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I. Session on Post-Traumatic Cystic Degeneration of the Cord

SYRINGOMYELIA AS A SEQUEL TO TRAUMATIC PARAPLEGIA

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Abstract. Ten cases of post-traumatic paraplegia are described in whom syringomyelia symptoms have supervened. Five patients have been operated upon after investigation. Operative results have been encouraging. A discussion of likely pathogenetic mechanisms is presented.

Key words: Paraplegia; Syringomyelia; CSF dynamics; CSF pressure; CSF pulsation.

PRIOR to World War II few patients with traumatic paraplegia survived for long. Survival of these patients has been helped by antibiotics and improved nursing care. In spite of improvements in the overall management of these patients, many complications and post-traumatic sequelae occur. Among these is the syndrome of progressive ascending post-traumatic syringomyelia. Although the reported incidence of this condition is low, 0.3 to 2.3 per cent (Barnett & Jousse, 1976), the effect on the patients can be disastrous. There are several varieties of cord cavitation of which post-traumatic syringomyelia is but one example (Barnett, Foster & Hudson, 1973).

Clinical Data

We have studied eight men and two women with syringomyelia following complete cord lesion. Age at the time of injury ranged between 16 and 47 with a median of 20 years. All patients had complete or almost complete paraplegia after traumatic fractures of the spine (Table I).

The onset of these symptoms from the syrinx varied from 9 months to 17 years after the fracture with a median of $4\frac{1}{2}$ years. There was no relationship between the latent period and the level of the initial injury (Table I).

Pain was the initial symptom in half our patients. Three of them could relate their symptoms to activities which involved straining (cases one, seven and nine). Nine of these patients showed signs of neurological deterioration during the course of follow-up which ranged from 2 to 11 years. Average length of follow-up since onset of syringomyelia was 6 years (Table I). None of these patients had neuropathic joints, bilaterality or brain stem involvement which have been reported (Jellinger, 1967; Rossier, Werner, Wildi & Berney, 1968; Barnett *et al.*, 1976). None had Horner's syndrome but two patients (Table I) had excessive sweating on the side of the lesion. The occurrence of sweating below the site of the paraplegia indicates that the syrinx has spread below the lesion as well as above (Stanworth,

TABLE

Case No.	1	2	3	4	5
Age at injury	M 16	F 20	M 17	F 47	M 17
Level	T 3	T 5	T 4	T 5	T 5
Interval before syrinx onset	9 yrs	1 yr	9 mths	1 yr	4½ yrs
Time since onset of syrinx	2 yrs	9 yrs	11 yrs	10 yrs	6 yrs
Presenting symptoms	Pain in right shoulder after pulling on a trapeze	Pain in right shoulder of insidious onset	Hyperhidrosis and analgesia of right arm. Weakness and contracture of right hand of insidious onset	Pain in right arm with dissociated sensory loss	Pain in right arm with dissociated sensory loss
Later symptoms	Dissociated sensory loss later	Dissociated sensory loss after 1 year	Progressive weakness of right arm with severe disability	Progression of weakness over 9 yrs with atrophy	Hyperhidrosis
Sensory loss
Pinprick loss shaded					
Total loss solid					
Contrast radiology	—	—	Iophendylate myelogram showed complete block at site of injury	Iophendylate myelogram showed cord not enlarged above the injury site	Iophendylate myelogram showed cord not enlarged above the injury site
Operative findings	—	—	—	Catheter was passed downwards below the fracture and left to drain	—
Result	—	—	—	Slight improvement for 2 years	—

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6	7	8	9	10
M 20	M 27	M 22	M 22	M 34
T 6	T 12	L 1	L 3	C 8
12 yrs	4 yrs	17 yrs	10 yrs	2 yrs
7 yrs	4 yrs	9 yrs	9 yrs	3 yrs
Hyperhidrosis of legs L-R	Pain in right arm after sneezing	Paraesthesia of the right arm	Pain and aching of the left arm after exercise	Paraesthesia and weakness of the right hand
Dissociated sensory loss. Sympathectomy for hyperhidrosis. Intermittent weakness after exercise	Dissociated sensory loss after 1 month. Difficulty in writing	Pain and weakness developing in the right arm. Nystagmus and vomiting attacks	Progressive weakness and dissociated sensory loss of arm and trunk	Dissociated sensory loss of left arm and trunk. Had to change hands to write

