NERVOUS SIGNS IN SARCOIDOSIS

BY

MICHAEL JEFFERSON, B.M., M.R.C.P.

Lecturer in Neurology, Birmingham University

The occurrence of neurological complications in sarcoidosis, though well established in the literature of the disease, is a fact perhaps still not very widely known. The signs most commonly met are those of lesions of cranial or peripheral nerves. Without any doubt the most frequent disturbance is paralysis of the facial nerve, but retrobulbar neuritis, papillitis, optic atrophy, cycloplegia, ptosis, external ocular palsies of one kind and another, trigeminal sensory loss, nerve deafness, weakness or paralysis of the palate or pharynx or of the vocal cords or tongue, loss of taste, or anosmia, are all more or less often encountered, and so too are motor, sensory, and reflex signs and symptoms, of nerve or root type, in the limbs and trunk. Any of these disorders may occur in isolation, or may be combined with others to present as a multiple neuritis. Rather less often there may be evidence of central nervous dysfunction, usually alone but sometimes in conjunction with peripheral lesions. Focal or generalized fits, hemiplegia, confusion, coma, extrapyramidal syndromes, cerebellar disorders, and symptoms indicating mischief in the brain stem or spinal cord have all been reported. In particular, diabetes insipidus from hypothalamic or pituitary damage has been recorded a number of times. Lastly, symptoms of meningeal involvement are occasionally seen, with headache, neck stiffness, a raised cerebrospinal fluid protein content and white-cell count, and sometimes reduction of the sugar and chloride content.

The four case records which follow are presented to exemplify some of these features: the first two patients had polyneuritic syndromes, the next a spinal cord disorder, and the fourth a combination of central and peripheral nervous lesions.

Case 1

A married woman aged 29, a patient of Dr. Ernest Bulmer, who kindly allowed me to see her, became ill just before Christmas, 1950, with bilateral swelling of the parotid glands, at first thought to be mumps. However, the swelling did not subside, and after six weeks severe iridocyclitis of both eyes supervened. It became clear that she was in fact suffering from the uveoparotid form of sarcoid, first described by Heerfordt in 1909.* Her eyes remained inflamed and her sight dim for about eight weeks, and the parotids were swollen for some four months in all. During this time she felt ill, with lack of energy, poor appetite, and dry mouth, and lost weight steadily at the rate of 1 to 2 lb. (450 to 900 g.) a week. She observed of her own accord (because they were tender to the touch) that the lymph nodes in various parts of her body were enlarged, and a chest *x*-ray film showed that the typical radiological signs of pulmonary sarcoid were also present.

In April, 1951, she had the first of what proved to be a series of neurological symptoms, which came on in turn until mid-August. The initial complaint was drooping of the left upper eyelid and fixed dilatation of the left pupil, which made it difficult to focus near objects with that eye. At the beginning of May she had diplopia, and a little later the left side of her face and inside of the mouth became numb. In early June pain developed in the left leg, and with its onset she became aware of constant tingling in the front of the thigh and shin, and a tendency for the limb to give way when walking. More or less simultaneously pain started in the left arm down its outer side from shoulder to mid-forearm level, and the hand felt constantly cold. At this time too the right axilla felt numb and dead for a short while. A few weeks later, at the end of June, the right arm and leg were affected with rather similar symptoms, which came on (she said) very abruptly in the space of about three hours. In July the skin over the right lower ribs felt frozen, and, finally, on August 12 she woke to find the right side of her face paralysed.

During the period of evolution of her nervous troubles she had first a fine nodular eruption on her forehead, which had apparently been more palpable than visible, and then crops of purplish-red lesions on the shins and extensor aspect of the forearms, which she likened to chilblains. Her family and social history and her previous medical record were blameless.

