"calcium" appeared within a few days of the administration of mercury. This is in marked contrast with our experience in producing experimental nephrocalcinosis, which was usually found to take 10 to 14 days. Since von Kossa's stain for calcium is not specific it is possible that at least some of the stainable "calcium" reported by these authors was in fact a phosphate mercury complex.

Specific glomerular changes other than the accumulation of eosinophilic material in Bowman's space have not been described following the administration of mercurial diuretics. However, shrunken glomeruli of the type described earlier were found in the kidneys of rabbits which had received mercuric chloride (Edwards, 1942). These shrunken glomeruli may be of considerable importance in this case, since it is recognized that in the nephrotic syndrome there is not only tubular dysfunction with some resultant failure to reabsorb protein, but also increased permeability of the capillary tufts in the glomerulus (Hardwicke and Squire, 1955). The nephrotic syndrome has not been a feature of previously recorded cases of fatal renal damage following mercurial diuretics, and this might be explained by the fact that the glomerular lesion is not a constant feature in these cases.

From the clinical angle mercurial diuretics are now widely used. The possible action of this drug as a nephrotoxin does not seem to be as fully recognized as it should. In this case the renal tubular damage had apparently become irreversible before steps to institute an alternative diuretic could be arranged. It is not easy to suggest methods for the recognition of the onset of renal failure. The one sign which may be of value is the onset of increasing proteinuria.

Summary

A case of the nephrotic syndrome following the administration of a mercurial diuretic is described.

The salient histological feature was necrosis and fatty degeneration without calcification of the epithelium in the convoluted tubules. In addition a number of the glomeruli were shrunken.

The significance of the dual lesion in the production of the nephrotic syndrome is discussed.

The observation of increasing proteinuria in patients under treatment with mercurial diuretics suggests that renal damage may be occurring, and that prompt steps should be taken to institute alternative diuretics.

Our thanks are due to the Medical Research Council for an expenses grant. We are grateful to Dr. J. C. Prestwich, under whose care the patient was for her final illness, for his permission to publish this case. We are also grateful to Mrs. S. J. W. East for the line drawing and to Mr. A. E. Clarke, F.I.M.L.T., for the photomicrograph.

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The Medical Research Council's medical mycology committee has recently revised *Nomenclature of Fungi Pathogenic to Man and Animals* (M.R.C. Memorandum No. 23, H.M.S.O., price 1s. 3d. net.). The memorandum lists the names for fungi and for fungal diseases which the committee recommends should be used in Britain; it also includes notes on why some of the names are preferred and a list of rejected names.

LARYNGEAL STRIDOR IN RHEUMATOID ARTHRITIS

BY

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[WITH SPECIAL PLATE]

Arthritis of the cricoarytenoid joint is one of the more unusual manifestations of rheumatoid arthritis and has been accepted by most observers as the cause of laryngeal stridor which sometimes occurs late in the disease. We have recently encountered five patients with unmistakable evidence of advanced rheumatoid arthritis who presented with stridor. The onset of this complication occurred when the generalized joint disease appeared to be quiescent, and tracheotomy became necessary in four cases. Clinical examination in three of the cases indicated a bilateral abductor paralysis of the vocal cords. As the difficulty of differentiating between an arthritis of the cricoarytenoid joint and a bilateral abductor paralysis with disuse fixation is well recognized, the death of two of our patients provided material for a detailed microscopical study of this problem.

Case 1

The patient worked in a rolling mill until the age of 57, when he developed rheumatoid arthritis. He received a variety of physical treatments at a local spa, but these did not include gold or other injections. Gradually the disability increased so that he had to find lighter work. In December, 1953, at the age of 62, and two years before his death, he developed a respiratory infection with hoarseness. At no time in the past had there been any laryngeal symptoms, and there was no local pain or dysphagia on this occasion. Three weeks later his breathing became very distressed owing to respiratory obstruction. Both vocal cords were touching, and an emergency tracheotomy was performed. After recovery from the operation the nature of the laryngeal lesion was more fully investigated. Indirect laryngoscopy showed a symmetrical appearance with a vocal gap of 2–3 mm. posteriorly. No movement occurred on inspiration or expiration, but the cords met on phonation. On straining, the larynx as a whole contracted. Direct laryngoscopy was performed under thiopentone and succinylcholine chloride anaesthesia, and the mobility of the arytenoids tested by grasping them in a pair of Paterson's forceps. Movement of the cricoarytenoid joint was found to be unrestricted. Because of these findings the lesion was regarded as a bilateral abductor paralysis rather than a cricoarytenoid arthritis. Neurological examination showed no cause for a paralysis of the vocal cords, so the case was finally diagnosed as idiopathic bilateral abductor paralysis of the larynx.