Iophendylate myelogram showed wide cervical cord	Air myelogram showed collapsing cord	Iophendylate myelogram showed severe obstruction at injury site, no cervical cord enlargement	Air myelogram showed collapsing cord	Air myelogram showed flattened cord
Big syringe going up 4 cm and down 13 cm	Moderate (4 ml) syringe going up for 30 cm. Downward extension was separate, only the upper syringe was drained	Moderate (12 ml) syringe going 35 cm upwards communicating at the top. Top only was drained	Small syringe 3 mm wide extending for 20 cm upwards only	—
Improved pain, improved tendon reflexes and power after 3 years	Subjective improvement only, stable for one year	Improvement of nystagmus and vomiting. Arm was stronger, less clumsy with lessening of paraesthesia. Bladder control was worse. After 6 months all symptoms had recurred except that the bladder remained worse than pre-op	Improvement of strength and tendon reflexes. Stable for 1 year	—

1979). One patient (Case 6) has had lumbar sympathectomy to relieve the sweating. This procedure improved the hyperhidrosis in the leg but did not relieve the sweating in the upper extremity.

Two patients suffered proprioceptive and motor loss in the hands severe enough to force them to change hands for writing (Cases 7 and 10). None of the others had detectable dorsal column loss.

Five patients showed depression or loss of tendon reflexes in the legs. This would seem to be evidence of a downward extension of the syrinx, which was verified in four of the cases at operation.

Myelography was performed in eight patients (Table I). Six had either an enlarged cord or a collapsed cord in the supine position, the results of myelography in two were inconclusive. Two patients have not had myelograms. They did not have any significant disability or motor weakness but showed dissociated sensory loss and asymmetrical deep tendon reflexes in the upper extremities. The five operated patients had detailed radiographic investigation of the foramen magnum and cisterna magna. In none of them was any related abnormality found. There was a large cisterna magna in Case 7, a finding usually without significance. Four of the patients have had Transmission Computed Axial Tomography (TCAT) scans of the cord. Only two of these demonstrated the syrinx. One case underwent surgery on clinical grounds alone, and was found to have a syrinx containing 12 ml. of fluid extending above the level of the injury (Case 8, Table I).

Sufficient fluid from the syrinx was difficult to aspirate uncontaminated and analysis was reported in only two patients. The high protein values in Case 6 may be related to the fact that this was the only cyst under raised pressure at the time of surgery. The values in Case 8 were compatible with a communication with cerebrospinal fluid.

Five patients have undergone surgery. The findings in all have included narrowing of the bony spinal canal, most usually symmetrical narrowing from front to back, and also an expansion of the cord which was adherent to the surrounding dura around all, or nearly all of the circumference. If a patent channel from the upper to the lower subarachnoid space was present it tended to be anterior and lateral. In Case 6 the cavity was prominent at the level of the paraplegia and extended freely both upwards and downwards. In Case 9 the small cavity found at the upper end of the fracture extended upwards only. A downward cavity was not sought. In the other cases the cavity was intermediate in size. In each case myelotomy was done, a silastic catheter was left between the syrinx and the subarachnoid space to allow the fluid to run out of the intramedullary cavity. Because of the upward thrust of CSF which occurs during straining the bottom end of the myelotomy tube was inside the lower part of the syrinx and the upper end was left outside the cord in the subarachnoid space (Fig. 1). In addition to giving a sensible flow preference it saved the cord from being at risk from an internal drainage tube and ensured that the upper end was in an area of subarachnoid space well away from the possibility of adhesions. The dura was closed in each case after the drainage tube had been sutured in place.

