

## SEVERE NEUROLOGIC COMPLICATIONS FOLLOWING SPINAL ANESTHESIA: REPORT OF SIX CASES \*

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Received for publication April 9, 1951

SEVERE neurologic complications following routine administration of spinal anesthesia, while noted in the literature, are rare when compared with the hundreds of thousands of such procedures that have been conducted without incident. Nevertheless, the danger of permanent paralysis is ever present, no matter how many the safeguards or meticulous the technic. To demonstrate the possible hazards following the use of spinal anesthesia, 6 cases are presented.

All of the anesthetics were administered during a six month period. Four were given on the surgical service and two on the obstetric service. The two services are physically separated, maintain separate supplies and nursing personnel and employ different drugs and anesthetists. During this period, spinal anesthesia was used in 10 per cent of 1700 surgical anesthetics, these being reserved for teaching cases and for those cases in which the technic was definitely indicated. Approximately 50 per cent of the obstetric deliveries were effected under "saddle" spinal anesthesia with "heavy nupercaine." One of the surgical patients was a teaching clinic case, anesthetized by the Chief of the Anesthesia Section, intended to demonstrate to the senior medical students all precautions necessary to prevent chemical or bacterial contamination of the ampules of anesthetic drugs, the spinal tray and the final anesthetic solution.

Cases 1 to 4 were anesthetized under the supervision of members of the Section on Anesthesia, with strict adherence to the following precautions: (1) storage of ampules in deeply-colored sterilizing solutions; (2) twenty-four hour presterilization of ampules in the same solution; (3) careful inspection of each ampule placed upon the spinal anesthesia tray, of the cerebrospinal fluid immediately after aspiration, and of the final anesthetic solution for foreign bodies, discoloration, or precipitation; (4) washing of needles and syringes with green soap, water and ether before autoclaving; (5) aseptic technic and (6) abandoning spinal anesthesia in the presence of bloody cerebrospinal

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fluid, precipitation of the solutions by the cerebrospinal fluid, or evidence of persistent contact of the needle tip with nerve roots.

Cases 5 and 6 were anesthetized by residents from the Department of Obstetrics, with precautions including colored sterilizing solutions, aseptic technic, and autoclaved spinal anesthesia trays.

The anesthetic drugs consisted of the ordinary commercial ampules of 1 per cent pontocaine hydrochloride, 10 per cent glucose and the usual 5 mg. ampule of nupercaine hydrochloride in 5 per cent glucose ("heavy nupercaine"). The lot number of the nupercaine-glucose ampules could not be obtained. Pontocaine ampules and glucose ampules from the lot used in Cases 1 to 4 were analyzed by the manufacturer and reported to be pure and as labeled. The sterilizing solution consisted of 1 per cent formaldehyde, 1 per cent gentian violet and 70 per cent isopropyl alcohol.

Suspecting the sterilizing solution to be at fault, pontocaine and glucose solutions from the lots involved in the complications were deliberately contaminated and injected into a 20 pound mongrel dog. One per cent pontocaine hydrochloride, 1 cc., 10 per cent glucose, 1 cc., and sterilizing solution, 0.5 cc., were injected at the eleventh thoracic interspace. The anesthetic solution had a definite purplish discoloration. Excellent anesthesia resulted, the animal presenting paresis of the forelegs, intercostal paralysis, profound abdominal relaxation and flaccid hindquarters within five minutes. There was no change after four hours. In six and one-half hours the animal was walking with some spasticity of the hind legs. Examination revealed no abnormalities eighteen hours after the onset of anesthesia. After three months, during which the animal was apparently normal, it was sacrificed and the spinal cord removed. Gross abnormalities could not be found. The dura was thin, white, transparent and nonadherent. The site of puncture could not be found, although it had been thought that the cord substance was pierced at the time of anesthesia. Microscopic sections of the cord were normal.

