PREVENTION AND TREATMENT OF HYPOTENSION DURING CENTRAL NEURAL BLOCK

A. F. McCRAE AND J. A. W. WILDSMITH

The management of hypotension occurring during central neural block is a subject of some contention, with proponents for the use of either volume loading or vasopressors expressing dogmatic views. It is important not to be too specific in the approach to this problem and to treat each patient on individual merit, recognizing that many factors may influence arterial pressure during anaesthesia and surgery.

CONTROL OF ARTERIAL PRESSURE

Arterial pressure is the product of cardiac output and systemic vascular resistance and both of these variables are influenced by many factors (fig. 1). Cardiac output is determined by venous return according to the Frank Starling law. Venous return is influenced by gravity, the calf muscle pump, intrathoracic pressure and the degree of venomotor tone, which is matched to circulating blood volume. Systemic vascular resistance is determined by sympathetic vasomotor tone and by the influence of hormones such as renin, angiotensin, aldosterone and antidiuretic hormone. Endogenous vasopressin (antidiuretic hormone) also contributes to maintenance of arterial pressure during extradural anaesthesia [51]. The vasomotor centre in the brain stem controls the degree of sympathetic tone in a feedback loop involving the baroreceptors. Other factors affecting arterial pressure such as pain, exercise and emotion act directly through the vasomotor centre, which also receives input from the chemoreceptors.

Changes within the microcirculation may also influence arterial pressure, although it is more usual to think of those factors as being responsible for autoregulation of flow. There are two main mechanisms: myogenic and chemical. Myogenic autoregulation acts via stretch receptors in vessel walls which cause them to constrict when pressure is decreased. Chemical autoregulation is mediated by the local concentration of vasoactive metabolites. In the presence of vasodilatation, as produced by sympathetic block, an increase in flow washes out the metabolites and produces reflex vasoconstriction.

ORGAN PERFUSION

Although it is arterial pressure that we measure and adjust, it is important to remember that organ flow is the vital factor. Compensatory mechanisms ensure that flow to vital organs is maintained over a wide range of pressures.

Coronary blood flow is autoregulated between 60 and 150 mm Hg. If coronary perfusion pressure decreases, the myocardium becomes relatively hypoxic and ADP accumulates. This is converted to adenosine, a potent coronary vasodilator, which restores local blood flow. A decrease in arterial pressure results in decreased left ventricular work and oxygen demand. Therefore hypotension may have a benign effect unless there is concomitant tachycardia or a significant pressure gradient across the aortic valve, both of which impair coronary artery filling during diastole.

Cerebral blood vessels are devoid of sympathetic nerve supply and autoregulation of flow occurs between 50 and 180 mm Hg by reflex arteriolar constriction in hypertension or vasodilatation in hypotension. Thus normal cerebral blood flow is maintained until mean arterial pressure is less than 50 mm Hg. At such a pressure, ischaemia occurs, leading to local hypercapnia and hypoxia, both of which stimulate the vasomotor centre chemically to increase sympathetic tone and systemic vascular resistance in an attempt to restore arterial pressure.

Renal autoregulation occurs between 80 and 180 mm Hg. The afferent glomerular arteriole has a myogenic response to stretch and constricts with hypertension and dilates with hypotension to maintain a constant renal perfusion pressure. This can be overridden by the sympathetic innervation of the kidney. A small decrease in arterial pressure produces sympathetic stimulation and causes vasoconstriction of the efferent arteriole to maintain filtration pressure in the glomerulus in the presence of decreased renal blood flow. A more marked decrease in arterial pressure and more intense sympathetic stimulation results in constriction of the afferent

KEY WORDS

Correspondence to A. F. McC.
The autonomic nervous system affects arterial pressure through various factors. These include heart rate, stroke volume, cardiac output, venous return, intra-thoracic pressure, blood volume, peripheral resistance, hormones, and metabolites. The level of sympathetic block is higher than the sensory block in general, but the situation is complex. The differential cephalad extent of block has been reported as two segments if loss of temperature discrimination is used as the measurement of sympathetic block [23] and six segments if thermographic imagery techniques are used to measure small changes in skin temperature [6].

