ANAESTHETIC PROBLEMS IN RESECTION OF ABDOMINAL AORTIC ANEURYSMS*

A. B. BULL, M.B., CH.B. (CAPE TOWN), F.F.A.R.C.S. (ENG.), F.F.A.R.C.S. (I), *Department of Anaesthetics, Groote Schuur Hospital and University of Cape Town*

During recent years surgery of the aorta for the treatment of aorto-iliac occlusive disease and for the resection of aneurysms has become common surgical practice.

Some of the anaesthetic problems which have arisen during the development of this type of surgery in our unit at the University of Cape Town, and which is based on the management of 83 patients who underwent infrarenal resection and grafting of the abdominal aorta during the past 8 years, are presented.

These patients presented as follows:

Age incidence: 40 - 49 11 patients
50 - 59 28 patients
60 - 69 37 patients
70 - 79 4 patients
80 - 89 3 patients

Sex incidence: The ratio of males to females was 6:1.

Race incidence: 86% of patients were European, 12% Coloured and only 2% Bantu.

The cardiac state: 60% had evidence of either existing or previous congestive cardiac failure, ischaemic heart disease or hypertension of over 160/100 mm.Hg with cardiomegaly. There were 18 patients with frank rupture of the aneurysm with severe circulatory collapse. The commonest pulmonary pathology was emphysema with chronic bronchitis, which is what is expected at the ages given.

This brief analysis shows that in this type of anaesthesia we are usually presented with an elderly patient with cardiovascular disease and a greater or lesser degree of pulmonary emphysema and infection.

The type of surgery to which these patients are subjected is inevitably accompanied by major interference with the circulatory dynamics.

The surgical approach requires profound muscular relaxation for prolonged periods of time.

Massive haemorrhage, requiring massive rapid blood transfusion, is a likely possibility, and in fact, this procedure is inevitable in all cases of a ruptured vessel.

The problems we encountered in this series of cases concern mainly these three circumstances and the way in which they affect anaesthetic management.

THE USE OF MUSCLE RELAXANTS

Firstly, it must be remembered that the most important single factor in determining the duration of neuromuscular block, namely the blood supply to the muscles,[1] is interfered with to a greater or lesser extent. This results from: (a) the period of relative ischaemia produced in a large part of the body during cross clamping of the aorta; (b) the vasoconstrictor effect of temperature drop during prolonged surgery and the transfusion of large quantities of blood; and (c) the probable existence in these patients of arteriosclerotic vascular disease in the smaller vessels impairing blood supply to the periphery.

Secondly, the effect of body cooling *per se* must be borne in mind with regard to the choice of a relaxant. The magnitude and duration of block with the depolarizing drugs is markedly prolonged with hypothermia. The intensity of block produced by d-tubocurarine is reduced by cooling, its duration remaining unaffected. Some degree of hypothermia may arise inadvertently during this type of surgery as a result of the time factor involved and the transfusion of large quantities of blood. Duration of operation in our series varied from 2 to 6 hours, the average time being 4 hours.

Thirdly, alterations in the acid base balance, owing to either metabolic causes, which may result from impaired tissue perfusion, or to respiratory causes, may play their part in modifying the effect of relaxants.

Fourthly, oliguria and acute renal failure is by no means an uncommon postoperative sequel in this type of surgery. An incidence of as high as 30% has been reported,[2] and in our series there were 15 cases. In this connection, certain relevant features appeared in our series. Three patients who all had gallamine as the relaxant, presented a picture of recurarization two hours after the end of the anaesthetic and a reversal of curarization after injection of neostigmine. Repeated doses of neostigmine over a period of 36 hours in one case, and 12 hours in another, were required to maintain normal ventilation. In the third case, tracheostomy and IPPR were resorted to. All three of these patients had anuria, and the re-establishment of urine secretion coincided with complete de-curarization and terminated the need for neostigmine. Of particular interest here is the fact that the patient who remained curarized for 36 hours was readmitted to hospital three months later with adhesive intestinal obstruction. On this occasion she was anaesthetized by the same anaesthetist, using precisely the same anaesthetic technique and drugs, and on this occasion de-curarization was completely uneventful.

That such a picture of persistent curarization after the use of gallamine in the patient with anuria or oliguria is an inevitable consequence, is, of course, not true, because in this series at least 6 patients anaesthetized by this technique, and who had oliguria during the postoperative period, did not show this clinical picture. Nevertheless, with the knowledge that gallamine is excreted almost entirely by the kidneys, we feel that it is unwise to choose this relaxant for these cases and our routine now is to use d-tubocurarine, because its fate in the body is not determined by renal function only.

