INFERIOR VENA CAVAL OCCLUSION IN LATE PREGNANCY AND ITS IMPORTANCE IN ANAESTHESIA

BY

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SUMMARY

The effects of posture upon circulatory haemodynamics in late pregnancy and their importance to the anaesthetist are discussed in relation to conscious patients, during general anaesthesia and following epidural or spinal blockade.

That the supine position can cause sudden and severe hypotension during late pregnancy has been known for many years (Brigden, Howarth and Sharpey-Schafer, 1950; Howard, Goodson and Mengert, 1953). These acute falls in arterial blood pressure have been ascribed to the weight of the gravid uterus pressing upon the inferior vena cava, and led to the term "supine hypotensive syndrome". Holmes (1960), studying 500 women in the last few weeks of pregnancy, found that, although this syndrome affected only 3 per cent of patients, there was some decrease in arterial pressure in up to 70 per cent.

In a study of the changes in pressure within the lower inferior vena cava during advanced pregnancy, considerable elevation of the pressure was a constant feature (Scott and Kerr, 1963). In non-pregnant women, the pressure in the inferior vena cava is normally 4–7 mm Hg in the supine position (Scott, 1963). In the last weeks of pregnancy, however, this pressure is 20–30 mm Hg, a figure only reached in non-pregnant cases by complete occlusion of the inferior vena cava above the level of the measuring catheter (fig. 1). In the pregnant cases, the high pressure in the lower inferior vena cava is accompanied by damping of the respiratory wave pattern. On turning the patient into the lateral position there is a sharp fall in pressure to around 10–15 mm Hg and the respiratory wave pattern becomes much more pronounced (fig. 2). These changes indicate that inferior vena caval occlusion is occurring in the supine position, even in the absence of postural hypotension, and is relieved, in part at least, by turning the patient on to her side.
Inferior vena cavogram in the supine position in late pregnancy. Injections of radiopaque dye are made simultaneously into each femoral vein. Little or no dye enters the inferior vena cava. The collateral circulation via the paravertebral veins is clearly seen.

If inferior vena cavograms are taken during the last weeks of pregnancy (Kerr, Scott and Samuel, 1964), it will be seen that little or no blood flows up the vein in the supine position (fig. 3), the main channel for the venous return being the paravertebral system of veins emptying into the vena azygos. Even in the lateral position, there is considerable narrowing of the vena cava in its distal 5–10 cm (fig. 4).

When the uterus is emptied at Caesarean section, the pressure in the lower inferior vena cava returns rapidly to non-pregnant levels (fig. 5) and flow along the cava becomes normal (fig. 6).

These studies clearly show that inferior vena caval occlusion was the rule rather than the exception in the supine position in late pregnancy.

They did not show, however, the importance of this occlusion on central haemodynamics, which could remain unaffected if the venous return via the collateral circulation was unchanged.

A detailed study of the central haemodynamics related to this problem has now been reported (Lees et al., 1967). Of six patients who were quite unaffected clinically by the supine position, three showed a significant fall in cardiac output when turned from the lateral to the supine position. Thus, a fall in output of 20 per cent was accompanied by no alteration in mean arterial pressure or heart rate, thus indicating a considerable rise in peripheral resistance (fig. 7). That the fall in output is due to reduced venous return is shown by falls in central venous pressure and the cardiopulmonary blood volume. It is noteworthy that in
Inferior vena caval pressure at delivery during Caesarean section. Note the rapid fall in pressure to normal non-pregnant values (10 mm Hg) and reappearance of normal respiratory wave pattern.

Lees and colleagues (1967) also investigated two cases of supine hypotensive syndrome. In these patients dramatic changes occurred as soon as the supine position was assumed (fig. 8). There was an immediate and large fall in cardiac output (up to 50 per cent) in spite of which the mean arterial pressure was maintained for 4–5 minutes. At this point the pulse rate changed from a moderate tachycardia to a marked bradycardia. This led to a sharp fall in arterial pressure and a further drop in cardiac output. The sudden appearance of a bradycardia, which in circumstances of reduced cardiac output can only lead to further circulatory impairment, is typical of a vasovagal attack. This concept, that the supine hypotensive syndrome is in part a vasovagal phenomenon, fits in well with the clinical features seen in these cases. Patients not only feel faint, they may also complain of visceral symptoms such as nausea and vomiting. Bradycardia is a common feature and loss of consciousness can occur, the systolic blood pressure often falling as low as 45–50 mm Hg. Turning into the lateral position leads to rapid relief of the signs and symptoms. Atropine might be expected to prevent these attacks but, in the dosage used for premedication, it is of little value, as supine hypotension with bradycardia can be seen, prior to induction of anaesthesia, 1 hour after 0.6 mg intramuscularly. Complete vagal blockade, of course, requires much higher dosage.

