CEREBROSPINAL FLUID PRESSURES DURING LABOR AND OBSTETRICAL ANESTHESIA

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DURING the latter stages of gestation, pregnant women require smaller subarachnoid doses of anesthetic drugs to achieve levels of spinal anesthesia. Assali and Prystowsky injected procaine solutions intrathecally into normal obstetrical patients, first prior to and again 36 to 48 hours following delivery of the infant.[1] They observed that, in the postpartum test, three to four times the quantity of drug was needed to obtain the same sensory level of anesthesia as previously. Changes in cerebrospinal fluid volume and pressure have been considered as causes of this altered response. To evaluate the effects of cerebrospinal fluid pressure changes, continuous measurements of this factor were made throughout labor in 20 normal parturients.

METHOD

When the cervix was at approximately 4 cm. dilatation, a polyethylene catheter (PE 50) was introduced into the intrathecal space between the second and fourth lumbar levels. Pressures were recorded by means of a strain gauge (Statham P23AA) and a Grass polygraph in both the right lateral recumbent and in the supine positions. While in the supine posture, the patients were placed on a foam rubber pad with a cut-out on one side which permitted the catheter to "float" freely at all times. At intervals, the patients were encouraged to bear down actively, both during periods of uterine contraction and of uterine relaxation. They were asked to put their chins on their chests or to lift up one and both legs at right angles to the body. These three exercises were repeated after delivery of the baby. Two of the parturients required no sedation; the other 18 received meperidine or meperidine with promethazine and scopolamine in varying amounts. Ten patients gave birth under spinal anesthesia (5, single injection; 5, continuous block) and 10 under inhalation anesthesia (3, nitrous oxide-oxygen; 2, nitrous oxide-oxygen-trichlorethylene; 3, cyclopropane-oxygen; 2, diethyl ether-oxygen). Seven deliveries were accomplished spontaneously, 12 with the aid of a low forceps, and one was a breech extraction with forceps on the aftercoming head. The duration of pressure recording ranged from 1½ to 6 hours. Nine male patients served as controls. The recordings were calibrated before, during and after the measurements with a mercury manometer.

Presented at the Annual Meeting of the American Society of Anesthesiologists, Inc., New York, New York, October 5, 1960, and accepted for publication December 29, 1960. The authors are in the Department of Anesthesiology, Bronx Municipal Hospital Center—Albert Einstein College of Medicine, Yeshiva University, New York 61, New York.

RESULTS

In the supine position, between contractions, the cerebrospinal fluid pressures ranged from 160 to 350 mm. of water (average 285 ± S.D. 55 mm. of water). Pressures below 200 mm. of water were recorded in two women, and pressures above 300 mm. of water in six. Control measurements from men revealed pressures from 205 to 350 mm. of water (average 295 ± S.D. 50 mm. of water).

In the lateral recumbent position, with the back perpendicular to the table, pressure readings were 70–120 mm. of water lower than in the supine position in both the obstetrical patients and the controls. Within the same patient, the pressure differential between the two positions was the same before and after delivery.

During uterine contractions, elevations in cerebrospinal fluid pressure occurred only when the patient responded to the contraction. When a contraction was depicted as perceptible but painless, and the patient remained tranquil, a rise in pressure was not recorded. When a spinal block was administered to a sensory level above the tenth thoracic level, and the pain of contraction was abolished,

increases in fluid pressure were no longer recorded in spite of normal advancement of labor (fig. 1). When cyclopropane or ether anesthesia was maintained at a plane commensurate with an electroencephalographic pattern of light anesthesia, elevations of cerebrospinal fluid pressure during uterine contractions were also absent (fig. 2). The three types of responses to the pain of uterine contraction most often observed during the first stage of labor were wiggling of hips and shoulders, whining, or tensing of the body backwards (fig. 3). Concomitant rises in cerebrospinal fluid pressure ranged from 110 to 390 mm. of water above normal. The degree of increase was found to be proportionate to the magnitude of skeletal muscle activity which, in turn, depended on the degree of pain perception and response pattern. During the second stage of labor, bearing down efforts and panting types of respirations were encountered in addition. The latter produced rapid large fluctuations in the cerebrospinal fluid pressure (increases of 250 to 350 mm. of water per breath).