BLOOD REPLACEMENT

The amount of blood loss requiring replacement in these cases is very variable. Blood lost pre-operatively in the case of leaking aneurysms can be extremely difficult to assess, and one is not infrequently presented with a somewhat hypovolaemic patient on this account. In the case of frank rupture of the aneurysm pre-operatively, gross peripheral vascular collapse will of course be evident, requiring

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immediate and energetic blood replacement in order to save the patient's life. During surgical treatment one has to contend, firstly, with the blood loss occasioned by any major operative exposure, and this, in our experience, amounts to approximately one pint of blood per hour in the average case, but this figure refers to the unheparinized patient. It is our practice to heparinize the patient fully using a dose of heparin of 90 mg. per square metre of body surface, given a few minutes before cross clamping of the aorta. Following heparinization, blood loss from what one might term 'normal oozing' is then greatly increased and the figure for blood loss per unit time increases from approximately 1 pint per hour to nearly double this figure. To this amount of blood loss by continuous ooze, one must add that lost by bleeding from major vessels, such as lumbar arteries; or from veins damaged in difficult dissection of the aneurysm; also the loss resulting from release of the aortic clamp or the clamps on the iliac vessels to wash out displaced atheroma or clot from the aneurysm.

Some idea of the amount of blood replacement required in 50 of our patients is given in Fig. 1. The range of loss is from 2 pints in simple, uncomplicated, straightforward resection and grafting of an abdominal aneurysm, to 20 pints or more in the difficult case. By far the commonest

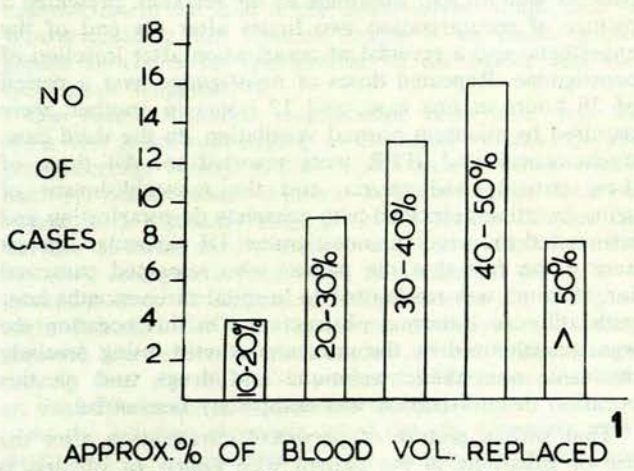

Fig. 1. The amount of blood required by 50 patients.

quantity of replacement is in the region of 6-7 pints of blood, a figure which represents up to 50% of the patient's normal blood volume. A very large percentage of cases then receive the transfusion of a relatively large percentage of their blood volume over a relatively short period of time. In a number of cases, replacement amounted to as much as the full blood volume of the patient, and in two cases, double the patient's normal blood volume was replaced.

Factors to Consider

Several factors must be considered in transfusions of this magnitude, relating to the composition of the donor blood:

1. Compatability.
2. The effect of transfused citrate and potassium ions on ionized serum calcium.
3. The acid base balance effect caused by the transfusion of blood of low pH.
4. The gas dissociation curves of donor blood.
5. The so-called post-transfusion haemorrhagic diathesis.

1. *Compatability*: The principal factor concerns ABO and Rh-compatability, and this must be strictly adhered to. Danger exists in assuming the 'universal donor'-quality of Group O blood and giving it in large quantities to group A or B or AB patients in an emergency, followed by subsequent transfusion of the patient's own group. The α and β-agglutinins of the transfused O blood may become sufficient under these circumstances to cause agglutination. We have had one such case.

A further factor is the presence of cold agglutinins, which may be considered of more than slight importance unless careful temperature control of the patient and of the blood given is maintained.