On examination (August 17) she was haggard and thin, her weight being nearly 3 st. (19 kg.) below its normal level. There were no clinical signs in the chest, the liver and spleen were not enlarged, and only a few small glands were palpable in the right supraclavicular fossa and in both groins. The salivary and lacrimal glands were not swollen, nor was there any sign of uveitis. The various skin eruptions of which she had complained had vanished, leaving no trace beyond some shiny pigmented patches, desquamating at their edges, over both tibiae. The salient neurological features were as follows: the left pupil was widely dilated and immobile, the right rather smaller and responding sluggishly to light and on convergence. There was bilateral ptosis, slight but definite on the right and obvious on the left, and diplopia in all directions of gaze, especially to the sides, without visible fault in eye movements. There was somewhat impaired touch and pain sense in all divisions of the left trigeminal nerve, most marked in the third, and the corneal reflex was depressed. Complete right facial palsy of lower motor neurone type was present. In the arms there was slight blunting of sensory acuity of root pattern, from the eighth cervical to the second thoracic dermatome on the right, and in the territory of the fifth and sixth cervical dermatomes on the left. Both legs were a little weak, the left slightly worse than the right; the left knee-jerk was greatly diminished, and the ankle-jerks were both absent. There was mild objective numbness from the fourth lumbar to the first sacral root inclusive in the right leg, and in the left from the second to fifth lumbar root.

I saw her again on October 15. She had made a great symptomatic improvement, and was scarcely recognizable as the same person. Her only spontaneous complaint now was of shock-like paraesthesia in the arms and legs, mostly at night, which made her limbs jump. The facial palsy had largely but not completely recovered, and her other signs had also become much less easily demonstrable.

Case 2

A 47-year-old widow was taken ill early in December, 1945, with a vague febrile malaise. Two or three weeks later her fingers gradually went numb one by one, starting with the middle finger of the left hand, and the grip of both hands became weak. Soon afterwards a constant tight feeling round the lower ribs came on, more on the left side than on the right, with recurrent girdle pains. She discovered that the skin felt peculiar and uncomfortable, if she touched it or her clothes rubbed it, in a band on either side from ribs to navel. The power in her legs became increasingly disturbed till she could scarcely walk or stand, and both big toes became numb. She also had intermittent pains in both arms and legs. These symptoms took some weeks to evolve, remained at full intensity for a further few weeks, and then slowly receded. They were already

*It is now generally recognized that uveoparotitis is simply a phase or variant of sarcoidosis (Bruins Slot, 1936; Longcope and Pierson, 1937; Bruins Slot *et al.*, 1938; Sandbacka-Holmström, 1940; Roos, 1940).

beginning to recover somewhat, when, in April, 1946, the sight in the right eye became dim and foggy; a fortnight later the left followed suit. On May 24 she was admitted to the Queen Elizabeth Hospital under the care of Professor P. C. P. Cloake. Examination at this time showed bilateral diffuse uveitis, with posterior synechiae, keratic precipitates, and vitreous haze—findings which accounted for the depression of her visual acuity. The pupils were dilated and fixed, the right slightly larger than the left, there was impaired sensation in the finger-tips of both hands and on the trunk in the distribution of the seventh to the ninth thoracic segments, and diffuse weakness of both legs of moderate severity. The arm-jerks were diminished, the knee-jerks very weak, and the ankle-jerks absent. The other systems were clinically normal.

A chest *x*-ray film showed increased hilar shadows with fine reticulation of the parenchyma, and serum protein estimation a relative increase of the globulin fraction, though the total was not definitely increased. A Mantoux test was negative at 1:1,000. C.S.F. examination gave a normal fluid with three lymphocytes per c.mm., 26 mg. of protein per 100 ml., negative Pandy test and Nonne–Apelt reaction, and Lange curve 0011000000. The C.S.F. and blood W.R. were both negative.

The patient was in hospital about five weeks, during which time she made rapid progress towards recovery. Ever since then she had been kept under observation as an outpatient. I saw her on September 6, 1951. She said that it was 12 to 18 months after the onset of her illness before she felt really well, but in the past four years her health had been good. Her only complaints were of occasional tight feelings round the trunk and sometimes of faint numbness in the finger-tips. Nothing significant could be discovered in her previous medical, social, or family history. On examination the pupils were still unequal and moderately dilated, and almost completely inactive to light and on convergence. The triceps-jerks and all the leg-jerks were still absent, and there was still a demonstrable band of hyperaesthesia on the trunk in the same distribution as before, though no abnormality could be found in the finger-tips even with two-point testing. The chest *x*-ray picture was now perfectly normal, and the plasma proteins were normal in total and ratio.

