

Studies in spina bifida

Part IV The frequency and extent of paralysis

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In earlier parts of this survey we have attempted to describe in broad outline the clinical progress of a series of rather more than 300 consecutive cases of spina bifida cystica first seen between 1949 and 1958 (Doran and Guthkelch, 1961), to indicate some considerations that seem important in deciding when to repair the spinal defect (Guthkelch, 1962a), and to demonstrate a previously undescribed seasonal variation in births of infants with spina bifida (Guthkelch, 1962b). The present communication is concerned with a more detailed examination of the neurological findings in these cases, for this is one aspect of the study of spina bifida cystica which seems to have received less attention than perhaps it deserves, most writers confining themselves to general statements, such as that there is always 'definite paralysis' in rachischisis (myelocoele) and so on; Warkany (1960), indeed, complained that nowhere in the literature was it definitely stated whether or not neural conduction ever occurs through such a lesion. MacNab (1957) has said that 93% of cases of 'myelomeningocoele' show 'clinical evidence of paralysis', presumably implying that in 7% there is no neurological deficit, but his classification of spina bifida cystica excludes all cases of rachischisis in which there is no true sacculaton, and although he notes that there are some cases in which the neural element 'forms part of the sac wall at its fundus, giving rise to the clinical appearance of ulceration . . . (and others in which) . . . true skin is separated from the lesion by a large fibro-fatty mass of tissue' he does not distinguish between the neurological findings in the two varieties. Consequently MacNab's work does not explicitly answer the point raised by Warkany, for it could have happened that those of MacNab's cases which were neurologically intact were the ones in which the cord was not exposed on the surface of the body and which to Warkany would not be cases of myelocoele. Laurence (1960a and b) analysed a series of 407 cases of spina bifida with respect to neurological deficit. He does not

make MacNab's (1957) rather arbitrary distinction between non-cystic and cystic spina bifida, but classifies all cases in which neural elements enter into the lesion as 'myelocoele'. In respect of these 'myelocoeles', amounting to 90% of his whole series, Laurence (1960b) notes that the pattern of disability 'was largely dependent on the position of the spinal lesions. The percentage of cases with limb involvement was greatest in those with the sac in the lumbar region, while those with the upper dorsal and sacral lesions less frequently showed severe paralysis. On the other hand the lower the sac along the cerebrospinal axis, the more frequent was the occurrence of sphincter paralysis'. Here again, however, no distinction is drawn between those cases in which the cord rudiment is and is not exposed on the body surface at birth.

Very recently, Sharrard, Zachary, Lorber, and Bruce (1963) recorded examples of preservation of function in, for example, the dorsiflexors of the feet of infants born with a lumbar 'myelocoele'; in their paper, the term 'myelocoele' seems to be used to describe only lesions with the cord exposed and this is in fact the first explicit statement that neural transmission occurs across such a lesion.

CLASSIFICATION

In an earlier communication (Doran and Guthkelch, 1961) we, like Laurence, regarded all cases of spina bifida with herniation of meninges through a defective posterior neural arch as properly defined by the general term 'spina bifida cystica', whether or not true sacculaton was present, for we felt that to exclude, as does MacNab (1957), the sessile myelocoeles introduced an unnecessary complication, quite apart from the fact that with changes in intracranial pressure a flat lesion may become cystic and vice versa. But because there seems to be a difference in neurological status between those cases in which the spinal cord tissue is exposed at birth and those in which it is already covered by skin, we have analysed our findings in these two

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groups separately, and use for the former the term 'open' and for the latter the term 'closed' myelomeningocele. All cases of complete rachischisis and of myelocoele as defined by MacNab (1957) are therefore included in the former group as open myelomeningoceles, though 'myelocoeles' as defined by Laurence (1960a and b) are divided into two separate categories. The use by Sharrard *et al.* (1963) of the term 'myelocoele' and ours of 'open myelomeningocele' is the same. For those of our cases in which only meninges and cerebrospinal fluid were found in the cyst, we, in common with most other writers, use the term meningocele. The necessity to classify the lesions in spina bifida yet again is regretted, but seems unavoidable, because it is evident that the expressions currently in use mean different things to different writers.

