PARAPLEGIA AND INTRACRANIAL HYPERTENSION FOLLOWING EPIDURAL ANESTHESIA

REPORT OF FOUR CASES

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There are, now, many reported cases of irreversible neurologic syndrome caused by spinal (subarachnoid) anesthesia [13, 21, 22, 25, 32] and some about the perhaps less frequent complications of epidural anesthesia [3, 4, 5, 28].

A very complete revision of 32 verified cases of arachnoiditis post spinal anesthesia was made by Schwartz and Bevilacqua [25]. Neurologic complications were present in 17 of 11.574 spinal anesthesia [19] in 84 cases of 10.440 [23] and in 37 of 10.098 anesthesias [7] in three different series, but only the last two series of patients were followed up. Severe, irreversible situations, however, are much less frequent and occurred in no patient of these three series.

Hellmann [10] reports no neurologic complication in 26.127 cases of epidural anesthesia in his obstetrical series, and two cases of severe neurological complications in his surgical series; again, follow-up of patients is not specified. Bonica et al. [2] describe one case of serious neurologic complication among 3.637 peridural blocks.

Even in more recent papers, the pathogenesis of neurologic sequellae of both epidural and subarachnoid anesthesias is discussed and not clear [27].

Chemical contaminants were experimentally shown [12] to be able to produce severe arachnoiditis and clinical paraplegia in monkeys and could be the causal agent in some clinical situations. Increased concentration of the anesthetic agent was also demonstrated to produce, in experimental animals, neurologic syndromes similar to the observed in clinical situations [16, 17]. Treatment of neurologic sequellae is usually unsuccessful and most cases are irreversible.

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During the last four years, six patients with severe neurologic disabilities following epidural anesthesia were seen at the Institute of Neurosurgery, Porto Alegre. The present report concerns four of these patients in whom good information was obtained about the anesthetic procedure and close observation about evolution was possible.

REPORT OF FOUR CASES

CASE 1 — F.Z., white, male, age 63, was in good health until July 24, 1970, when operated on for inguinal hernia, under epidural anesthesia. He was premedicated with Valium 10 mg and put in the left lateral position. Puncture was made in L2-L3, by the resident, under supervision of the anesthesiologist, with T-16 needle. Ascertainment of epidural space was reported to be difficult, but no arachnoid perforation was reported. Epidural tray was cleaned in water and autoclaved. The anesthetic agent was Lidocaine 2%, 400 mg, with adrenaline, 1:160.000. The operation was uneventful and no hypotension occurred during surgery. Immediately after surgery, the patient felt tired and slept, just to awake, 2 or 3 hours later, with very severe pains in both legs. He required several analgesic injections during the next 10 hours and next morning he complained of numbness in both legs and chiefly in saddle area, retention of urine, constipation and weakness in both lower limbs.

At this time, neurologic examination showed reduction of all kinds of sensation from L3 to L5 and anestthesia from L5 down to S5, bilaterally absent ankle jerks, extensor plantar responses; motor strength was reduced in extensors of toes and feet and external rotation of feet. No fasciculations and no pains. Lumbar puncture revealed colourless fluid under normal pressure, containing 120 cells per ml, and 300 mg of protein per 100 ml. Bacteriologic examination was negative. On the assumption this was an aseptic spinal cord inflammation secondary to epidural anesthesia, oral corticoid treatment was started with Decadron 12 mg a day, progressively reducing dose until December 1970. In August, 7, 1970, cerebrospinal fluid (CSF) examination showed: protein 149 mg per 100 CU. MM., 15 cells per ml, increased albumin/globulin relation in Cellogel proteinogram. The patient's situation improved slowly during the next four months: on the beginning of September the urethral catheter could be taken off and he was able to pass urine with some difficulty, by abdominal compression. His gait improved and he was able to drive his car. Sensation was normal to pinprick to L5, and reduced from L5 to S5. Vibration was reduced at the ankles and proprioception showed some errors at the toes.