It has become clear that inferior vena caval compression is the rule rather than the exception in the supine position in advanced pregnancy. Only occasionally, however, does it result in a gross reduction of arterial blood pressure. Nevertheless, large changes may be occurring in haemodynamics in spite of a normal blood pressure. As almost all obstetric operations are performed in
Haemodynamic pattern in a patient in late pregnancy. Note that when lying supine, there is a marked fall in cardiac output with little change in arterial pressure or pulse rate. Peripheral resistance is increased during these periods of reduced cardiac output.

Effects of caval compression in conscious patients.

Few women in advanced pregnancy lie flat on their back from choice, and the supine hypotensive syndrome may be missed because the patient lies in a slightly lateral position while the blood pressure is being taken. If made to lie completely supine, many patients show either a reduction in arterial blood pressure or a fall in pulse pressure.

Before inducing anaesthesia, the blood pressure should be taken in both the lateral and supine positions. If supine hypotension is detected, the patient should be turned into a semi-lateral position, this being sufficient in most cases to restore the blood pressure before the anaesthesia is begun. Even when there is no great reduction in systolic blood pressure there is sometimes a fall in pulse pressure in the supine position and such
Haemodynamic pattern in a patient exhibiting the supine hypotensive syndrome. Note the large fall in cardiac output on turning into the supine position. The arterial pressure and heart rate are maintained for 5 minutes, at which point sudden bradycardia occurs with marked hypotension. All parameters return rapidly to normal on the resumption of the lateral position.

Cases should be watched carefully as severe hypotension may ensue during anaesthesia. Anaesthetists may be asked to see patients suffering from severe antepartum haemorrhage. In assessing these cases, the patients should be turned on their side because, in the supine position, if the postural effect is marked, the degree of shock can be exaggerated and inappropriate treatment given. For this reason all such cases should be nursed in the lateral position.

Effects of caval compression during general anaesthesia.

Although it is now common practice to use only the lightest forms of general anaesthesia during obstetric anaesthesia, hypotension is not an
uncommon occurrence soon after induction (Crawford, 1962). This can happen in patients who have shown little or no signs of supine hypotension when conscious. As stated above, in conscious patients who become hypotensive, a vasovagal reflex with bradycardia supervening on caval occlusion is the probable mechanism. Under anaesthesia, hypotension often occurs without bradycardia and a vasovagal reflex is less likely to be responsible. The most likely cause is the widespread vasodilatation that occurs even with light anaesthesia. This effectively reduces the peripheral resistance which is usually raised in order to maintain normal arterial blood pressure in conditions of reduced venous return. The dilated veins of the lower limbs are capable of holding large quantities of blood, thus effectively reducing the circulating blood volume. As the pressure in the veins below the level of occlusion rises, the collateral circulation becomes more effective and some improvement in the level of arterial pressure may result, thus explaining the spontaneous reversal of severe hypotension described by Crawford (1962). Often, however, the circulation is only substantially improved when the caval compression is corrected.

It must be remembered that hypotension may occur at any time up to the emptying of the uterus and not only during the immediate postinduction period. Small movements of the uterus during manipulation or positioning of the patient can lead to a more complete, or a higher, level of caval compression. The fact that light anaesthesia is being used makes any other cause of hypotension most unlikely. Pulse and blood pressure should be observed carefully throughout and the obstetrician warned of circulatory embarrassment.

Expeditious removal of the foetus is the most logical treatment for postural hypotension. As soon as the baby is delivered the venous return to the heart is greatly augmented and leads to considerable changes in the central haemodynamics. Removal of the placenta from the circulation also increases the venous return to the heart, though this is offset by the loss of blood that occurs at this time. In the first few minutes after delivery it is not uncommon to see a sharp rise in the arterial blood pressure, often to quite high levels, usually settling rapidly to normal (fig. 9). Ergometrine, which is itself a vasopressor, especially in pre-eclamptic patients (Baillie, 1963), is usually given at the moment of delivery, thus augmenting the hypertensive effect.