2. *The serum potassium* of stored blood is high and ranges from 5 mEq./l. in fresh blood to 25 or even 30 mEq./l. after 3 weeks of storage.[3] In addition, citrate is present in varying concentrations of 500-600 mg. of citrate per unit of blood. The effect of this upon potassium/calcium ion ratio has formed the basis of much conflicting opinion in the literature.[4] In our experience, however, electrocardiograph monitoring has shown changes compatible with potassium/calcium imbalance during rapid transfusion, as reflected by T-wave spiking and prolongation of the corrected QT interval. These changes can be enhanced by hypothermia, by acidosis, and in patients with poor cardiac function and impaired renal potassium elimination. Impaired liver function may further interfere with citrate metabolism, especially when the necessity for very rapid transfusion arises. The state of patients undergoing this type of surgery often fulfils, to a large extent, most of these conditions, and it is almost certain that rapid transfusions can be the final precipitating factor in the genesis of severe cardiac disability on the above basis. When the ECG-change of spiking of the T-wave appears, we have found it valuable to administer calcium gluconate in 10 ml. doses of a 10% solution. This is usually followed by an immediate reversion of the ECG changes to normal, or at least to their pre-transfusion state.

3. *Blood of low pH and its effects.* The pH of stored blood varies from about 6·5 to 7·0, depending on the length of storage. Transfusion of blood of such low pH is likely to enhance an already existent metabolic acidosis, which results from the 'tourniquet effect' of clamping of the aorta, and the presence of a greater or lesser degree of oligaemic shock. Here one must emphasize that metabolic acidosis, when it is compensated, is compensated by respiratory alkalosis. For this reason the ventilatory state of the patient is of vital importance as well as for purposes of oxygenation. If it is considered that metabolic acidosis is severe, resort may be had to the intravenous use of sodium bicarbonate, and we have used this with success in an empirical dosage of 1 mEq./kg. body weight, in the absence of actual measured serum pH, P CO_2 and bicarbonate levels.

4. *Oxygen dissociation.* Valtis and Kennedy in 1953,[5] and subsequently other workers,[6, 7] demonstrated that the oxygen-dissociation curve of stored blood undergoes an increasing shift to the left with duration of storage. This shift is aggravated by hypothermia and is a further factor in the production of acidosis and tissue anoxia. This state of affairs in the transfused cells persists for up to 24 hours or longer after transfusion into the recipient.

5. *The haemorrhagic diathesis.* Stored blood is deficient in haemostatic factors too and may give rise to uncontrollable oozing, which can be confused with persistent heparin action, and lead the misguided to the excessive use of heparin antagonists. A full, clotting-factor assay is necessary to diagnose this and to know how to counteract it.

When massive transfusion is necessary, it is preferable to use the most physiological form of transfused blood available. This, as far as we can determine, is fresh, warm, heparinized blood which will avoid, to a large extent, many of the disadvantages referred to above.

OLIGURIA AND RENAL FAILURE

Many satisfactory resections and graftings of the aorta are complicated by oliguria and acute renal failure. In our

series, acute renal failure or marked oliguria occurred in at least 15 cases.

Renal complications may arise from emboli dislodged in cross-clamping the aorta. Spasm of the renal arteries has been incriminated. Oligaemic hypotension is undoubtedly a major factor, and more recently it has been shown that the molecular structure of certain batches of the heparin antagonist, polybrene, is capable of producing tubular damage. The use of ganglion blockers to maintain renal blood flow has been advocated, but it is felt that the accompanying hypotension in these elderly patients with cardiovascular disease carries too great a danger of myocardial and cerebral vascular complications. In this series we have had one patient who developed a postoperative hemiplegia and three patients in whom myocardial infarction was diagnosed postoperatively. In all 3 of these, varying periods of hypotension, owing to rapid blood loss, had occurred.

The periarterial infiltration of the renal vessels with local anaesthetic has, however, been tried by us to combat spasm, but without convincing effect. Barry *et al.*[8] described the use of 'mannitol' as an osmotic diuretic in maintaining urine flow in this type of surgery. It is now our practice to use a modification of their technique. At the commencement of operation the patient is given a water load of some 500 - 800 ml. Between 100 and 200 ml. of a 10% mannitol solution is next given intravenously over a period of 20 - 30 minutes, the infusion ending just before cross-clamping the aorta. All patients have an indwelling catheter. Diuresis usually commences within an hour or two of the mannitol infusion. We have used this technique on our last 10 patients and so far have not encountered oliguria or anuria. It is also our practice now to use protamine sulphate rather than polybrene as a heparin antagonist.

BLOOD PRESSURE

Major fluctuations in blood pressure are the rule with this type of surgery. The patient with a frank rupture of his aneurysm, of course presents as an emergency with acute peripheral circulatory collapse. Under these circumstances we have found it wisest to employ resuscitative transfusion cautiously and to urge immediate laparotomy and control of the aorta above the aneurysm. After this, the most vigorous transfusion to replace blood volume is proceeded with. Attempts to achieve full restoration of blood pressure, in the case of rupture before the aorta is controlled, may serve only to aggravate the break in the aneurysm.