**Circulatory Effects of Central Block**

Hypotension during central neural block is mainly a result of a decrease in systemic vascular resistance which occurs by block of preganglionic sympathetic fibres. This allows vasodilatation and an increase in venous capacitance with venous pooling, unless gravity is used to maintain venous return. Hypotension is more likely to occur in dehydrated or hypovolaemic patients.

The major factor in the development of hypotension is the level of block. The sympathetic outflow is between T1 and L2, so that a block below that level has no effect on arterial pressure, but one to T8 "sympathectomizes" the lower half of the body. The sympathetic supply to the adrenal medulla is from T8 to L1 and central neural block alters arterial pressure by inhibition of the systemic release of catecholamines. Fibres from T2 to T4 provide the sympathetic supply to the heart, and decreased contractility and bradycardia (from unopposed vagal activity) ensues if they are blocked. Although vagal overactivity may cause severe hypotension in individual patients, changes in arterial pressure relate more usually to the level of block than to vagal efferent activity as measured by ECG R–R intervals [13]. A block as high as T1 totally removes the ability of the body to compensate for other circulatory changes in addition to producing extensive vasodilatation.

The level of sympathetic block is in general higher than the sensory block, but the situation is complex. The differential cephalad extent of block has been reported as two segments if loss of temperature discrimination is used as the measurement of sympathetic block [23] and six segments if thermographic imagery techniques are used to measure small changes in skin temperature [6]. Sympathetic block may also be incomplete. Only 60% of patients undergoing spinal anaesthesia had complete sympathetic block as demonstrated by skin conductance response measurements [41]. Such measurements correlate with changes in conductance resulting from sweating with sympathetic nervous system activity, although other workers have cast doubt on the use of this method as a measure of the completeness of sympathetic block [31]. The duration of sympathetic block may not be as long as previously thought, and one study has shown that motor and sensory block outlast it [3]. However, this may simply indicate that hormonal and capillary reflexes compensate for the sympathetic block and obscure its effects.

**Type of Central Block**

There are differences between spinal and extradural block. The degree of sympathetic block tends to be greater, and the onset of hypotension tends to be faster, after spinal than after extradural injection, because the speed of onset of the block is faster than the development of physiological compensation [14]. Individual variations in technique such as speed of injection, the use of barbotage in spinal or an incremental technique in extradural block [15] may cause different degrees of hypotension by altering the height of block obtained and the rate at which it develops.

Alterations to the local anaesthetic solutions, such as changing the baricity of the agent used in spinal anaesthesia to manipulate the height of block, may influence the development of hypotension [1]. Adding adrenaline tends to produce a more profound central block and therefore greater decreases in mean arterial pressure and systemic vascular resistance, but the changes may be modified further by the direct effect of adrenaline on peripheral resistance, heart rate and cardiac output. The use of alkalinized
Lignocaine in extradural block is associated with both a greater degree and more rapid onset of hypotension than non-alkalinized solutions [50]. Finally, the systemic effect of a large dose of local anaesthetic is myocardial depression and this may also influence the arterial pressure.

The Obstetric Patient

The obstetric patient presents additional problems. In the supine position, the inferior vena cava may be occluded completely by the pregnant uterus. Unless adequate venous return occurs through the collateral system of extradural and ayzygous veins, decreased arterial pressure and uterine blood flow result [35]. The aorta may also be compressed and, although this may not produce symptoms in the mother, uterine blood flow decreases, resulting in fetal distress and neonatal depression. This syndrome of supine hypotension by aortocaval compression may occur from 20 weeks onwards [8] and is relieved by the mother adopting the left lateral position. Although delivery of the baby also relieves the situation, it may be associated with an acute loss of circulating volume and compensation may be impaired seriously if the block is extensive.