In December, 1970, CSF contained 5 cells per CU. MM. and 120 mg protein per 100 ml. CSF culture was negative, E.S.R. 8, normal hemogram and serum proteinogram; irrelevant lumbar and sacral spine X-Ray. Urine infection was present since August and not yet cured. As his situation was stationary the patient decided to see another neurologist, by whom Pantopaque myelography was indicated and done, and later said to have shown "cord atrophy".

Comment: acute lumbo-sacral myelopathy with inflamatory findings in CSF, occurring immediately after epidural anesthesia. Partial improvement during next four months.

CASE 2 — M.B.R., 27 years old, white female, was in good health until September 1970 when she had epidural anesthesia for her second vaginal delivery. Pregnancy was normal. Puncture made by anesthesiologist, in lateral position, L3-4, first tap, 10-T needle, epidural space ascertained by Dogliotti technique. During puncture, patient reported sensation "like shock in the whole spine", but

no blood or CSF came. Epidural tray prepared by autoclavation, including Lidocaine. Syringes and needles previously washed and cleaned with detergent and then washed in water. Lidocaine 1.5%, 300 mg, with adrenaline 1:320.000 injected in 5 minutes. Immediately on being turned to supine position, patient was unable to move legs and trunk, and felt numbness up to upper limbs. She also reported difficulty in swallowing and got extremelly anxious. Blood pressure 90 x 60, no respiratory depression. Three to four hours after anesthesia, she complained of pricking and tingling sensations, from head down to lower limbs, immediately followed by very intense pains in both legs and trunk, during the next 6 hours.

Some days later her legs seemed to be weak and she fell frequently. By November, she started noticing numbness in her feet, which increased very slowly during the next four months. In February, 1971, she had ankle clonus and difficulty in passing urine.

She was first seen in March, 10, 1971. Neurologic examination showed: marked reduction in flexion and extension of the toes and internal rotation of the feet. Slight reduction of external rotation and dorsal flexion of both feet. Bilateral extensor plantar responses and ankle clonus and +++ symmetrical knee jerks. Upper superficial abdominal reflexes present and lower abdominals absent. Pinprick sensation markedly reduced from T10 to L4 but spared in L5 and perineum. Vibration reduced in both knees and ankles and proprioception absent in the toes.

The situation was indicative of a myelopathy with upper level in T10, but sparing of pain sensation from L5 drown suggested a central cord lesion, possibly related to anterior spinal artery obstruction. A lumbar puncture was performed but no fluid obtained. A myelography with positive contrast was done by suboccipital route and showed a partial stop in T9 and complete stop in T10. Suboccipital CSF was normal. Chiefly in view of the posibility of a preexisting central cord tumor, a laminectomy was performed in March, 1971, and disclosed an intense proliferative arachnoiditis, containing some whitish cysts of fluid; cord was narrowed, pale, and completely impossible to be liberated from the arachnoid. Both cord and roots were enmeshed by a thick, fibrous tissue. After surgery, patient's neurologic situation continued to deteriorate, slowly but progressively. She was given oral Prednisone, starting with 60 mg a day and reduced slowly to 10 mg in June.

In August 1971, she was taken to Boston (U.S.A.) and seen at the Mass. General Hospital. At this time she presented a complete spastic parapleglia with bilateral ankle and knee clonus, extensor plants and paralysis from the lower abdominals down; sensory anesthesia with level in T10 for all modalities of sensation. She was still able to control bladder and bowels. Suboccipital fluid showed normal chemical and cytologic examinations. In view of the progressively worsening situation, methyl prednisolone acetate (Depo-Medrol) 40 mg, was injected at the suboccipital level in August 27, 30 and September 3. At that date, only 1 cc of faintly turbid CSF could be aspirated and there was a question of loculation with adhesions at that point. It was felt that further instillations of Depo-Medrol were not likely to be of value.

After completing paraplegia with sensory and motor level in T10 the patient's situation stabilized and is much the same after 2 and 1/2 years. She is still able to control urine. She developed urinary infection, controlled with difficulty during last year.

Comment: slowly progressive thoraco-lumbar arachnoiditis and consequent myelomalatia, with level in T10, stabilized after complete paraplegia; insidiously developped after epidural anesthesia in which probable total spinal anesthesia occurred.