The tincture of green soap used for washing the needles and syringes was also a possible contaminant. A second mongrel dog, weighing 39 pounds, was given a spinal anesthetic solution consisting of 0.7 cc. of the 1 per cent solution of pontocaine hydrochloride, 0.7 cc. of the 10 per cent solution of glucose, and sufficient (approximately 0.1 cc.) tincture of green soap from a bottle in general use to make the final solution nontransparent and milky-white in color. The site of the subarachnoid puncture was the twelfth thoracic interspace. Paralysis of all four limbs was evident after ten minutes. After 105 minutes, function of the fore-limbs had returned. In four hours and forty-five minutes the animal was walking, although there was slight spasticity of the left hind leg. Eighteen hours after the onset of anesthesia, normal conditions returned and remained for seven weeks. The cord was not examined.

## REPORTS OF CASES

*Case 1.* J. H. P., a 69 year old colored man, was admitted to the surgical service on November 9, 1949, for repair of bilateral inguinal hernias which had been present for twenty-five years. The patient had the appearance of a man about 50 years of age.

On November 12, 1949, bilateral inguinal herniorrhaphy was performed under spinal anesthesia. The anesthetic solution consisted of 12 mg. of 1 per cent pontocaine, 120 mg. of 10 per cent glucose, 5 mg. of 1 per cent neosynephrine hydrochloride, and cerebrospinal fluid to make the total volume 6 cc. The subarachnoid puncture was performed without difficulty, and no paresthesias were elicited. Anesthesia developed to the sixth thoracic segment. The blood pressure, which was 160/100 at the onset of anesthesia, was supported with 50 mg. of ephedrine hydrochloride in the skin wheal, but dropped to 80/60 after eighty minutes, at which time nausea occurred. Following the administration of oxygen by mask and 5 mg. of neosynephrine hydrochloride subcutaneously it rose to 210/140. At the completion of the operation the blood pressure was 200/100. The anesthetist was a surgical resident who had recently completed a period of six months in the Section on Anesthesia at this hospital.

The patient moved his legs by the afternoon of the operative day but complained of pains from his hips to his feet. On the second postoperative day overflow incontinence of urine was noted and a retention catheter was inserted. On this day he first attempted to walk but complained of pain and weakness of the legs and on the fifth postoperative day had an involuntary stool in bed.

The patient was examined by the neurologic resident on the ninth postoperative day because of persistent complaints of pain and weakness in the lower extremities, obstipation and inability to initiate urine flow. The significant findings at this time were: 75 per cent weakness of the lower extremities, absence of the stretch reflexes, bilateral plantar extensor responses upon plantar stimulation, poor anal sphincter tone, absence of the anal reflex, and stocking type of hypalgesia extending to the level of the knees. The upper extremities and cranial nerves showed no abnormalities except the presence of the suck and snout reflexes.

A spinal puncture was done on November 23, 1949. The initial pressure was 110 mm. of cerebrospinal fluid. Inflation of a neck cuff to 40 mm. of mercury caused a rapid rise of the pressure to 450 mm. of cerebrospinal fluid and upon release it fell to the initial level. Removal of 10 cc. of clear, faintly yellow fluid, caused the pressure to fall to 50 mm. of cerebrospinal fluid. The fluid contained 25 lymphocytes per cubic millimeter, 460 mg. per 100 cc. of total protein, was negative to the Kahn test and the colloidal gold curve was 3322110000.

On November 24 the only movements of the lower extremities were of the toes. There was a vague area of hypalgesia to the level of the eleventh segment. By December 1, 1949, difficulty developed with the use of the upper extremities when the patient fed himself and he became drowsy and apathetic. The paralysis of the upper extremities progressed and was complete by December 6. On that date, there were bilateral complete sixth and seventh nerve palsies and a diaphragmatic type of respiration. Lumbar punctures were attempted at several levels on December 5, 1949 and December 6, 1949, but no fluid could be obtained. The patient became weaker and died on December 6, the twenty-fourth postoperative day. Until the time of death there was no change in the sensory abnormalities. Autopsy was not obtained.

In summary, this patient presented the picture of radiculitis followed by subacute ascending myelitis, adhesive arachnoiditis, bulbar involvement and meningo-encephalitis.

*Case 2.* R. N., a 38 year old white man was admitted to the surgical service on November 8, 1949, for the repair of bilateral inguinal hernias.

