

by different observers by slightly different symptomatic measures, as well as in some cases by different antibiotics in addition to cortisone or A.C.T.H. administered by different routes in varying doses for varying periods—came to the conclusion that cortisone was beneficial in diminishing pain in and swelling of the testicle, when compared with a group, not necessarily equivalent, treated symptomatically only. However, in their properly controlled trial Klemola and Somer (1956) found an absence of the effect of steroids on the symptoms of orchitis similar to that in our study, but in their cases the temperature did remain raised for a little longer. Mongan's (1959) findings were similar.

The fact that cortisone appeared to have no effect on the later history of the affected testicles which may be related to the duration of the original swelling agrees with its absence of effect on the latter. It was of interest to observe changes in testes not affected to our knowledge at any time by an acute process, and it would have been of great interest to examine the testicles of all the men in the depot on arrival and follow them up at intervals throughout the epidemic and after, whether or not they had had clinical mumps.

Abnormality of liver-function tests was mentioned by Petersdorf and Bennett (1957), and some possible disturbance was found in 6 of our 11 patients in whom it was sought. These patients were selected only in so far as they were among the last 13 patients in our series.

Finally, although no testicular biopsies or sperm counts were performed on these patients, it is hoped that they may all be followed up for some years so that further testicular changes and their effect may be observed.

#### Summary and Conclusion

During an epidemic of mumps in an Army depot all the 35 cases of orchitis were admitted to hospital and treated with bed rest and simple analgesics. Half, selected at random, were also given a six-day course of cortisone as described above. One case was later excluded because of the absence of serological evidence of mumps, which was present in the other 34 cases. All the patients were examined daily, and observations were charted on a previously prepared form by one of two observers. All the patients were seen again approximately three, five, and seven months later by one or other of the observers.

The following observations were made: (1) At the time of the acute illness cortisone in the dosage administered had no statistically significant effect on the duration of the swelling, the pain, or the tenderness of the affected testicle, even in cases treated within 48 hours of onset. (2) In the cortisone-treated group the duration of the pyrexia was slightly reduced from a mean of 2.6 days to one of 1.1 days; this reduction was found to be statistically significant ( $P < 0.01$ ); the mode of termination of the pyrexia had no relation to treatment or to the sequelae. (3) Testicular degeneration, as evidenced by a loss of consistency and a decrease in size leading in some to complete atrophy, was found to be progressive for at least five months after the acute attack; it did not appear to have been influenced by previous cortisone therapy. (4) The ultimate state of the testicle some seven months after the acute attack did not appear to be related to the duration of the pain, the tenderness, or the pyrexia, but may perhaps have been influenced by the duration of the swelling at the time of the attack. (5) Cortisone appeared to depress the forma-

tion of complement-fixing antibody, but the level of the latter did not seem to be related to the symptoms, signs, or sequelae of the attack. (6) The serum globulin and thymol turbidity were slightly raised in 6 of the 11 patients in whom they were determined.

It is thought that neither the previously reported cases nor the present trial show any evidence of the efficacy of steroids in the treatment of mumps epididymo-orchitis.

We are grateful to Brigadier M. F. H. Kelleher, late R.A.M.C., for his enthusiastic support, and to Lieutenant-Colonel A. D. Young, R.A.M.C., for his encouragement throughout. The trial would have been impossible without the continuous co-operation of Drs. Frank Scott and P. J. Murphy, formerly Captains, R.A.M.C., at the Gurkha Depot, Sungei Patani, to whom we are indebted. We thank Lieutenant-Colonel Hinton J. Baker, M.C., for the virus studies performed at the U.S.A.M.R.U.; Drs. B. L. Elisberg and P. A. Webb for much stimulation; and Dr. David B. Evans, formerly Captain, R.A.M.C., for the routine investigations.

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## CAUDA EQUINA LESIONS ASSOCIATED WITH ANKYLOSING SPONDYLITIS REPORT OF THREE CASES

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Ankylosing spondylitis is a not uncommon disease which has been extensively studied and described. Recent reviews of the disease include those of Baird *et al.* (1955), Hart (1955, 1959), Baird (1956), Swezey *et al.* (1957), Lefkovits and Thomas (1958), and Graham (1960). A search of the available literature has revealed no description of an association between this disorder and lesions of the cauda equina.

We have observed three patients with long-standing, quiescent spondylitis who developed cauda equina lesions many years after the active disease process had settled. Investigation revealed no cause for these defects. There is no direct evidence that the neurological abnormality is caused by the ankylosing spondylitis, but the similarity of the clinical picture in these three patients is such as to suggest that a causal relationship may well exist.