CASE 3 — L.M.M., 25 years old, white female, was in perfect health until December 1972, when received epidural anesthesia for cesarean delivery. This was uneventful. Premedicated with Valium 10 mg. Puncture in L3-L4, with number 10 Crawford needle. Patient seated. Identification of epidural space by loss of resistance. No back flow of fluid when needle turned to the four angles. Epidural tray prepared by autoclavation; syringes and needles previously washed with detergent and then with water. Lidocaine 2%, 360 mg, with adrenaline 1:160.000, was slowly injected. On being turned to supine position, patient reported numbness in both lower limbs. As hypotension was observed, she was put in Trendelemburg position and treated with Metaraminol and oxygen. A few hours after cesarean, she had intense pains in both legs and low back, which alleviated only after the third analgesic injection.

She was well for about thirty days, when she started feeling pins and needles, and then numbness in both feet, which gradually involved her legs and thighs. In beginning of March she started with daily, intense headaches, accompanied by sweating and dizziness. At about the same time she noticed gait disturbances and loss of power in both lower limbs. No bowel or bladder disturbance. For 10 days before admission, headache episodes were also accompanied by transitory bilateral blindness lasting 1 to 3 minutes.

She was admited to the Department of Internal Medicine, Hospital de Clínicas, Porto Alegre, on March, 27, 1973. She had no systemic complaints or disturbances; weight 73 kg. Neurologic examination: slow, horizontal nystagmus to both directions; isochoric pupils reacting normally. Hypoesthesia for pain and temperature in the region of the right maxillary nerve. Power markedly reduced in both lower limbs, chiefly on the right, where there was complete paralysis of extension of toes and foot and of external rotation of the foot; loss of power was less marked in the same groups on the left; flexion of both legs and thighs was bilaterally reduced whereas extension was normal. Sensation was reduced for touch and pain from T4, and absent from T6 down; vibration absent from T4. Deep reflexes in the upper and lower limbs +++ bilaterally; extensor plantar reflexes bilaterally. Intense bilateral papiloedema was present.

Evidently, this was a gradually progressing myelopathy, with a more severe lesion in T4. The history made clear that it was an ascending lesion, and she had signs of involvement of intracranial structures and intracranial hypertension as well. The situation was very suggestively related to epidural anesthesia and ascending arachnoiditis was an obvious diagnosis. Lumbar puncture was performed but no fluid obtained. Cysternal puncture was then made, with patient in seated position, and clear, colourless fluid showed normal cytological and chemical findings. Other laboratory examinations: hemogram, E.S.R., serum proteinogram, were normal. V.D.R.L. and tuberculin test negative.

The decision was taken to give her both oral and intra-thecal steroids. Oral Prednisone was started with 60 mg a day, and 40 mg of methyl-prednisolone acetate injected suboccipital on the third day in Hospital. Headache stopped immediately after the first injection, and so the episodes of transitory blindness; the nystagmus, and papilloedema were absent by the end of the first week, but there was no change in the motor and sensory findigs. By the end of the second week, headache and transitory blindness reappeared and a second injection was made of 40 mg methyl-prednisolone: the symptoms cleraed now for only three days. A third injection was made but useless. Her situation deteriorated rapidly during the fourth week and in April 23 she was transferred to the Neurosurgery Institute. At this time she presented a complete paraplegia with sensory level in T5 and bilateral papilloedema with multiple hemorrhages in both dysks and retinas. Episodes of bilateral blindness recurred several times a day, lasting sometimes for more than 5 minutes; fundi examination showed no additional abnormality in

these occasions. A carotid angiography was done and showed symmetrical hydrocephalus.

A ventriculo-peritoneal shunt was placed in April 27, with a medium pressure Raimondi catheter, coupled to a Portnoy ventricular catheter. Headache disappeared the same day and she had no more episodes of blindness so far. Ophtalmoscopic examination in May 16, showed "almost complete reabsorption of retinal haemorrhages and marked reduction of protrusion of the dysks". Normal fundi in June 6.