TREATMENT

Defining hypotension

A variety of options is available for both prevention and treatment of hypotension caused by central block. These have been researched extensively, but there are many problems in comparing studies in this field. Although the variability of “resting” arterial pressure is widely recognized, it is unusual for the status of the control figure to be defined clearly. Second, arbitrary definitions of hypotension have been chosen by different authors: systolic arterial pressure < 100 mm Hg or < 90 mm Hg, 20% or 30% decreases from baseline or any 30-mm Hg decrease in systolic pressure have been used. In some studies, changes in mean rather than systolic arterial pressure were analysed and relatively few studies have been fully randomized. Frequently, only patients who became hypotensive were studied.

Such factors have favoured the preconceived notion that all patients become hypotensive during central nerve block and as a result there are few studies with true control groups in which no intervention was used. Figure 2 [38] relates the percent change in systolic pressure to the level of block in 100 non-obstetric patients who received a variety of regional techniques, but no prophylaxis for hypotension other than a 5° head-down tilt. The risk of hypotension increased with height of block, but many patients with extensive blocks had minimal changes in arterial pressure.

General points

Because a high level of block is a major determinant of the development of hypotension, choosing a block limited to suit the planned procedure prevents unnecessary decreases in arterial pressure. Haemorrhoidectomy does not require a block higher than S1, whereas abdominal hysterectomy needs a block of T10 at least. In any patient receiving a central nerve block, venous return should be maintained by a slight (5–10°) head-down tilt to eliminate venous pooling in the legs, and by the use of left-lateral tilt in obstetric patients.

Infusions and combined blocks have been advocated as being less likely to produce significant hypotension. When continuous extradural infusions are used in labour, the majority of episodes of hypotension occur in relation to the initial loading dose [20]. If a bolus of the dilute solution intended for the infusion is used as a loading dose rather than...
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The combined spinal-through-extradural needle technique decreases the incidence of hypotension by producing rapid establishment of a spinal block of the lower segments followed by careful extension of the block using bolus extradural injections. During Caesarean section using the combined technique, the incidence of hypotension was 33% compared with 53% in a conventional extradural group. Another advantage was a reduction in the total dose of local anaesthetic to approximately 33% of that used in the conventional group [56].

The use of combined spinal-through-extradural needle techniques have not produced problems of accidental high block with associated severe hypotension [11, 18, 33]. However, spinal block performed after extradural anaesthesia may result in a very extensive block and severe hypotension [2, 64]. Possibly, local anaesthetic passes through the puncture wound in the dura or, more likely, the subarachnoid space is compressed by the large volume of local anaesthetic present in the extradural space [2].

Hypotension during spinal anaesthesia may be reduced using a catheter technique. A retrospective study of elderly patients undergoing orthopaedic surgery found that hypotension occurred less frequently, and fewer doses of vasopressor were required during continuous spinal anaesthesia than during continuous extradural block [65].

Mechanical methods

Several mechanical methods of compressing the lower limbs, in order to encourage venous return, have been used with varying degrees of success. In studies of Caesarean section under spinal anaesthesia, the application of Esmarch bandages from ankle to mid-thigh decreased the incidence of hypotension from 83% to 16%, [4], and inflatable full length leg splints (normally used for fracture stabilization) from 83% to 48% [22], but inflatable boots produced no significant effect [27]. However, a different volume of fluid was given in each of these studies: 20 ml kg\(^{-1}\) in the first, 15 ml kg\(^{-1}\) in the second and 10 ml kg\(^{-1}\) in the third. During extradural Caesarean section, elastic bandages were found to reduce the incidence of hypotension from 83% to 13% [21], but graduated compression stockings made no significant difference, the incidence of hypotension being 30% in both groups [34]. However, the volumes of fluid administered were different; Ringer’s lactate solution 15 ml kg\(^{-1}\) in the first study and only 500 ml in the second. It would appear that none of these methods is entirely reliable in preventing hypotension.