All of the patients have had immediate subjective improvement in sensation. None of these five patients were made worse by surgery excepting bladder function in Case 8. Three out of five demonstrated objective signs of improvement by the return of reflex activity or strength (Cases 6, 7 and 9). Both of the patients without objective signs of improvement were subjectively improved (Table I).

Four out of the five operated patients have remained stable at review after 6 months to 3 years, with a median time of 1 year after surgery. This is admittedly

FIG. 1

Operative findings and procedure: (a) the cord is usually visibly abnormal under the dura. A longitudinal midline incision shows the dura to be adherent to the cord which is enlarged at least in its lateral dimension. (b) A longitudinal myelotomy may come upon the syrinx cavity almost immediately or at a depth of some millimetres. (c) A silastic drainage catheter may be placed from the inside of the syrinx to the subarachnoid space, preferably the lower end inside and the upper end outside; this was not possible with Cases 7, 8 and 9.

too short to determine whether or not the benefit of surgery will be lasting. Case 8 initially showed a marked improvement in the arm symptoms but began to deteriorate after some months and 1 year after operation had regressed to the state where the arms were as pre-operative, but the bladder function which had deteriorated after laminectomy had not recovered.

Discussion

Gardner (1973) pointed out that the commonest form of syringomyelia is accompanied by a foramen magnum abnormality and that decompressive operations on the craniovertebral junction are helpful in improving patients with this condition. The term 'communicating' was introduced (Williams, 1969) accepting the assumption made by Gardner that there was a communication along the central canal from the floor of the fourth ventricle in the region of the obex to the inside of the syrinx. It was supposed that there were mechanisms causing filling of the syrinx directly from the ventricular system. This communication is only present in about 10 per cent of patients with 'communicating' syringomyelia at the time that they present for investigation (West & Williams, 1980).

The theories of Gardner have been somewhat marred by his attempt to include

a wide variety of disorders into the same spectrum of disease which he calls 'dysraphism'. This is certainly unreasonable and it is not sensible to assume, as Gardner has done, that cases of post-traumatic syringomyelia have an abnormality at the foramen magnum. None of these cases have had such an abnormality radiologically and it seems unlikely that any of the reported post-traumatic cases have had such an abnormality (Rossier *et al.*, 1968; Nurick, Russell & Deck, 1970).

Some differences therefore must be proposed between post-traumatic syringomyelia and the most common type. Because of the length of time which separates the injury from the subsequent syringomyelia syndrome, the disease process is likely to have at least two components. It seems probable that there is an initial cause of cavity formation in the region of the fracture and that the factors which lead to expansion of this at a later date may be secondary.

There are several factors which may be responsible for initial cavitation of the cord. Lesions remote from the primary cord injury have been reported apparently related to arterial injury. The role of ischaemia in producing small areas of infarction followed by cavitation has been suggested (Mair & Druckman, 1953; Jellinger, 1967). Recently there have been reports dealing with cyst formation on a biochemical basis and there is evidence that lysosomes and other cellular enzymes play a role in cyst formation after trauma (Kao & Chang, 1977; Kao, Chang & Bloodworth, 1977). Liquefaction in a prior haematoma seems most likely, occasional intracord haematomata occur both clinically and experimentally and certainly occur of sufficient size often enough in 2 per cent of cases. Many early reports described cavities resulting from haemorrhage within the cord followed by necrosis and associated with myelomalacic cores (Cushing, 1898; Holmes, 1915). The objection raised by Rossier, Werner, Wildi and Berney (1968) that the syrinx does not spread immediately can be explained on the assumption that the blood in the cord clots and that only after it liquefies and becomes able to move do the cavities extend under the influence of other factors.

The mechanisms of extension of these cavities must explain the site which tends to be in the grey matter between the dorsal horn and posterior columns. This area is between the dorsal and ventral arterial supply and has caused some authors to suggest vascular factors in the production of these lesions (Mair & Druckman, 1953; Scher, 1976). This area of the cord is also weak in supportive or connective tissue which may help breakdown. If fluid is forced into the central regions of dogs it dissects upwards along these planes, usually on one side of the incomplete septum provided by the ventral fissure and the posterior columns. The cavity tends to be maximal in the cervical and lumbar regions and breaks freely in and out of the central canal which becomes dilated thereby. The cavity may burst through along the central canal to the fourth ventricle as the case of McLean *et al.* (1973) appears to have done. For detailed description of experimental methods and a discussion of sites of weakness see Williams and Weller (1973). There are other factors which may be involved in the extension of these lesions. Secondary haemorrhage into a cyst is a possibility but this usually produces sudden severe deficits and is probably the exception.