MATERIAL AND METHODS

Our original group of 307 cases (308 lesions) comprised 65 simple meningoceles and 243 myelomeningoceles. Thirty of the latter, however, were judged to be unsuitable for inclusion in the present more detailed study for various reasons: for example, the infant was moribund when first seen, the picture was clouded by established sepsis, or neurological examination was insufficiently precise. We are satisfied that the exclusion of these cases has not altered the general trend of our observations.

In considering neurological status we have used the terms 'partial' or 'complete' paralysis to indicate loss of function below a certain level of the cord rather than in part or the whole of the lower limbs since the former convention is more in accord with standard neurological practice. Consequently a child with a sacral spina bifida cystica and complete loss of motor and sensory function in the second to the fifth sacral segments is classified as a case of 'complete paralysis' even though function in both legs may have been excellent and only the sphincters affected.

The spinal defect, and consequently, in open lesions, the exposed part of the cord, often stops short of the sacral segments and there is not infrequently reflex activity below the level of the lesion. The anal skin reflex, in particular, is often active in cases of open thoracolumbar or lumbar myelomeningocele with paralysed legs, but this does not imply that there is conduction of neuronal impulses across the defective area, and care has been taken to avoid such assumptions. In respect of the assessment of motor function nothing has been counted as voluntary movement short of either motor activity of a purposive character or occurring in circumstances where no reflex contraction of muscle would

be expected, or else a response in the muscle groups supplied by spinal segments at or below the level of the myelocoele to a stimulus, *e.g.*, handling or tickling, applied above that level. Conduction of sensory impulses has been judged to occur on the basis of a constant response to pin prick, *e.g.*, by crying, struggling, or puckering of the face. Where the infant has survived, the original findings have been checked by re-examination.

RESULTS

Table I shows the distribution of the cases in the three categories already defined. The absence of any cases of open myelomeningocele at cervical or thoracic level should not be taken to indicate that none occur but rather that they rarely, if ever, survive for more than a few hours after birth. Examination of the records of the necropsy findings in the last 50 cases of stillbirth or neonatal death occurring in spina bifida babies delivered at St. Mary's Hospital in Manchester showed that every case of open cervical or thoracic myelomeningocele was associated with anencephaly. In our own series most of the 19 cases of closed myelomeningocele which occurred in the upper part of the spine conformed to the pattern of the so-called syringocoele, to which attention was drawn in the first part of this study (Doran and Guthkelch, 1961). It should be noted, however, that occasionally pressure ulceration of the fundus of the sac gave rise to an appearance somewhat similar to that of open myelomeningocele but distinguishable by the fact that in syringocoele the ventral part of the cord lies beneath the ulcerated area and separated from it by a hydromyelic dilatation of the central canal.

It might be expected that the most extensive spinal lesions in spina bifida would also be cases of the most extreme degree of failure of fusion and Table I brings out this point. All the thoraco-lumbo-sacral, almost all the thoraco-lumbar, and a definite excess

TABLE I
DISTRIBUTION OF THE VARIOUS FORMS OF SPINA
BIFIDA AT DIFFERENT LEVELS OF THE SPINE

Site	Simple Meningocele	Closed Myelo- meningocele	Open Myelo- meningocele	Total
Cervical	6	6	0	12
Thoracic	7	12	0	19
Thoraco-lumbar	3	1	23	27
Thoraco-lumbo- sacral	0	0	10	10
Lumbar	23	14	48	85
Lumbo-sacral	13	18	60	91
Sacral	13	7	14	34
Total	65	58	155	278

