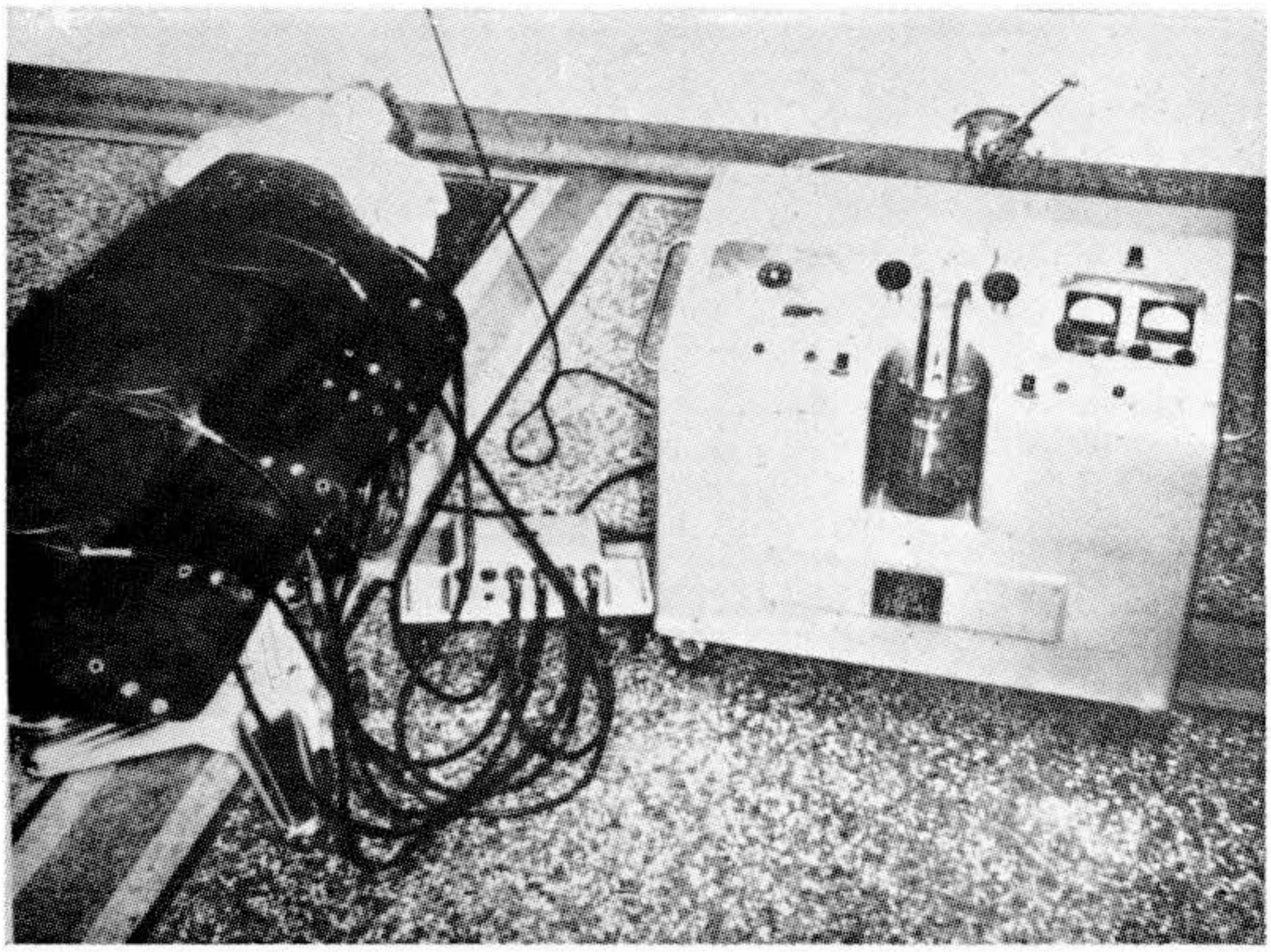

Fig. 7

consideration is whether it is advisable to use chlorpromazine to assist the cooling. If the cardiac lesion includes a stenosis restricting the cardiac output the vasodilatation of the chlorpromazine may produce an excessive fall in blood pressure before the cooling can offer any protection. If there is a right to left shunt as in Fallot's tetralogy a lowered peripheral resistance increases the shunt away from the lungs and so contribute to underventilation. Bearing these considerations in mind the following premedication is suggested.