The most important mechanisms are probably connected with changes in the pressure in the venous system. Not 'venous back pressure' but rather the direct effect of intraspinal veins on CSF. In any activity which causes raised pressure in the thorax and abdomen, such as a cough, the epidural veins become distended because of high pressure in the azygos venous system. They then compress the dura and squeeze the CSF forcing it upwards towards the head. This phase, in an acute cough, lasts about 0.1 to 0.2 of a second and is accompanied by energetic

movement of fluid (Williams, 1976). Such movement causes two phenomena of importance which occur together but which may be described separately.

a. 'Slosh'.

If there is fluid in the cord, particularly if the cord is somewhat enlarged, then the fluid inside will be moved by undulations in the walls of the cord cavity. Such pulsatile surges of combined pressure and movement may be called 'slosh'. A diagram is given in Fig. 2. This can be screened if Myodil is introduced into a syringomyelia cavity, if there is room for the intrasyrinx fluid to move it will pulsate. The energy thus possessed by the intrasyrinx fluid tends to break down the sites of structural weakness at the extremities of the cavity; this is one of the mechanisms which causes syringomyelia of all kinds to progress and is responsible, for example, for syringobulbia. The energy which in the normal passes freely up and down the subarachnoid space of the spine in the form of CSF pulsation may be concentrated at the level of a fracture (Fig. 3). Theoretical analysis of the dis-

FIG. 2

The mechanism of 'slosh' in syringomyelia. On the left the effect of compression applied from the dura is represented as squeezing the cord and making the fluid inside the cord move upwards in the same way as the fluid outside the cord. When this upward surge of fluid drops downward again it may still possess some destructive energy, as suggested by the right-sided diagram.

FIG. 3

The anatomical situation is more complex than in Fig. 1. There is a partial or almost complete blockage of the subarachnoid space at the site of the fracture. There may be a sizable intracord cavity there and a venturi effect plus adhesions around the site of injury may result in most of the energy of pulsation being inflicted on the intrasyrinx fluid.

sipation of energy at the sites of partial block has come from Lockey, Poots and Williams (1975). Clinically the initiating symptoms are commonly brought on by sneezing or strenuous activities. This is true of the common form of 'communicating' syringomyelia (Williams & Turner, 1971) but is especially true of syringomyelia above a paraplegia. (Williams, 1970; Barnett *et al.*, 1973, 1976; McLean *et al.*, 1973). Three patients in this series had such a history. Pressure recordings at rest are given in Fig. 4 and during coughing in Fig. 5. The amplitude of the pulses associated with coughing are 20 times bigger than cardiac pulses and energy transmission to the walls of the syrinx. For a discussion and an account of recording techniques see Williams (1974, 1976). It is also interesting to note that all of the patients in this series had complete or almost complete cord lesions although Barnett *et al.* (1976) report a high incidence in partial lesions also. Totally paralysed patients may have more straining during daily activities than less

FIG. 4

CSF pulsation recorded at rest above and below the fracture site of Case 8. The vertical scale is pressure in millimetres of mercury. The similarity of wave from above the level of fracture to the arterial wave form indicates that this comes from the arterioles and arterial end of the capillary bed in the brain. This 'arterial' pulsation produces a small degree of 'slosh' which may be seen on radiographic screening.

FIG. 5

Pressure recordings during five consecutive coughs in Case 8. The fluid does not have time to get past the area of arachnoiditis and there is no 'suck' visible i.e. the differential never goes below zero. Note that the amplitude of the pulse is 20 times that of the arterial pulsation. This phenomenon produces violent 'slosh' in intrasyrinx fluid.

severely disabled patients (Scher, 1976). The importance of slosh may account for the comparative rarity of this complication in cases with quadriplegia.

b. 'Suck'.