Case 3

A 39-year-old joiner sought medical advice in 1944 on account of chronic cough, with sputum occasionally blood-stained, and shortness of breath on exertion. He was at first suspected of having phthisis, but in 1948 a generalized enlargement of lymph nodes and an enlarged liver and spleen were discovered. Lymph node biopsy was then carried out, which showed the characteristic histological features of sarcoid. From 1948 onwards he had had recurrent inflammation of the eyes of variable duration, one attack of apparently simple pneumonia, and a transitory swelling of the left testicle, which lasted about eight weeks. His nervous complaints dated from April, 1950, when he noticed that his feet and legs felt continuously cold, and that their strength was beginning to fail a little. The enfeeblement of his legs progressed, and it was gradually borne in on him that feeling was blunted not only in the legs but on the trunk up to about waist level, and his sphincters became difficult to control.

On December 5, 1950, he was admitted to the neurosurgical unit of the Queen Elizabeth Hospital under Mr. J. M. Small. I saw him a few days later. Neurological examination then demonstrated a profound spastic paraplegia, with cutaneous sensory loss in the trunk and lower limbs, the upper border of which was at about the sixth thoracic segment on each side, and gross defect of postural and vibration sense. The signs were essentially those of a central cord lesion. General examination revealed moist sounds in both lungs, palpable liver and spleen, mild widespread lymphadenitis, bilateral chronic phlyctenular conjunctivitis, and a curious rash round the mouth and over the lower jaw, consisting of many closely placed flat-topped yellowish little papules set upon an erythematous background.

Various investigations were made. Chest *x*-ray examination demonstrated a heavy fibrosis of both lungs. The sputum was negative for tubercle bacilli on several occasions, the Mantoux reaction was negative, and the serum proteins were normal. A full blood count was normal, but the E.S.R. (Wintrobe) was 28 mm./hour. The C.S.F. protein was elevated (236 mg. per 100 ml.). C.S.F. and blood W.R. were both negative. Myelography was quite normal, and a diagnosis of a sarcoid lesion in the mid-thoracic segment of the spinal cord was made.

Through the kindness of Dr. I. Gallant, the patient was seen again at St. Chad's Hospital, Birmingham, early in October, 1951. There was little change in his neurological or general physical state. His legs were spastic, and, except for a flicker of movement in the toes, completely paralysed. There was knee and ankle clonus with briskly upgoing toes. Postural and vibration sense were grossly disturbed in the lower limbs, and there was still a sensory level for touch, pain and temperature at the sixth thoracic dermatome, with relative escape of the sacral segments bilaterally. The arm-jerks were somewhat exalted on both sides, a finding which had been noted the previous December, but otherwise the arms and cranial nerves were normal.

Case 4

A housewife aged 41 was first admitted to the Queen Elizabeth Hospital under Professor W. H. Wynn on May 25, 1944. She then gave a history of having had Bazin's disease of the legs from the age of 14 until she was 35. Two years before admission to hospital she had developed a cough with sputum, and had begun to lose weight steadily and to feel unwell. Twelve months later her hands and feet had been numb for some time, and since then recurrent small lumps had appeared in the skin of the face and limbs. For six months she had been excessively thirsty and had to pass urine very often.

On examination she was thin, being some 2½ st. (15.9 kg.) underweight. There were numerous old ulcer scars on both legs, relics of her earlier erythema induratum, palpable glands in the axillae, an easily palpable spleen, moist sounds in both lungs, and slight hypertension (B.P. 170/100). There were also some small raised rose-red lumps 1 to 1.5 cm. in diameter on both forearms, and a slight but definite weakness of the left side of the face of peripheral type with deviation of the tongue to the left on protrusion. The chest *x*-ray picture showed a little hilar enlargement and bilateral basal fibrosis. The serum proteins were normal. She was observed to have an irregular low fever, and it was confirmed that her fluid intake and output were abnormally high. A diagnosis of sarcoidosis with diabetes insipidus from hypothalamic involvement was made, and the patient was sent out of hospital on June 10.