During bearing down exercises, elevations in cerebrospinal fluid pressure demonstrated individual variations relative to the force of straining. Within the same patient, there was no difference between bearing down during the first stage of labor—whether with or without a contraction—or after delivery (fig. 4). The average rise in fluid pressure during these

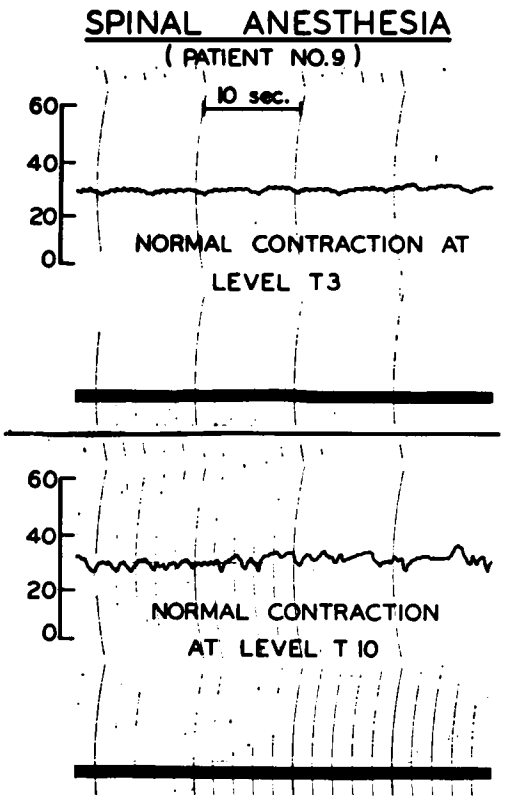

FIG. 1. At a sensory level above the tenth thoracic, patient had no pain and cerebrospinal fluid pressure fluctuations were absent (upper tracing). Levels below the tenth thoracic were associated with pain and cerebrospinal fluid pressure fluctuations (lower tracing). Uterine contractions continued normally throughout (heavy marker line).

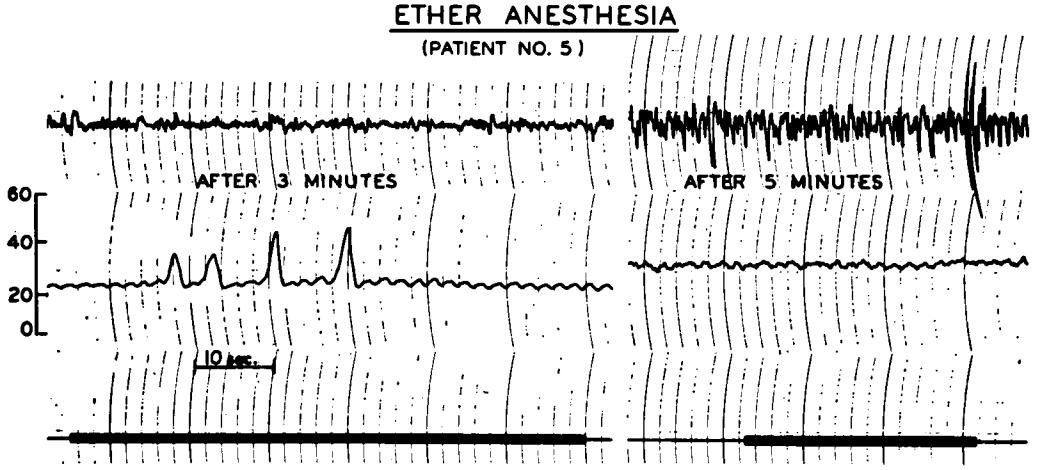

FIG. 2. During high frequency low voltage electroencephalographic activity, the patient's reaction to uterine contraction is reflected in cerebrospinal fluid pressure changes (left tracing). During the phase of mixed waves, uterine contractions are not associated with cerebrospinal fluid pressure changes (right tracing).

bearing down efforts was 530 mm. of water above normal. The male controls exhibited similar elevations during straining (average increase 435 mm. of water above normal). During the second stage of labor, bearing down efforts concomitant with uterine contractions were more intense and, thus, associated with higher cerebrospinal fluid pressures; the average increase at this time amounted to 710 mm. of water above normal.

