

some action, other than that at the neuro-muscular junction, which is as yet unknown.

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CONTROL OF THE BLOOD PRESSURE AND CONTROLLED HYPOTENSION

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Control of the Blood Pressure during Anaesthesia

Blood pressure is the lateral pressure exerted by the blood on the vessel walls; our considerations are especially directed to the arterial system. The difference between systolic and diastolic pressure is the pulse pressure which depends on the stroke volume of the heart and the volume, elasticity and length of the arterial tree. In order to study the various factors influencing the arterial pressure, it is convenient to consider the mean pressure which is proportional to the product of the cardiac output and the peripheral resistance.¹

1. Cardiac Output

This is the product of the stroke volume and the heart rate and depends on the force of the heart and the cardiac filling.

(a) *The Force of Cardiac Contraction* affects the efficiency with which the heart muscles produce emptying. It depends on the initial length of the muscle fibres (Starling's law), so that an increased venous return leading to distension of the heart enhances the force of the beat. In a failing heart, however, overdistension occurs and increasing the venous pressure causes a further fall in output, conversely in such cases venesection may improve the circulation.

The force of the heart is also dependent on its nutrition and oxygen supply, which are affected by such factors as anoxaemia, the state of the coronary vessels and the arterial blood pressure. Excessive potassium ions cause increasing relaxation of the heart, finally bringing about arrest in diastole; for this reason stored blood is not advised for aortic transfusion during cardiac surgery.³⁸ Cardiac contraction is also influenced by body temperature and by nervous effects from

the vagus and sympathetic, it can be improved by humoral agents (adrenaline) and inotropic drugs, and is readily depressed by certain anaesthetic agents which produce dilatation.

(b) *Cardiac Filling*: This depends on the length of diastole and the effective venous pressure.

Venous pressure: The venous system accounts for 70 per cent. of the total volume of the vascular bed; it is evident that considerable circulatory adjustment can occur in this region. Surprisingly little is known of any venomotor mechanism:^{2,3} it is usually stated that there is insufficient muscle in the walls of the veins to produce any important effect. In the case of the larger veins it is probable that this thin layer of smooth muscle functions in a manner characteristic of hollow organs having a relatively thin wall compared with the size of the lumen, the purpose being maintenance of tone in order to allow considerable variation in capacity or filling with only slight changes in pressure.⁴ This tone is presumably regulated from the vasomotor centre: anaesthesia will diminish it, digitalis also can reduce venous pressure, noradrenaline can produce constriction of the veins and improve the venous return. A number of factors influence filling of the venous system, notably the blood volume.⁶ This may be varied in contrary directions by haemorrhage and transfusion. Closely connected with this is the state of the arterial bed, which accommodates a proportion of the blood volume and provides a through pressure to promote the venous return. The state of the body cavities, respiration, muscular activity and especially posture,⁹ mechanically influence the return of blood to the heart.

Apart from these effects on venous filling the venous pressure depends on the efficiency with which the heart empties the great veins. For this

reason attempts to correlate central venous pressure with cardiac output can give inconsistent results, for increased cardiac output can operate to produce a fall in venous pressure and vice versa. This is unfortunate, because the venous pressure is easily measured and would provide valuable information if it were a reliable index of cardiac output. As a guide to the onset of shock a falling venous pressure is an earlier sign than arterial hypotension; the degree of its elevation by transfusion provides a guide to replacement.^{5, 6, 7}