On August 3 she was readmitted, complaining that since going home her limbs had become stiff and weak, and her gait restricted, and that one week previously the right side of her face had become paralysed. On examination there was complete infranuclear palsy of the right seventh nerve, but the other cranial nerve signs previously described had gone. There was some weakness of the arms and legs, with rather variable spasticity, increased tendon-jerks, and extensor plantar reflexes, but no objective sensory disturbances. In less than a month the facial palsy had almost completely recovered, and the signs of spinal cord dysfunction were thought to have diminished. Treatment with pituitrin snuff controlled the patient's diabetes insipidus very satisfactorily, and she was sent home on this drug.

On September 26, 1945, she was admitted for a third time with complaints of lassitude, intermittent pains in the limbs, and twitching of the legs. There were no cranial nerve signs, but a mild spastic tetraparesis was still evident, with flexor spasms in the legs. General physical examination was essentially as before. The C.S.F. protein and cell count

were normal, also the Lange curve. The C.S.F. and blood Wassermann reactions were negative. A chest *x*-ray film showed increased basal lung markings as before, and the serum protein ratio was now abnormal (albumin 3.5 g., globulin 3.3 g.).

She was again in hospital in the summer of 1946, under Dr. J. M. Malins. She now had to use a stick to get about even in her own home. Six months previously she had had bilateral uveitis, which recovered slowly, and a week before admission a left peripheral seventh-nerve paralysis had come on. The pyramidal signs in her limbs were unchanged or perhaps a little more severe. In April, 1947, when examined once more in hospital, the liver and spleen were both easily palpable, there were firm glands in the neck and armpits, and a fading sarcoid lesion of the skin on the right wrist and another over the proximal phalanx of the right middle finger; her blood pressure had risen to 185/105. Neurologically she was *in statu quo* so far as her limbs were concerned; the left facial palsy had made only imperfect recovery, and the right pupil was now dilated and fixed. *X*-ray films of the hands showed no sign of osteitis multiplex cystica. The sedimentation rate was 20 mm./hour (corrected). Serum proteins: albumin 3.7 g., globulin 3.8 g.

Six months later she was in congestive heart failure, and while being treated developed occlusion of the popliteal artery, which necessitated amputation of her left leg below the knee. A year later (November, 1948) she had to be treated, again in hospital, for heart failure; her neurological signs were stationary. In January, 1949, she was transferred to a home for incurables, and her subsequent history is unknown.

Comment on Cases

In contrast to Case 1, in which nervous derangements appeared only at a relatively late stage, Case 2 actually presented with a neurological symptomatology.

Cases 1 and 2 are perfectly typical in their course and symptomatology of polyneuritic sarcoid, and can be matched by a score or more of published examples. Case 3, on the other hand, is one of sarcoid involvement of the central nervous system.

In the context of Case 3 it may be remarked that the prognosis for recovery of nervous function is very much better with peripheral than with central lesions, a point which is also attested by Case 4, in which the spinal cord signs showed little or no improvement, though there was recovery from more than one attack of infranuclear facial palsy. The notes on Case 4 were taken from the records of the Queen Elizabeth Hospital, and this patient never came under my observation at any stage of her illness.

In Case 4 there were signs and symptoms of both peripheral and central nervous involvement, as witnessed by the recurrent facial palsies of lower motor neurone type and spastic tetraparesis; the diabetes insipidus which was a prominent feature at one stage was presumably caused by a sarcoid deposit either in the hypothalamus or in the pituitary itself. The early history of Bazin's disease suggests the possibility that the latter may be a form of cutaneous sarcoid: descriptions of the histology of the lesions of erythema induratum are not contrary to such a view.

