COMPLICATIONS AND MORBIDITY OF CONTROLLED HYPOTENSION

M. J. LINDOP

The surgeon may praise the anaesthetist for a bloodless field and the patient may awaken with a good operative result; but, has any particular risk been taken? Indeed, can the risk of using deliberate hypotension be assessed?

A major problem is that many papers give an account of groups of patients safely undergoing surgery facilitated by controlled hypotension. However, each paper contains one or two cases where death has occurred attributable to the hypotensive technique employed (Enderby, 1961; Hugosson and Högström, 1973; Prys-Roberts et al., 1974; Rollason and Hough, 1960; Warner, Shumrick and Caffrey, 1970; Way and Clarke, 1959).

The areas of risk of complication and morbidity may be categorized:

(A) Risks of major organ underperfusion.

(B) Surgical complications following hypotension.

(A) Risks of Major Organ Underperfusion

Blood-flow carries oxygen to an organ to meet its metabolic requirements. When the flow is reduced, the available oxygen is reduced (available oxygen = the product of blood-flow and blood oxygen content). Blood-flow (I) is related to perfusion pressure (V) by the vascular resistance (R). By analogy with Ohm’s Law, I = V/R. As the perfusion pressure decreases, the flow will decrease unless there is a parallel decrease in the vascular resistance. The resistance must decrease, or the oxygen need must be reduced, if an organ is to avoid hypoxia.

The clinical detection of inadequate blood-flow will be discussed and related to reported complications of controlled hypotension.

(1) Brain

Adequacy of cerebral blood-flow during controlled hypotension has been assessed by the following methods:

(a) level of consciousness

(b) jugular bulb venous oxygen tension

(c) electroencephalography (e.g.)

(d) higher cerebral function after surgery

(e) cases with cerebral damage after operation

(f) biochemical indices

(a) Conscious level. Finnerty, Witkin and Fazekas (1954) used conscious subjects rendered hypotensive by hexamethonium and head-up tilt. Cerebral hypoxia was diagnosed with the onset of sighing, yawning, staring, or confusion. They suggested that the minimum cerebral flow tolerated without symptoms of hypoxia is 30 ml/100 g/min (normal 50–60 ml/100 g/min), which occurred in normal conscious subjects at mean arterial pressures of 35 mm Hg. Hypoxic symptoms developed at higher mean arterial pressures in the hypertensive subjects as shown in table I. In a similar study, Moyer and Morris (1954) found that hypoxic symptoms were associated with mean arterial pressures of 55 mm Hg. These pressures suggest an absolute value (about 60 mm Hg systolic) below which cerebral perfusion may be dangerously impaired even in the fittest of conscious patients. It should be noted that at light planes of general anaesthesia there is little or no reduction in cerebral metabolic rate (Smith and Wollman, 1972). Conscious level can be monitored when controlled hypotension is used in the awake patient under regional analgesia.

<table>
<thead>
<tr>
<th>TABLE I. Tolerance of hypotension by hypertensive subjects.</th>
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<td>Mean arterial pressure at rest (mm Hg)</td>
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Data reported by Finnerty, Witkin and Fazekas (1954).

(b) Jugular bulb venous oxygen tension. In patients with normal cerebral vasculature, a jugular venous oxygen tension (PjO2) greater than 20 mm Hg (haemoglobin saturation ≥ 35%) makes cerebral hypoxia unlikely (Lennox, Gibbs and Gibbs, 1935). Eckenhoff and colleagues (1963) found no values...
below 27 mm Hg, and no cerebral damage in their study when 15 patients had arterial pressures less than 60 mm Hg and six had pressures less than 50 mm Hg. The most significant finding was that \( P_{jvO_2} \) correlated better with arterial carbon dioxide tension than with mean arterial pressure. In the presence of carotid arterial disease, satisfactory \( P_{jvO_2} \) values have been associated with cerebral damage following anaesthesia (Larson et al., 1967). In one patient after carotid artery occlusion, ipsilateral \( P_{jvO_2} \) was at all times greater than 47 mm Hg, yet after operation the patient developed contralateral limb weakness. It is possible that some of the blood may have drained from better-perfused contralateral hemispheres.