The night before operation: phenergan 25-50 mgm.; pentobarb. 0.2 gm.

The day of operation: phenergan 25 mgm.; pethidine 50 mgm.; scopolamine 0.4 mgm., intramuscularly, 2 hrs. before the anaesthetic.

If chlorpromazine is to be used it can be added to this intramuscular injection in a dose 25-50 mgm.

The induction of anaesthesia is with thiopentone a little less than twice the sleep dose. 300-400 mgm. proceeding through nitrous oxide oxygen and ether to light third stage anaesthesia. When quiet regular respiration is established tubarine 15 mgm. is given. After the jaw has relaxed the patient is intubated with a large cuffed endotracheal tube. Control of respiration is established with carbon dioxide absorption.

The patient is now ready for the operating theatre where an intravenous transfusion of 4 % Dextrose and 1/5 normal saline is started. Two separate cannulae are advisable for if one is displaced in the movement of the patient it would not be easy to start a fresh transfusion after cooling had commenced. If there is any reaction to the cooling the liver may be deprived of glycogen and so become more susceptible to damage. So in surface cooling the dextrose drip seems a wise precaution. Electrocardiographic leads are fitted to assist in monitoring the heart both in cooling and during the operation. Two thermometers are placed in position one in the oesophagus about 30 cms. from the teeth to lie behind the heart and the other in the rectum. In our early cases we had some difficulty in obtaining reliable thermometers accurate for the scale we required and I can assure you that it engenders considerable anxiety if you find that you cannot trust the thermometers. This has now been solved by using a thermistor in a simple balanced circuit. The thermometer behind the left auricle gives a reading of the temperature of the circulating blood, whilst that in the rectum approximates to that in the tissues. In cooling the oesophageal temperature will fall first and the rectal will follow it down. Some assis-

tance may be had from studying this lag in estimating the amount of after cooling to be expected.

The patient is now ready for immersion in the bath, but first a supplementary dose of tubarine is given unless the lung inflates very easily. The patient is lowered into the cold bath. The head is supported clear of the water by a webbing strap under the neck. The arms and wrists are strapped to the side clear of the water but the feet are immersed. The temperature of the bath at the start is about 17°C. When the condition of the patient after the move has been assessed and if it is satisfactory cooling begins in