The upward and downward movement of CSF in the neuraxis may produce temporary differences in pressure. The upward surge, being the most energetic and accompanied by dilatation of the dura, tends to force fluid upwards more readily than it can drop downwards again. This is responsible for temporary low pressure below a partial blockage, the more severe the blockage the longer the fluid takes to seep back below the obstruction and the pressure below the blockage may become permanently low. If the blockage is at the foramen magnum it commonly consists of a hindbrain hernia, a conical downward extension of the cerebellar tonsils which forms a valve. This valve may become jammed in the foramen after coughing and straining, particularly during the post-Valsalva manoeuvre and be responsible for hindbrain symptoms and 'communicating' syringomyelia as well as further progression of the hindbrain hernia itself. With blockage at the foramen magnum the pressure thus measurable has been called cranio-spinal pressure dissociation (Williams, 1974, 1976, 1977, 1980). It is immaterial whether the pressure above in spinal blockage is high or that below is low, the pressure difference exerts the same effects and thus the ponderous term 'cranio-caudal intraspinal pressure dissociation' as well as 'craniospinal pressure dissociation' may be replaced by 'suck'. Once fluid is present inside the cord at a fracture site it may be sucked downwards inside the cord and produce downward extension of a syrinx. Fig. 6 shows the degree of suck in Case 6 after a 66 second Valsalva manoeuvre. For technical methods and discussion see Williams (1977). This is unimpressive when compared with a typical hindbrain hernia problem but this kind of result occurs after every strain. The total damage to a precarious spinal cord after

FIG. 6

Valsalvas' manoeuvre in Case 6 showing 'suck'. The patient blew into a mouthpiece which recorded a pressure of 30 mm Hg for 6 seconds. During this time the lumbar pressure was higher than the thoraco-cervical spinal pressure by some 5 mm Hg or so. When the strain was over the lumbar pressure dropped below that in the thoraco-cervical region by around 2 mm Hg. The post-Valsalva rebound phenomenon was more marked in the cranial end of the neuraxis and pushed the pressure difference ('suck') up to 7 mm Hg at the peaks of cardiac output.

transferring from chair to bed or even after opening the bowels by external, manual compression is not inconsiderable. 'Suck' is a likely mechanism of extension of cavities of all kinds, but only in 'communicating' syringomyelia is it likely to be a cause of the initial cavitation.

The importance of venous events in both 'suck' and 'slosh' may cause the value of excessively strenuous and competitive exercises during rehabilitation to be called into question. Case 6 used to suffer severe weakness of the right deltoid muscle after playing wheelchair basketball and Case 9 noticed his weakness particularly if he went on canoeing holidays. It seems reasonable, if syringomyelia supervenes, to limit exercise to arm power endeavours such as archery even in operated cases. Even arm exercises may need to be moderated, the frequency of Charcot's joints of the upper limb is higher in post-traumatic syringomyelia than in 'communicating' syringomyelia, possibly related to stress in the arm joints. An incidence of one in three was reported by Barnett and Jousse (1976).

The source by which the fluid within these cysts is replenished is questionable. In this series the results of fluid analysis have been sparse and of no value in determining the source of the fluid, some reports indicate a high protein in the fluid on occasions. One possible source is from the subarachnoid space via a hole at the level of the cord injury. This type of communication has been confirmed twice (Savoirdo, 1976; McLean *et al.*, 1973).

Obviously a myelotomy to the subarachnoid space may allow fluid to enter the syrinx readily and although it may relieve fluid pressure which is being added by transudation it is possible that under some circumstances a subarachnoid to syrinx myelotomy may make the patients worse. It seems likely that the laminectomy itself and the change in the form of the fracture site, particularly widening of the spinal canal, may produce the principal benefit. Drainage of the syrinx to the peritoneum has fewer disadvantages (Edgar, 1976).