Putting the chin on the chest caused a moderate rise in cerebrospinal fluid pressure before or after delivery. The average increase was 110 mm. of water prepartum and 115 mm. of water postpartum.

Raising either leg led to insignificant elevations in cerebrospinal fluid pressure at all times. Lifting both legs simultaneously produced somewhat higher levels prepartum as compared to postpartum. The average rise amounted to 260 mm. of water before delivery, to 170 mm. of water after parturition, and to 190 mm. of water in the controls.

During the birth of the infant, the cerebrospinal fluid pressure declined from 15 to 95 mm. of water (average 30 mm. of water) in 14 patients, but returned to the predelivery level within three to five minutes. No change was noted in the other 6 patients. Decreases greater than 50 mm. of water were recorded only four times (1 spontaneous delivery under cyclopropane-oxygen anesthesia, 1 low forceps delivery under spinal block, 1 low forceps delivery under diethyl ether-oxygen anesthesia, and 1 breech delivery under cyclopropane-oxygen anesthesia) (table 1).

Twenty to 40 minutes after delivery, with the patient completely recovered from either spinal or inhalational anesthesia, the cerebrospinal fluid pressure was the same as during the first stage of labor in 5 patients, slightly higher (average 55 mm. of water) in 12 patients, and slightly lower (average 25 mm. of water) in 3 patients. No correlation could be ascertained between the final pressure readings and duration of labor, mode of delivery or type of anesthesia.

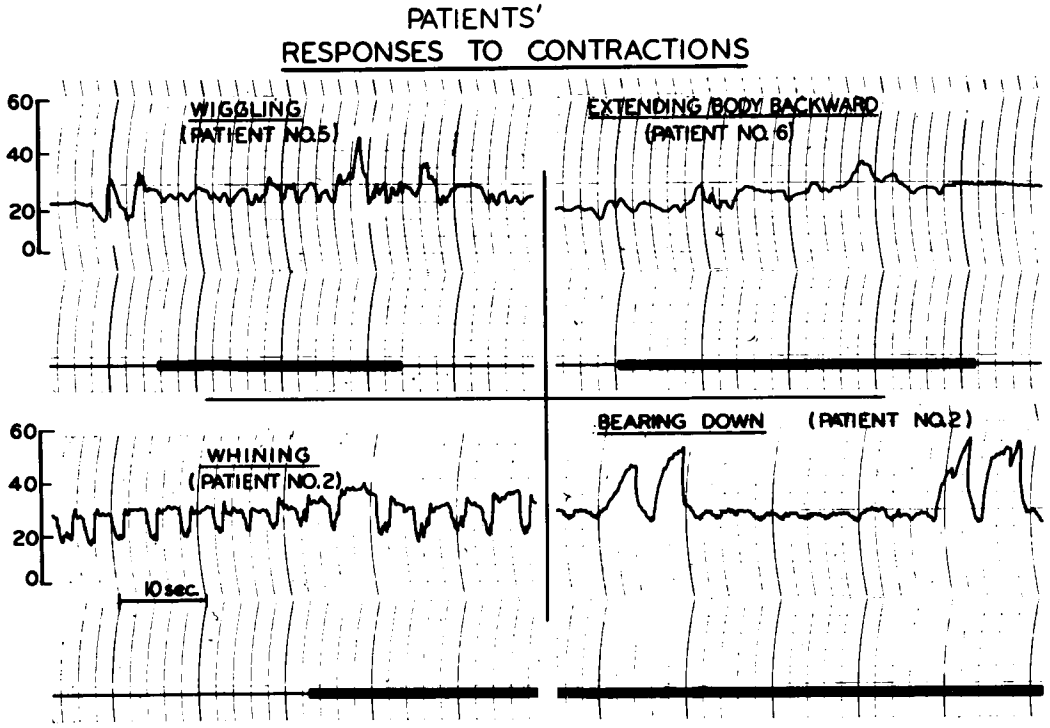

FIG. 3. During the first stage of labor, three different responses to uterine contractions were encountered. "Bearing down" was performed on command.