This study casts doubt whether \( P_{jvO_2} \) can indicate subtle deficiencies in regional blood-flow to critical areas of the cerebrum. The measurement cannot be relied upon to detect clinically dangerous hypoxia. However, values of \( P_{jvO_2} \) below 20 mm Hg have been measured in patients who were found later to have no obvious cerebral damage (Prys-Roberts et al., 1974).

(c) Electroencephalography. Rollason, Dundas and Milne (1964) observed no significant changes in the e.e.g. during hypotensive anaesthesia. The appearance of delta waves and burst suppression are features of the e.e.g. during general anaesthesia. It has been questioned whether these changes, which are associated also with hypoxia, are detectable before cerebral hypoxia has become intolerably severe (Cohen et al., 1967).

(d) Higher cerebral function after surgery. Sensitive tests of higher cerebral function are difficult to control in the abnormal situation of the period following surgery. Berg, Nilsson and Vinnars (1957), using the flicker fusion test, found impairment in 50\% of a group of hypotensive patients. Various techniques were used to achieve an un-declared degree of hypotension, and the control series was not strictly comparable. The observed impairment was unrelated, interestingly, to the age of the patient. Gruvstad, Kebbon and Lof (1962), in prone, head-down, spontaneously ventilating patients anaesthetized with trichloroethylene, found some impairment of mental function 6 days after operation, but concluded that it was "of no practical importance". Eckenhoff and colleagues (1964) compared two matched groups of young patients, one of which was subjected to 90 min hypotension (average 63 mm Hg systolic). A battery of six psychometric tests chosen to elicit minimal brain damage showed no significant difference between the groups. Rollason and colleagues (1971), also using six psychometric tests, randomly assigned 27 patients into two groups. They were given subarachnoid cinchocaine, and general anaesthesia. One group had a mean systolic pressure of 66 mm Hg, the other by use of vasopressors averaged 119 mm Hg. The tests revealed no difference between the groups, although in both groups performance was depressed 5 days after operation. This is a good example of the importance of studying a comparable control group. Enderby (1961), reviewing 9,107 hypotensive anaesthesetics, noted changed mental state occasionally in elderly patients, but "not significantly different from non-hypotensive patients". Rollason and Hough (1960) noted two patients with personality changes in a series of 40 elderly patients.

(c) Cases with overt cerebral damage after operation. Cerebral thrombosis may presumably be precipitated when the flow in cerebral vessels becomes critically reduced. Little (1955) records 29 (0.1\%) non-fatal, and 18 (0.60\% of the series, 18.8\% of all deaths) fatal cases of cerebral thrombosis. Enderby (1961) records one 25-year-old girl who developed hemiplegia after surgery, despite arterial pressure readings always greater than 70 mm Hg during the operation. She was nursed in the sitting position after the operation, while showing a persistent low arterial pressure, and the damage may have occurred in this less intensively monitored period. Prys-Roberts and colleagues (1974) record two cases of cerebral damage in 15 patients. One 27-year-old patient developed hemiplegia following surgery. A 56-year-old patient did not recover consciousness and was found to have extensive cerebral and cerebellar infarction. Way and Clarke (1959), reporting on 50 cases, mention one patient who died of middle cerebral artery thrombosis. Grace (1961) used controlled hypotension in 40 cases with a history of cerebro-vascular disease, and three (7.5\%) died of extension of their disease. Of another 960 cases, only two (0.2\%) died of cerebral thrombosis. Linacre's (1961) 1,000 cases suffered no cerebral complications.

(f) Biochemical indices. These are discussed by Nilsson, Norberg and Siesjö (1975).