**Case 1**

A 53-year-old man was admitted to Green Lane Hospital with staphylococcal pneumonia. At the age of 17 years he developed ankylosing spondylitis which progressed with pain and increasing rigidity until he was 32. In spite of treatment with physiotherapy over this period, the end-result was a rigid, moderately kyphotic spine and a fixed thoracic cage. He did not receive x-ray therapy. Both hips were ankylosed at an angle of 90 degrees, so that he walked with difficulty. When he was 43 bilateral osteotomies enabled him to walk erect with crutches. At the time of writing he was able to work as a clerk and to drive his own car.

For two years he had complained of frequent bowel motions, occurring four or five times daily. They were well formed and were accompanied by profuse mucus. Rectal sensation had been impaired for 18 months but control was adequate. Sigmoidoscopy one year after the onset of symptoms revealed a granular proctitis.

For 12 months he had been troubled by urinary frequency of increasing severity until he was passing urine seven or eight times by day, and as often by night. While awake, bladder sensation was normal except that the feeling of a full bladder came on precipitately. There was stress and urge incontinence. At night he was often incontinent. Urine flow was intermittent with a poor stream.

Two years before admission he noticed a sensation of pins-and-needles over the lateral aspect of the left foot. This persisted, and 12 months later an area of numbness appeared on the back of the left thigh.

Other symptoms were related to his current pneumonia and to a previous history of lung abscess and bronchopleural fistula.

On examination his spine, neck, and hips were fixed in a functionally adequate position. There was an area of diminished sensation to touch, pin-prick, temperature, and deep pain sensation over the area supplied by all the sacral and coccygeal nerve roots on the left side. A poorly defined area of increased sensitivity to pin-prick was present over an area on the right buttock. Position sense was normal. There was an indolent ulcer in the left buttock crease (Fig. 1). Gross fasciculation was present in the left calf muscles. The left ankle-jerk and plantar response were absent, but all other reflexes were normal. There was no muscle-weakness; tone and co-ordination were normal. No other neurological abnormalities were found. On digital rectal examination the prostate was small and of normal consistency, but the external sphincter was lax. Examination of the other systems revealed considerable lung disease but no other relevant findings.

**Investigations.**—Peripheral blood showed: haemoglobin, 13.8 g./100 ml.; leucocyte count, 5,500/c.mm.; sedimentation rate, 15 mm. in one hour. The alkaline phosphatase content of the serum was 6 King-Armstrong units. The Wassermann and Laughlen reactions were negative in the blood. The cerebrospinal fluid was under a pressure of 110 mm. The manometric responses were normal. The protein content was 38 mg./100 ml.; 166 red cells and 2 leucocytes per c.mm. were pre-

sent. The Lange curve read 000000, and the Wassermann reaction was negative. X-ray examination of the spinal column showed complete ankylosis throughout, and similar changes in the sacro-iliac joints and symphysis pubis. No abnormality of the neural foramina was detected. Lumbar myelography outlined prominent diverticula along the lumbar nerve-root sheaths but was otherwise normal.

**Case 2**

A 50-year-old man was admitted to Cornwall Hospital for treatment of his ankylosing spondylitis. This first appeared at the age of 20 years, and, despite treatment with physiotherapy and deep x-ray therapy to the spine, the disease progressed until after two years the hips were ankylosed and the lower spine was rigid. There was little movement in the upper spine, but the neck and shoulder-joints were unaffected. With crutches he could walk slowly but was not able to work. His condition remained unaltered until one year before admission, when he complained of further stiffening of the thoracic spine.

For six months his usual tendency to constipation had become more marked, so that instead of passing a motion every two days, he passed only one weekly. For two months he had experienced increasing difficulty in beginning micturition. The urine flow was poor, and he had a sensation of incomplete emptying of the bladder. There was some increase in frequency and he was often incontinent while asleep.

He had not noticed any sensory changes in his legs, but a cup of tea spilt over the back of his left leg caused a deep burn with little pain.