Changes in the thorax may have important effects on the blood pressure. The lungs during full inspiration hold 120 cc. more blood than during expiration; it follows that with the smaller vascular bed available during expiration there is increased resistance to blood flow. The calibre of the vessels may also vary reflexly, but little information is available on pulmonary vasomotor phenomena. Reflexes initiated by deep inspiration or inflation produce generalized cutaneous vasoconstriction. Apart from these vasomotor effects, inspiration by reducing intrathoracic pressure and by the descent of the diaphragm improves the venous return—the respiratory pump effect. In expiration the converse is true: expiration against resistance (Valsalva manoeuvre) impedes the venous return and the cardiac output and blood pressure (especially pulse pressure) fall.⁸ During expiration some blood is expelled from the lung vessels and the outflow into the arteries is thereby increased; there is also a direct transmission of changes of thoracic pressure to the arterial system. In view of the lag of two or three beats for venous return changes to be manifest as arterial pressure fluctuations, it is not surprising that mixed effects upon the blood pressure are reported.¹⁰ These phasic variations can be detected by means of direct electromanometry, and during average respirations are minimal. As most examples of protest to surgical stimuli during inadequate anaesthesia take the form of an increase in expiratory tone with decrease in size of the thorax, it is evident that relaxants by preventing such voluntary muscular activity will obviate blood pressure fluctuations of this type. Passive ventilation reverses the normal pressure phases in the chest. During inflation the venous return is impeded, especially if there is active muscular resistance due to inadequate anaesthesia, and so the arterial pressure will tend to fall. With the chest open such changes are less striking. Recent investigation indicates that cardiac output is greatest with a rapid build-up of inflation pressure followed by a total release of pressure in expiration, the duration of the expiratory phase being longer than inspiration. If these conditions are not fulfilled cardiac output may be reduced, especially

if the circulation is already impaired (e.g. haemorrhagic shock, constrictive pericarditis).¹¹ As passive ventilation is now undertaken during many anaesthetics, these facts merit serious consideration.

2. *The Peripheral Resistance.*

Peripheral vascular resistance depends upon two factors: (1) the viscosity of the blood, and (2) the total cross-sectional area of the available exit vessels, which varies according to (a) the anatomical state of the vessels, (b) nervous control and (c) peripheral chemical and humoral effects on the vessels.

Nervous Control: The most important part of this mechanism is the medullary vasomotor centre, the function of which is to adapt the volume of the vascular bed to the volume of blood available; this it does by regulating the calibre of the vessels, notably the arterioles which constitute the most important part of the peripheral resistance. Nervous control has also been demonstrated in the larger arteries and on the venous side of the circulation. All these changes in calibre are mediated through constrictor and dilator nerves of various types. Numerous factors act on the vasomotor centre to produce these effects, including the hypothalamus and higher centres. Raised carbon dioxide tension and oxygen lack exert a pressor effect. Afferent impulses from the lungs, muscles and abdominal organs produce varied results. Experimentally demonstrable reflexes involving the centre may be pressor or depressor according to the strength and frequency of the stimulus and the type of nerve fibre excited; for example, 'C' fibre stimulation characteristically produces a fall of blood pressure. Under anaesthesia many of these effects are abolished, but presumably reflex changes in pressure resulting from traumatic stimuli are often observed during light anaesthesia.

The most important impulses acting on the vasomotor centre emanate from baroreceptors in the carotid sinus, aortic arch and certain other areas. The higher the arterial pressure the greater is the rate of discharge of these special end organs. The resulting impulses have a depressor action on the vasomotor centre: by this mechanism it has been said 'the blood pressure regulates the blood pressure.' Pressure on the carotid sinus may stimulate these nerve endings and lead to a depressor effect associated with vasodilatation and cardiac inhibition.¹² Anaesthesia has been said to depress this sinoaortic apparatus; this will not, however, serve as an explanation for hypotension because depression of the carotid sinus will reduce inhibition of the vasomotor centre and so should lead to a rise in

blood pressure. Creyssel has blocked the sinus nerves with procaine in shocked patients and described clinical improvement with a rise in the arterial pressure.¹³

A fall in blood pressure can result from block of vasoconstrictor reflexes at any level: chlorpromazine affects the hypothalamus, the vasomotor centre may be depressed by anaesthetic agents, the sympathetic nerves may be blocked (e.g. by spinal or epidural anaesthesia) or the block may be at ganglionic level (T E A B, hexamethonium, etc.).

Local Chemical Influences can act directly on blood vessels: CO₂, histamine and metabolites generally produce vasodilatation, noradrenaline and numerous pressor drugs are vasoconstrictors.