In May 5, she started with movements in left foot; intensive physiotherapy was started after ventriculo-peritoneal shunt was placed. By August 4, she had flexion and extension of both legs and thighs. In October 19 she was able to walk with canadian canes and long braces in both lower limbs, and in December 26 she was able to walk 10 steps without canes and braces. Motor power, by this time, was only reduced bilaterally in extension of the toes and in dorsal flexion and external rotation of the feet. Vibration was felt normally in both legs, but pain sensation was still reduced, although present, from T6 down. Ankle clonus was now absent. She stopped oral Prednisone in October. In April 23, 1974, patient is walking alone, without canes, and motor power is practically normal.

Comment: slowly progressive ascending myelopathy with chief level in T4; increased intracranial pressure and episodes of transitory blindness. Ascending spinal and intracranial arachnoiditis starting one month after "epidural anesthesia". Short lasting improvement after intra-cysternal methyl-prednisolone injection, followed by deterioration until complete paraplegia and bilateral choked dysks with hemorrhagic foci. Immediate disappearance of intracranial hypertension and episodes of transitory blindness after ventriculo-peritoneal shunt. Steady, marked progressive recovery from paraplegia during next 12 months after ventriculo-peritoneal shunt.

CASE 4 — H.M.M., 25 years old, white female, was in perfect health until October 21, 1973, when received epidural anesthesia for vaginal delivery. This was her first pregnancy and first experience with epidural anesthesia. Delivery was uneventful. Puncture was done in L3-L4 interspace, with a T-16 needle, by a midline approach. Epidural space has been located after one attempt, and confirmed by Gutierrez method. Xylocaine 1.2% with adrenaline 1:200.000 20 ml has been injected, after negative aspiration for fluid. About 8 min. later the patient was markedly dyspneic and felt lower limbs paresthesias. She received oxygen, there was no need for vasopressors and the delivery was uneventful with a newborn in excellent conditions.

Less than one hour after delivery, patient started feeling pins and needles in both lower limbs, and then very intense pains irradiating from the pelvic girdle to lower limbs. The pains were only alleviated by the third injection of Propoxiphene, about 8 hours after delivery. Influenced by the knowledge of other cases, the anesthesiologist decided to call a neurologist at this time. Fourteen hours after epidural injection, the patient was well and neurologic examination was normal, showing not even meningeal signs. Lumbar puncture showed a faintly turbid fluid under normal pressure.

In view of the turbid aspect of the fluid, this was allowed to drop until 75 ml were taken out, stopped by patient's headache; 40 mg of methyl-prednisolone were dissolved in 10 additional ml of fluid and reinjected slowly.

Next six days she was submitted to 5 lumbar punctures, until normal fluid was obtained. Apart from transitory hypotension headache during punctures, patient was well and had no symptoms. Two additional injections of Depo-Medrol were done during this week (Table 1).

	Oct.	November						December					January				Feb.	Apr.
	22	23	24	25	27	28	30	1st	3	5	21	29	1st	6	13	30	15	15
CSF cells		80	32	26	7	1.4	0.3											
% lymphocites		46	62	64	83													
mg protein		173	143	118	68	26	91	68	58	60								
glucose		87	84				75											
chloride		605	610				710											
CSF pressure		28	22				30	14	22									
CSF taken out		75	50	40	35	30	6	10	20	20								
Depo-Medrol inj.		40	40		20			40	40									
Radicular pains	+++																	
Headache						+++	+++	+++	–	–	–	+++	–	–	–	–	–	–
Papilloedema						+	+	+		–	–	+++	+++	+	–	–	–	–
Ophtalmoplegia												+++	+++	++	+	–	–	–
Paraplegia													+++	+++	+++	+++	++	+
Vent-perit shunt																		

Table 1 — Case 4: clinical-CSF correlation.

The patient was sent home in October 29 and was well until November 27, when started with headache, chiefly occipital, sometimes accompanied by dizziness. Neurologic examination was normal and so were ocular fundi. Two days later, headache was strong and incipient bilateral papilloedema was seen. Lumbar puncture showed clear fluid under pressure of 30 cm water. Methyl-prednisolone was again injected two times, 40 mg each, until normal fluid came. Both headache and papilloedema cleared immediately. She was kept under oral Dexamethasone 4.5 mg a day, until December 21, when she stopped Decadron.