Additional mechanical methods have been used in obstetric patients to eliminate supine hypotension, by displacing the uterus, either by a special device or by adopting the full left-lateral position. In pregnant women at term, a comparison between the supine or left-lateral “wedged” positions with either the Colon–Morale or Kennedy uterine displacement device found that the wedge was more effective than mechanical displacement of the uterus [19]. One interesting study looked at the inter-relationship between fluid loading and mechanical displacement in patients undergoing Caesarean section with a spinal anaesthetic. In elective patients, the incidence of hypotension was 92% in an untreated control group, 57% after 1000 ml of crystalloid and 53% with mechanical displacement and crystalloid [10]; in a group of emergency Caesarean sections, the incidences were 50%, 46% and 15%. The lesser frequency of hypotension in the labouring groups was possibly the result of contractions autotransfusing blood from the uterine vessels to the general circulation. It seems that mechanical devices have little to offer, as they merely displace the centre of gravity, whereas tilt alters the direction of force.

Volume loading

The effect of i.v. fluid loading is to increase stroke volume and cardiac output [58], although these return to control values after institution of a central block. Both crystalloid and colloid have been used to compensate for these changes and arguments have been advanced for the use of each. In general, the use of crystalloid requires a larger volume than colloid and this might be expected to cause pulmonary oedema in an at-risk patient, but one study found this not to be the case [68]. Colloid may stay in the intravascular compartment longer, but is more expensive and some of the solutions confer a small, but significant, risk of anaphylaxis [63].

Large volumes of fluid may not be desirable in some circumstances, and may be frankly dangerous in elderly patients with poor cardiac function, in whom there is a risk of pulmonary oedema and cardiac failure. Routine volume loading may be unnecessary in these patients unless they are dehydrated. Ringer’s acetate solution 0, 8 or 16 ml kg\(^{-1}\) was given to elderly patients having lower limb or lower abdominal operations under spinal anaesthesia, but the incidence of hypotension (24–32%) was not significantly different between the groups [12]. Obstetric patients are also at increased risk of pulmonary oedema, because interstitial lung water is increased during the puerperium [39]. A possible further complication of large volume administration is the development of urinary retention. Sympathetic vascular tone returns before bladder sensation as a block regresses, so that overdistension occurs while the patient is unaware [7].

In a small study of spinal anaesthesia for Caesarean section or vaginal delivery, pretreatment with 1000 ml of 5% glucose in Ringer’s lactate solution prevented any significant decrease in arterial pressure from baseline, whereas the control group all became hypotensive (to a greater extent in the patients with a higher block for Caesarean section) [71]. A larger preload (2000 ml) of Ringer’s lactate solution was used in an open study of Caesarean section under extradural block and only 6.7% of patients had a decrease in arterial pressure greater than 20% from baseline [36]. Therefore volume loading was concluded to be a satisfactory method of preventing hypotension. In contrast, a recent study of Caesarean section under spinal anaesthesia com-
pared volume loading with crystalloid 20 ml kg\(^{-1}\) given over 20 or 10 min. The incidence of hypotension was still 60%, and 70%, respectively, and three of 10 patients in the latter group had unacceptable increases in central venous pressure [60].

It has been argued that colloid produces a more sustained and more effective expansion of the intravascular space and several studies have compared colloid with crystalloid in the prevention of hypotension during central neural block.

In extradural Caesarean section, a regimen of Ringer's lactate 500 ml + gelatin 500 ml solutions proved superior to 1000 ml of Ringer's lactate solution and reduced the incidence of hypotension from 45% to 10% [25]. Similarly, Dextran 70 solution 7.5 ml kg\(^{-1}\) was more effective than Ringer's lactate solution 15 ml kg\(^{-1}\), reducing the incidence of hypotension from 25% to 5% [70]. Using thoracic electrical bioimpedance, the latter study also demonstrated a smaller thoracic fluid index, indicating less lung water in the patients given colloid. In patients having lower limb operations under extradural block, volume loading with 500 ml of low and medium molecular weight hetastarch, Dextran 70 and balanced salt solution were compared [32]. The requirement for ephedrine (ethyl-norphenylephrine) to maintain arterial pressure was not significantly different between the groups, although there was a trend towards greatest usage in the balanced salt solution group, less in the hetastarch groups and least in the Dextran group.