On examination his spine and hips were ankylosed but there was full shoulder and neck movement. Over the distribution of all the sacral and coccygeal nerve roots in the right leg, touch, pin-prick, and temperature sensation were severely impaired. There was an inconstant diminution of all forms of cutaneous sensation over a similar distribution on the left side. A healing burn on the left thigh had

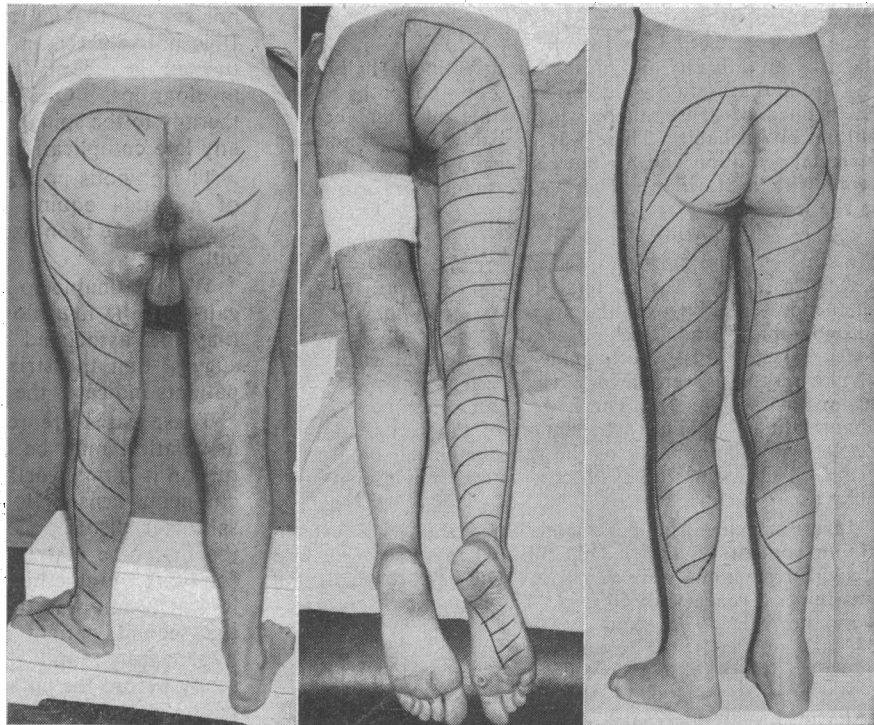


FIG. 1

FIG. 2

FIG. 3

FIG. 1.—Distribution of cutaneous sensory loss in Case 1, and ulcer on left buttock. FIG. 2.—Distribution of cutaneous sensory loss in Case 2. The bandage on the left thigh covers a comparatively painless healing burn. FIG. 3.—Distribution of cutaneous sensory loss in Case 3.

been comparatively painless (Fig. 2). There were no trophic changes in the legs. Muscle power was full and tone was normal. There was no muscle-wasting and no fasciculation was seen. The right ankle-jerk was absent and the left diminished. Both plantar responses were absent. No other neurological abnormalities were found. Rectal palpation revealed a moderately enlarged prostate of normal shape and consistency. The tone of the sphincter was fair. Examination of the other systems showed nothing contributory.

*Investigations.*—The haemoglobin content of the peripheral blood was 13.8 g./100 ml., and the leucocyte count 5,000/c.mm. The sedimentation rate was 32 mm. in one hour. The alkaline phosphatase content of the serum was 12 King-Armstrong units. The Wassermann and Laughlen reactions were negative in the blood. The cerebrospinal fluid contained 15 mg. of protein per 100 ml., 380 red cells per c.mm., and 1 leucocyte per c.mm.; the Lange curve read 000000; the Wassermann reaction was negative. X-ray examination of the spine showed a striking "bamboo spine" with ossification of the interspinous ligaments. No abnormality of the neural foramina was seen. Myelography, screened in the supine position owing to his inability to lie prone, showed a series of indentations at each vertebral level, pooling the "myodil" between them. These findings were regarded as an exaggeration of the normal appearances seen in the supine position, and were probably not of significance. There was no obstruction to the flow of contrast medium in the lumbar theca.

### Case 3

A 51-year-old man was admitted to Auckland Hospital for investigation of urinary incontinence. Ankylosing spondylitis first appeared at the age of 35. Over the course of several years his spine and neck became completely rigid in spite of deep x-ray therapy, but his hips and shoulders escaped. He was able to walk freely without sticks, in a good position.

Four months before admission he had gradually developed incontinence of urine at night and occasionally by day. Over the same period his stream had become poor. It was started with difficulty and finished in dribbling. His bladder and urethral sensation were normal. For six months he had complained of constipation, which came on after years of normal bowel habit. There was probably some diminution of rectal sensation and his bowels tended to move involuntarily with micturition. There were no sensory symptoms in the legs.