Hypertension During Anaesthesia

The Effects of Carbon Dioxide

Anoxia and carbon dioxide retention may both produce hypertension. In so far as these factors are due to maladministration of the anaesthetic, such changes in pressure are avoidable. Increase in carbon dioxide tension affects the blood pressure in two directions: acting on the vasomotor centre it causes vasoconstriction, peripherally dilatation is produced by a direct action on the vessels. In the anaesthetized patient carbon dioxide accumulation, due for instance to soda lime exhaustion, frequently leads to a rise in pressure. In patients under extensive spinal anaesthesia this pressor effect is annulled due to the sympathetic block, and in these patients the peripheral dilator effect predominates leading to hypotension. Similar considerations may apply after ganglioplegics or during deep anaesthesia. Under cyclopropane, carbon dioxide retention, due to hypoventilation, may maintain the blood pressure at a relatively high level despite major surgical trauma. (Yandell Henderson's carbon dioxide therapy of shock will be recalled.) When the anaesthetic is discontinued the carbon dioxide level falls with resulting hypotension—'cyclopropane shock.'¹⁴ It is obvious that any anaesthetic technique which may cause hypoventilation (e.g. the relaxants) can produce a similar effect. When such methods are employed assisted or passive ventilation must be undertaken, ensuring an adequate minute volume, to avoid carbon dioxide retention. There is an urgent need at present for the development of some indicator to place 'controlled respiration' on a basis other than empirical. Carbon dioxide balance is far more difficult to assure than adequate oxygenation. The post-operative hypotension following such hypercapnia may be of long duration; its severity appears to be directly related to the degree of hypercapnia but not to the duration of the hypercapnic state.¹⁵ In so far as ganglioplegics (e.g. pentamethonium) prevent the pressor

effect of anoxia or hypercapnia,¹⁶ their administration may obviate hypotension following carbon dioxide retention: such an action may be partly responsible for the improved post-operative state observed when these drugs have been given.

Reflex Hypertension: Reflex rises in blood pressure due to stimuli from the field of operation are occasionally seen; the type of stimulus and depth of anaesthesia especially conducive to this reaction are uncertain.¹⁷ Distension of the bladder, and injection of solutions into the capsule of the prostate often lead to a transitory hypertension. In paraplegics the response to such stimuli can be very dramatic: hypertensive crises result from the operation of a mass autonomic reflex; this complication can be controlled by the administration of a ganglioplegic drug.^{18, 19} Of especial interest to anaesthetists is the hypertension reflexly produced by laryngoscopy and intubation during light anaesthesia.^{20, 21}

Adrenaline and Pressor Drugs: Handling the adrenal gland during adrenalectomy (even when the gland is normal) commonly produces a brief hypertension. This response can be prevented by the anti-adrenaline drug, chlorpromazine (12.5 mg. to 25 mg. intravenously). When a phaeochromocytoma is present such hypertensive crises may be very severe, even fatal,²² and must be controlled during extirpation of the tumour by the intravenous injection of adrenolytic drugs,²³ e.g. Benzodioxane 15 mg., Regitine 3 mg., or chlorpromazine 25 mg.; these doses may need to be repeated. Hypotension following removal of the tumour may need to be counteracted by 1 noradrenaline given in a drip (4 mg. 1 noradrenaline to 1 litre of normal saline).²⁴

1 noradrenaline is the most powerful overall vasoconstrictor available (being the physiological agent).²⁵ Adrenaline produces a marked increase in cardiac output while acting as an overall vasodilator.²⁶ Chlorpromazine reverses the pressor effect of adrenaline and if this drug be given beforehand infiltration of 10 cc. of 1:10,000 adrenaline can be used during the fenestration operation to produce local ischaemia combined with hypotension.