However, other studies of extradural Caesarean section have shown no benefit of colloid compared with crystalloid. One compared Ringer's lactate solution 1200 ml, with similar volumes of Ringer's lactate solution combined with albumin and found no significant difference in mean arterial pressure or ephedrine requirement [55]. Another study compared Ringer's lactate solution 2000 ml with Hetastarch solution 1000 ml and also found no difference between the groups [45]. During Caesarean section under spinal anaesthesia, Ringer's lactate solution 15 ml kg\(^{-1}\) with 5% glucose was compared with the same solution containing 5% albumin; 50% of the patients in each group were then given ephedrine 25 mg by i.m. injection. No patient given albumin developed significant hypotension, whereas hypotension occurred in approximately 30% of the crystalloid groups, with or without ephedrine [42].

Taken overall, these studies are contradictory, even in the incidence of hypotension in the “control” groups. Infusion of large volumes of fluid has been shown to overload the right side of the heart [46] and requires that all patients are “treated” before the spinal or extradural needle is sited. This may result in a significant proportion of patients who would not have become hypotensive receiving a large fluid load. Perhaps a more physiological approach to the treatment of hypotension induced by sympathetic block is to counteract it as it occurs, by using sympathomimetic drugs.

**Vasopressors**

A large proportion of the studies which have been performed have involved obstetric patients. However, there has been concern over the use of vasopressors in obstetric practice, particularly methoxamine, which may decrease uteroplacental blood flow [53]. A possible added complication is the development of severe hypertension if prophylactic vasopressors are used in conjunction with oxytocic drugs [5], particularly in a mother with pre-eclampsia or essential hypertension. This is less likely since synthetic oxytocin replaced the pituitary derivative “Pitocin” which may have been contaminated with vasopressin.

Without volume loading or lateral tilt, the incidence of hypotension in Caesarean section under central neural block exceeds 90% [10] and most studies of vasopressors in obstetric patients include preliminary volume loading of the patient. This makes evaluation of the effect of vasopressors difficult.

**Ephedrine** is the most commonly used vasopressor. It has direct and indirect mechanisms of action and stimulates both alpha and beta receptors to increase cardiac output, heart rate, systolic and diastolic arterial pressure. Coronary, cerebral and muscle blood flow are increased, while renal and splanchnic blood flow are decreased [69]. Ephedrine was used first in 1927 to counteract hypotension caused by spinal anaesthesia [47], and early administration to prevent a decrease in arterial pressure was advocated, rather than treating hypotension after it had developed. In a double-blind study during Caesarean section under spinal anaesthesia, patients were given crystalloid 1000 ml and an i.m. injection of ephedrine 50 mg or placebo before the block [24]. All patients in the placebo group became hypotensive (in spite of the volume load), compared with only 25% in the ephedrine-treated group. An increase in systolic arterial pressure occurred in all the ephedrine-treated patients, but significant hypotension did not develop.

A larger, randomized study of prophylactic i.m. ephedrine in extradural Caesarean section compared placebo and i.m. ephedrine 25 mg or 50 mg after volume loading with 1000 ml of crystalloid. The incidence of hypotension was less in this study (8–12%) and was similar in all the groups. Persistent hypertension was observed in 66% of the group given ephedrine 50 mg and the study was discontinued for this reason [59].

In non-obstetric patients, development of hypotension is related to the general physical condition of the patient. Patients in ASA grades I–III undergoing spinal anaesthesia for lower body surgery and preloaded with isotonic saline–glucose solution 7 ml kg\(^{-1}\) received either placebo or ephedrine 12.5 mg i.v. + 37.5 mg i.m. [26]. No patient given ephedrine became hypotensive, but in the control groups hypotension occurred in 0%, 12.5% and 50% of patients graded ASA, I, II and III, respectively.

