expansion of the lung volume can be accompanied by opening of the silent alveolar–capillary units, thus creating temporarily hypoventilated but perfused alveoli or actual venous admixture. Although this could account for the transient nature of the observed Pa_{O_2} change in 17 patients, we are unable to explain why five of our significantly heavier patients did not show the same Pa_{O_2} pattern.

The changes in monitored circulatory variables were minimal and variable when the abdomen was opened. The low-cardiac-output state, however, could not be ruled out as a cause for the Pa_{O_2} change, as cardiac output was not measured. Similarly, transient pulmonary congestion remains a possibility.

The average Pa_{O_2}'s in individual patients varied widely. Our finding, the transient decrease of Pa_{O_2}, further indicates that the monitoring of arterial blood gases is essential for adequate oxygenation of morbidly obese patients undergoing laparotomy.

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Prevention of Spinal Hypotension Associated with Cesarean Section

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Maternal hypotension during spinal anesthesia for cesarean section is a persistent problem. Without preventive measures, the incidence of hypotension (defined as a decrease in systolic pressure to less than 100 torr) can rise as high as 82 per cent,[1] and there is evidence that fetal deterioration can result.[2,3]

Various methods are used in the treatment and prevention of spinal hypotension. Administration of a vasopressor before[1,2] or after administration of spinal anesthesia has been utilized. Greiss and Crandell[4] have demonstrated that intravenous fluid administration improves uterine blood flow. Phenylephrine, although effective in returning maternal pressure to normal, is ineffective in improving uterine blood flow. The pre-spinal infusion of a liter of lactated Ringer's solution has been shown to prevent a decrease in maternal mean blood pressure.[5] Finally, several devices and methods have been advocated to displace the uterus off the inferior vena cava (Colon-Morales,[6] Kennedy,[7] Finley,[8] Eckstein and Marx,[9] etc.[10–12]) to reduce hypotension by improving venous return.

We have studied the effects of fluid loading and left uterine displacement in the prevention of hypotension under three conditions: 1) no fluid loading or left uterine displacement prior to administering spinal anesthesia; 2) fluid loading only; 3) both fluid loading and left uterine displacement by means of a "Sluder" (sustained left uterine displacer[7]).

Materials and Methods

ASA class I patients who were at or near term and had agreed to the use of spinal anesthesia for cesarean section were studied, indications being either repeat cesarean sec-

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tion or cephalopelvic disproportion. There was no maternal disease such as hypertension, diabetes, or toxemia, nor were there instances of fetal complications such as erythroblastosis, prolapsed cord, or fetal distress.

Blood pressure and pulse were recorded, and the intravenous line was checked for competency before administering tetracaine (usually 8 mg). None of the patients received prophylactic ephedrine, and all patients breathed room air. The spinal anesthesia was administered with the patient in the lateral or sitting position, after which she was laid supine. Blood pressures and pulse were checked frequently, and the anesthetic level was determined by pin prick. Apgar scores of the infants were determined 1 and 5 minutes after birth. None of the mothers had folded towels, sandbags, or wedges placed under the right hip.

Group I received neither fluid loading nor left uterine displacement. If hypotension (systolic pressure less than 100 torr) occurred, the slow infusion of 5 per cent dextrose in water iv was stopped and rapid 1,000-ml infusion of 5 per cent dextrose in lactated Ringer's solution was begun, along with tilting the table to the left. Ephedrine was injected iv if these procedures did not reverse the hypotension. Incremental injections were given when necessary. The table was returned to the level positon before operation, and ephedrine used to treat any hypotension that subsequently developed.

The preceding therapy was designed to keep the systolic pressure above 100 torr, ephedrine being used when necessary to raise the maternal blood pressure and return the uterine blood flow to near normal limits.[13,14] Since many of these patients were in early labor, two subgroups evolved: those patients sectioned while not in labor (Group I E) and those sectioned while in early labor (Group I L).

Group II received a 1,000-ml infusion of 5 per cent dextrose in lactated Ringer's solution 30 minutes prior to the administration of spinal anesthesia.[5] If hypotension occurred during preparation of the patient, the table was tilted to the left. If the hypotension persisted, ephedrine was given. The table was leveled again before the incision was made. Ephedrine was given if hypotension occurred beyond this point. As in Group I, two subgroups evolved: those patients not in labor (Group II E) and those patients in early labor (Group II L).