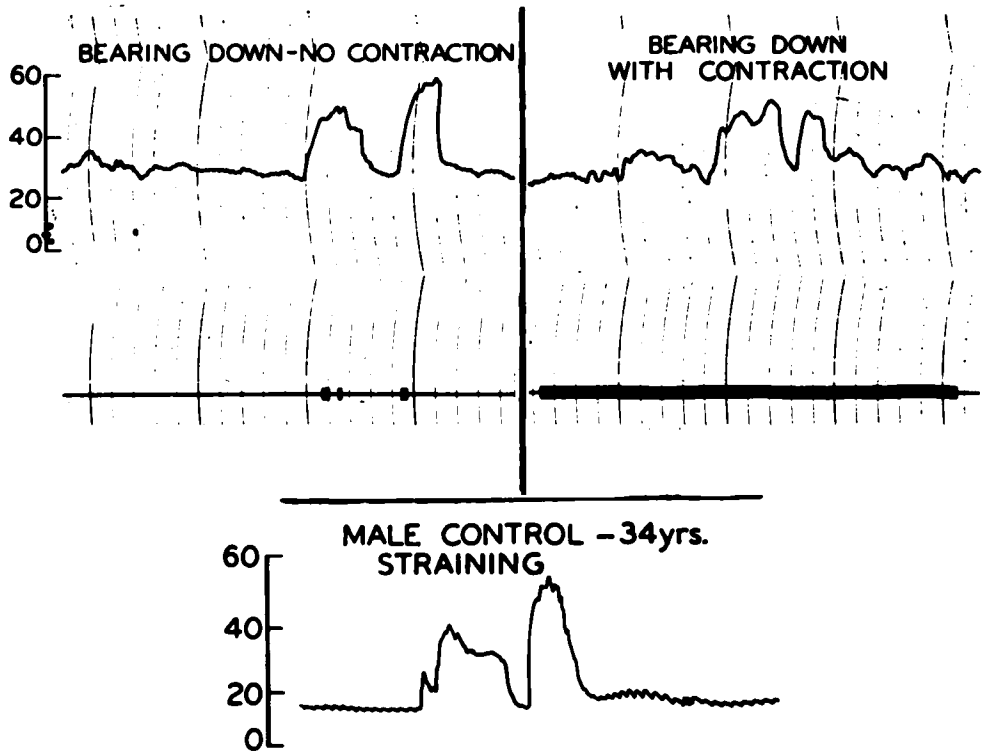

FIG. 4. The influence on cerebrospinal fluid pressure of bearing down efforts with and without a contraction during first stage of labor (upper tracings—patient 3) and in a male control (lower tracing).

DISCUSSION

The amount of cerebrospinal fluid normally present in the ventriculosubarachnoid space varies considerably among individuals. Estimates are based on the quantity of fluid that can be removed by drainage at lumbar puncture or replacement with air at encephalography. Encephalograms were performed on 34 adults in whom the ventriculosubarachnoid space appeared to be of normal size roentgenologically. The amount of fluid removed from these 34 patients varied from 65 to 180 ml. The rate of formation of cerebrospinal fluid is related to changes in the osmotic and hydrostatic pressures of the blood and to variations in the venous pressure. There is no definite evidence as to the volume of fluid formed under normal conditions. On the basis of pressure measurements, it was found that, after removal of small amounts, the fluid reformed in humans at the rate of 0.3 ml. per minute (432 ml. per 24 hours). However, this figure does not allow for vascular compensation to the initial lowering of the pressure nor for the fluid reabsorbed during the period of observation.[2]

Cerebrospinal fluid pressures vary widely in normal man. Merritt and Fremont-Smith measured cerebrospinal fluid pressures in 1,033 patients with normal blood pressure and without evidence of inflammatory or expanding lesions in the central nervous system. With the subjects in the lateral recumbent position, the extreme range of values was 30 to 220 mm. of water with the peak of the distribution curve at 150 mm. of water.[3] In the absence of disease, the fluid pressure is influenced principally by cerebral venous pressure, the osmotic pressure of blood and the CO_2 tension in blood.[4]

The function of cerebrospinal fluid is mainly a mechanical one. It serves as a water jacket for the brain and spinal cord helping to support the weight of the brain, and acts as a lubricant between the cord or brain on one