TABLE II
FREQUENCY OF LIMB AND SPHINCTER PARALYSIS
(PARTIAL OR COMPLETE)
IN ALL MYELOMENINGOCELES

Site	Paralysis of		Total
	Limbs	Sphincters	
	Number of Cases	Number of Cases	
Cervical	3 (50%)	0 (0%)	6
Thoracic	2 (17%)	1 (8%)	12
Thoraco-lumbar	23 (96%)	16 (67%)	24
Thoraco-lumbo-sacral	10 (100%)	10 (100%)	10
Lumbar	53 (85%)	48 (79%)	62
Lumbo-sacral	68 (87%)	60 (77%)	78
Sacral	10 (48%)	14 (67%)	21

of the lumbo-sacral lesions, *i.e.*, those in which more than one region of the spine was involved, were examples of open myelomeningocele.

In respect of percentage involvement of limbs and sphincters at different levels of the spine our findings show a fair measure of agreement (Table II) with those of Laurence (1960a, Fig. 4).

There are two main points of difference: 1 We found less frequent spinal cord involvement in the thoracic cases, and 2 our estimate of sphincter involvement was higher in the thoraco-lumbar, lumbar, and lumbo-sacral ones. The first may be due simply to the fact that thoracic myelomeningocele is uncommon, so the groups of cases were small in both series, but it is also possible that some lesions classified by the surgeons who operated on Laurence's cases as simple meningocele would have been regarded by us as syringocele without neurological signs. This would have the effect of increasing, in Laurence's series, the proportion of cases with neurological deficit amongst his thoracic myelocoeles.

Our higher estimates of sphincter involvement is possibly due to our having assessed all patients who died in early infancy as having sphincter involvement if there was bilateral sacral anaesthesia and a patulous anus, for no surviving baby with these signs has normal bladder and bowel control. If for similar fatal cases in Laurence's series, the situation was regarded as indeterminate, the discrepancy is explained, for it is the cases with paraplegia at higher spinal levels which have the heaviest mortality and this is precisely where Laurence's figures and ours differ most.

SIMPLE MENINGOCELES There was no case of impaired function of the central nervous system associated with cervical or thoracic meningocele. Nine out of 23 cases of lumbar meningocele were associated with some abnormality on neurological

examination—at first sight a remarkably high proportion—but in three of these a diastematomyelia with bony spur was present and in a further two the only abnormality was a unilaterally absent ankle jerk without any loss of function. The other four cases all showed wasting of the calf, absent ankle jerk, pes cavus, and an extensor plantar response on one side, but in only two was the weakness and deformity of the foot sufficient to require orthopaedic correction.

Three out of 13 cases of lumbo-sacral meningocele showed abnormal neurological signs, namely, partial paralysis and deformity of one foot in two and incontinence in one. The proportion of cases of sacral meningocele with neurological abnormality was the same and comprised simple absence of one ankle jerk in one case and incontinence in two.

It was not thought right to extend the surgical exploration in these cases further than was necessary to exclude such gross lesions as diastematomyelia with bony spur, but there is no doubt that various other anomalies of the cord and nerve roots, of the type to which James and Lassman (1962) have recently redirected attention in spina bifida occulta, can and do occur in simple meningocele as well. Finally there may be anomalous development of the spinal cord in cases of simple meningocele though the cord itself has no anatomical connexion with the cyst and the affected part may be remote from it (Cameron, 1956). It is of course the associated anomaly and not the simple meningeal hernia which is responsible for any neurological deficit which may be present in these cases.

CLOSED MYELOMENINGOCELES Next in seriousness, from the point of view of neurological deficit, was the group of closed myelomeningocele. Of 58 cases studied, there was no spinal cord or root involvement in 31 cases. There was partial paralysis in 21 cases and a complete loss of function below the level of the lesion in six (Table III).