Group III also received 1,000-ml infusion of 5 per cent dextrose in lactated Ringer's solution 30 minutes prior to spinal anesthesia. Each patient was placed in the supine position following administration of the spinal anesthesia and the uterus was pushed to the left with the "Sluder" and held in that position. Hypotension, when it occurred, was treated with ephedrine. The "Sluder" was removed when the uterus was incised. There were the subgroups of patients not in labor (Group III E) and patients in early labor (Group III L).

Results

There were 247 patients, each receiving one of the three treatments of the protocol. As there were patients undergoing section while not in labor and patients in early labor in all three groups, six subgroups evolved. Table 1 summarizes the results.

Group I E. There were 27 patients who were not in labor and received no fluid loading or left uterine displacement prior to spinal anesthesia. Of these, 25 (92 per cent) became hypotensive: 13 were given ephedrine; tilting the table and infusing fluid restored the pressure in the other 12 patients, making ephedrine administration unnecessary.

Group II E. There were 76 patients who received fluid loading only and were not in labor; 43 (57 per cent) of these became hypotensive. This is a significant reduction in the incidence of hypotension compared with Group I ($P \leq .005$§). Thirty-two of the 43 required ephedrine despite the table tilt.

Group III E. There were 53 patients not in labor who received both fluid loading and left uterine displacement by means of the "Sluder." Despite this combination of preventive measures, 28 became hypotensive. This is not a significant reduction in the hypotension rate (52.8 per cent) when compared with Group II E. Twenty-three of the 28 hypotensive patients were given ephedrine.

Group I L. There were 18 patients who were

§ Chi-square test.

TABLE 1. Results

	Patients Not in Labor (Elective Section)			Patients in Early Labor (Primary or Repeat Section)		
	Group I E No Prevention	Group II E Fluid Loading Only	Group III E Fluid Loading Plus "Slinder"†	Group I L No Prevention	Group II L Fluid Loading Only	Group III L Fluid Loading Plus "Slinder"
Number of patients	27	76	53	18	39	34
Hypotension incidence (systolic pressure less than 100 torr)	25/27 (92 per cent)	43/76 (57 per cent) $P \leq .005$*	28/53 (52.8 per cent) NS*	9/18 (50 per cent)	18/39 (46.1 per cent) NS*	5/34 (14.7 per cent) $P \leq .005$*
Number receiving ephedrine, iv	13/25 (52 per cent)	32/43 (74 per cent)	23/28 (82 per cent)	4/9 (44 per cent)	13/18 (72 per cent)	5/5 (100 per cent)
Mean total dose of ephedrine (hypotensive patients who received ephedrine)	21.8 mg	20.1 mg	26.2 mg	27 mg	27 mg	22.5 mg
Mean induction–delivery interval	27.5 min	23.5 min	25.6 min	28.1 min	27.9 min	24.0 min
Number of infants with Apgar scores of 1–6 (1 minute)	0/27 (0 per cent)	3/76 (3.9 per cent)	1/53 (1.9 per cent)	2/18 (11.1 per cent)	0/39 (0 per cent)	0/34 (0 per cent)

* Statistical significance compared with preceding group by Chi-square test.
† Sustained left uterine displacer.

in early labor and received no fluid loading or left uterine displacement prior to the spinal; nine (50 per cent) became hypotensive. Although this group had the same treatment as Group I E, the incidence of hypotension in the group in early labor was significantly lower than that of the group not in labor ($P \leq .005$§).

Group II L. There were 39 patients in the group that received fluid loading only and were in early labor when sectioned; 46 per cent became hypotensive. This is not a significant reduction in the incidence of hypotension when compared with Group I L.

Group III L. Of the 34 patients in early labor who received both fluid loading and left uterine displacement, only five (14.7 per cent) became hypotensive. This is a significant reduction in hypotension rate when compared with Group II L ($P \leq .005$§). Ephedrine was given to all five of the patients who became hypotensive.

The most common anesthetic dermatome level in each group was T6; dermatome levels ranged from T2 to T10, except for the single incidence of a C4 level. Overall, the patients with anesthetic levels of T4 and higher had a significantly greater incidence of hypotension ($P \leq .005$§) than those patients with dermatome levels below T4. There was no significant difference* in the distributions by dermatome levels for the six subgroups. Therefore, we believe the differences in hypotension incidence observed between subgroups to have been due to the presence or absence of preventive measures, since the distribution by dermatome levels was not biased toward any given subgroup.