The patient with a leaking aneurysm often presents with a normal or near normal blood pressure, which may fall precipitously when induction of anaesthesia removes the vasomotor tone which is compensating for his loss of blood volume. These patients require careful judgement in the choice of anaesthetic agent used for induction, and this refers especially to the use of barbiturates.

During surgery, the pattern of blood pressure changes is a fairly constant one and is illustrated in Fig. 2, which is, in fact, the record of a patient with a leaking abdominal aortic aneurysm. The drop in blood pressure just following induction is probably due to the vasodilator effect of the anaesthetic on a patient who has lost blood into the retro-

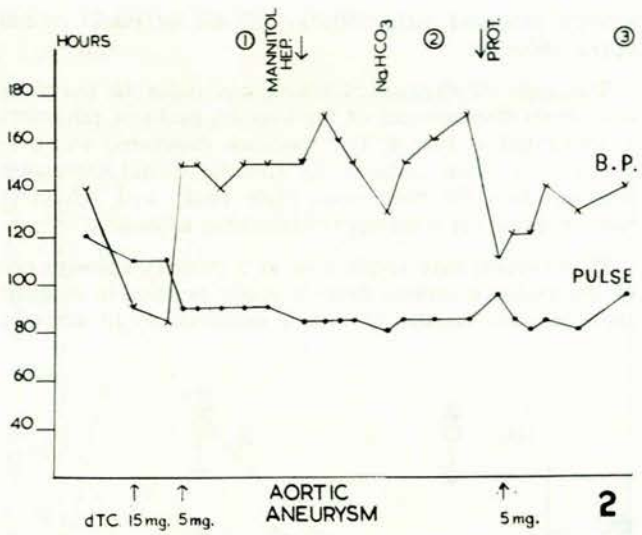

Fig. 2. Blood-pressure changes in a patient undergoing surgery for a leaking aortic aneurysm.

peritoneal tissues. It is this type of picture that one would expect in a patient with poor cardiac reserve. In this event the blood pressure is restored by rapid transfusion and the administration of 0·5 mg. of phenylephrine intravenously. The next constant feature in the blood pressure chart of these cases is the change which occurs when the aorta is clamped. The common response is a slight rise in blood pressure, which may be maintained or may drop slightly in approximately the next 20 minutes (Fig. 3).

Removal of the aortic clamp heralds the next critical period when blood is allowed to flow through the graft to the lower limbs. At this stage one must be prepared to face a precipitous fall in blood pressure, caused by the reopening of a fairly large portion of the vascular bed which has been largely deprived of circulation during the period of clamping of the aorta, which is then subject to the vasodilator effect one sees following the release of a torniquet. In order to minimize this falling blood pressure, it is our practice to place pneumatic cuffs around both thighs and to inflate them just before releasing the aortic clamp. This has the effect of diminishing the 'area run-off' from the graft and allowing some circulatory readjustment to take place, before the cuffs are deflated one by one, to open up the whole vascular bed. At this time also a certain amount of blood loss from leak through the suture lines of the graft and seepage through the graft material is likely to take place, so that vigorous blood replacement must be carried out very often simultaneously.

When the aortic clamp is applied initially, instead of a rise in blood pressure, some patients experience a sharp fall as illustrated in Fig. 3. The reason for this is, most probably, that the venous return from the lower part of the body becomes interfered with at this stage of the operation, and so diminishes the cardiac output in a patient with poor cardiac reserve. The question of vena-cava obstruction is quite an important one, and is of course not infrequently met with in many types of surgery in-

volving extensive retractions or the use of packs in the upper abdomen.

The type of obstruction which can occur to the vena cava from displacement of the liver by packs or retractors is illustrated in Fig. 4. The specimen illustrated was prepared in a fresh cadaver by running dental-impression material into the vena cava while packs and retractors were in place for wide upper abdominal exposure.