TABLE III
CLOSED MYELOMENINGOCELES
CLASSIFIED BY SITE AND BY SEVERITY OF DEFICIT
IN CENTRAL NERVOUS SYSTEM

Site	Normal Central Nervous System	Partial Paralysis	Complete Paralysis	Total
Cervical	3	3	0	6
Thoracic	10	2	0	12
Thoraco-lumbar	1	0	0	1
Thoraco-lumbo-sacral	0	0	0	0
Lumbar	7	5	2	14
Lumbo-sacral	6	8	4	18
Sacral	4	3	0	7
Total	31	21	6	58

The nature of the deficit in the cases with incomplete paralysis is interesting. One thoracic case was unusual in that closure of the defect had been attempted elsewhere and the child was referred on account of continued sepsis with a transverse myelitis. He was eventually left with spasticity of both legs. With this exception there was no instance of involvement of the long tracts in any case of closed myelomeningocele occurring above the lumbosacral junction; plainly, therefore, the extensive hydromyelia which is often noted in these cases is compatible with normal neural transmission: bilateral root involvement was found in two (one cervical and one lumbar); in seven the lesion was unilateral. Of eight partially paralysed cases of lumbo-sacral closed myelomeningocele, four had unilateral root lesions (L4 or L5 to S1 or S2); there was bilateral anaesthesia (L4 to S2 on one side and L5 to S1 on the other) with very little loss of motor power in one case and bilateral motor and sensory root lesions of L5 and S1 in another, while in two there was incomplete bilateral motor and sensory deficit with sphincter involvement. The three sacral cases all showed a patchy sensory loss in the saddle area with imperfect sphincter control. The closed myelomeningoceles of the lower end of the spine were therefore rather more frequently associated with bilateral neurological signs, and this is perhaps to be expected since at lower levels the character of the lesion is rather different. Instead of there being a syringoid protrusion from the posterior aspect of the cord into the sac, the bulk of the cord itself remaining in the spinal canal, which is the commonest type of closed lesion at higher levels, the whole lower end of the cord, except sometimes for the extreme tip of the conus, lies within the sac and is attached to the under-surface of a fibro-fatty mass sometimes containing teratoid elements—cartilage, various glands, and even intestinal mucosa—which itself may be covered by naevoid or hairy skin. At lower levels also, the attachment of the cord to the fundus of the sac was sometimes found at operation to be associated with a visible thinning immediately above the point of fixation, very suggestive of the effects of traction.

There was no case of complete paraplegia complicating closed myelomeningocele above the lumbar region, and even at lower levels, complete paralysis was uncommon. Of the six cases in which it was found, one became paralysed in the post-operative period and the onset of paraplegia was associated with a prolonged and serious attack of septic meningitis, doubtless complicated by inflammation of the cord itself. In the other five cases the swellings were unusually large; one contained no less than 5 pints of yellow cerebrospinal fluid; in

two others the size of the swelling is recorded as 8 in. \times 6½ in. (the height is not stated) and 7 in. \times 6 in. \times 4 in. respectively. The significance of these findings will be discussed later.

OPEN MYELOMENINGOCELES No instance was found of an open myelomeningocele in which cord function was perfect, but there was evidence of some degree of conduction across the lesion in 22 cases (14%), the remainder being paraplegic. The distribution of the two groups is shown in Table IV. Six

TABLE IV
OPEN MYELOMENINGOCELES
CLASSIFIED BY SITE AND BY SEVERITY OF DEFICIT
IN CENTRAL NERVOUS SYSTEM

Site	Partial Paralysis	Complete Paralysis	Total
Cervical	0	0	0
Thoracic	0	0	0
Thoraco-lumbar	6	17	23
Thoraco-lumbo-sacral	0	10	10
Lumbar	10	38	48
Lumbo-sacral	5	55	60
Sacral	1	13	14
Total	22	133	155

of the patients with significant preservation of neurological function survive, and their present condition is set out in detail below. In each case the operative procedure followed the lines set out in our earlier paper.