On November 12, 1949, the patient received a spinal anesthetic solution consisting of 12 mg. of 1 per cent pontocaine hydrochloride, 120 mg. of 10 per cent glucose, and 1.2 cc. of spinal fluid through the third lumbar interspace. Fifty milligrams of ephedrine hydrochloride was injected into the skin wheal. Sub-arachnoid puncture was accomplished on the second attempt. No paresthesias were noted. The anesthesia level rose to the sixth thoracic segment. The course of anesthesia and operation was uneventful except for nausea, which was treated with oxygen inhalations. The blood pressure and pulse remained stable. The anesthetist was the surgical resident who had anesthetized Case 1.

The patient complained of pain in the hips, back and lower abdomen during the first two postoperative days, but was allowed out of bed on the third day and discharged ambulatory on the fourth day. On the tenth day when examined in Surgical Clinic, he was asymptomatic. On November 30, 1949, the patient noted mild weakness of the legs. On December 21, 1949, he appeared in the clinic complaining of severe occipital headaches, nausea and vomiting, and he was admitted to the Medical Service three days later because of intensification of his symptoms.

When examined by the neurologic consultant on December 27, 1949, the patient was confused and disoriented, could hear only when the examiner approached either ear and shouted, and was unable to stand or walk unaided. The degree of weakness in the lower extremities was estimated at 60 per cent. The stretch reflexes were hyperactive and there were bilateral sustained ankle clonus and plantar extensor response to plantar stimulation. There were no sensory abnormalities. Otherwise, the cranial nerves and upper extremities appeared normal to examination. The patient had an overflow incontinence of urine.

From December 24 to December 28, 1949, spinal punctures were attempted daily in various interspaces but no fluid could be obtained. A cisternal puncture was done on December 28, 1949, and revealed an initial pressure of 250 mm. of cerebrospinal fluid. The fluid was crystal clear, contained no cells, total protein of 8 mg. per cent and negative to the Kahn test.

The patient's condition became progressively worse and on January 1, 1950, a retention catheter was inserted when voluntary urination became impossible. On that date, complete deafness, complete paraplegia and a vague loss of pain appreciation to the ninth thoracic dermatome were present. By January 7, 1950, the patient was lethargic, difficult to rouse and did not see well. Bilateral papilledema of 2 diopters was noted. Within the next two days paralysis of the upper extremities and respiratory embarrassment developed. The patient died on January 12, 1950, the sixty-first postoperative day and permission for an autopsy was not obtained.

Although this case resembled Case 1, the course was slower and the encephalitic signs initiated and predominated the neurologic complications. In addition, the involvement of the eighth cranial nerves was of interest, as well as the development of increased intracranial

pressure. The latter was probably due to obstruction of the free flow of cerebrospinal fluid or its absorption.

*Case 3.* C. H., a 26 year old colored woman, was admitted October 30, 1949, for medial meniscectomy of the left knee. History, physical examination and laboratory studies were normal except for mild hypochromic anemia.

November 2, 1949, the patient received a spinal anesthetic consisting of 10 mg. of 1 per cent pontocaine hydrochloride, 100 mg. of 10 per cent dextrose and 1.0 cc. of spinal fluid. The subarachnoid puncture was performed without difficulty and no paresthesias were elicited. This anesthetic was administered by the chief of the anesthesiology section. Anesthesia was established to the ninth thoracic segment, and both the operative and anesthetic courses were uneventful. On postoperative examination the patient had no complaints and was discharged in a wheel chair (non ambulatory) on the ninth postoperative day.