Many other drugs (with varying activity on cardiac output and peripheral resistance) can be given to raise the blood pressure: most of these are allied chemically to adrenaline and many produce dangerous types of tachycardia during anaesthesia. Evidently beneficial results from these agents can be expected when hypotension is associated with vasodilatation (e.g. during spinal or hypotensive anaesthesia): during oligoemic shock when the blood pressure is falling despite intense vasoconstriction such drugs are not indicated, but in shock when massive blood replacement fails to

restore the pressure (vascular paralysis) these agents have a place.²⁷ It is important to note that restoration of arterial pressure by these agents, e.g. after hexamethonium, is the result of generalized vasoconstriction. The capacity of the body for physiological redistribution of blood by vasomotor reflexes is not restored. Furthermore, the effect of the pressor drug, especially when given intravenously, may wane before the ganglioplegic and hypotension will then recur. Prolonged indiscriminate vasoconstriction maintained for days, e.g. by 1 noradrenaline drip, leads to tissue anoxia and liver necrosis has been described as a result of such therapy. Examples of useful pressor drugs are: ephedrine (50 to 100 mg.), Phenylephrine (5 to 10 mg.) and methyl-amphetamine or methedrine (10 to 30 mg.)—the last agent has a marked analeptic effect, and may cause post-operative restlessness and insomnia.

Arterial Hypotension

From physiological considerations it follows that a fall in arterial pressure may be due to a reduced cardiac output, to reduced peripheral resistance or to a combination of these. These disturbances may be classified:

1. Reduction in vasomotor tone.
2. Reduction of cardiac output
 - (a) Reduced venous return
 - (b) Reduced force of contraction
 - (c) Changes in heart rate.

Under normal conditions a reduction in cardiac output or peripheral resistance will be compensated reflexly and so long as this is adequate no marked change in blood pressure will be observed. The most important compensatory mechanisms against hypotension are increasing the heart rate and vasoconstriction. Inglis²⁸ has suggested that the pulse rate is the key to the treatment of hypotension occurring during anaesthesia. Hypotension associated with tachycardia is usually due to reduction of the cardiac output from a fall in the venous return: treatment is to increase the venous return by posture or transfusion. Cardiac output may also fall owing to reduced force of cardiac contraction, which may be due to anaesthetic overdosage: treatment should be directed to maintaining perfect oxygenation and promoting elimination of the anaesthetic—it has been suggested that cardiac and medullary stimulants may be given.

Hypotension with an unchanged pulse rate is probably due to reduction in peripheral resistance: vasomotor tone may be increased by pressor drugs given intravenously, e.g. noradrenaline 4 mg. per litre given as a drip, methylamphetamine 10 to 30 mg., or phenylephrine 10 mg. Hypotension with bradycardia may be due to vagal reflex action:

treatment consists in removing the precipitating cause and blocking the vagal efferents with atropine gr. 1/100 to 1/50 intravenously. During anaesthesia many variable factors are implicated,²⁹ so that it will not always be possible to determine the cause of any blood pressure change according to these simple rules; therefore, further discussion seems merited.

Induction of Anaesthesia is usually accompanied by depression of the vasomotor apparatus both centrally and peripherally. This is evidenced by the usual signs of vasodilatation (loss of temperature gradient in the limbs, dilatation of the superficial veins and increase in limb blood flow).³⁰ Some authorities hold that consciousness of itself has a pressor influence.³¹ Many agents, notably the barbiturates and chloroform also depress the myocardium and lead to cardiac dilatation; these factors reduce the circulatory reserve, but hypotension does not invariably occur in young people. In the elderly, compensation is less effective and one report suggests combining methylamphetamine with thiopentone to avoid hypotension in such patients.³² Ability to compensate against hypotension resulting from changes in posture is lost at a very light level of anaesthesia: in one investigation 60 per cent. of men over forty years of age who had previously received morphine gr. $\frac{1}{4}$ had hypotension and fainted when they were abruptly tilted into the head-up position. Burstein suggests that such falls in pressure can be avoided by inducing anaesthesia in the position desired for operation. Vasoconstriction is still possible during light anaesthesia and blood loss may be well compensated thereby. Pallor of the ears which develops during major operations is evidence of this. On rubbing with the hand, the ear becomes red due to vasodilatation as long as any circulatory reserve remains and to this it provides a guide. Blood oxygenation can be assessed by this test and the rapidity and intensity with which the pallor recurs is a guide to the state of the circulation.