She was admitted to our Service as an urgency, on December 29, after being treated at home, by another physician, for some days. During this time, headache reappeared and was then accompanied by vomiting, lassitude, numbness and weakness in both legs, trunk, and, in rapid progression, urinary retention, paraplegia, tremors in both hands, diplopia, respiratory difficulty and clouding of consciousness. On examination, she had bilateral sixth nerve and unilateral right, complete third nerve paralysis, with mydriasis and ptosis; clouded consciousness, diaphragmatic breathing and complete flacid paraplegia with sensory level probably in T2. Severe bilateral papilloedema with numerous, large, hemorrhagic foci.

A ventriculo-peritoneal shunt was urgently placed, the same day, with medium pressure Raimondi catheter coupled to a Portnoy ventricular catheter.

From next day she had no more headache and was in perfect state of consciousness. Papilloedema disappeared during next two weeks. In January 28, ocular movements and pupils were normal, and she was able to feel pinprick down to L2 and to localize tactile stimuli in both legs and feet. In February 23 she had flexion and extension of both feet and toes, but proximal muscles of lower limbs were still paralysed. In April 20, power was slightly reduced in flexion and extension of both feet, and moderately reduced in flexion and extension of both legs. Sensation was normal. In Sept. 13, patient walks alone, without canes.

Comment: acute aseptic meningitis immediately following "epidural anesthesia". Treatment with repeated lumbar punctures and intra-thecal injections of Methyl-prednisolone acetate until normal fluid obtained. Increased intracranial pressure, one month later, normalized after a second series of lumbar punctures and intra-thecal Methyl-prednisolone. Acute increased intracranial pressure, oculomotor disturbances and complete paraplegia in T2 one month later. Complete normalization of intracranial pressure and partial recovery of paraplegia after ventriculo-peritoneal shunt.

DISCUSSION

In probably three of our patients (cases 2, 3 4) the high level of anesthesia obtained is indicative of spinal (subarachnoid) anesthesia [2]. The rapid onset of neuromuscular weakness and sensory level to a very high dermatome could be interpreted as possible misplacement of the anesthetic solution into the subdural space [26]. This could not be excluded as no fluid was obtained in these instances, but would be hard to demonstrate.

The immediate neurologic deficit in case 1 is suggestive of radicular — cauda equina — lesion, but the presence of unequivocal extensor plantar responses made it clear that a cord lesion was also present, in L3.

Ischaemic lesions of the cord are a likely cause of some of the neurologic problems occurring after epidural anesthesia. In some cases [5, 28] it is sugges-

ted that the combined vasoconstrictive effect of the adrenaline and the hypotension produced by the epidural blockade, by occluding the radicular vessels to the spinal cord, are sufficient to deprive the cord of an adequate blood supply. Cord ischaemia is certainly involved in many other cases and possibly in most cases of myelopathy secondary to spinal (subarachnoid) anesthesia. Vascular changes in radicular and anterior spinal artery were present in most cases whose pathologic material was reported [25]. Intrathecal adrenaline was demonstrated to cause paraplegia in dogs, but in doses corresponding to 2,5 mg per kg [4]. It is possible that the immediate myelopathy observed in case 1 was caused by cord ischaemia. A vascular — anterior spinal artery — syndrome, was also present in our case 2.

CSF cytology was normal in the case reported by Urquhart-Hay [28] and not reported in the patients of Davies and Solomon [5], Braham and Saia [3] and Bonica et al. [2]. CSF examination in our cases 1 and 4 (the two who were examined in the first 24 hours after epidural anesthesia) are characteristic of an acute inflammatory reaction, a chemical meningitis.

Many of the reported cases of arachnoiditis post spinal (subarachnoid) anesthesia are atributed to the presence of contaminants in the spinal anesthetics [18, 22, 30]. Contaminants were proved [12] to be able to cause severe arachnoiditis in experimental monkeys and the lesions and conditions of anesthesia were very similar to those in human cases pathologically studied.