Central venous pressure, arterial pressure and inferior vena caval pressure at delivery during Caesarean section. This patient showed a very marked rise in arterial pressure in the first few minutes following delivery, accompanied by a raised central venous pressure and a decreased inferior vena caval pressure. Note the increase in pulse pressure after delivery.
While the heart is almost always capable of dealing with this considerable increase in venous return this, on occasion, may not be the case. In patients with severe cardiac disease, the danger is obvious and the third stage of labour is well known as a difficult period for these cases. In severe pre-eclamptics with hypertension, the sudden increase in arterial pressure, which may last much longer than normal, can give rise for concern. The vasoconstricted state of the circulation in these patients accentuates the hypertensive effects of increasing the venous return. In a similar way, patients to whom vasoconstrictors have been given may also develop cardiac embarrassment. Unfortunately, many anaesthetists, faced with sudden and unexplained hypotension, administer vasoconstrictors to restore the pressure to normal. In conditions of severe reduction in venous return and cardiac output, the restoration of a normal systolic pressure represents an intense degree of generalized arterial vasoconstriction. With the relief of the venous obstruction, the left ventricle may have difficulty in ejecting the increased cardiac output into the constricted systemic circulation. These circumstances can lead to the development of acute pulmonary oedema.

The indiscriminate use of vasopressors to treat hypotension regardless of its causation is to be deprecated. Although these drugs raise the blood pressure, there is no good evidence that they increase cardiac output. Indeed, methoxamine, a very commonly used drug, may have the reverse effect (Shimosato and Etsten, 1965).

Effects of caval compression during epidural and spinal blockade.

There are numerous reports in the literature of sudden death following the administration of spinal analgesia in advanced pregnancy and many of these have been reviewed by Holmes (1957). The explanations advanced to account for these deaths have been numerous. Intercostal paralysis from high spread of the spinal block in the presence of a „splinted“ diaphragm is believed to be of importance (Macintosh, 1949), but the suddenness with which cardiac arrest occurs makes acute failure of the venous return a much more likely explanation (Holmes, 1957). A marked fall in blood pressure is very common after epidural and spinal blockade in late pregnancy. It is unlikely that this is due solely to sympathetic blockade even though epidural blocks have an enhanced “spread” in pregnancy (Bromage, 1962), because it is difficult to lower the arterial blood pressure to any serious extent in young, non-pregnant, conscious, supine patients, even with high spinal block. If, however, inferior vena caval occlusion is impeding the venous return, vasodilatation of the lower limbs, which is especially marked with these types of regional analgesia, can lead to cardiovascular collapse. Another contributory factor which must not be overlooked is the common occurrence of bradycardia following high spinal and epidural block. As has been stated above, bradycardia in association with a decreased venous return can lead to an acute failure of the circulation.

As in the case of general anaesthesia, expeditious delivery is the most effective way of treating this form of hypotension. Unfortunately, the blood pressure often falls to very low levels before analgesia is adequate for surgery to begin. At the first sign of developing hypotension, the patient should be turned into the lateral position and kept there until analgesia is complete. The operation can commence with the patient in a semi-lateral position or, alternatively, the obstetrical assistant can pull the uterus away from the posterior abdominal wall. Acute hypotension in conscious patients can lead to unpleasant symptoms such as faintness, nausea and vomiting. The patient should be encouraged with the assurance that these symptoms will quickly disappear once the baby is delivered. If bradycardia is present, atropine 0.6 mg intravenously may be given.

The use of vasopressors is to be avoided. As mentioned above, these drugs do not always improve cardiac output even though they elevate the blood pressure. Moya and Smith (1964) found that the routine use of these drugs in spinal anaesthesia was associated with an increase in the incidence of foetal depression. Greiss and Crandell (1965) found that maternal hypotension decreased uterine blood flow during spinal anaesthesia, but this was not improved by vasopressors. Although many of these drugs cause an intense arterial vasoconstriction, they have little or no effect upon veins. Thus, peripheral resistance is increased without any appreciable improvement.
in venous return. In these circumstances, the sudden release of caval occlusion can lead to severe hypertension.

