primiparas and have had uniformly good results. ... I have used this method of anesthesia without preliminary administration of nembutal and find patients are a bit restless, especially if the family is with them during labor. ... I prefer, however, to give nembutal early in the first stage of labor and then use metycaine when the cervix is dilated from 1 to 3 fingerbreadths." 4 references.

J. C. M. C.


"Low spinal anesthesia of 50 mg. of novocain crystals in the fourth lumbar interspace has been used with gratifying results in 200 of a series of 310 consecutive deliveries during the last two years. There were no maternal deaths nor were there any 'close calls.' A few patients complained of a minor headache which was relieved easily with aspirin. The fetal mortality was less than 2 per cent and had no relation to the type of anesthesia used. ... The most opportune time to give the anesthetic was found to be after effacement had taken place. ... Advantages of low 50 mg. spinal anesthesia for delivery are thought to be numerous. As evidenced by this series of cases they are enumerated as follows: 1. Technical difficulties associated with administration were reduced to a minimum. 2. Due to the relaxation of the lower uterine segment and the vaginal canal the length of labor is definitely shortened. 3. There is no interference with the vital mechanisms of the child as seen with general anesthesia. 4. Delivery of the placenta and involution of the uterus are not retarded. 5. Obstetric maneuvers, such as rotation of occiput posterior presentations, are carried out with greater ease and less danger to the child. Breech deliveries are made infinitely less tedious. 6. Blood loss is no greater than with general anesthesia. In most cases there is probably less blood loss. 7. Postpartum complications are reduced to a minimum. In this series of 200 spinal deliveries it was found necessary to catheterize only one patient, while several of the patients who had been given general anesthetics had to be catheterized. There were no postpartum infections in either group. ... It is felt that in selected cases this method is safe." 6 references.

J. C. M. C.


"Thousands of spinal anesthetics have been used without resultant damage to the central nervous system. ... It seems inescapable that any process that produces such extensive clinical involvement of the nervous system as spinal anesthesia must produce in order to be effective, must result also in some anatomic alterations to these tissues in spite of apparently complete functional recovery. Many reports are now available in the literature emphasizing the potential damage of this type of anesthetic. ... It is apparent ... that though the nervous system involvement resulting from spinal anesthesia appears to be reversible and hence harmless, there are instances in which irreversible and progressive changes occur. ... Case report.—Mr. O. A., a sixty-six year old white man, was admitted to the hospital on August 5, 1941, because of hematuria of ten days' duration. ... The general physical examination showed no significant abnormalities. ... The first spinal anesthetic consisted of 80 mg. of metycaine given in order to facilitate a cystourethroscopic examination. ... Recovery from the effects of this anes-
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The second spinal anesthetic of 240 mg. methacaine [sic] dissolved in 4.2 cc. of water was given one week later, at which time a suprapubic prostatectomy was performed. . . . The operation was uneventful and patient left the operating room with the usual flaccid paraplegia but otherwise in good condition. On the following day he was unable to move his legs and examination by the urological resident showed flaccid paraplegia with abolition of all reflexes in the lower extremities. There was hypesthesia from the umbilicus and complete anesthesia to the level of the iliac crests. . . .

"Two days after the second spinal anesthetic . . . the cranial nerves and upper extremities were normal. Lower extremities showed a complete flaccid paraplegia with absent deep and plantar reflexes. There was upward deviation of the umbilicus on contraction of the abdominal recti muscles. Touch and pain sense was impaired from the ninth dorsal to the second lumbar segment bilaterally and all forms of sensation were absent below the third lumbar cord segment. Three days later spinal taps were made at the third, fourth, and fifth lumbar regions. The upper tap brought clear fluid which was slightly yellowish. The middle tap brought xanthochromic fluid which coagulated in a few minutes. The fluid from the lower tap was bloody. Four days later (ten days after the operation) the patient was again examined and now had anesthesia below the first lumbar dermatome on the right, twelfth dorsal on the left and hypesthesia to the ninth dorsal (original level). There was some power of flexion in the toes of the right foot. . . . At this time it was felt that there was a blocking mechanism between the two needles due to arachnoiditis. Treatment consisted of daily intramuscular injections of thiamin chloride and large doses of the other vitamins by mouth. Daily massage and passive motion were carried out and an indwelling catheter was kept in the bladder. Bowel incontinence persisted. Aside from some return of motion in the left toes and slight return of sensation in the right leg there was very little change in the neurologic status during the next few weeks. Spinal taps were again made three weeks later or one month after the operation. . . . It was felt that there had been some decrease in the subarachnoid block and that the patient was suffering from a degenerative condition of the conus and cauda as well as from an arachnoiditis. During the next six months there was improvement in the neurologic conditions. . . .

"Seven months after the operation, the patient's temperature rose to 99.4° and he complained of chills. On several previous occasions there had been transient fever, presumably due to urinary sepsis. That night he became cyanotic, drowsy, and vomited several times. The following morning he had a convolution involving the head, arms and upper half of the body. Temperature rose to 105.2° F. axillary. Examination showed the patient to be comatose and not responsive to painful stimuli. The pupils were equal but miotic and not reactive to light. The right eye deviated outward and the right arm was spastic and held in flexion. No reflexes were obtained. The condition of the lower extremities was flaccid as on previous examinations. The patient gradually failed, temperature rose to 108° F. axillary and death occurred sixteen hours after the convulsion. . . . The brain and its meninges were grossly normal and were not studied microscopically. Upon gross examination the cervical cord was of essentially normal size. In the thoracic levels the meninges were firmly adherent to the cord, particularly in its posterior and anterior aspects. In the upper lumbar regions
there was a fine layer of subarachnoid discoloration, probably due to old bleeding. The mid-thoracic levels of the cord were markedly shrunken and were covered with a thin layer of old hemorrhagic discoloration. The meninges were entirely free in the cervical region and around the cauda equina. Cut sections through the thoracic levels of the cord revealed a complete obliteration of the normal architecture. No divisions could be made out between the gray and white matter. The cord substance was extremely soft from the level of the mid-thoracic to the upper lumbar region. The lumbar segments contained numerous petechiae but the cord architecture appeared to be preserved. The posterior columns in this region presented a whitish discoloration suggesting a tissue necrosis. This posterior column involvement was visible throughout the cord although it tended to decrease in extent and intensity in the higher levels. Microscopic studies demonstrated a most extensive involvement at almost all levels of the cord, these being most marked in the lower thoracic and lumbar regions. . . .