The patient managed to get about at home fairly well for the next 18 months until his dyspnoea returned, eventually being present even at rest. When examined at this time he showed undoubted changes of advanced burnt-out rheumatoid arthritis, with permanent deformities in the hands. In addi-

tion there were marked finger-clubbing and the characteristic features of a diffuse interstitial fibrosis of the lungs, with tachypnoea at rest, hyperventilation, central cyanosis, and persistent loud rales over both lungs. His general condition was fair and there was no evidence of vitamin deficiency, peripheral neuritis, or other neurological disorder, nor of generalized arteriosclerosis. There was no disease of the skin. The temporo-mandibular joints were normal.

A radiograph of the chest showed a diffuse interstitial fibrosis which had advanced since an earlier radiograph taken a few days after the tracheotomy. Barium swallow was normal. The Wassermann and Kahn reactions were negative. A blood examination showed a moderate anaemia. The plasma proteins were normal. An electrocardiogram suggested right ventricular hypertrophy.

Treatment in hospital included antibiotics to control the mild respiratory infection and oxygen to relieve the cyanosis and dyspnoea. These measures proved of no avail, and he died rather suddenly four weeks later from bronchopneumonia.

Post-mortem Examination

The immediate cause of death was a recent myocardial infarction of the anterior wall of the left ventricle due to a thrombus in the left descending coronary artery. The heart weighed 360 g. and the right ventricle was hypertrophied. The pericardium was adherent. There was obliteration of both pleural sacs from old adhesions. The lungs were small and indurated as the result of an advanced interstitial fibrosis with some superficial cystic changes. The lower lobes revealed a patchy bronchopneumonia. There was no evidence of an old mediastinitis or of fibrosis around the larynx. The other internal organs were normal. The cricoarytenoid joints were fully mobile. The brain and spinal cord were removed and the vagus with its laryngeal branches was dissected out. These nerves appeared much thinner than normal. Sections were taken from representative levels throughout the central nervous system and from these nerves.

Microscopical examination showed a thickened, chronically inflamed synovial lining of the cricoarytenoid (Special Plates, Figs. 1 and 2) and cricothyroid joints and a thick-walled, chronically inflamed subhyoid bursa. Several foci of chronic inflammatory cells were present in the subcutaneous tissues of the neck. The laryngeal muscles showed a neural atrophy. The superior and recurrent laryngeal nerves showed severe demyelination and degeneration of the axis cylinder, and this was present but less marked in the vagus (Special Plate, Fig. 3). No obvious cause for the neuropathy could be seen in the sections examined. The nerve cells of the nucleus ambiguus and other cranial-nerve nuclei appeared intact and there was no evidence of motor neurone disease. The brain showed marginal gliosis and scattered small focal areas of old infarction consistent with arteriosclerotic brain atrophy.

Case 2

A woman of 65 years was first seen by us in 1955 during an attack of bronchitis. She stated that she had rheumatic fever when she was 30 years of age, and for more than 20 years had suffered from rheumatoid arthritis with progressive disability. When she was 54 hoarseness and stridor developed, without any associated pain or dysphagia. Indirect laryngoscopy showed appearances which were considered to be due to bilateral abductor-nerve palsy. A tracheotomy was done to relieve the respiratory obstruction, and she has worn a rubber tube ever since. Following the operation she has been subject to recurrent attacks of asthmatic bronchitis. She had never received gold injections.

Clinical examination in November, 1955, showed good general nutrition and unmistakable signs of severe rheumatoid arthritis with gross deformity. There was no cyanosis or clubbing of the fingers, but persistent rales were audible over the left lower lobe with occasional scattered rhonchi. The blood pressure was 190/120 mm. Hg. All superficial pulses were present, although there was some generalized arteriosclerosis. There were no lesions of the skin. The voice was weak and croaking, and accomplished by closure of the tracheotomy opening with the finger. Indirect laryngoscopy showed hypertrophy of the false cords and a symmetrical appearance. The vocal chink was 1–2 mm., with no movement occurring on respiration, but slight adduction on attempted phonation. The larynx as a whole contracted on straining. Under thiopentone and succinylcholine chloride anaesthesia some reduction in the normal mobility of the arytenoids was found. Radiographs of the skeleton showed general osteoporosis with mild subluxation of the cervical vertebrae and advanced rheumatoid changes in the small joints of the hands. A chest radiograph revealed partial collapse of the left lower lobe. An electrocardiogram showed inversion of T waves. There was a mild anaemia with an eosinophilia of 14%. The Wassermann and Kahn reactions were negative and the plasma proteins normal.