TABLE 1

SUMMARY OF CEREBROSPINAL FLUID PRESSURE CHANGES IN 20 HEALTHY PARTURIENTS

Parity	Delivery	Anesthesia	Cerebrospinal Fluid Pressure (mm. H_2O)			
			First Stage Labor Supine	Pre-Delivery Lithotomy	Post-Delivery Lithotomy	Fourth Stage Supine
I	Forceps	N_2O-O_2	340	445	405	395
II	Spont.	C_3H_6-O_2	270	475	405	270
II	Spont.	Spinal	270	380	380	350
II	Forceps	Spinal	350	435	405	395
I	Forceps	Spinal	300	325	245	300
I	Forceps	Ether-O_2	350	380	330	350
III	Spont.	Spinal	160	190	190	215
II	Spont.	Spinal	245	270	245	220
I	Forceps	Spinal	300	410	410	380
III	Forceps	Spinal	270	355	325	340
V	Spont.	N_2O-O_2	325	410	410	350
II	Forceps	N_2O-O_2-Trilene	285	410	380	300
I	Forceps	N_2O-O_2-Trilene	300	410	380	285
II	Forceps	Ether-O_2	350	475	380	300
I	Forceps	Spinal	300	310	310	325
I	Breech	C_3H_6-O_2	245	460	370	285
III	Spont.	C_3H_6-O_2	190	310	285	270
IV	Spont.	N_2O-O_2	300	315	315	300
V	Forceps	Spinal	245	315	300	245
I	Forceps	Spinal	340	410	395	395
Average			285	355	345	315

side and spinal column or skull on the other.[2] The cranial vault and spinal column, together with the almost inelastic dura, form a semirigid chamber which is completely filled by the brain and cord, the cerebrospinal vascular bed and the cerebrospinal fluid. Because of its semirigidity and because of the incompressibility of its tissue and fluid contents, this chamber offers great resistance to swift movement or change in volume of any single constituent. There must be an almost exactly equal and opposite effect on the remaining constituents.[5] Changes in the amount of fluid in the ventriculosubarachnoid space occur relatively slowly to protect the central nervous system from sudden alterations in the amount of blood entering it. Adjustments may take place through modified rates of formation and reabsorption of fluid, or both.[4]

The altered response to spinal anesthetic drugs in pregnancy has been explained by the hypothesis that "women at term have in the subarachnoid space a volume of cerebrospinal fluid that is considerably less than that normally present." This reduction in the capacity of the spinal subarachnoid space has been assumed to occur due to venous engorgement in the peridural and subarachnoid spaces secondary to obstruction to normal venous drainage by the mass of the gravid uterus.[6] Such venous obstruction has been demonstrated in the lower extremities by means of radioactive tracer substances which revealed a marked retardation in venous flow rate.[7] Measurements of venous pressure in the supine position have shown no change in antecubital pressure during the course of gestation, but have disclosed a steady rise in femoral pressure from 11 cm. at the beginning of the second trimester to 24 cm. at term. The stagnation of circulation is due entirely to mechanical pressure of the uterus on pelvic veins as evidenced by the fact that the elevated venous pressure declines markedly after removal of the infant and lies somewhat below the average control level during the puerperal period of bed rest.[8] Any significant degree of venous engorgement of the peridural and sub-

arachnoid spaces secondary to obstruction by the pregnant uterus should subside with the birth process at the same fast rate and to the same marked extent as demonstrated by femoral venous pressure tests. Rapid re-expansion of the subarachnoid space should then follow resulting in a sharp and prolonged decrease in cerebrospinal fluid pressure. The results of our study are not in accordance with such findings. We observed the cerebrospinal fluid pressure to be within normal limits in 20 healthy parturients. The average pressure differential between the lateral and supine positions was the same before and after delivery and in the male controls. This would indicate that cerebrospinal fluid pressure is not influenced by the postural relationship of the uterus to the inferior vena cava. During the delivery process, no change in cerebrospinal fluid pressure was recorded in 6 women and insignificant decreases in 10; only 4 patients showed a moderate drop. However, in minutes following parturition, the pressure had returned to its pre-delivery level in all patients. This suggests that changes in respiration and/or cerebral venous pressure are the probable causes for the short-lasting small decreases. We may assume, therefore, that venous engorgement of the peridural and subarachnoid spaces plays but a minor role in changing the effect of spinal anesthetic drugs in advanced pregnancy. This supposition seems to be rational in the light of our knowledge that the cerebrospinal fluid serves to keep "the relationships of the intracranial contents in their rigid skeletal container relatively constant." [9]