Discussion

A reading of the more easily available literature on sarcoidosis suggests that the incidence of nervous complaints in the uveoparotid form of the disease is so high that it constitutes an almost regular feature of the syndrome. All three of the cases upon which Heerfordt (1909) based his original account of "subchronic uveoparotid fever" had such troubles: the first (a boy of 11) had optic neuritis; the second (a boy of 14) facial palsy and difficulty in swallowing; and the third (a man of 27) facial palsy, difficulty in swallowing, and sensory upset in hands and trunk. He cited from earlier writers two further cases, which he grouped with his own: one had facial paralysis and loss of taste in one-half of the tongue and the other retrobulbar neuritis. Other examples with a wide variety of complications—mainly polyneuritic—were later published by numerous observers (Mackay, 1917; Maitland Ramsay, 1921; Feiling and Viner, 1921–2; Macbride, 1923; Critchley and Phillips, 1924; Parker, 1926; Rogers and Bodman, 1926; Merrill and Oaks, 1931; Mohn, 1933; Garland and Thomson, 1933, 1934; Lloyd Davies, 1934; Savin, 1934; Tait, 1934; Levin, 1935; Waldenström, 1937; Bruins Slot *et al.*, 1938).

On the other hand, nervous symptomatology in the Boeck–Schaumann variants seems to be a relative rarity. Thus Bodley Scott (1938) described eight cases, none of which had nervous signs; and Longscope (1941), who published observations derived from experience with 31 cases, had met them very seldom. Their absence is also a striking feature of Alsop Riley's (1950) series of 52 personal cases. According to Salvesen (1935), who reported a patient with optic neuritis, nervous lesions had previously been observed only by Winkler in 1905, by Mazza in 1908, by Urban in 1910, and once by Boeck himself (who was, however, inclined to regard his case as due to coincident leprosy). Schaumann (1936) made passing reference to sarcoid lesions in the nervous system as an infrequent part of the disease. In the past 10 to 15 years a number of further case descriptions have accumulated (Roos, 1937; Bruins Slot *et al.*, 1938; Lindau and Löwegren, 1940; Colover, 1948; Williams, 1950). It would appear, therefore, that there is a differential frequency of occurrence of nervous signs within the group of sarcoid disorders which would bear further investigation. It is possible that the difference may be no more than the result of a bias in medical reporting, but, at least superficially, it seems too large to be thus explained away.

With regard to pathology, neurological symptoms are caused by the presence in the neural substance of epithelioid and giant-cell follicles without caseation, just like those found in other tissues. How the lesions come to be present in the nervous system is a point which deserves brief consideration. Schaumann (1936), who did much to unify the diverse clinical manifestations of sarcoid, always insisted that it was primarily and electively a system disease, an affection of the reticulo-endothelial system, and it is now generally accepted as a reticulosis. The primitive mesenchymal cells of Maximow, which (as in other reticuloses) are the target of attack, are present in every tissue of the body, though more richly in some than in others, and it is their proliferation and differentiation which gives rise to the characteristic lesions. Sarcoid follicular systems in the cerebrospinal axis are thus to be looked upon simply as a sign of local reaction by indigenous mesenchymal cells, part and parcel of a widespread reticular response to whatever is the cause of the disease.

What the aetiology of sarcoidosis may be is still unknown. In the past there has been much discussion of its relationship to infection with the tubercle bacillus. It has been postulated that it is the result of an anomalous response (anergy) on the part of the host's tissues to invasion by the organism, or that the organism itself is of attenuated virulence, or that both factors act simultaneously. However, no really satisfactory proof of the causal nature of Koch's bacillus has ever been given. Though it seems that in something like 10% of cases frank tuberculosis—for example, pulmonary, renal, or meningeal—is either the immediate or a contributory cause of death, a similar incidence is to be met with in other conditions, such as Hodgkin's disease or silicosis. In each instance the coincidence of the primary illness with tuberculosis can more easily be interpreted as meaning that the former predisposes the subject to infection with the latter than to suppose that tubercle was, all along, the secret cause which only declared itself terminally.

It would seem wisest to account sarcoid a syndrome—a malady, that is, which may perhaps be set in motion by any one of a host of factors, acting singly, in concert, or in sequence, rather than to hold it a disease in the old strict sense of a morbid process having a unitary and unvarying

cause. There is already apparent some tendency to adopt such an attitude, and even to look upon it as yet one more example of failure of bodily adaptation to stress (Selye, 1951).

As to its treatment, recent reports of initial success with cortisone or A.C.T.H. (Siltzbach *et al.*, 1951 ; Lovelock and Stone, 1951 ; Small, 1951) are encouraging, but it is too early yet to assess their real value. No remedy used in the past has proved of an unequivocal value.