On January 9, 1950 (sixty-eight days after operation) it was learned that the patient had been admitted to another hospital and was receiving x-ray therapy for a supposed pontine tumor. The interim history was obtained from the patient and members of her family. A severe, generalized headache had kept the patient awake the night after operation but there were no recurrences of headaches until December 15. On her arrival at home, both legs were weak and numb, the patient was unable to walk unaided and had no control of bowel or bladder function. In mid-December 1949, her eyes became crossed, and severe generalized headaches occurred. Complete paraplegia and weakness of the upper extremities developed by January 9, 1950, at which time pain in the thighs and legs occurred and continued until her death. When examined on January 27, 1950, she was well oriented and was able to give a good history. She complained of severe intermittent occipital headache and was emotionally labile. Bilateral papilledema of 2 diopters was found with some constriction of the visual fields. She was unable to count fingers at 3 feet. There was complete bilateral paralysis of the sixth nerve. The lower extremities were completely paralyzed and showed marked decrease in resistance to passive manipulations. It was estimated that the weakness of the upper extremities was 50 per cent. All stretch reflexes were absent and there was bilateral extensor response to plantar stimulation. Appreciation of pain and vibration sense was absent to the level of the fifth thoracic segment. Various laboratory studies, including blood counts, urinalysis and roentgenograms of the skull and spine, were normal. Spinal punctures were attempted several times between January 9 and January 27, 1950, but fluid could not be obtained.

The patient was discharged from the hospital on January 30, 1950, and died at home February 15, 1950. She was not observed during the final weeks of illness. Autopsy was not obtained.

This case was similar to the preceding two in that initial myelitis, encephalitis, radiculitis, adhesive arachnoiditis, ascending myelitis and finally increased intracranial pressure were present.

*Case 4.* E. J., a 36 year old colored man, was admitted on October 3, 1949, following severance of the left femoral artery by a bullet. A spinal anesthetic solution consisting of 10 mg. of 1 per cent pontocaine hydrochloride, 100 mg. of 10 per cent dextrose, 5 mg. of 1 per cent neosynephrine, and 1 cc. of spinal

fluid was given. No difficulty was encountered during subarachnoid puncture, and paresthesias were not elicited. The course of anesthesia and a three-hour reparative operation was uneventful. On October 5, 1949, left lumbar sympathectomy was performed under spinal anesthesia, using 7.5 mg. of 0.25 per cent of nupercaine hydrochloride in 5 per cent dextrose (the preparation routinely used for obstetric "saddle spinal"). On October 18, 1949 a left mid-thigh amputation was performed under spinal anesthesia; the anesthetic solution consisted of 10 mg. of 1 per cent pontocaine hydrochloride, 100 mg. of 10 per cent dextrose, 1.5 cc. of spinal fluid and 5 mg. of 1 per cent neosynephrine. The spinal anesthetics were administered by members of the anesthesia section. There were no postoperative complaints and the patient was discharged on November 9, 1949. On November 22, 1949, he was readmitted with a diagnosis of anemia and arterio venous aneurysm in the stump and up to January 10, 1950 received general anesthesia five times for various minor surgical procedures, revisions of the stump and femoral arteriograms.

On December 22, 1949, urinary retention developed, and by January 10, 1950, the patient was incontinent. On examination, January 19, 1950, the wound on the medial aspect of the left thigh had opened, and there were infected bedsores over the sacrum and right ischial tuberosity. The anal sphincter was relaxed with involuntary discharge of feces. The cranial nerves and upper extremities were normal and there was an estimated 80 per cent weakness of the lower extremities. All stretch reflexes were absent and there was no response to plantar stimulation of the right foot. Anesthesia of the perianal, scrotal and penile regions was present. Although spinal puncture was attempted several times at different levels, fluid was obtained on only one occasion and this by aspiration. By this means, 0.5 cc. of clear yellow fluid was obtained on January 25, 1950, and showed 5 lymphocytes and a strongly positive globulin reaction.

The patient continued to be incontinent. The blood pressure ranged from 90 to 100 mm. systolic and 50 to 60 mm. diastolic. The only pain was that produced by coughing and consisted of a sharp lancinating pain throughout the course of the right sciatic nerve. When last examined on February 1, 1951, he had about 50 per cent weakness of the lower extremities, absent stretch reflexes and no response to plantar stimulation on the right side. Anesthesia of the saddle, penile and scrotal regions was present and there was an anesthetic area on the lateral aspect of the right foot. Further examination of spinal fluid was refused.

This case represents the longest survival in this series, a total of more than thirteen months following evidence of the first neurologic sequelae. The complications, clinically, are limited to a myeloradiculitis of the lower segments of the spinal cord and involvement of the cauda equina. In addition, there is evidence of an adhesive arachnoiditis.