(2) Heart

Unlike the brain, the oxygen consumption of the heart varies and is related to myocardial work, a function of cardiac output and resistance (cardiac
work = stroke volume × mean arterial pressure). Although the coronary circulation shows some autoregulation, changes in resistance are less than in the cerebral circulation. When mean arterial pressure decreases coronary flow decreases. When this is parallel with a decrease in cardiac work, cardiac ischaemia is unlikely. During controlled hypotension, electrocardiograph monitoring has been used to detect this. E.C.G. signs associated with ischaemia are depression of the S-T segment, or flattening or inversion of the T-wave. Rollason and Hough (1960) noted that mild coronary ischaemia was likely at pressures below 60 mm Hg systolic. The previously hypertensive patient was more susceptible to ischaemia. A faster rate of pressure decrease is associated with earlier onset of ischaemic e.c.g. signs (Rollason and Hough, 1969). Eckenhoff (1962) noted no e.c.g. changes in lead II, in the East Grinstead series of patients.

Myocardial infarction is a relatively uncommon complication in the reported series. Grace (1961) mentions only seven cases (0.7%) of myocardial infarction in 1,000 elderly patients, 176 of whom were suspected, before operation, of having heart disease. Little (1955) mentions 26 cases of coronary thrombosis out of 27,930 cases (0.1%), but if his categories for cardiac arrest and cardio-vascular collapse are added to these, the total becomes 98 (0.35%). In neither series was routine use made of e.c.g. monitoring, a factor which would preclude early diagnosis of reversible cardiac ischaemia.

(3) Lung
The oxygen content of arterial blood must be maximal when a possibility of reduced flow exists. Hypotension markedly decreases pulmonary efficiency, measured predominantly as an increase in alveolar deadspace (Askrog, Pender and Eckenhoff, 1964). Enderby (1961) places hypoventilation during operation as the cause of death in two of 9,107 cases. An increase in ventilatory deadspace may have hidden benefit, when oxygenation can be maintained by increasing the inspired oxygen concentration. The combination of hypocapnia and arterial hypotension is particularly deleterious to cerebral blood-flow (Harp and Wollman, 1973). The extent of hypocapnia caused by inadvertent hyperventilation will be limited by the increase in deadspace. No pulmonary complication following surgery has been attributed to the use of controlled hypotension.

(4) Kidney
Renal blood-flow decreases during anaesthesia, but early studies showed this decrease to be less during ganglion blockade (Miles et al., 1952). Urine flow ceases when arterial pressure decreases below about 70 mm Hg. In a series of patients subjected to controlled hypotension, where urine output was measured, a decrease was consistently found (Warner, Shumrick and Caffrey, 1970). Urine microscopy (Evans and Enderby, 1952; Hugosson and Högström, 1973), creatinine clearance after operation (Hugosson and Högström, 1973), and blood urea (Warner, Shumrick and Caffrey, 1970), have failed to show evidence of renal damage. The larger series of Little (1955) reported 54 cases of oliguria (0.19%) and 62 cases of anuria (0.22%). Nine deaths following surgery (9.4% of all deaths) were due to anuria. It should be noted that the technique of controlled hypotension was then in vogue for retropubic prostatectomy, in a group of patients likely to have abnormal kidneys before operation and therefore susceptible to renal complications. Other large series (Enderby, 1961; Linacre, 1961) mention no incidence of serious renal complications in a total of 10,000 cases.

(5) Liver
The hepatic circulation probably is not capable of autoregulation, and hepatic perfusion decreases pari passu with mean arterial pressure. Bromsulphthalein retention tests (Greene et al., 1954), serum bilirubin (Rollason and Hough, 1960), and serum aminotransferases (Hugosson and Högström, 1973) failed, after operation, to show a significant difference between controlled hypotensive and normotensive general anaesthesia. No morbidity or mortality has been ascribed to impaired hepatic perfusion during controlled hypotension.

(6) Eye
Retinal artery thrombosis occurred in three out of 27,930 cases reported to Little (1955). This serious complication is not mentioned in other large series.