I.m. injections of ephedrine before central neural block have unpredictable absorption and peak effect, and do not reliably prevent hypotension. In addition, large bolus doses may cause unacceptable hypertension, so it may be preferable to await the onset of hypotension and then to administer treatment. In a
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non-random study during Caesarean section with spinal anaesthesia, patients were given crystalloid 1500 ml and allocated to three groups after the block. The control group were those in whom no decrease in arterial pressure occurred (37%). The remainder were treated with either bolus doses of ephedrine when systolic pressure decreased to less than 100 mm Hg, or as soon as any decrease in arterial pressure from baseline was observed. Episodes of nausea and vomiting caused by hypotension were significantly less frequent in the patients treated early [17].

Vasopressor therapy may be adjusted to the degree of hypotension using an infusion. In a study during Caesarean section with spinal anaesthesia, patients received volume loading with Ringer’s lactate solution 15 ml kg⁻¹ and were treated with either ephedrine 20 mg i.v. followed by 10-mg doses if arterial pressure decreased to 80% of baseline, or with ephedrine 10 mg i.v. followed by an infusion of ephedrine adjusted to maintain systolic pressure at 90–105% of baseline [29]. Total ephedrine dosage was similar in the two groups, but nausea was more frequent in the bolus group (36%) than in the infusion group (5%).

Etilerine, used in central Europe and Scandinavia, was compared with ephedrine in a randomized study in elderly patients undergoing hip surgery with spinal anaesthesia. Preliminary fluid administration was small, and all patients whose mean arterial pressure decreased by at least 25% were included in the study. Hypotension was treated with repeated i.v. bolus doses of vasopressor. Both were equally effective in restoring systolic pressure, but ephedrine was more effective in restoring mean and diastolic arterial pressures [66].

Metaraminol has direct and indirect sympathomimetic actions which increase cardiac contraction and venous tone and hence increase arterial pressure. It has a prolonged action (4 h) after i.m. injection [69]. Metaraminol has been studied in patients having transurethral prostatectomy under spinal anaesthesia using a variety of local anaesthetic agents and doses [16]. There were four groups of patients who received a preload of 5% glucose 500 ml, mephentermine 30 mg i.m., a combination of both, or no treatment. Hypotension occurred in 44% of both the control and the volume loaded groups, but was significantly reduced, to 24% by mephentermine and to 4% with combined therapy.

Mephentermine has direct and indirect alpha effects which produce vasoconstriction which may affect peripheral vessels selectively, thus increasing systolic and diastolic pressures and producing a reflex bradycardia [69]. Dobutamine has pure beta₁ sympathomimetic effects, increasing the force of myocardial contraction [69]. Metaraminol and dobutamine infusions have been compared in patients having major abdominal surgery under extradural anaesthesia [28]. Both agents increased systemic and pulmonary pressures and cardiac work and appeared to be equally effective in preventing hypotension. Mephentermine was preferred because of its selective action on the heart and lack of effect on total peripheral resistance.