*Case 5.* G. J., a 27 year old white woman, multigravida, bipara, was admitted to the hospital at 6:00 p.m. on January 2, 1950 in active labor. The prenatal course of this pregnancy was complicated by severe vomiting in the first trimester and an increase in weight of 39 pounds. The patient's history revealed three previous pregnancies, two of which were carried to term and one which was terminated by spontaneous abortion at two months. All of the pregnancies were complicated by severe vomiting in the first trimester. The patient had previously had spinal anesthesia on two occasions without complications.

At 12:15 a.m., January 3, 1950, an attempt was made to induce "saddle block" spinal anesthesia using 2.5 mg. of nupercaine in 1 cc. of 5 per cent glucose ("heavy nupercaine"). Anesthesia did not result. After twenty minutes, a second attempt was made, using the same dosage. No note was made regarding technical difficulties or paresthesias. Satisfactory anesthesia resulted, the blood pressure remained stable, and delivery was completed at 12:45 a.m.

Thirty-two hours after delivery the patient complained of pain in the neck and back and inability to void. Catheterization yielded 300 cc. of urine. Thirty-six hours after delivery she had two epileptic seizures (grand mal) in rapid succession, with bilateral plantar extension on plantar stimulation as the only resultant neurologic abnormality.

On January 5, 1950, lumbar puncture was done which showed an initial pressure of 150 mm. of cerebrospinal fluid. The fluid was clear but slightly yellow and contained 113 leukocytes, 104 of which were lymphocytes. The Pandy reaction was markedly positive. The total protein measured 1640 mg. per 100 cc., sugar 160 mg. per 100 cc. and chlorides 643 mg. per 100 cc.

The patient's subsequent course was characterized by complaints of headache, low back pain, evidence of progressive paraplegia, cranial nerve involvement, sphincter difficulties, spinal fluid block, seizures, orthostatic syncopeal reactions and finally death on May 2, 1950.

By January 13, 1950, listlessness, apathy intermittent severe bifrontal headaches, lancinating pain in the back, soreness of the calves and brief sharp, stabbing pains in the right subscapular area were present. Spinal puncture at that time showed an initial pressure of 50 mm. of cerebrospinal fluid. The fluid was slightly yellow, contained 33 lymphocytes and had a strongly positive reaction for globulin. All subsequent attempts at spinal puncture were futile.

Weakness developed in the lower extremities by January 10, 1950, and on January 20 there was an estimated 80 per cent loss of strength. Because of urinary retention, catheter drainage was required subsequent to January 28.

On February 5, 1950, the patient's speech was indistinct, nasal, dysarthric and explosive. Nystagmus appeared, as well as left sixth nerve palsy with associated diplopia. Any attempt to assume a sitting or standing posture produced unconsciousness within five seconds. On February 20 she had a tonic seizure. Subsequently, repeated daily episodes of confusion occurred with sudden onset and cessation, lasting two to three minutes. There were no objective sensory abnormalities at any time and her hospital course was afebrile. The patient received, among other drugs, streptomycin, penicillin and dilantin. From January 5., 1950 to March 29, 1950 dicumarol was given in doses of 50 to 100 mg. per day. The prothrombin time varied from 30 to 50 per cent of normal.

The patient insisted on leaving the hospital on April 19, 1950. At home, complete paraplegia occurred, the episodes of confusion increased and the headaches became continuous. She died at home following a seizure on May 21, 1950.

Autopsy was performed sixteen hours after death. Other than mild splenomegaly, suppurative cystitis and pyelitis, the important abnormalities were limited to the nervous system. The surfaces of the brain were somewhat flattened. The arachnoid was opaque and thickened over the inferior surfaces of the cerebellum, medulla, pons and midbrain, and at the base of the brain in the vicinity of the optic nerves and chiasma. The ventricular system, partic-

ularly the fourth ventricle, was dilated and estimated to be three times normal size. The ventricular walls were smooth.

The spinal cord was of average size and shape. In many places there were white fibrous adhesions between the dura and pia-arachnoid, which were so dense that separation was impossible.

Hematoxylin and eosin, myelin, Perdrau and hematoxylin and van Gieson stains were made of several segments of the spinal cord, cauda equina, medulla, pons, midbrain, cerebellum, thalamus, hypothalamus and various cortical areas.