Among the contaminants possibly responsible for such reactions, there are some detergents commonly used in many Hospitals for cleaning of surgical and anesthetic material. As long ago as in 1954, Paddison and Alpers [22] emphasized that caution should be exercised in the use of detergent cleaned apparatus for intrathecal injections and that detergent materials should not be employed in the preparation of those items used in mixing and injecting spinal anesthetic agents. In two of our patients (cases 2 and 3) detergents were certainly used in preparation of the peridural tray. They were probably not used in case 1. If detergents were the responsible agent of chemical meningitis and arachnoiditis after spinal anesthesia, they would certainly be able to cause the same reactions after introduction of a larger amount, even in epidural space. Much of the anesthetic material is diffused, through the arachnoid villi, to the subarachnoid space [26]. In fact, there is not an experimental study to demonstrate diffusion of detergents through arachnoid villi. Actually, at these times, detergents should never be used for preparation of anesthetic trays and disposable syringes and needles should be used whenever possible.

In our case 4 we are sure that no detergent or any other possible contaminant was used in the preparation of the anesthetic material.

Bonica et al. [2] report that in 19 cases of epidural anesthesia a subarachnoid block was produced unintentionally, but in 17 of these cases the spinal block resulted from the test dose and was recognized before the full amount

of solution was injected. In one patient in whom massive subarachnoid block occurred, serious neurologic sequelae developed; soon after the injection, the patient began to complain of severe lancinating pain.

Tissue reactions have been observed for the usual local anesthetics in concentrations only somewhat higher than those recommended for anesthesia [15, 24]. Experimentally, paraplegia was provoked in animals by increased concentrations of anesthetic agents [16, 17].

There would be strong evidence that increased amount of anesthetic in the subarachnoid space is the cause of cord lesion in our case 4. Even if we cannot rule out the action of detergents in cases 2 and 3, the similarity of our four cases suggests that they were possibly caused by the intrathecal (subarachnoid or subdural) injection of large amounts of Lidocaine.

Dural perforation is not so rare on epidural anesthesia, and this would still leave the possibility for an hiperergic reaction to Lidocaine in the patients where the reaction occurs. All four anesthesias were done by respectful, experienced anesthesiologists, and in no case was CSF obtained before injection of the anesthetic.

Even if the test dose is injected, the possibility of neurologic complications to develop still remains, and its early detection would be desirable if therapeutic measures were able to change the course of the disease.

All four patients had very intense pains, that must be interpreted as radicular, in the first 8 to 10 hours after anesthesia, and these ceased with analgesics, not to return. In both patients — cases 1 and 4 — who had a lumbar puncture performed during the first 24 hours, CSF was typical of an aseptic meningitis. It could be supposed that the radicular syndrome of the other two patients was also the expression of an aseptic meningitis.

In at least one patient (case 4) and, with all probability, also in cases 2 and 3, the disease run a three stages course: aseptic meningitis, followed by an asymptomatic period, and then the adhesive, fibrous, thickening of the arachnoid, causing obstruction to CSF flow and compression of cord vessels.

Intracranial subarachnoid obstruction occurred in two of our cases. It is very much rarer than the spinal lesions. Thickening of the leptomeninges of the base of the brain was demonstrated in some cases [15, 22, 30] after spinal anesthesia. Because of the severity of intracranial hypertension, carotid angiography instead of pneumoencephalography was made in case 3 and symmetrical hydrocephalus was shown. No radiologic demonstration of hydrocephalus was considered necessary in case 4 in view of the urgency to reduce intracranial pressure and of the obvious clinical diagnosis. In both cases, immediate reduction of intracranial hypertension syndrome by ventriculo-peritoneal shunt was demonstrative that it was caused by hydrocephalus.

Howland at at.[11] studied the effect of intrathecal corticosteroids in preventing the development of paraplegia and arachnoiditis after experimental injection of Pantopaque and blood in subarachnoid space. Even if the study was made with a limited number of animals, neurologic sequellae were less conspicuouis in the treated group. Davis et al.[6] suggested the addition of methyl-prednisolone to the contrast medium Conray (meglumine iothalamate) to minimize the risk of arachnoiditis. Positive evidence of the benefit of this procedure is still lacking.[1]. Intrathecal methyl-prednisolone was demonstrated by Lehrer et al.[14] to achieve markedly increased concentrations of the steroid in nervous structures close to the injection when compared to administration by systemic route. They propose that more effective local soluble steroid levels within the central nervous system can be obtained with intrathecal than with systemic administration.