B.H. (49/4021), born in April 1949, had a small sacral lesion of which the area medullovasculosa was still exposed at the age of 13 days. This was allowed to epithelialize, and an operative repair performed at the age of 2 months. She is now (1963) attending a normal school. The neurological abnormalities comprise a complete sensory and motor loss below the fourth lumbar segment on the left side only with loss of the ankle jerk, plantar response, and anal skin reflex on that side. The child occasionally voids her bladder without warning when she is excited but otherwise has no sphincter difficulties.

C.B. (50/4291), born in April 1950, was first seen at the age of 14 weeks. There was a recently healed lumbo-sacral myelomeningocele, the hospital records accompanying the patient making it clear that this had been an open, granulating area at birth. At this time there was noted to be a paralytic deformity of the left foot, which was flail. The right foot was normal, the pelvic muscles appeared to be working well, and sensation was present in the saddle area. The spine was repaired within the next few days and an uncomplicated recovery followed. Her present (1963) status is good. There is a complete motor and sensory loss in the fourth and fifth lumbar and first sacral segments on the left side only; the right leg is normal and sphincter control is perfect. Following

elongation of the tendo achillis and subsequently a triple arthrodesis, the left foot, though smaller than the right and deformed, is stable and painless.

J.S. (52/3425), born April 1952, had a lumbo-sacral myelocoele of which an area 1.5 cm. in diameter was still raw when she was first seen at the age of a fortnight. There was then paralysis of the left leg below the knee and a complete anaesthesia in and below the fifth lumbar segment on the same side. She was operated upon as soon as the lesion was soundly epithelialized, in June 1952, and after some months in which the head circumference increased rather rapidly, the hydrocephalus became arrested and has not given further trouble. Now (1963) she is of normal intelligence but attends a school for crippled children and has a completely paralysed left lower leg and foot as before, with no change in the degree of sensory loss. The right leg is of normal size and undeformed, an anal skin reflex is present on the right side, and sphincter control is quite normal.

E.W. (53/6877), born October 1953, was seen a few hours after birth with a large thoraco-lumbar myelocoele, and multiple deformities of the vertebral bodies. The area of spina bifida extended from the eighth thoracic to the fifth lumbar vertebrae, but the exact length of exposed cord rudiment is not recorded. There was paralysis of the lower abdominal muscles on the left side with a paralytic lumbar hernia. There was vigorous movement in the right leg, the left was moving weakly; the anal skin reflex was present on both sides. The defect was repaired the same day, the abnormal cord being separated from the surrounding membranes and returned to the spinal canal after its exposed surface had been thoroughly washed with saline. The dura was closed over it and the repair reinforced by fascio-muscular flaps. Recovery was complicated by a long period of wound sepsis and a cerebrospinal fluid fistula, during which time there seemed to be some deterioration in function in both legs. Now 9 years old, she is able to walk with elbow crutches. The right leg is normal but the left is weak and wasted, there being little power at hip or knee and a flail foot. She has no sphincter control and the formation of an ileal-loop bladder is contemplated.

K.L. (57/3489), born August 1957, was seen at the age of 17 days with a leaking lumbo-sacral lesion. At this stage the left leg was weak and there was sensory loss on this side below the fifth lumbar segment. The left knee and ankle jerks were absent.

The exposed area medullovasculosa was slow to heal and repair of the spinal defect was not performed until the baby was 11 months old. The operation was not followed by any substantial change in the neurological state except that the anal skin reflexes disappeared for a few months. At 4 years he began walking with the aid of a below-knee caliper for his left foot, which has a moderate paralytic calcaneus deformity, the calf being wasted. The left knee jerk and both ankle jerks are absent; the right plantar response is extensor, the left absent. This little boy is still incontinent at night but can control his bladder for up to two hours at a time during the day. There is definite sacral sensation on the right side, but not on the left, and despite the mild neurological abnormalities in the right leg, its function is normal.