Another possible source of fluid could be through a communication between the 4th ventricle and the cyst. This idea stems from the concept of syringomyelia described by Gardner in a series of publications from 1957 onwards; his conception is outlined above and is summarized in his monograph of 1973. Several reports suggest this mechanism (Rossier *et al.*, 1968; Nurick, Russell & Deck, 1970) but there is no convincing evidence in its favour. An alternative source of fluid could be directly from the cells lining the cyst wall. There is considerable gliosis surrounding the cysts along with prominent vasculature which may produce a transudate particularly if injured by slosh or by shearing due to suck or to movements of the spine in relation to adherent cord (Martin & Maury, 1964; McLean, Miller, Allen & Ezzedin, 1973).

Aboulker (1979) has championed two additional mechanisms, venous stasis and the arrival of fluid into a syrinx along the perivascular spaces.

The venous drainage of the spine is particularly good because it provides an extensive anastomotic channel used particularly when the thoraco-abdominal pressure is high. Additionally, although it is possible to envisage venous blockage and thrombosis at a fracture site in the acute stage, the revascularisation which accompanies healing must surely dispose of any raised venous pressure within the $4\frac{1}{2}$ years which was the median time for our cases. Venous stasis seems best regarded as a possible contributory mechanism in the acute phase only.

If the perivascular spaces can freely transmit fluid between the syrinx cavity and the subarachnoid space then this is more likely to result in drainage of the syrinx than filling. It is inconceivable that by raising the pressure outside the spinal cord it might thereby be inflated. Raised pressure outside the cord would

have a tendency to flatten it. It is possible that once a cavity is developed it might be maintained through this route; this remains to be proven.

Post-traumatic syringomyelia is distressing and threatens upper extremity functions of individuals who are already severely disabled. Surgery is likely to be the only means of effecting a cure or halting its progression. There are no large series of patients of long duration to allow an accurate long term prognosis. Although there are reports questioning the benefit of surgical treatment for this condition (Martin & Maury, 1964; Lacert, Trottier, Durand, Pannier & Grossiord, 1977) most authors report improvement in the majority of patients following surgery and few complications (Freeman, 1959; Zdrojewski, Werner & Rossier, 1969; Barnett *et al.*, 1973, 1976; Edgar, 1976). In competent hands, laminectomy and myelotomy are uncommonly associated with significant neurological deterioration.

The history of this disorder in this series was variable. The evidence for spontaneous regression is unclear although two cases were reported as improving spontaneously by Scher (1976). Our present series suggests that the majority of patients will eventually show signs of progression. If this is the case, it makes sense to drain these cavities before the onset of irreversible myelopathy. The occurrence of slosh in particular is more likely in cases with big syrinxes. For a discussion of this and other points see Williams (1980b).

For these reasons, we feel that surgery is justified in patients who demonstrate an ascending lesion with a positive myelogram or TCAT Scan. Even in the presence of negative investigational studies, patients with clinical evidence of a lesion should be considered for surgical treatment (Case 8). Sizable syrinxes may be asymptomatic, really disabling syrinxes may show a normal external diameter to the cord. Since the lesion is always accessible at the site of fracture, myelography becomes something of a luxury for those with an investigational bent. In patients with only a dissociated sensory deficit and little disability, at least frequent follow-up examinations are necessary. Once surgery is decided upon, treatment should be directed at establishing permanent drainage of the cyst. Only long-term studies of larger numbers of these patients and careful study of the mechanical aspect of 'suck' and 'slosh' as well as the biology of the fluid and precise morphological evaluation most probably involving the post-operative use of TCAT scans will lead to successful management of this condition.