Summary

Neurological complications in sarcoidosis are occasionally met with, more commonly in the uveoparotid form of the disease than in other variants. Most often they are of the peripheral neuritic type, but disorder of central nervous function also occurs, with symptomatology proper to the region involved. Four clinical case records are presented by way of example. Sarcoid lesions in the nervous system, whether in nerves or cerebrospinal axis, are the same in histological structure as those seen in other tissues, and, like the latter, presumably represent reaction of indigenous mesenchymal elements to whatever may be the cause (or causes) of the syndrome of sarcoidosis.

I wish to express my thanks to Professor P. C. P. Cloake, Professor W. H. Wynn, Dr. Ernest Bulmer, Dr. J. M. Malins, and Mr. J. M. Small for access to their case records and for the opportunity of examining patients under their care.

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Attention has once again been drawn to the relatively high cost of German medical and scientific periodicals (Bonser and Russell, *Nature*, 1952, **170**, 446). As long ago as 1928 Dr. W. Bonser remarked on the exorbitant cost of German periodicals, and his efforts achieved a reduction of some 20% by 1933. While there is a slight rise in the cost of British and American journals since 1933, due to increased cost of production, the ratio of increase of German periodicals compared with British is over 4 : 1. Before the war complaints were not only of the excessive cost but also of the impossibility of computing the annual cost, owing to irregularity in the number of volumes per year. This same state of affairs exists to-day. "We are sure that it will be agreed that their (German scientists') output is as essential to research as that of other countries, and curtailment of its circulation owing to prohibitive costs is to be deplored."

A NEW DIABETIC CHART

BY

PHILIP L. ROBINSON, M.D., M.R.C.P.
Senior Medical Registrar

AND

E. T. BAKER-BATES, M.D., F.R.C.P.
Consultant Physician
Clatterbridge General Hospital, Bebington, Cheshire

Practically every physician having charge of diabetics uses a different form of chart for recording the results of tests and treatment given during the course of standardization in hospital. The amount of detail given on the chart, and the guidance given to those using it, will depend not only on the ideas of the physician in charge but also on the circumstances in which it is used. Our problem is the treatment of a large number of diabetics, many of whom are scattered in numerous non-medical wards throughout a large general hospital. In these wards the nursing staff, and in many cases the medical staff, have had little experience in the practical management of diabetes, and it is left to us to supervise the treatment. Partly to meet this difficulty, but also for use in the numerous medical wards, we have devised a new chart, which we feel is worth publishing as it embodies several principles, as well as details, which we have not previously seen. Our aim has been to make it an easy chart to fill in and to read, and at the same time to allow flexibility so that it may be easily adapted to meet the varying requirements of different patients, different stages of standardization, different meal-times, and out-patient use.

Diet Panel

For most of our cases we use a simple unweighed diet, as devised by R. D. Lawrence (1950). This allows an average intake of protein and fat (except in obese cases), and detailed restriction is of carbohydrate only. The carbohydrate is allowed in "portions," each containing approximately 10 g. The number of portions allowed at each meal is recorded in the first column of the diet panel, and if the diet is altered during the period covered by the chart the revised allotment is recorded in the second column, the date of change being written at the top of the column. The revised allotment is, of course, put in the first column of the succeeding chart.

The size and isolation of the panel make it stand out clearly, so that doctors, nursing staff, and patients can see at a glance the carbohydrate allowance at each meal and the time at which it is taken.

Times of Urine Specimens

The most important innovation on the chart is that it draws the attention of all concerned to the importance of *the time over which any specimen of urine is actually secreted.* Blank spaces are provided for recording not only the time at which specimens are passed but also *the time at which the bladder was previously emptied.* We have found this a much more satisfactory method than having fixed times printed on the chart, as the physician may then choose the time of routine urine tests to fit in with whatever meal-times occur in the different wards. He may also arrange to observe either "short-period" or "long-period" specimens, depending on the type of case with which he is dealing. As a routine we arrange for short-period specimens to be tested before the main meals of the day, the bladder being completely emptied half an hour before the