"Most of the alterations [in the cervical level of the cord] were limited to the columns of Goll where the myelin sheaths were markedly swollen and the myelin finely granular. In many areas the myelin sheaths had ruptured to form large vacuolated spaces. The axis cylinders were swollen, fragmented and occasionally entirely absent. Numerous dilated vessels were scattered throughout this partially destroyed tissue. Some of the nerve cells, especially of the anterior horns were swollen and partially chromatolytic. Their nuclei, however, were invariably intact. . . . The appearance [of the upper thoracic level of the cord] was very similar to that in the levels above. . . . In [the mid-thoracic] . . . region of the cord the intramedullary changes were similar to those already described except that scattered petechiae were now present within the gray substance. . . . The most striking alterations, however, were not within the cord substance but within the meninges, especially along the posterior and lateral aspects of the cord. Both the subarachnoid and subdural spaces in these regions were replaced by a relatively acellular, fibrous membrane which was composed of interlacing bands of collagenous fibers. . . . The inner portions of this membrane were loose and reticular in structure and were attached to the thickened pia. . . . In some areas this membrane had become completely hyalinized. The posterior rootlets were enmeshed within this fibrous tissue but were not compressed or altered structurally. In the anterior aspects of the cord this new membrane became narrowed to a thin band, involving only the arachnoid.

"[In the] lower thoracic [levels] the meningeal alterations were very similar to those observed in the mid-thoracic cord level. The cord, however, was much more extensively involved. Both posterior columns were partially replaced by a large central area of softening which was round, irregular, but did not extend to the surface of the cord. The center of this softened area was filled with fragmented cord tissue while its periphery was composed of less severely injured elements which had already begun to be invaded by glia. The gray substance was also extensively damaged. Most of the anterior horn cells were shrunken, rounded and appeared homogenous on staining. Their nuclei were often eccentrically placed or entirely absent. Many of the nerve cells had disappeared or faded out to form ghost cells. Large areas of hemorrhage appeared within the regions of the lateral horns and had produced some destruction of the surrounding tissues. Disrupted and fragmented
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Tissues could be seen adjacent to many of these large hemorrhages. The intramedullary changes at [the upper lumbar] cord level were similar but less extensive than those observed within the lower thoracic regions. The posterior column injury at this level had extended laterally on the left side to almost completely destroy the posterior horn and the adjacent structures. The anterior horn cells were swollen and rounded, showing complete chromatolysis. Large petechiae had produced partial destruction of the gray matter on the right side of the cord. The meninges on the anterior circumference of the cord appeared intact but the subarachnoid space was filled with a fine, rather loose membrane. In the lateral and posterior aspects the entire subarachnoid and subdural spaces were still completely replaced by a very fibrous, relatively acellular membrane whose meshes were filled with a small group of erythrocytes. No inflammatory elements were visible except for a few scattered mononuclear cells. In the lateral regions this membrane was partially hyalinized. The rootlets were completely surrounded but were structurally intact. The arachnoid could not be identified and probably became incorporated within the membrane. The pia was markedly thickened, but the dura remained relatively uninvolved.

"[In the] lower lumbar [level]... the destruction to the posterior columns extended to the surface of the cord. The tissue damage appeared to have extended laterally, completely destroying bilaterally the fasciculus dorsolateralis and the substantia gelatinosa of Rolando. Only a few posterior horn cells could be detected scattered among the fragmentated injured tissues. Some of the anterior horn cells were swollen and chromatolytic with eccentrically placed nuclei. The meninges were not so extensively involved. Posteriorly a fine membrane still filled the subarachnoid space and the enmeshed posterior rootlets showed a moderate involvement consisting of a decrease in the number of axons and some swelling and fragmentation of the myelin sheaths. The involvement at...[the sacral] level was confined to the left posterolateral aspects of the cord where there was a moderate destruction and fragmentation of the tissues. The nerve cells appeared intact. The nerve rootlets for the first time showed most extensive changes. All the posterior rootlets, especially those on the left side, showed almost complete destruction of all their elements, no normal axons or myelin sheaths being present. The entire nerve bundle appeared homogenous. The anterior rootlets were relatively intact. There was no involvement of the meninges at this cord level.... Since this complication is unpredictable the possibility of its occurrence must be kept in mind whenever a spinal anesthetic is given." 23 references.

J. C. M. C.


"In the past year I have seen two cases [of peripheral arterial embolism] treated by spinal anaesthesia and immediate operation. On each occasion when the vessel was exposed at the judged site of the embolus it was found that the embolus had moved peripherally owing to vasodilatation. I therefore believe a better method is to give a spinal anaesthetic in the ward, leave the patient for at least half an hour, and then redetermine the site of the embolus. By so doing it is possible that it may have moved sufficiently far distally for operation to be unnecessary."

J. C. M. C.