(b) Surgical Complications of Hypotension
Cut vessels may not bleed during intraoperative hypotension. When the arterial pressure increases after operation, bleeding may begin. Reactionary haemorrhage and haematoma formation have not been a problem, as bleeding can usually be prevented simply by the application of pressure dressings, and by the avoidance of sudden return to higher
pressures following surgery. However, pressure dressings are inappropriate after thoracotomy, prostatectomy or hysterectomy. Tough (1960) recorded bleeding after operation in 10.4% of 268 thoracotomies performed with hypotension, but this was the same incidence as in a normotensive control group. Following gynaecological surgery, Linacre (1961) recorded only 3.0% haemorrhage after operation. In Little's (1955) series, the incidence was 0.9%. After prostatectomy, bleeding is not noted to be increased (Bodman, 1964; Boreham, 1964), but a controlled study is required.

Stasis is not associated with low pressures produced by vasodilating techniques. There is no evidence that the incidence of venous thrombosis is increased.

GENERAL REVIEW

The validity of conclusions drawn from large series such as Little's (1955), must be questioned. It was a postal questionnaire with a 50% response rate from anaesthetists using various techniques, variable levels of hypotension, and monitoring techniques to a variable extent and achievement. The problem was identified, but not accurately assessed. Criteria of assessment varied in this series: for instance, delayed awakening occurred in 193 British cases, but in only three U.S. cases.

Series from single investigators offer better opportunity for comparability, but numbers are necessarily limited and the range of ages and types of operation may not be comparable between different series. Enderby's 9,107 cases include many younger patients undergoing plastic surgery. Linacre's 1,000 cases (1961) were middle-aged and older women undergoing gynaecological surgery. Bodman (1964) reported 506 cases in the older age group undergoing retropubic prostatectomy. It is often not possible to distinguish the complications attributable to the hypotensive technique, and those which would have occurred under normotensive anaesthesia. No series of sufficient number to give a significant incidence of infrequent complications has reported on a comparable control group. Series using control groups (Warner, Shumrick and Caffrey, 1970; Hugosson and Högström, 1973), have been able to show no significant difference attributable to the use of the technique.

CONCLUSIONS

Arterial pressure must be carefully recorded, either by intravascular manometry (which also allows ease of access for blood-gas estimations), or by a reliable artefact-free indirect method effective at low arterial pressures (e.g. oscillotonometry), or ultrasound pulse detection (Poppers, Epstein and Donham, 1971). It is chastening to note that complications cannot be avoided with certainty by the closest monitoring of intravascular pressure, if that pressure happens to be inadequate for a particular patient (Prys-Roberts et al., 1974; Warner, Shumrick and Caffrey, 1970).

The pooled series reviewed here suggest non-fatal complications in one in 39 cases, and fatal complications in one in 167 cases. The above discussion emphasizes that, in the absence of control series, no conclusion can be drawn from such figures as to whether these complications are directly caused by the hypotensive technique. The small incidence of complications which are self-evidently as a result of arterial hypotension, need not be interpreted in terms of comparative figures. No matter how many cases are uneventful, this risk remains.

The risks are greater in the presence of: (a) hypertension before operation, (b) cerebro-vascular disease before operation, (c) hypocapnia, (d) mean arterial pressures less than 70 mm Hg, (e) rapid reductions of arterial pressure, (f) cardiac output reduced by hypovolaemia, or by cardiac depressant drugs, (g) anaemia and (h) indifferent monitoring techniques. An increased risk has not been clearly established for ischaemic heart, pulmonary, and renal disease, although many anaesthetists would consider these to be relative contra-indications. If the risks of deliberate hypotension are justified by the surgical indication, a technique must be chosen to avoid any of these increased risk factors. Vasodilating techniques seem most logical. It is to be hoped that future progress will allow cerebral function to be monitored so effectively as to detect minimal hypoxia. At present, minimum monitoring should include reliable arterial pressure recording, and continuous electrocardiography. It should be noted that the bandwidth of an electrocardiograph machine is different in one which is intended for monitoring only, from one which is intended for diagnostic purposes. Also, usually there are fewer leads. Consequently, changes which occur during myocardial ischaemia may not appear on a monitoring instrument.

REFERENCES