Blood and Fluid Loss associated with the operation lead to decreased venous return and a fall in cardiac output. Compensatory widespread vasoconstriction may mask blood pressure changes but after brisk or severe haemorrhage there is usually marked hypotension, the systolic pressure falling by a greater degree than the diastolic. More gradual loss of an even larger volume may often be effectively masked in respect of blood pressure changes, but anaesthetics impair the homeostatic mechanisms in varying degrees.³³ It is certain that modern balanced anaesthesia, employing nitrous oxide-oxygen supplemented by analgesics such as pethidine and relaxants as indicated, allows these circulatory mechanisms to act to a greater extent than during ether anaes-

thetia.³⁵ It is not clear that these influences are entirely beneficial, vasoconstriction by reducing blood flow leads to tissue anoxia—a stage in surgical shock. It is possible that ganglioplegics (and also chlorpromazine), by blocking these changes, may prevent shock during severe surgical operations, provided that any blood loss is promptly replaced by transfusion. Observations have been made of the amount of blood lost during various surgical procedures and it has been noted that the measured loss is the minimal amount that must be replaced because additional blood is lost into traumatized areas by post-operative oozing, etc.³⁴ When the arterial pressure has fallen due to haemorrhage mere restoration of this pressure to normal by transfusion is not the index of adequate therapy, for persisting vasoconstriction plays a part in this rise. Transfusion cannot be considered adequate until the tone of the vessels is restored to normal and good peripheral circulation is re-established. Examples of increase in the arterial pressure above normal due to excessive transfusion are rare, the usual sign being a rise in the venous pressure as evidenced by distension of superficial veins. Recent investigation of the arterial route for transfusing blood shows that in desperate cases (e.g. patients in moribund condition following profound blood loss) this method may produce dramatic resuscitative effects. Lives may be saved which would be beyond the reach of intravenous transfusion.³⁶ The aortic route may be the most rapid and effective in such emergencies;³⁷ the coronary flow is improved immediately the transfusion into the arterial system is begun and improvement in the arterial pressure is more rapid than that produced by intravenous transfusion.

Neurogenic Hypotension: It is widely accepted that hypotension can occur as the result of neurogenic or reflex activity. Conclusive proof of this mechanism is lacking. Presumably nervous impulses due to trauma travel up afferent nerves to produce inhibition of the vasomotor centre. It was commonly believed that anaesthesia of adequate depth could prevent this type of fall. It is doubtful if this view can be sustained since reflex hypotension rarely occurs during light general anaesthesia accompanied by relaxants. Direct recordings of arterial pressure show that traction on mesentery can produce an immediate fall in pressure with instant recovery on the release of traction;¹⁰ these reactions are prevented by nerve block. The conclusion that such changes are due to a reflex seems inescapable. One disturbance of this type is of great interest and has been referred to as the coeliac plexus reflex;¹² it may be encountered during various upper abdominal procedures.⁷¹ When operative stimulation in the

coeliac region is maximal the pressure suddenly becomes unassessable by auscultation. The pulse remains palpable, however, and is not materially changed in rate. With careful auscultation, allowing the cuff pressure to fall slowly, a reading can be obtained in the region of the previous diastolic level: the pulse pressure is greatly diminished, e.g. the reading may be 90/85. Direct manometry has confirmed that such bizarre readings are not due to artefact associated with the Riva-Rocci method. Such fantastically low pulse pressure can only be caused by a great reduction in cardiac output. It is not known how this is produced reflexly; one report suggests that the phenomenon is due to vagal cardiac inhibition and advises treatment with intravenous atropine.³⁹ In confirmation of this view high right auricular and venous pressures have been observed during the operation of this reflex.^{4, 7} This disturbance is unlikely to occur when adequate dosage of relaxant has been given. An alternative explanation is a fall in the venous return due to pressure by the surgeon on the great veins (by packs or retractors). Extension of the spine (e.g. by a rest or bridge) or the lateral jack-knife posture may produce similar effects to stimulation in the coeliac region.¹⁰ Should such posturing lead to serious hypotension, a prompt return to a more normal position is indicated: elevation of the legs and head-down, tilt may further improve the venous return.