Dopamine, the precursor of noradrenaline, causes vasoconstriction through alpha receptor stimulation when used in large doses [69], and it may be an ideal agent to use in the prevention of hypotension because of its rapid onset and short duration of action. In a study during Caesarean section under spinal anaesthesia, 78% of the patients became hypotensive and were allocated randomly to receive an infusion of dopamine 2–10 µg kg⁻¹ min⁻¹ or i.v. boluses of ephedrine 10 mg [9]. The remaining 22% of patients who did not show any decrease in arterial pressure formed the control group. Although dopamine proved effective, it was no more so than intermittent ephedrine. Dopamine 4 µg kg⁻¹ min⁻¹ has been shown to increase mean arterial pressure, cardiac output and heart rate during thoracic extradural anaesthesia in which volume loading with 5% albumin 500 ml with crystalloid 1000 ml was not effective in preventing hypotension [37].
Adrenaline is a potent alpha and beta stimulator. Its predominant effect is to increase systolic pressure by direct myocardial stimulation, increased heart rate and vasoconstriction in the skin, mucosa and kidneys [69]. In a study in anaesthetized, splenectomized sheep, extradural analgesia was performed and decreases in arterial pressure and peripheral resistance were observed. Administration of Dextran 70, 10 mg kg\textsuperscript{\text{-1}} produced a small increase in arterial pressure secondary to increased stroke volume, but this was counteracted by a further decrease in total peripheral resistance. Administration of adrenaline approximately 1 \mu g kg\textsuperscript{\text{-1}} min\textsuperscript{\text{-1}} reversed the hypotension by increasing cardiac output secondary to improved contractility and venous return. There was no effect on peripheral resistance [49]. This study showed that plasma expansion alone was insufficient to counteract systemic hypotension, but that adrenaline can restore arterial pressure without adverse effects on regional perfusion. In elderly patients undergoing total hip replacement with hypotensive extradural anaesthesia, low dose adrenaline infusion was used effectively to maintain arterial pressure at 50–55 mm Hg by increasing cardiac index and stroke volume without change in heart rate [61].

Prenalterol, a beta\textsubscript{2} receptor agonist has been used in a small study of thoracic extradurals for aortic aneurysm surgery. Hypotension occurred in all patients and was treated with prenalterol 10 mg by i.v. infusion. Arterial pressure was rapidly restored as a result of increased cardiac output without any increase in heart rate or vascular resistance [57].

SUMMARY

Hypotension during central neural block may occur by three main mechanisms: decrease in venous return (in turn influenced by posture, bleeding and inferior vena cava compression), vasodilatation and decreased cardiac output. It is also important to recognize that, occasionally, other factors play a part. Bladder distension during central nerve block has been shown to produce hypotension inappropriate to the level of block [48, 62] and vagal overactivity may contribute in the unsedated patient. Preventive measures to reduce the likelihood of hypotension include correction of hypovolaemia, restriction of the upper level of block, use of a slight head-down tilt to maintain venous return and judicious use of sedation, especially in anxious patients. In the obstetric patient, the single most important factor in eliminating hypotension is the use of full left-lateral tilt. Mechanical methods to improve venous return by compressing the legs are not particularly helpful. Volume loading does not guarantee maintenance of arterial pressure and excessive fluid may be harmful in patients with bladder neck obstruction or at risk of pulmonary oedema. The administration of up to 1 litre before surgery may be particularly advisable if significant blood loss is expected (no matter what the anaesthetic technique), but colloid solutions do not have clear proven benefit over crystalloid.

The prevention or treatment of hypotension induced by central block by administration of large volumes of fluid is a more contentious subject, although the practice is widespread. Review of the literature has shown that many studies have been poorly designed and the results have often been contradictory, even in such basic principles as the incidence of hypotension in control groups. There is a need for evidence based on correctly designed studies comparing tightly designed treatment regimens. One such study [72] has recently shown the benefit of ephedrine compared with both volume loading and methoxamine in the prophylaxis of cardiovascular changes during combined extradural and general anaesthesia. In the treatment of hypotension, vaspressors can be much more rapidly effective and directly reverse the physiological changes produced by the block. Early intervention with small i.v. doses (e.g. ephedrine 3–6 mg or methoxamine 1–2 mg) if arterial pressure decreases towards an unacceptable value provides better control than large i.m. doses given before the block. Finer control can be achieved by using a suitable drug in an infusion. Ephedrine is probably the most useful drug in terms of safety and efficacy, but a drug with predominantly alpha agonist activity (e.g. methoxamine) may be more appropriate if heart rate is increased, especially if the response to ephedrine is poor. The decision on what degree of hypotension requires treatment can be made only for individual patients, but there are few who cannot tolerate a decrease of 25–30% quite safely.

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