D.M. (57/3944), also born in August 1957, with a large, thin, open lumbo-sacral myelomeningocoele, was first seen when 5 days old, and was operated upon immediately because rupture of the sac appeared imminent. At 4 years of age he has a paralysis of the right foot (except for some voluntary movement in the tibialis anterior) with a normal left foot. Sacral sensation is present on both sides and there are no sphincter difficulties.

Of the 16 fatal cases, there is none in which it appears likely that, had they lived, there would have been less neurological deficit than in the survivors. All appeared to have at least a complete motor and a partial sensory loss at and below the level of the lesion on one side.

DISCUSSION

Two points of interest arise from the analysis of neurological function in those cases of open myelomeningocoele which have survived. First, there was no particular age at which operation on the spine yielded a better post-operative result in terms of spinal paralysis. Two cases out of five operated upon during the first week of life have done well but so have four out of 98 operated upon later (the difference is not formally significant, $\chi^2 = 2.21$; $P \sim 14\%$) and against the possible advantages of immediate operation (Sharrard *et al.*, 1963) must be set the risk of turning surface infection into meningitis and myelitis (Guthkelch, 1962a). The figures of Sharrard *et al.* (1963) make it clear that sepsis is in fact still a frequent and serious complication of open spina bifida. The second point is that when paralysis is incomplete in open myelomeningocoele, one leg is always less affected than the other and this raises the possibility that one half of the cord may perhaps have become covered by skin during intra-uterine life while the other has remained open and degenerated further. However, no direct evidence of this has been found in the notes of the operative findings in these cases, and many types of congenital deformity of the spinal cord show asymmetrical loss of function in the lower limbs.

There is abundant evidence, both from examination of malformed human embryos (Patten, 1952) and from experimental studies (Fowler, 1953; Warkany and Takacs, 1959), that the more severe degrees of myeloschisis are associated with local overgrowth of embryonic nervous tissue in the affected area. But the exposed position of this tissue on the external surface of the embryo renders it liable to degenerate, the process being often far advanced at birth (Warkany, 1960). Consequently one would expect that in open myelomeningocoele loss of motor and sensory function below the level

of the lesion would be common, as indeed has been known for centuries (Ruysch, 1691).

Our observations, however, confirming those of Sharrard *et al.* (loc. cit.), show that function can sometimes be preserved below the level of the lesion in open myelomeningocele.

In closed myelomeningocele the malformed nervous tissue is in no danger of degeneration from intra-uterine exposure to amniotic fluid, and much less from birth trauma, or from post-natal infection, being insulated from the external body surface by a layer of skin and fibro-fatty tissue and sometimes by cartilaginous and even teratomatous elements as well. In such cases the occurrence of sensory and motor deficit might be explained on the basis of anomalies of the primary development of the nervous system, passive distension of the central canal resulting from hydrocephalic states, or traction. It will be convenient to consider these possibilities in order.

IMPERFECT DEVELOPMENT There is no reason to suppose that degeneration of the nervous system following its exposure on the external body surface for an abnormal period of foetal life is the only cause of failure of full functional development of the cord in spina bifida, for abnormalities of the spinal cord and nerve roots are found even in cases of simple meningocele (Cameron, 1956). The fact that in cases of closed myelomeningocele occurring at all levels of the spine it is the nerve roots issuing from the affected area which are most commonly and most severely paralysed suggests a primary failure of connexion between the cord and its surroundings. Were the cord itself mainly at fault, the long tracts would presumably suffer equally with the roots, and the clinical picture would be different.