Illustrative case reports.

Case 1. In this case caval compression with maternal hypotension was unsuspected until foetal bradycardia was noted. Correct diagnosis avoided the necessity of Caesarean section.

A 27-year-old para-1 was admitted to hospital with slight antepartum haemorrhage and kept at rest for two weeks until the 38th week of pregnancy. At this time an examination under anaesthesia was performed in the operating theatre to exclude placenta praevia. This was done in the lithotomy position under light general anaesthesia. There was no evidence of placenta praevia and the membranes were ruptured prior to artificial induction of labour with an oxytocin drip. At the completion of this manoeuvre the foetal heart was checked and found to be beating at the rate of 80/min. The theatre staff were alerted to the possibility of an immediate Caesarean section. The maternal blood pressure was noticed to be 85/50 mm Hg. Suspecting a postural effect, the patient was turned on her side. This caused an immediate rise in pressure to 110/70 mm Hg and the foetal heart rate increased to 120/min. Labour was allowed to proceed and vaginal delivery occurred without complication.

Case 2. This case shows the harmful effects of a vasopressor given to raise blood pressure when hypotension was, in fact, due to caval compression.

A 36-year-old para-1 having her second elective Caesarean section for disproportion was found to be hypertensive (systolic blood pressure 70 mm Hg) soon after induction of anaesthesia (thiopentone 250 mg, suxamethonium 50 mg, and nitrous oxide/oxygen with controlled respiration). The radial pulse became difficult to palpate and methotrexate 8 mg was given intravenously. The blood pressure was quickly restored to 140/100 mm Hg. Following delivery of a normal baby it was noticed that inflation of the lungs became progressively more difficult and within 20 minutes frank pulmonary oedema was present with froth issuing from the endotracheal tube. The arterial pressure at this time was 180/115 mm Hg. This patient required tracheostomy and positive pressure ventilation for 24 hours before the pulmonary oedema disappeared. There had been no evidence of cardiopulmonary dysfunction before operation.

In most cases of pulmonary oedema developing during obstetric anaesthesia, inhalation of acid gastric contents is rightly suspected. This explanation is most unlikely in this case, however. Not being an emergency, it may have been assumed that the stomach was virtually empty. Induction was smooth and the larynx and pharynx were seen to be free of gastric contents on intubation. The fact that pulmonary oedema developed immediately after emptying the uterus supports the hypothesis that failure of left ventricle resulted from a combination of vasoconstriction and sudden release of caval occlusion.

Case 3. Continuous epidural analgesia is widely used for the relief of pain in labour. Once the block is effective, the patient is particularly susceptible to postural hypotension. If vasopressors are used to elevate the pressure, severe hypertension may result when the caval compression is relieved at delivery.

A 26-year-old para-1 was given a continuous epidural block to provide a painless labour. Analgesia to T10 was maintained for 8 hours with repeated injection of 1.5 per cent lignocaine. On three occasions it was noted that the systolic blood pressure had fallen to 80 mm Hg and this was restored with methylamphetamine 5 mg intravenously. The last such episode occurred 10 minutes before forceps delivery, and the blood pressure following the methylamphetamine was 130/95 mm Hg. Shortly after delivery the patient complained of headache and the blood pressure was found to be 220/140 mm Hg. Within a few minutes the headache became extremely severe and was only partially relieved by pethidine 100 mg and chlorpromazine 50 mg intravenously. It took 2 hours before the blood pressure became normal and the headache disappeared.

Conclusion

Compression of the inferior vena cava by the weight of the gravid uterus in advanced pregnancy is very common in the supine position. The possibility that this is responsible for changes in the circulation should always be borne in mind by the anaesthetist. Even in the absence of gross hypotension, cardiac output may be seriously affected and sudden collapse may occur at any time up to the delivery of the baby.

If hypotension develops, relief of the inferior vena caval occlusion is urgently required. This may be accomplished by rapid delivery, by repositioning of the patient, or by pushing the uterus away from the posterior abdominal wall. Vasopressors should be avoided.

Following relief of the caval compression, the effects of the augmented venous return must be recognized.

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**REFERENCES**


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