The dominant pathologic changes were an extreme degree of fibrosis of the pia-arachnoid and destruction of portions of many of the roots and of the tracts of the spinal cord. The arachnoid was three to four times thicker than normal and a large amount of collagen, moderate numbers of fibroblasts and an increase in reticular fibrils were demonstrated. Sparse infiltration of lymphocytes and mononuclear leukocytes (histiocytes) was present. The arachnoid and pia were fused into a single thick membrane in many places. The fibrous thickening was equally pronounced over all spinal segments and the cauda equina, and the cellular infiltrates were evenly distributed. In many of the roots, both anterior and posterior, of the lumbosacral segments particularly, all or part of the medullated fibers had disappeared. The roots were small and in the devastated zones there were large numbers of histiocytes, fibroblasts and an occasional lymphocyte. Scattered pigment-filled macrophages and others devoid of pigment but lying in a pink precipitated material were seen. There was ascending degeneration of the columns of Goll and the dorsal spinocerebellar tracts and descending degeneration of the corticospinal tracts, the latter below the upper thoracic levels. In addition, there was a loss of medullated fibers in the peripheral subpial zone of the lumbar, thoracic and cervical spinal cord segments. In these regions the tissue was loose, the astrocytes were increased in number and size and there were a few microglial phagocytes. Some of the anterior horn cells were swollen. The small subarachnoid arteries had thickened cellular walls and somewhat narrowed lumina.

The same fibrous thickening and mononuclear infiltration of the meninges were noted over the medulla, cerebellum, pons and midbrain. There were rare subependymal glial nodules in the floor of the fourth ventricle. Two glial clusters were seen in the inferior olivary nucleus, probably representing degenerated neurones. The Purkinje cells in the most superficial folia of cerebellum had disappeared and were replaced by a thick band of astrocytes. There were no significant changes in the cerebral cortex, thalamus, or basal ganglions except for rare perivascular infiltrates of lymphocytes and mononuclear leukocytes.

The pathologic diagnoses were • (1) chronic cerebrospinal meningitis (chronic adhesive arachnoiditis); (2) meningomyelitis and (3) hydrocephalus.

There were several features of special interest in this case. The pathologic findings corresponded to what had been expected clinically and had been seen to a lesser or greater degree in the other cases. The protean involvement of the central nervous system was striking. The rationale for the use of dicumarol is not too clear. It was given with the view that the pathologic changes were possibly secondary to progressive venous thromboses. The results of its use are not clear out and need further study.

\* We are indebted to Dr. Raymond D. Adams, Harvard Medical School, for his pathologic studies of this case.



*Case 6.* A. K., a 25 year old colored primipara, was admitted to the hospital on April 10, 1950, in active labor, and was taken to the delivery room several hours after admission. Spinal anesthetic which consisted of 2.5 mg. of nupercaine hydrochloride in 5 per cent glucose was administered, using the "saddle" technic, and satisfactory anesthesia was obtained. No note was made regarding technical difficulties or paresthesias.

Routine examination several hours after delivery revealed that the patient had not urinated and had no desire to do so; however, there was an overflow incontinence and upon catheterization a large residual collection of urine was found. This incontinence of urine and feces persisted and required indwelling catheter drainage. About April 19, 1950, there was numbness in the perianal region. Neurologic examination showed only equivocal plantar responses and hypalgesia in the fourth and fifth sacral segments.

Spinal puncture on April 24, 1950, showed a pressure of 95 mm. of cerebrospinal fluid, which contained 1 lymphocyte and a total protein content of 59 mg. per 100 cc.

April 25, 1950, there was an estimated 35 per cent weakness in the lower extremities which increased to 80 per cent by May 2, 1950. There were no complaints of pain or paresthesias.

Spinal puncture was repeated on May 3. The pressure was 70 mm. of cerebrospinal fluid and the dynamics were normal. There were no cells and the total protein was 29 mg. per 100 cc. Further spinal punctures were forbidden by the patient.