On examination his spine and neck was rigid in a functionally good position and he was able to walk without assistance. There was bilateral diminution of all forms of cutaneous sensation over the areas of skin supplied by the second, third, and fourth sacral nerve roots and the coccygeal segments. Both ankle-jerks were absent. Both plantar responses were flexor. There was no muscle wasting or weakness (Fig. 3). There were no other neurological abnormalities. Digital examination of the rectum did not disclose any abnormality of the rectal sphincter or prostate gland. The findings on cystoscopy and sigmoidoscopy were normal.

*Investigations.*—The peripheral blood showed a haemoglobin content of 13.3 g./100 ml. The Wassermann and Laughlen reactions were negative. The cerebrospinal fluid was under a pressure of 60 mm. The manometric responses were normal. The fluid contained 37 leucocytes and 353,000 red cells per c.mm., attributed to a traumatic puncture. The protein content was 84 mg./100 ml. The Lange curve read 110000. X-ray examination of the lumbar spine and sacrum revealed only the complete ankylosis of all joints of the lumbar spine and sacro-iliac joints characteristic of ankylosing spondylitis. No abnormality of the neural foramina was seen. Lumbar myelography showed no abnormality.

### Discussion

The striking similarity of the clinical pictures in these three patients is apparent. The onset of ankylosing spondylitis occurred between 16 and 26 years before the appearance of neurological symptoms, the active phase of the rheumatic process having long passed. The presenting symptom in each case was a disturbance of sphincter control characteristic of a bilateral cauda equina lesion, with nocturnal incontinence of urine, poor stream, and diminished bladder and rectal sensation. Changes in cutaneous sensation and ankle tendon reflexes were found in each patient, indicating a bilateral lesion of the cauda equina affecting the first-sacral-nerve roots at the highest level. All the roots below this level were clearly affected on both sides in one patient, while in the other two the lesion was asymmetrical but with evidence of some involvement on the relatively unaffected side. The onset was insidious in each case, and unassociated with pain, the course of the neurological symptoms being apparently slowly progressive. The cerebrospinal fluid was normal in two patients, and in the third the increased protein content could be attributed to the contamination with red blood cells owing to the difficulty in carrying out the lumbar puncture.

Myelography revealed no evidence of compression of the nerve roots in their intrathecal course, and no radiographic abnormality was found in the sacral neural exit foramina.

The pathogenesis of the progressive cauda equina syndrome in these patients remains unexplained. The normal myelographic appearances exclude compression of the nerve roots by a posterior prolapse of a lumbar intervertebral disk, or by proliferative bony or cartilaginous changes on the posterior aspect of the lumbar vertebrae. The myelographic appearances did not suggest that an adhesive arachnoiditis was present. It is unlikely that a cauda equina or conus medullaris tumour is present in three such patients with normal myelograms. One patient has not had deep x-ray therapy to the spinal column, and this appears to exclude any late complications of this treatment.

The obvious possible explanations for the association of a cauda equina lesion and ankylosing spondylitis seem to have been excluded by the investigations carried out.

We are unable to offer any suggestion regarding the cause of the neurological lesion or the way in which it may be associated with the rheumatic disease, but suggest that the striking similarity between these three patients indicates the possibility of a causal relationship.

These cases are reported in the hope that a similar association may be observed by others. In this connexion it is noteworthy that no complaint relating to the cutaneous sensory loss which showed the nature of the sphincter disturbance was made by these patients. In the first case rectal incontinence had been investigated for some months before it was recognized that a neurological lesion was the cause; the urinary symptoms of the second patient had been attributed to prostatic hypertrophy; and the third patient underwent cystoscopy before the cutaneous loss was recognized. In two patients the signs were discovered during hospital admissions for other reasons. It is possible that neurological examination of a series of patients with ankylosing spondylitis may reveal the presence of a cauda equina lesion in other cases.

**Summary**

Three cases are described in which a cauda equina lesion of gradual onset and progression was associated with long-standing inactive ankylosing spondylitis. No cause for the neurological lesion was found, and its possible causal relationship to the spondylitis is discussed.

We are indebted to Dr. J. L. Reynolds, Dr. R. A. Barker, and Mr. J. M. Langham for permission to report the patients admitted to hospital under their care.