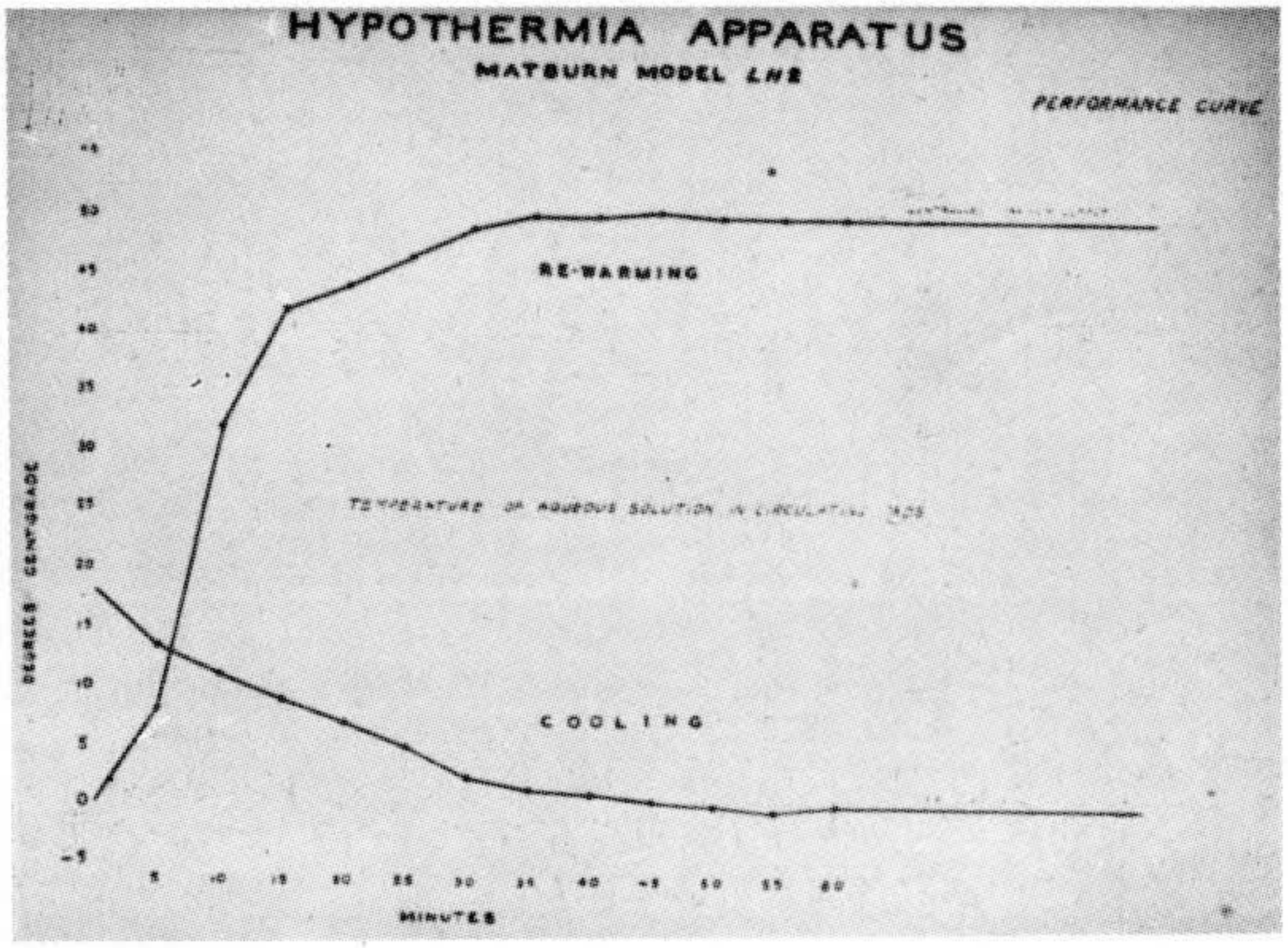

Fig. 8

earnest. Broken ice is added to the bath which is kept constantly stirred to prevent the stagnation of warmer water near to the patient. More even cooling is promoted by massaging the muscles and subcutaneous tissues from time to time. This is rather painful to the hands once the temperature of the bath has fallen to about 6°C. and a relay of volounteers is required to keep it up. Cooling of the limbs occurs more rapidly than the body and to prevent damage by cold, the hands are kept out of the water and the feet are lifted clear after about 15 mins. cooling. The rate of cooling will depend on the build of the patient and the amount of subcutaneous fat but after ten minutes the oesophageal temperature will begin to fall.

As the temperature drops the pulse slows. The response of the blood pressure is more variable but a marked hipotension is usual by the time that 30°C. has been reached. Owing to the low pressure and constriction of the artery it is usually impossible to continue to record it with a stethoscope over the brachial artery. When the oesophageal temperature drops to about 31°C. the patient can be removed from the bath because after cooling can be expected to continue for another half an hour. The patient is lifted from the bath on to a stretcher covered with absorbent towelling and is carefully dried. He is then transfered to the table and the preparations for rewarming are commenced, for generally in these relatively short operations the rewarming starts as soon as the operation begins. The patient is on a mattress through which warm water circulates at a temperature of 40°C. Gentle warmth is applied to the limbs by draping them with electric blankets but these are disturbing to the E. C. G. and they may have to be switched off.