DISTENSION OF THE CENTRAL CANAL Gardner (1960, 1961) has recently written in support of the hypothesis, originally propounded by Morgagni, that rachischisis should be regarded as a bursting through of the already closed neural tube onto the body surface in consequence of an internal hydrocephalus, and, if this were the case, the process of rupture might well cause destruction of nervous tissue at the point of its occurrence whether the end-result were a closed or an open myelomeningocele. There are, however, objections to Gardner's hypothesis. Patten (1952) has described and illustrated lumbo-sacral myeloschisis in an 8 mm. embryo, yet telencephalic choroid plexus has never been found in embryos of below 13 mm. in length and not always even in those of 17 mm. (Kappers, 1958), while Weed's (1917) illustrations show little sign of even the myelence-

phalic choroid plexus in embryos below 15 to 16 mm. in length though the latter plexus may slightly precede the former in its appearance during ontogeny. Indeed Kappers doubts whether the human choroid plexus is capable of producing cerebrospinal fluid before the fourth month of foetal life, basing this opinion on observations of the histological structure of the plexus which in the earlier stages of development is quite different from that of the adult. Granted that the ventricular system must be filled with some sort of fluid from the time that it appears, even so the pulse wave emanating from the choroid plexus, which is an essential postulate in Gardner's hypothesis of secondary rupture of the neural tube, cannot be operative at the earliest stage of development at which spina bifida has been described.

TRACTION In their demonstration that traction on the cord in a case of spina bifida cystica with neuroectodermal continuity (to use Gunberg's, 1956, convenient phrase) was inadequate to explain the genesis of encephalo-cranial disproportion, Barry, Patten, and Stewart (1957) were careful to point out that traction might have local effects on the cord and nerve roots, and it is common, at operation on a large myelomeningocele, whether open or closed, to observe that the cord is thinned, apparently by traction, for several segments above its point of attachment to the sac. Except for one case of post-operative infection, all examples of complete paraplegia occurring in closed myelomeningocele were seen in infants with unusually large swellings, and in each case the diameter of the cord was greatly reduced for some way above the lesion. In two of these cases, indeed, there is some reason to believe that cord function deteriorated between birth and the time when they were first seen by us, *i.e.*, at the time when the sac was increasing in size.

These observations suggest that traction on the cord due to undue distension of the sac may in fact cause progressive paraplegia in some cases of myelomeningocele but that when no element of traction is present the outlook for preservation of cord function, when the lesion is a closed one, is good. We would therefore advise that in any case of spina bifida cystica where the sac is tense or increasing rapidly in size, intracranial pressure should first be reduced by one of the standard procedures, *e.g.*, the insertion of a Holter valve, which are now available for this purpose, after which the spinal defect should be repaired in the standard way.

SUMMARY

The degree of paralysis encountered in cases of spina bifida cystica varies with the anatomical

characteristics and situation of the lesion. Three types of lesion are distinguished and termed simple meningocele, closed myelomeningocele, and open myelomeningocele respectively.

In simple meningocele any degree of spinal paralysis is uncommon, and due to associated abnormalities.

In closed myelomeningocele some degree of paralysis is seen in about half the cases but it is suggested that complete paraplegia is only found when the large size or rapid expansion of the cyst causes extreme traction on the cord, and that these complications are therefore an indication for immediate surgical intervention, comprising first a reduction in intracranial pressure and later a repair of the spinal defect.

In open myelomeningocele most of the cases show complete loss of function below the level of the lesion but a few have survived with one normal lower limb and half of these have sphincter control as well. It is therefore certain that neural conduction can occur across such a lesion of the cord, both before and after surgical repair.

Considering all viable cases of myelomeningocele, spinal paralysis tends to be maximal when the lesion is situated at or near to the thoracolumbar junction. Indeed, extensive involvement of this region was almost invariably associated with some degree of paralysis of the lower limbs and four cases out of five had sphincter troubles as well. The incidence of both these handicaps was found to diminish sharply as the site of the spina bifida deformity approached the upper end of the spine,

though in half of the cervical cases the arms were affected. At the caudal end the difference was less striking, there being limb involvement in half the sacral cases and incontinence in two-thirds.

The significance of disordered function of the nervous system in spina bifida is discussed with special reference to the reason for its occurrence in closed myelomeningocele.

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