Searching for other possibilities to explain the altered response to spinal anesthetic drugs, two thoughts occur. Since the pia mater is a highly vascular membrane, could there be alterations in cerebrospinal fluid circulation due to the increased capillary vasomotor activity which is evidenced late in the third trimester in capillaries of other organs, especially the eye? [10, 11] Or, since cerebrospinal fluid is a "selective transudate" of serum, could there be alterations in its content of solids paralleling the physiological changes occurring in the blood of pregnant women? Little is known about the first possibility but in reference to the second, it is well known that the plasma concentration of the total proteins and of the albumin and gamma globulin fractions is reduced during pregnancy.[12] A similar decrease in cerebrospinal fluid concentration may be present. Papper and Rovenstine, evaluating the extent and duration of spinal anesthesia subsequent to injection of procaine dissolved in spinal fluid as compared to procaine dissolved in human plasma, observed increased duration of sensory anesthesia and decreased motor paralysis when plasma was used as diluent.[13] They stated that this altered response may be due "largely to retarded absorption of the drug from the plasma solution because of the presence of relatively large amounts of protein." Conversely, a lower than normal protein content of cerebrospinal fluid may result in less "binding" of a local anesthetic drug and, thus, a larger than normal amount of "free" drug. We consider that this availability of free drug explains the rapid and inordinate spread of spinal anesthesia in the obstetrical patient. This aspect is being investigated at present.

The level to which a spinal anesthetic drug is distributed following injection at a conventional lumbar puncture site depends on various factors. Macintosh wrote that turbulent currents are induced if an injection is made rapidly into the subarachnoid compartment, and that this motion influences the extent of the spread of the injected solution beyond the site of puncture.[14] In our study, marked fluctuations in cerebrospinal fluid pressure were recorded during painful uterine contractions. These were observed to be due to the patient's reaction to the pain of contraction and not to the contraction itself, since they could be mitigated by any means affording relief of pain. "Spiking" (∧∧∧) increases occurred as the result of wiggling, whining and panting, and rises with a sustained "plateau" (∧⌒∧) were seen during bearing down efforts and during opisthotonus-like extension of the body. We believe that these marked fluctuations in cerebrospinal fluid pressure act similarly to the turbulent currents initiated by rapid injections and that they promote the spread of anesthetic solutions in the same manner. Empirically, it has been recognized that spinal blocks performed during uterine contractions tend to result in high levels of analgesia. The above

explanation appears to be plausible and warrants emphasis on the importance of injecting spinal anesthetic drugs following a uterine contraction in women in active labor.

Summary

Cerebrospinal fluid pressures were electrically recorded throughout labor in 20 normal parturients. Measurements were made in the lateral recumbent and supine positions, during uterine contractions and between contractions while the patients were bearing down, placing their chins on their chests or raising one or both legs. These exercises were reported after delivery. Male patients served as controls. The results showed that resting cerebrospinal fluid pressures are within normal limits in the normal obstetrical patient. The pressure differential between supine and lateral positions is in the same range before and after delivery as in the male controls. Elevations in cerebrospinal fluid pressure noted during uterine contractions are not related to the contraction of the uterine musculature, but are the result of skeletal muscle contractions which occur in response to pain.

During the birth of the infant, the cerebrospinal fluid pressure declined in 14 patients, but returned to the predelivery level within a few minutes; no change was noted in the other 6 patients. After the patient had recovered from delivery and anesthesia, the cerebrospinal fluid pressure was the same as during the first stage of labor in 5 patients, slightly higher in 12 patients, and slightly lower in 3 patients. The implications of these data on the administration of spinal anesthesia to patients in advanced pregnancy are discussed.

This study was supported in part by Grant B-1886 from the National Institute of Neurological Diseases and Blindness, Public Health Service, National Institutes of Health.

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