As the cooling has proceeded the ether concentration has been steadily reduced and by the time the patient is on the table maintenance is by equal volumes of nitrous oxide and oxygen. More curare is given before the incision is made. At about this time the heart often changes from sinus rhythm to auricular fibrillation and a constant watch is kept lest ventricular fibrillation should occur. We think that a contributory use of ventricular fibrillation is too rapid cooling so that the heart is childed before its load has been reduced. But may be precipitated by movement or surgical stimulation. The management of this accident has already been described and it in no way differs at the lower temperature.

When the chest is opened and the temperature has reached its lowest level of about 29°C. in both oesophageal and rectal thermometers the nitrous oxide is stopped, the ventilation continuing with pure oxygen. After the heart has been prepared for the occlusion of the circulation by passing nylon tapes around the venae cavae and the aorta has been prepared for the application of a clamp a sterile syringe is prepared for the surgeon to give intracardiac neostigmin. The dose is of the order of 0.25 mgm. but is varied according to the speed of the heart. More being given if the pulse is rapid. This is directed into the coronary circulation by clamping across the aorta before injecting. The heart is slowed by the neostigmin and so suffers less from anoxia in the period of circulatory arrest. During this period it is obviously pointles to continue the artificial respiration so the field can be completely still for the surgeon. The period of arrest that is safe at this temperature is about 12 minutes so that 10 minutes can be regarde as giving a good margin. Although we have not had to do so, if more time were required it would be possible to close the opening in the

auricle with a clamp, release the tourniquets and the aortic clamp and so restore the circulation. After 10-15 minutes of re-oxygenation the operation could recommence.

When the defect has been sutured and the circulation restored artificial respiration with oxygen is restarted. The restoration of circulation washes the neostigmin out of the heart and the cardiac rate picks up. As the chest is closed nitrous oxide is added to the oxygen to obviate a premature return to conciousness. The wound is closed and sealed and more zealous efforts at rewarming can commence. The patient is transfered to the bath at 40°C. and allowed to rewarm. Again the oesophageal temperature responds first and when it reaches about 35-36°C. the patient can return to bed. The first steps in warming should be slow because to produce a surface vasodilatation by warmth before the heart was ready to increase its output would produce shock. Post operatively the patient needs special care to ensure that there is not a hyperthermic reaction; otherwise the management is as for other cardiac cases.

Sumário

O A., que é dos anestesistas que mais experiência tem no assunto, relata com simplicidade a técnica que usa quase rotineiramente em cirurgia cardíaca.

Pequenas diferenças de tática em relação às lesões valvulares e às lesões congênitas, mas procurando sempre tornar simplificada a interferência do anestesista.

No capítulo da hipotensão aplicada a êste campo da cirurgia, fez ressaltar o extremo cuidado com que age nos raros casos em que a indica. Descreve sucintamente todos os processos, desde sangria e reinfusão, passando pela Raqui, Epidural — hipotensores de ação frusta, até os methoniuns, que usa mais freqüentemente, porém com extremo cuidado.

No capítulo referente à hipotomia, cita os diferentes processos em uso, porém dá a entender que não é adepto do uso da clorpromazina em cirurgia cardíaca. Prefere pura e simplesmente o "Surface cooling" que, pôsto que pareça um pouco mais trabalhosa, prescinde de colchões próprios, compressores e máquinas, nem sempre fáceis de obter. Descreve com minúcias a técnica que segue, bem como a conduta posterior, para aquecimento progressivo, e cuidados pós-operatórios.