SUMMARY

Ten cases of post-traumatic syringomyelia are described, five have been operated upon with four good results. None of the patients have had complete remission of symptoms. It is suggested that the clinical course and morphology of the disease is best explained by assuming that the basis of the initial cavitation is established at the time of the injury, most probably by an intracord haematoma, and that the subsequent enlargement of the cavity and progression both upwards and downwards is likely to be due to pulsatile action of cerebrospinal fluid under principally venous influences. Fluid inside the cavities may 'slosh' up and down as the patient strains and pressure differences developed across the fracture site may 'suck' fluid within the syrinx or may further traumatise the site of injury. Fluid in the cavity may initially come from liquefying haematoma, venous congestion or damaged cells and in the progressive phase of the disease may be replenished from the traumatised cells or from the bloodstream by transudation;

alternatively the cerebrospinal fluid pathways may refill the syrinx by either sizable fistulae at the injury site or by seeping along the perivascular spaces.

It is suggested that cases with syringomyelia symptoms should limit exercise likely to cause excessive pressure changes in the trunk cavities and that early surgery to drain syrinx fluid is advisable. Further study of the biology of the syrinx fluid and the mechanical stresses involved as well as improved clinical studies and analysis of the morphology are necessary to improve management of this condition.

RÉSUMÉ

Dix cas de syringomyélie posttraumatique sont rapportés. Cinq malades ont été opérés, quatre avec un bon résultat. Une rémission complète n'a pas été enregistrée dans aucun cas. La cavité syringomyélique se forme au temps du trauma sur la base d'un hématome intrarachidien. L'enlargissement de la cavité peut être déterminé par l'action pulsatile du LCR, qui est soumis aux influences de la congestion veineuse. Quand le malade presse, tousse etc. LCR est dispersé rapidement et alternativement vers la direction de la crâne et du cul-de-sac. Les différences de la pression qui s'établissent au niveau de la fracture favorisent l'aspiration du liquide dans la cavité et une traumatisation progressive du tissu rachidien. L'enlargissement secondaire de la cavité s'accomplit par une transsudation de produits du sang ou par le chemin de fistules au niveau de la lésion ou autour de la voie des espaces perivasculaires.

Au cours des l'exercices physiques chez malades avec les symptômes de syringomyélie posttraumatique il faut tenir compte de la nécessité d'éviter tels mouvements, qui produisent un changement brusque de la pression intrathoracale ou intraabdominale. La recherche extensive sur la biologie du liquide de la syrinx, la tension mécanique, l'évolution clinique, ainsi que les études morphologiques sont nécessaires pour améliorer le traitement de cette condition.

ZUSAMMENFASSUNG

Es wird über 10 Fälle von posttraumatischen Syringomyelien berichtet. 5 Patienten wurden operiert, 4 davon mit gutem Ergebnis. In keinem Fall kam es zu einer vollständigen Remission der Symptome. Die Klinik und Morphologie der Krankheit werden erklärt auf der Basis einer primären Kavität, die zum Zeitpunkt der Verletzung, am wahrscheinlichsten als Rückenmarkhämatom entstanden ist. Sekundäre Vergrößerung der Kavität sowohl nach oben als auch nach unten, scheint, verursacht zu sein durch die pulsatile Wirkung des Liquor cerebrospinalis unter venösem Einfluß. Die Flüssigkeit in der Kavität bewegt sich schwallartig hin und her, wenn der Patient preßt. Druckgradienten entstehen auf beiden Seiten der Frakturebene, wodurch die Flüssigkeit in die Syrinx eingesaugt wird. Die Flüssigkeit in der Kavität kann das Endprodukt der Verflüssigung des Hämatoms, venöser Stauung und zerstörter Zellen in der progredienten Phase der Krankheit sein. Spätere Zunahme der Flüssigkeit entsteht durch Transsudation aus dem Blutstrom oder durch die Fistula an Stelle der Verletzung oder die perivaskulären Räume entlang kommt es zum Einfließen des Liquor cerebro spinalis.

Kranke mit posttraumatischer Syringomyelie sollen vermeiden solche Übungen, die zu größeren Druckschwankungen im Bereich der Hohlräume (Thorax, Bauch) führen, durchzuführen. Frühe chirurgische Drainage der Syrinx ist indiziert. Weitere Untersuchungen der Biologie der Syrinxflüssigkeit und des mechanischen Stresses sowie langzeitige klinische Beobachtungen mit Analyse der Morphologie sind notwendig.

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