Spinal Anaesthesia: Arteriolar dilatation resulting from vasoconstrictor paralysis in the anaesthetized part of the body is the principal cause of hypotension during spinal anaesthesia. The ratio between the area of vasodilatation and that of compensatory vasoconstriction in the rest of the body determines the degree of blood pressure fall. When sympathetic block is complete such compensation cannot occur. It is probable that some reduction in cardiac output results from the increased circulatory bed during widespread vasodilatation.⁴¹ The above considerations apply equally to epidural block. Gillies⁴⁰ has advocated total spinal block anaesthesia leading to a hypotension of 40 mm. to 70 mm. systolic, to reduce haemorrhage and facilitate surgery during lumbodorsal sympathectomy. He pointed out that the pressure at the arterial end of the capillaries is normally 40 mm. and stated that in cases thus anaesthetized the major site of resistance is removed by dilatation of the arterioles so that the arteriolar capillary pressure approximates the existing arterial pressure of 40 mm. to 70 mm. With the patient in the head-down posture and breathing plentiful oxygen, tissue blood flow and oxygenation in important organs is adequate. Coronary blood flow being

dependent on the diastolic pressure may suffer, but this effect is mitigated by the reduced cardiac work lowering oxygen need. Other methods of producing controlled hypotension include the use of ganglioplegics such as pentamethonium and arterial bleeding. The ganglioplegics act in a similar fashion to spinal anaesthesia, producing, however, not only a sympathetic block but also a block of parasympathetic ganglia. When arterial bleeding is used to produce hypotension, vasoconstriction occurs which is likely to produce harmful tissue anoxia (haemorrhagic shock). On the other hand, a dry field is likely to be obtained at a relatively higher blood pressure level, furthermore the patient's own blood can be reinfused promptly to produce restoration of pressure immediately if it is indicated. This technique and the apparatus required are fairly complicated and reference should be made to the original articles.^{42, 43, 44} The excellence of the results obtainable with ganglioplegics even in neurosurgery have caused arteriotomy to fall into desuetude.

Controlled Hypotension

The aim of this technique is to provide the surgeon with an ischaemic operating field and to protect the patient against shock. Indications for controlled hypotension have not been rigidly defined and differ in various centres. Justification must be a product of the risk, the pathology and the possible advantage to be gained.⁴⁵

Three main methods are available as indicated:

1. Arteriotomy.
2. Spinal or epidural block. These methods carry the advantage that not only the bloodless field but analgesia and muscular relaxation are provided by the same manoeuvre. For further details reference should be made to published work on these applications of spinal block.^{31, 46}
3. Ganglionic blockade in which the hypotension is produced by intravenous agents, e.g. pentamethonium iodide.^{47, 48} Reduction in peripheral vascular resistance, gravitational pooling of blood in the veins of dependent parts and some reduction in cardiac output may all play a part.⁴¹ When the subject has been deprived of the nervous control of his circulation, changes in posture have a profound effect. When the body is tilted gravity induces a gradient of arterial pressure (30 mm. Hg. for every 15 in. vertical height from heart level).⁴⁹ This fact, combined with the hypotension, enables the anaesthetist to provide a remarkably dry operative field. For adequate control of surgical haemorrhage it may be necessary to reduce the blood pressure at the site of operation to 35 to 45 mm. A pressure of at least 60 mm. must always be maintained at heart level. The