Intrathecal injections of Depo-Medrol were administered to cases 2, 3 and 4. In case 2 their effect cannot be judged because they were made after parapleglia was complete. Both in cases 3 and 4 a remarkable improvement of the intracranial hypertension was observed after some Depo-Medrol injections. Improvement lasted for three and four weeks, respectively, and was very brief after a second injection in case 3.

Nicholson and Eversole[21] advised irrigation of the subarachoid space with isotonic saline solution when a cauda equina syndrome was observed after spinal anesthesia. Removal of large amounts of CSF was made in several consecutive days in our case 4 and this proved to be unsatisfactory. It may still be possible that energic washing of subarachnoid space or continuous drainage of CSF, coupled or not with more intensive intrathecal corticosteroid therapy would be effective in preventing the ulterior development of arachnoiditis when the initial meningo-radicular syndrome is present and disclosed. Lumbar puncture should be performed whenever radicular pains are very severe after peridural anesthesia.

Surgical liberation of arachnoid adhesions is a difficult task and usually unefective, although rare reports of good results in chronic arachnoiditis justify a trial in more localized lesions[8, 29]. Neurologic deficit, however, is some times relieved only temporarily and then, subsequently, progress to ccmplete paraglegia[31].

The time relation of development of paraplegia and unexpected recovery after placement of ventriculo-peritoneal shunt, in cases 3 and 4, suggest a causal relation. Recovery would not be atributed to intra-thecal Depo-Medrol, for neurologic deterioration continued after the injections and no other therapeutic measure was used at this time.

Nelson et al.[20] reported 2 cases of adhesive arachnoiditis occurring as suposed complication of intrathecal methyl-prednisolone therapy for multiple sclerosis. However, one patient received 23 injections of Depo-Medrol and the other had a myelogram before intrathecal steroid was started. An

increase in protein was the only significant change noted in spinal fluid in their cases. CSF protein decreased in successive examinations in our cases, transiently during and after methyl-prednisolone injections.

SUMMARY

Four patients who received epidural anesthesia presented sustained myelopathy; three of them had complete paraplegia and one a lumbo-sacral myelopathy with urinary retention. All four patients complained of very intense radicular pains immediately after the analgesic effect of Lidocaine was over. Two patients in whom lumbar puncture was done in the first 24 hours presented an aseptic meningitic reaction in CSF. Paraplegia completed in two to ten months in three patients and in two of them severe intracranial hypertension developped at this time. It is proposed that the disease runs a two-stages course, at least in some cases, characterized by an aseptic meningitis, followed, after a silent period of some months, by signs of adhesive spinal and intracranial arachnoiditis. Intracranial hypertension was controlled by ventriculo-peritoneal shunt; in two patients a transitory effect of intrathecal injections of methyl-prednisolone acetate was observed. Two patients recovered almost completely from paraplegia.

RESUMO

Paraplegia e hipertensão craniana após anestesia epidural. Relato de 4 casos.

Quatro pacientes que receberam anestesia epidural apresentaram mielopatia de longa evolução; em três ocorreu paraplegia completa e um apresentou uma síndrome medular lombo-sacra com retenção urinária. Todos os pacientes se queixaram de intensas dores radiculares imediatamente após a cessação do efeito analgésico da lidocaína. Dois pacientes apresentaram uma reação meningítica asséptica no líquido cefalorraqueano nas primeiras 24 horas. A paraplegia tornou-se completa em 2 a 10 meses após a anestesia; dois pacientes tiveram hipertensão craniana severa. Em alguns casos, senão em todos, esta afecção apresenta uma evolução em duas etapas, caracterizadas por meningite asséptica imediata, seguida, depois de um período silencioso de poucos meses, de sinais de aracnoidite adesiva espinal e intracraniana. A hipertensão intracraniana foi controlada por derivação ventrículo-peritoneal; em 2 pacientes houve melhora transitória pela administração de metil-prednisolona intratecal. Em dois pacientes houve recuperação quase completa da paraplegia.

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