Hypotension may result, too, as a primary consequence of the patient's cardiac state. It is our practice to monitor these patients constantly with a cardioscope. In addition

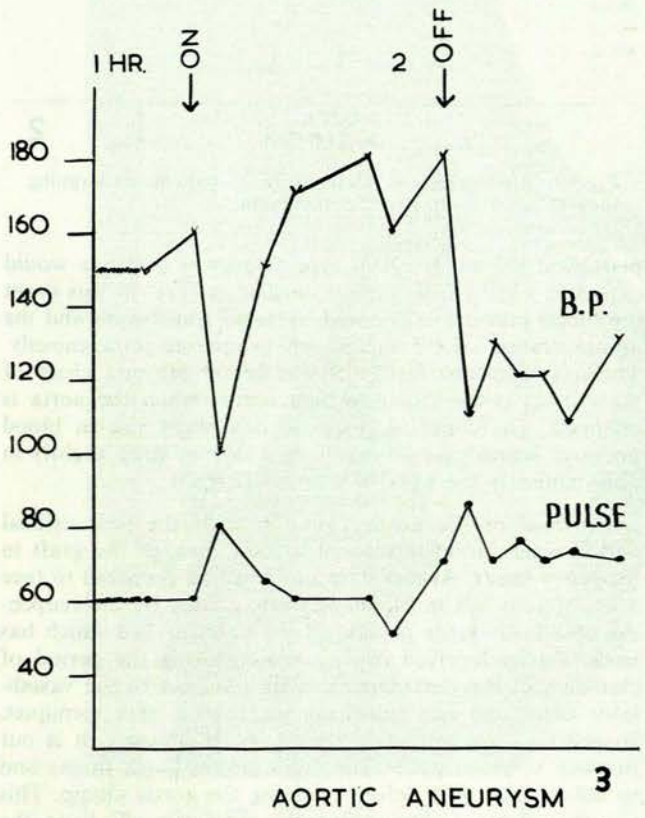

Fig. 3. The effect on the blood pressure of clamping the aorta.

to assisting in early recognition of the citrate effect during rapid transfusion which I have referred to already, it makes it possible to identify the type of arrythmia when one occurs. In this respect, the appearance of multiple ventricular extrasystoles is noteworthy, since this may lead to a ventricular tachycardia with precipitous fall in cardiac output, requiring immediate treatment. We have on occasions, too, noted varying degrees of heart block which may necessitate the use of isoprenaline.

Body temperature should, finally, also be checked, particularly if the procedure is a prolonged one. The wide abdominal exposure of viscera, together with the need for rapid transfusions of cold blood, are the two main factors which tend to affect this. In our series temperature monitoring has by no means been constant, but we have noted a drop in rectal temperature of 5°C in at least 6 cases, while in one the temperature droped as low as 28°C.

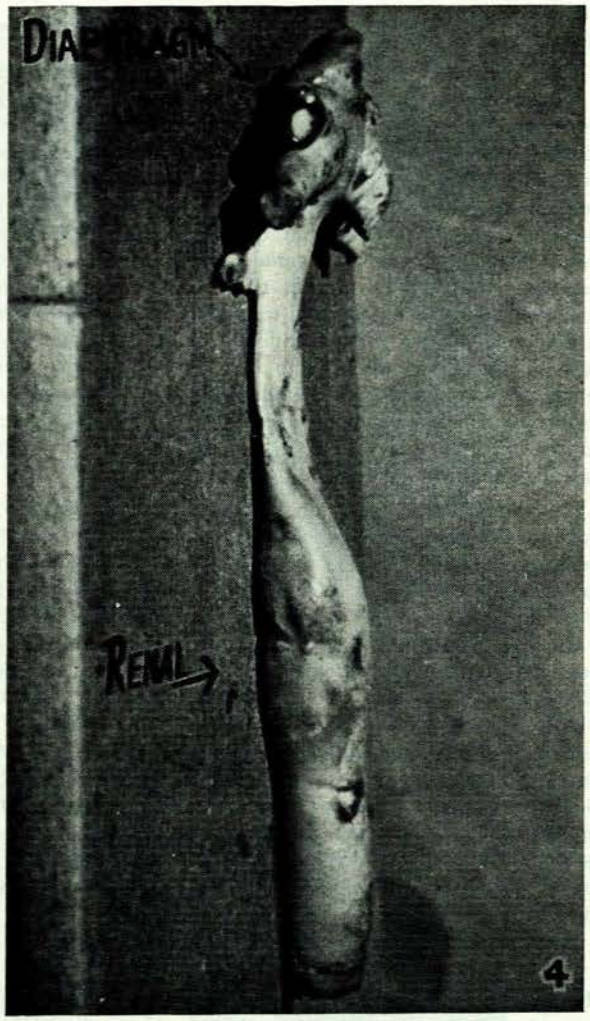

Fig. 4. The type of obstruction occurring in the vena cava owing to displacement of the liver by packs or retractors.

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