question obviously arises as to whether such hypotension can be tolerated or whether it endangers the life of the patient. The work of Gillies⁴⁰ appears to show that adequate tissue oxygenation can be maintained provided the blood is well oxygenated and that vasodilatation is ensured. The low blood pressure associated with vasodilatation is not, of course, of the same serious significance as a similar fall in pressure due to haemorrhage with which is associated extreme vasoconstriction, low tissue circulation, anoxia and capillary damage. In a 30 to 35° head-up tilt the cerebral blood pressure has been calculated to be 35 to 40 mm. when the heart pressure is 60 mm. Hg. but other vital organs are at a pressure higher than heart level. Studies during hexamethonium-induced hypotension have shown an increase in the cerebral arteriovenous oxygen difference which implied a decrease in cerebral blood flow.^{16, 50} There is, however, a decrease in cerebral oxygen need during anaesthesia and the increased oxygen utilization which the above researches demonstrated appeared to be adequate; other reports have suggested that cerebral blood flow may not, in fact, be reduced because there is a fall in cerebrovascular resistance.⁵¹ (Cerebral oxygen needs may be further reduced by hypothermia, which has been proposed as an adjunct to increase the margin of safety.) Electroencephalograms have shown that virtually all cortical activity ceased when the pressure was reduced to 55 mm. systolic.^{16, 52, 53} Some alteration in cerebral function has also been shown by the flicker fusion test of Berg.⁵⁴

Renal blood flow may not be greatly decreased;^{55, 56, 57} naked eye changes in the liver have been observed during laparotomy but their significance may have been misinterpreted.⁵⁸

Technique.^{47, 59, 60} A well-adjusted blood pressure apparatus (auscultatory or oscillometric) must be fitted from the start and the blood pressure must be checked frequently. Access to a vein must be ensured by means of a diaphragm needle or an intravenous drip. Respiratory obstruction must be avoided and a high level of oxygenation must be maintained; therefore some authorities suggest that endotracheal intubation should be the rule. It does not greatly matter which agents are used, but since most general anaesthetics produce a tendency to postural hypotension very deep planes of anaesthesia should be avoided. The combination usually employed has been thiopentone, nitrous oxide with abundant oxygen and a relaxant or supplement as necessary. Apnoea, though not to be feared provided oxygenation is maintained, is perhaps better avoided, firstly because spontaneous respiration provides a valuable continuous indica-

tion of the patient's condition, and secondly because controlled respiration may cause a further lowering of the pressure. It must be noted, however, that controlled respiration is at all times mandatory in preference to inadequate spontaneous ventilation.

The patient is placed in the appropriate posture as soon as possible after the induction of anaesthesia, in order to assess the lability of the blood pressure and to give as long a time as possible for blood to drain away from the operation site. The first dose of ganglioplegic is then given intravenously; the aim is to obtain a systolic pressure of 60 mm. Hg. because the best results are obtained at this level. A lower pressure is physiologically undesirable and renders observation of the level difficult. Should excessive hypotension occur minute doses of a pressor agent are given to raise the pressure to 60 mm. Hg. or, in the case of Arfonad, the drip is temporarily stopped and the adverse tilt reduced.

Several factors are available as guides in assessing the dosage of hypotensive agent:

1. The body weight.
2. The metabolic rate. When this is high, as in thyrotoxicosis, very large doses may be needed for successful results.
3. The initial blood pressure.
4. The lability of the circulation as assessed by the hypotension resulting from induction of anaesthesia plus posturing. When this fall is profound (as is commonly observed in hypertensive subjects) small doses of ganglioplegic suffice.
5. Age. This is the most valuable guide. Young patients require much larger doses than the aged. The young commonly develop tachycardia which tends to prevent the fall in blood pressure.

Bearing these factors in mind one may use the following initial doses.

Average robust young adults:

Pentamethonium Iodide	..	50 to 200 mg.
Hexamethonium Bromide	..	20 to 60 mg.
Pentapyrrolidinium Bistartrate		10 to 20 mg.
Arfonad: drip solution	1 mg. per 1 mil—	60 drops per minute. ⁶⁰

Older patients:

Pentamethonium Iodide	..	10 to 50 mg.
Hexamethonium Bromide	..	10 to 30 mg.
Pentapyrrolidinium Bistartrate		5 to 20 mg.
Arfonad: 1 mg. per 1 mil—		10 to 30 drops per minute.

Subsequent doses are given as indicated by the blood pressure readings, aiming to keep the systolic pressure in the 60 to 70 mm. Hg. range.