A associação antibiótica de amplo espectro antibacteriano

DICRISTICINA

PENICILINA PROCAINA REFORÇADA MAIS
ESTREPTOMICINA E DIIDRO-ESTREPTOMICINA

Eficaz - **A associação de penicilina e estreptomicina tem efeito aditivo ou sinérgico contra a maioria das bactérias comuns — tanto Gram-negativas como Gram-positivas — como ficou demonstrado com experimentações "in vitro" e em animais de laboratório. Dicristicina é notàvelmente eficaz no tratamento das infecções comuns mistas ou resistentes.**

Segura - **Dicristicina contém partes iguais de estreptomicina e diidro-estreptomicina. O perigo de ototoxicidade é grandemente reduzido, sem diminuição do efeito terapêutico. Reações de hipersensibilidade são raras.**

Flexivel - **Dicristicina é apresentada em 4 concentrações, com diferentes proporções dos componentes, a fim de satisfazer às necessidades terapêuticas dos vários tipos de infecção.**

	Penicilina procaina reforçada	*Estreptomicina e diidro-estreptomicina*
DICRISTICINA	**400.000 u.**	**0,5 g**
DICRISTICINA REFORÇADA	**400.000 u.**	**1 g**
DICRISTICINA "800"	**800.000 u.**	**0,5 g**
DICRISTICINA REFOR. "800"	**800.000 u.**	**1 g**

SQUIBB PIONEIROS NA PESQUISA E MANUFATURA DE PENICILINA E ESTREPTOMICINA

"DICRISTICINA" É UM NOME REGISTRADO

LAB. CLINICO SILVA ARAUJO S.A. RIO

NOVIDADE NA TERAPÊUTICA DA DOR

DOLCSONA

Sinergia medicamentosa de duas potentes substâncias de ação analgésica e antiespasmódica:
metadona e *papaverina*

★ Alivia a dor sem provocar narcotismo

★ Poder analgésico 3 vêzes maior que o da morfina e sem os seus inconvenientes

★ Ação terapêutica constante e uniforme quer pela profundidade quer pela duração da analgesia

★ Não afeta o coração nem a pressão arterial

★ Menor depressão respiratória que os opiáceos

★ É particularmente ativa nas dores provocadas, mantidas ou exaltadas por espasmos da musculatura lisa.

Ampolas - de 1cm3, em caixas com 5, 25 e 100
Comprimidos - tubos com 10

MEDICAMENTO ENTORPECENTE

VENDA SOB PRESCRIÇÃO MÉDICA

DOLCSONA

KONDROCURARE

SOLUÇÃO a 0,25 % do CLORHIDRATO do DIMETILETER
da METIL-BEBEERINA

PROPRIEDADES:

1.º — Introduzido por via parenteral, produz diminuição do tonus muscular e paralisia flácida que atinge sucessivamente os vários grupos musculares. Os últimos músculos atingidos são sempre os da respiração e o último, o diafragma.

2.º — Introduzido por via venosa, os sintomas surgem quase imediatamente; quando introduzido no músculo, de 10 a 20 minutos após.

3.º — A duração dos sintomas depende da dose e da via de introdução. E' relativamente curta (15 a 30 minutos), quando injetado na veia; 1 ½ a 3 horas, quando introduzido por via intramuscular.

4.º — A eliminação do **KONDROCURARE** se processa pela urina. E' completa em menos de 24 horas, não se observando fenômenos de acumulação.

5.º — Não produz baixa acentuada da pressão arterial e brônquio-espasmos, complicações observadas com alguns curares.

6.º — Bloqueia primeiro os impulsos nervosos de maior freqüência de emissão, donde a sua ação eletiva sôbre o tonus e sôbre o hipertonus, quando êste existe.

7.º — Sua ação é periférica e se explica pela quebra do isocronismo entre nervo e músculo (Lapicque), conseqüente do aumento da cronaxia dêste; pelo aumento do limiar de excitabilidade do músculo à ação da acetilcolina, se aceitarmos a teoria da transmissão química dos impulsos nervosos.

8.º — A prostigmina e a fisostigmina são antagônicos do curare e fazem desaparecer os sintomas da curarização.

INDICAÇÕES:

Coadjuvante da anestesia durante as operações abdominais. No decorrer da convulsoterapia, para evitar as complicações traumáticas. Nas síndromes espásticas e atetósicas. Medicação auxiliar do tétano.

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