

Anesthesiology

THE JOURNAL OF THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

VOL 20

MARCH-APRIL 1959

NO. 2

HYPOTENSION DURING OBSTETRICAL ANESTHESIA

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WHEN women in the latter part of their pregnancy lie supine obstruction of the inferior vena cava from compression by the uterus may occur.¹ As a result, the venous return to the heart is reduced which in turn lowers cardiac output. Typically, the heart rate increases, the blood pressure drops and the patient assumes a shock-like appearance. This has been called hypotensive syndrome of late pregnancy.¹ Simply turning the patient on her side relieves the obstruction and the circulation is restored.

Just prior to delivery when the parturient is placed in the lithotomy position, the situation again exists for the production of the supine hypotensive syndrome. Since the patient cannot be turned on her side at this time, we have manually displaced the uterus to the left relieving the abdominal venous compression. This has produced prompt results with the blood pressure returning to satisfactory levels by the time subsequent determinations of blood pressure could be made.

Spinal anesthesia (modified saddle block) is the type of anesthesia most frequently used for obstetrics at our institution. The question arose: how often is the hypotension formerly thought to be due solely to spinal anesthesia really a manifestation of the supine hypotensive syndrome? A review of the literature

failed to reveal information of this syndrome complicating obstetrical anesthesia. As a result we studied 600 consecutive obstetrical deliveries with spinal anesthesia to determine the incidence and causes of hypotension.

METHOD

Blood pressure and pulse were recorded in all obstetrical patients having spinal anesthesia. This was determined initially prior to anesthesia and subsequently every three to five minutes. If hypotension developed determinations were made at one minute intervals or less until the blood pressure was restored. Cesarean sections were not included because of the greater extent of anesthesia required. All patients with a systolic pressure below 100 mm. of mercury were considered to be hypotensive. In no patient of this series was the blood pressure found to be less than 100 mm. of mercury prior to anesthesia if left uterine displacement were employed or the patient turned on her side. The methods of treatment were used in the following sequence: (1) Displacement of the uterus to the left (LUD). When this was ineffective the second method was employed. (2) Elevation of the legs or placing the patient in lithotomy position. (3) Methoxamine 2 to 4 mg. was given intravenously if the preceding methods failed to correct the hypotension.

Tetracaine in doses of 4 to 6 mg. with 0.4 to 0.6 ml. of dextrose 10 per cent was used in

Accepted for publication December 4, 1958.
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TABLE 1
RESULTS IN TREATMENT OF HYPOTENSION DURING OBSTETRICAL ANESTHESIA

Reduction Systolic Blood Pressure (mm. Hg)	Number Patients	Pulse Rate 100+	LUD	Leg Elevation or Lithotomy	Vasopressor	LUD + Pressor
Tetracaine						
60	6	0	5	0	1	0
61-70	9	0	8	0	1	0
71-80	31	3	26	1	1	3
81-90	40	4	40	0	0	0
91-100	8	0	8	0	0	0
514 Cases	Total 94	7	87	1	3	3
Piridocaine						
60	0	0	0	0	0	0
61-70	1	0	1	0	0	0
71-80	6	4	6	0	0	0
81-90	4	2	4	0	0	0
91-100	1	1	1	0	0	0
86 Cases	Total 12	7	12	0	0	0
Combined	Total 106	14	99	1	3	3

LUD = left uterine displacement.

Left uterine displacement was tried first, then leg elevation after failure of LUD, and a vasopressor if LUD and leg elevation failed.

The vasopressor was methoxamine 2 to 4 mg. administered intravenously.

514 patients. Piridocaine hydrochloride (Lucaine) 20 mg. administered with 1 ml. of dextrose 10 per cent and 1 ml. of spinal fluid was used in 86 patients. When hypesthesia by pin prick reached the tenth thoracic segment, the head of the table was elevated to prevent further rise of the level of anesthesia. At no time was a vasopressor used prophylactically prior to the administration of anesthesia.

RESULTS

Of 600 patients 106 (17.7 per cent) exhibited a systolic blood pressure lower than 100 mm. of mercury after injection of the spinal anesthetic (table 1). Of the 514 patients given tetracaine 94 (18.3 per cent) developed hypotension. Of the 86 patients given piridocaine 12 patients (14 per cent) developed hypotension. In 81 of the 106 hypotensive patients the blood pressure fell to between 71 and 90 mm. of mercury. Left uterine displacement (LUD) was effective in restoring the blood pressure to a level of 100 mm. of mercury or higher in 99 patients (93.4 per cent). Elevation of the legs (lithotomy)

was effective in only one patient after LUD had failed. In 3 patients (2.8 per cent) vasopressors alone were required and in three other patients (2.8 per cent) the combination of uterine displacement and vasopressors was required. Tachycardia, with a rate in excess of 100, was noted in only 7 of the hypotensive patients given piridocaine and in 7 of those given tetracaine, a total incidence of 13.2 per cent.

COMMENT

The maintenance of a normal blood pressure prior to delivery is necessary to prevent fetal anoxia from inadequate circulation of the placenta. That the supine hypotensive syndrome is related to placental abruption has been demonstrated experimentally in dogs² and in humans.³ The mechanism includes a significant increase in pressure in the pelvic veins producing increased retroplacental pressure.

The supine hypotensive syndrome was by far the most important factor producing hypotension after the onset of spinal anesthesia. Left uterine displacement was effective in

restoration of normal blood pressure in 93.4 per cent of the cases. Vasopressors were seldom required. It was our experience that vasopressors were relatively ineffective in combatting hypotension of this type. Immediately after delivery, the vena caval obstruction was relieved and if vasopressors had been used, their pressor effect became evident. This effect coupled with additional vasoconstriction from the almost routine administration of ergonovine following delivery produced frequent hypertension. This was associated with headache and nausea.

Tachycardia occurred infrequently in those patients exhibiting hypotension. This may be due to the added factor of spinal anesthesia with its sympathetic blockade extending to an undetermined extent above the sensory level. The increased incidence of tachycardia in those patients given piridocaine may be an indication of the less intense block characteristic of this drug. Howard, Goodson and Mengert¹ in their original description did not report the incidence of tachycardia occurring in the supine hypotensive syndrome but merely gave examples of typical cases. There were many patients in this series who developed tachycardia with the systolic blood pressure remaining above 100 mm. of mercury. In many of these the heart rate would slow with left uterine displacement.

Forthman and Adriani⁴ reviewed the blood pressure changes during cesarean sections. They expressed doubt that the development of hypotension following the administration of

spinal anesthesia for cesarean section was due to the compression of abdominal veins since hypotension does not develop during general or local anesthesia and vasopressors were effective in restoring the blood pressure. We have noted, however, that in many cases uterine displacement did correct the hypotension prior to cesarean section. Pressors were unnecessary in these cases.

SUMMARY

Hypotension occurred in 17.7 per cent of 600 patients following spinal anesthesia prior to delivery. Left uterine displacement (LUD) alone was effective in restoring the blood pressure to normal in 93.4 per cent of the patients. The administration of vasopressors was seldom required to correct the hypotension. When ergonovine was administered after a vasopressor had been given, hypertension with headache was likely to occur.

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