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**COST OF DIETS**

BY

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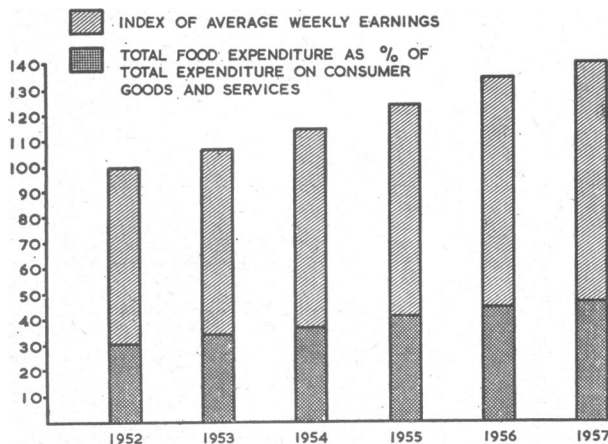
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The prescription of a special diet raises the possibility of greater individual expenditure on food. This has prompted us to study the costs of special diets prescribed and to compare these with the cost of an average normal diet. Normal diets vary greatly and rapid changes in food prices and incomes permit only



Change in earnings and consumer expenditure 1952 to 1957 to show that, while weekly earnings have increased, money spent on food as a proportion of the total expenditure on consumer goods and services has remained at 30%.

an overall view. The National Food Survey Committee (1959) of the Ministry of Agriculture, Fisheries and Food has made a detailed study of average normal diets: the average weekly individual expenditure on food of 28s. in 1957 is influenced by age, social class,

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family size, and proportion of protein, carbohydrate, and fat consumed. Between 1952 and 1957 the percentage of energy value derived from protein and carbohydrate decreased while that derived from fat increased. With increasing prosperity and increasing individual earnings the proportion of income spent on food appears to have remained at about 30% of the total expenditure on consumer goods and services, as is shown in the Chart which is computed from the *Ministry of Labour Gazette* (1958) and the *Monthly Digest of Statistics* (1959).

During 1957 the Middlesex Hospital Department of Dietetics prescribed 1,108 special diets to new out-patients. The different types of special diet used are shown in Table I. The cost of each type of diet was

TABLE I.—Distribution of Special Diets Expressed as a Percentage of All Special Diets Supplied During One Year

Diabetic ..	58.0%	Low-sodium	1.4%	High-protein	0.5%
1,000-calorie	21.0%	Low-fat ..	1.4%	Others ..	9.2%
Gastric type	8.5%				

calculated by taking the October, 1959, market price of constituent foods and working on the basis of one individual buying for herself rather than for a family. The table in Appendix I gives the prices taken and illustrates the findings of two independent observers in different shopping areas. By this method the British Medical Association's (1950) recommended normal diet for a woman doing sedentary work cost 33s. 8d., while that for a man doing medium work cost 44s. 6d.

**Results**

The composition and cost of the diets studied is given in Table II and in detail in Appendix II. The most frequently prescribed diet was the diabetic diet, accounting for 58% of all the special diets supplied (Table I);

TABLE II.—Composition and Estimated Weekly Cost (to the Nearest Shilling) of Various Therapeutic Hospital Diets Compared with the British Medical Association Recommended Normal Diet (a) For a Woman Doing Sedentary Work and (b) for a Man Doing Medium Work

Diet	Calories	Carbo-hydrate (g.)	Protein (g.)	Fat (g.)	Cost
Normal { <sup>a</sup> .. .. .	2,000	280	58	80	34s.
Normal { <sup>b</sup> .. .. .	3,000	350	87	135	44s.
Diabetic .. .. .	1,000	80	53	50	33s.
" .. .. .	1,500	150	73	69	35s.
" .. .. .	2,000	200	95	95	38s.
" .. .. .	2,000	150	106	110	41s.
" .. .. .	2,500	150	129	151	45s.
1,000-calorie .. .. .	1,000	100	55	51	38s.
Fluid type of gastric .. .. .	2,000	260	72	79	30s.
Convalescent gastric .. .. .	2,000	270	70	72	35s.
Low-sodium (0.5 g./day) .. .. .	2,000	273	77	73	34s.
High-protein .. .. .	2,200	220	100	103	44s.
Low-animal-fat .. .. .	1,000	110	58	35	40s.
Low-animal-fat + 60 g. corn oil daily .. .. .	1,500	110	58	95	43s.

the 1,000- and 1,500-calorie diabetic diets accounted for approximately two-thirds of all diabetic diets. The cost of most diabetic diets did not exceed that of the British Medical Association's recommended normal diets. The less commonly used 2,000-calorie diabetic diets were more costly; those with protein contents of 95 g., 106 g., and 129 g., cost 38s., 41s., and 45s. respectively. Among the non-diabetic diets, the 1,000-calorie reducing diet worked out at 38s. a week, salads and fruit accounting for 15s. 7d. of this. The cost of two average gastric diets—a fluid type of diet and a convalescent diet—was estimated. The first cost 30s. a week and the second 35s. a week. The lower cost of the fluid type of diet