It seems probable that the danger of overdosage is not great, the theoretical aim being *complete* sympathetic block such limit is unlikely to be

exceeded. In some patients such blockade produces excessive hypotension, in others a 'pressure floor' at a level higher than 60 mm. Hg. may be observed. Increased and additional doses of ganglioplegic produce no further fall in these resistant patients and only serve to prolong excessively such hypotension as is attained. The phenomenon of tachyphylaxis is frequently observed with these drugs, being least marked with Arfonad.^{60, 61} In resistant patients deepening the anaesthetic, increasing the adverse tilt, and administration of procaine amide⁶² may all help to secure satisfactory hypotension. In one centre a negative pressure is applied to the legs as part of the hypotensive technique.^{51, 63}

Surgical Technique: Infiltration with adrenaline solutions is unnecessary and (unless preceded by chlorpromazine) will produce a pressor effect leading to failure of the method. Haemostasis must be meticulous, even the smallest points being secured. Severe haemorrhage (e.g. damage to a major vessel) promptly leads to a further fall in blood pressure, the patient having been deprived of his compensatory mechanism. It is essential if much blood is lost, that prompt equivalent replacement be made; the blood pressure then returns rapidly to its former level.⁵⁹ As buffering reflexes are not active, such restoration of pressure is an accurate guide to transfusion. At the end of the operation the pressure should be rising and preferably should be in the 80 to 100 mm. region. Any acute rise is undesirable and may provoke haemorrhage. Frequent post-operative checks on the pressure should be made for the first 24 hours. Should the hypotension persist unduly long cautious antagonism with pressor drugs is indicated.

Operative Shock: The method has been used in certain operations usually regarded as liable to lead to operative shock, e.g. abdominoperineal resection, pelvic evisceration, etc., but provided adequate blood transfusion was undertaken shock did not occur. To what extent this phenomenon is due to prevention of blood loss or to the obtundation of circulatory reflexes is uncertain.⁶⁴ This observation has been corroborated⁶⁵ and is in accordance with physiological studies. Wiggers⁶⁶ states that recent studies favour the concept that the net effect of compensatory vasoconstriction is harmful and that the transition from impending to irreversible shock can be retarded or prevented by abrogation of sympathogenic vasoconstriction.

Contraindications to Hypotensive Technique are:

1. Inexperienced anaesthetist.
2. Unsettled anaesthetic conditions. When smooth anaesthesia cannot be secured or when an

anoxic episode has marred the induction hypotensive methods are contraindicated.

3. Established shock.
4. Recent coronary infarction or other severe cardiac disease.
5. Severe renal damage: elimination of the drugs may be retarded.
6. Respiratory inefficiency, severe anaemia or anoxia from any cause.
7. Severe diabetes.⁶⁷

Dangers of the Method: Obvious dangers include acute circulatory failure during the induction of hypotension; this has been reported but fortunately appears to be rare in the absence of cardiac disease or anoxic incident during anaesthesia. In deaths of this type the hypotensive technique seems directly implicated.

Deaths due to thrombosis, reactionary haemorrhage, renal failure, etc., were by no means unknown prior to the introduction of hypotensive methods: in one series the incidence of such complications was approximately equal in similar cases anaesthetised with or without hypotension.⁶⁸ Deaths due to anoxia of vital organs due to the hypotension have not been proven as a complication of the method. Opponents of this technique have suggested various complications, arguing from hypothesis and hearsay but unsubstantiated by case reports.

In one report⁶⁹ based on questionable evidence a death rate of 1 in 450 is suggested, but examination of the various causes of death reveals that many were not directly due to the hypotension, other complications were well known before hypotensive methods were introduced and the attributability of others is doubtful. Another valuable and interesting series of fatal case reports⁷⁰ reveals in almost every instance the danger of anaesthetic 'incidents' when hypotensive methods are used. It appears certain that the reserve or margin of safety is greatly lessened in the hypotensive state, any mismanagement of the anaesthetic or infringement of the rules will be paid for at the highest price—the life of the patient. For this reason induced hypotension is only permissible when there are direct and positive indications, when the anticipated benefit to the patient outweighs the risk and when first-class anaesthetic conditions are assured.

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