DISCUSSION

We have shown that the incidence of spinal hypotension can be greatly reduced by fluid loading alone or by the combination of preqsl spinal-anesthesia fluid loading and left uterine displacement, depending upon whether the

patients sectioned are in early labor or not. Despite preventive measures, hypotension still occurs, and ephedrine was necessary in some instances.

It was thought that the problem of spinal hypotension was solved when the report on fluid loading by Wollman and Marx[5] was published, as no significant change in mean pressure occurred in their treated group. In our experience with the method, hypotension still occurred, although the incidence was reduced. The use of mean pressure in their study may have obscured the fact that systolic pressure could fall below 100 torr.

It was hoped that hypotension might be eliminated by combining fluid loading with uterine displacement. Although further improvement resulted, the use of ephedrine was still necessary in many instances. The total dose of ephedrine was probably less than if neither measure had been used. When conservative measures fail, a vasopressor must be given; hence, the high incidence of need for ephedrine in groups III E (82 per cent) and III L (100 per cent).

When no preventive measure was used (Group I E), hypotension occurred in 92 per cent of the patients. This incidence was even higher than the 82 per cent reported by Shnider.[1] Fluids and left uterine displacement did reduce this figure to 52.8 per cent in Group III E ($P \leq .005$§). Surprisingly, Group I L (patients in early labor, no fluids, no left uterine displacement) had a hypotension rate of 50 per cent, lower than that of Group I E (patients not in labor receiving the same treatment) ($P \leq .005$§). The combined use of fluids and left uterine displacement reduced the incidence in the early-labor group (50 per cent) to the very manageable rate of 14.7 per cent ($P \leq .005$*).

The reason patients in labor are less prone to hypotension may be related to the continuance of uterine contractions. About 300 ml of blood are pumped into the maternal circulation with every contraction, supporting the circulation.[15] Another possibility is that descent of the fetal head reduces the development of caval compression and leads to less hypotension.[9,16] On the other hand, aortocaval compression occurs with each contraction (Poseiro effect[17]), so increased caval compression at this time would seem to promote hypotension.

This study utilized only one type of uterine displacement, that recommended by Kennedy.[7] One of the many other methods[6,7,9,10,11,12,18] may be more effective. It has been shown that the brachial-artery pressure is better maintained with the Colon-Morales or Kennedy displacers. The aortic component of aortocaval compression is better alleviated by hip elevation,[8] but this in turn is associated with an increase in A-aD_{O_2}[19]. Although the most effective method of uterine displacement is still not clear, the majority of obstetric anesthesiologists in this country use some type of right-hip elevation rather than a mechanical displacer.[20]

Conclusions

It is not apparent why the fluid infusion alone was as effective as the fluids combined with the "Sluder" in reducing hypotension in patients not in labor, whereas only the combination of fluids and the "Sluder" was effective in patients in labor. Cosmi and Marx[21,22] have attested to the choice of prevention of hypotension over the treatment after occurrence; preventive measures continue to be desirable. We therefore recommend the additive effect of infusion of 5 per cent dextrose in lactated Ringer's solution combined with left uterine displacement when spinal anesthesia is given for cesarean section.

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Serum Cholinesterase Activity Following Pancuronium and Antagonism with Neostigmine or Pyridostigmine

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We observed a patient who had prolonged neuromuscular blockade (more than three hours) following succinylcholine (SCh) when pyridostigmine had been administered an hour earlier to reverse pancuronium-induced paralysis.[1] The patient had responded normally to SCh before pancuronium and pyridostigmine. Both pancuronium[2,3] and anticholinesterase drugs[4] have been reported to inhibit serum cholinesterase (pseudocholinesterase) activity. Conceivably, such inhibition could have contributed to the prolonged SCh paralysis observed in our patient. Since the extent and duration of the reduction of serum cholinesterase activity after administration of anticholinesterase drugs, as used by anesthesiologists to antagonize competitive neuromuscular blockade, had not been established, we measured serum cholinesterase activity following pancuronium administration and reversal with either neostigmine or pyridostigmine.

METHODS

Eighteen adult patients without known hepatic or renal disease, undergoing elective operations requiring neuromuscular blockade, were studied. Preanesthetic medication was with morphine (8–12 mg) and scopolamine (0.4 mg). Anesthetic induction was with thiamylal, 4 mg/kg, plus pancuronium, 0.1 mg/kg, to facilitate tracheal intubation. Ventilation was mechanically controlled and anesthesia maintained with 60 per cent nitrous oxide and fentanyl. Additional pancuronium, 0.01 to 0.02 mg/kg, was administered as indicated by tetanic response to ulnar-nerve stimu-

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