Pain appeared for the first time on May 4, 1950, in the region of the fifth thoracic spine. It was continuous, radiated under the right breast, and was accentuated by flexion of the head. By May 11 there was complete paralysis of the left lower extremity, and 90 per cent paralysis of the right lower extremity. Both ankle jerks were depressed and the anal reflex was absent. There was a mild return of muscle strength in the lower extremities during the next few weeks and a return of the ankle jerks with ankle clonus bilaterally. On June 9, 1950 the patient complained of numbness in her legs and at that time there was a vague sensory loss to the level of the eighth thoracic segment. Subsequently, complete paraplegia developed. There was never any evidence of involvement of the upper extremities or the cranial nerves. She was discharged from the hospital on July 12, 1950 and was examined periodically in the outpatient department. The neurologic status remained unchanged. The patient was kept on catheter drainage and was incontinent of feces. The bladder could be emptied by abdominal compression. There was spastic paraplegia and the sensory level was present but patchy in distribution from the eighth dorsal segment caudad. There was marked saddle anesthesia. On December 18, 1950, a vesical calculus measuring 1.5 by 1.0 by 0.5 cm. was crushed.

This case was similar to Case 4 in that there was a limited ascending myelitis, radiculitis and cauda equina syndrome. Although there was no proof of the formation of an adhesive arachnoiditis it can be inferred that this process had taken place. The patient has survived nine months following the onset of neurologic complications. She was given dicumarol therapy from April 28, 1950, through July 15, 1950. What effect dicumarol had on the progression of the signs and symptoms is not too clear.

## COMMENT AND CONCLUSIONS

Six cases of severe neurologic complications following spinal anesthesia are reported. The complications in all cases were similar and consisted of the clinical and pathologic picture of radiculitis, cauda equina syndrome, ascending myelitis, adhesive arachnoiditis, bulbar involvement and evidence of meningo-encephalitis. Death occurred in Cases 1, 2, 3 and 5, in twenty-four, sixty-one 105 and 139 days respectively; Cases 4 and 6 are living, nine and thirteen months after the onset of the neurologic complications. These 2 cases have thus far shown evidence of limitation of the neurologic involvement to the level of the mid-thoracic cord and below. The incubation period, from the time of administration of the spinal anesthetic to the first evidence of spinal cord or radicular involvement, was within twenty-four hours in 3 cases, two days in one case, eighteen days in one case, and indefinite (although less than eighty days) in another. This time interval had no apparent importance in the determination of the severity of the sequelae.

The spinal anesthetics in all 6 cases were administered within a time period of six months, 4 of the 6 within twenty-two days of one another. Four of the complications occurred on the surgical service at a time when 10 per cent of the anesthetics included subarachnoid puncture. Two cases appeared on the obstetric service which, at the time, "anesthetized" 50 per cent of their patients by this means. The two services are separated physically from one another and each has its own personnel, equipment and drugs. Three of the surgical patients received pontocaine, while "heavy nupercaine" was used by the obstetricians. One surgical patient received two pontocaine anesthetics and one of "heavy nupercaine." In two cases, vasoconstrictors were added to the anesthetic solution but it was not thought that their contribution was significant. Glucose was used in all of the anesthetic solutions, in concentrations of 5 per cent in 2 cases and 3.3 per cent or less in the remaining 4 cases. Its role, if any, in the pathogenesis of the complications, was not determined. The sterilizing solution was the same in all cases.

The complications appeared in cases anesthetized by residents in surgery, obstetrics, anesthesiology and by the chief of the anesthesiology section.

Treatment of the complications seemed to have no effect except possibly to prolong life. Dicumarol was administered with the hope of hindering progressive thrombosis in the spinal veins and may hold some promise in the prevention of the development of progressive ascending myelitis and encephalitis, but needs further study.

Attempts to reproduce experimentally the clinical and pathologic picture in 2 dogs were unsuccessful. The pathogenesis of the syn-

drome, like that of many complications of spinal anesthesia, is not understood. It is our impression that contamination of the anesthetic solutions occurred but we are forced to conclude that such "contamination" cannot be detected or prevented with precautions available at the present time and that spinal anesthesia should be reserved for those patients who present definite contraindications to any other form of anesthesia.