During the last 18 months her condition has changed little, and it is considered that the laryngeal findings could equally well be those of a cricoarytenoid arthritis or of a bilateral abductor paralysis with disuse fixation.

Case 3

A married woman aged 61 was first seen by us in 1955 on account of severe respiratory obstruction due to laryngeal stridor. She had suffered from severe rheumatoid arthritis for the previous 30 years. At the age of 40 an ulcer developed on the inner side of the lower third of the right leg, which broke down repeatedly over the next 20 years. Ulceration developed between the first and second toes of the right foot, and this ultimately became gangrenous. The feet were otherwise warm, while the arterial pulses were normal. Investigations at the age of 54 showed a severe iron-deficiency anaemia, a histamine-fast achlorhydria, and granulocytopenia with hypoplasia of the granulocyte series of the bone marrow. A year later difficulty in breathing was noted, and laryngeal examination by Mr. J. H. Cobb showed a bilateral abductor-nerve palsy. Tracheotomy was not considered necessary, for her activities were severely limited by her arthritis. On her final admission she was frail, wasted, and crippled with chronic rheumatoid arthritis. The stridor, which had been caused by a mild respiratory infection a week previously, rapidly became worse, and an emergency tracheotomy was performed under general anaesthesia, preliminary intubation having been accomplished under local analgesia. The vocal cords were closely approximated, and remained so when the patient was breathing freely through the tracheotomy tube and also when this was momentarily closed. The arytenoids appeared to be freely mobile. The patient died 16 hours later.

Post-mortem Examination

Death was due to a patchy bronchopneumonia in both lungs, with a small recent infarct in the right upper lobe. The heart weighed only 200 g. The coronary arteries were patent and there was only minimal atheroma of the aorta. The other internal organs were free from gross disease. Examination of the larynx showed no evidence of fixation or of limitation of movement of the cricoarytenoid joints. The brain, spinal cord, vagus, and superior and recurrent laryngeal nerves were removed for microscopical examination. The nerves appeared much thinner than normal. The cricoarytenoid joint was free from any arthritic process. The laryngeal muscles showed a severe neural atrophy (Special Plate, Fig. 4). The vagi, superior laryngeal, and recurrent laryngeal nerves showed demyelination with varicose, swollen axis cylinders. These changes were associated with severe reduction of the lumen of the vasa nervorum due to intimal proliferation (Special Plate, Fig. 5). The inferior laryngeal branch of the inferior thyroid artery, which accompanies and supplies the recurrent laryngeal nerve, showed thombosis with recanalization by two minute channels. The internal elastic lamina was fragmented, and

calcification had occurred in this and in the proliferated intima (Special Plate, Figs. 6 and 7). The nerve cells of the nucleus ambiguus and other cranial-nerve nuclei appeared intact, and there was no evidence of motor neurone disease.

Case 4

The patient, a man aged 65, used to work as a springer in a steelworks. Rheumatoid arthritis developed at the age of 34, and within three years he was severely disabled. Aspirin and frequent courses of physiotherapy did not halt the disease, although gradually the pain in the joints became less. He did not receive gold injections. During the three years preceding his examination by us in 1956 his voice had become husky at times, although there was no local pain or dysphagia, and his breathing was not embarrassed until a few weeks previously, when he had begun to have alarming choking attacks at night.

Examination in hospital in May, 1956, showed the general features of severe chronic rheumatoid arthritis. There was mild respiratory stridor, but no cyanosis or clubbing. Moderate general arteriosclerosis was present, affecting particularly the arteries of the upper limbs. Indirect laryngoscopy showed a symmetrical appearance with a vocal gap of 2–3 mm. There was no movement on respiration, but the cords met on phonation and the larynx as a whole contracted on straining. Examination under thiopentone and succinylcholine chloride anaesthesia showed that the mobility of the arytenoids was limited. There was roughening of the cords at the anterior commissure which was suspected to be neoplastic. This appearance became more clearly that of a carcinoma, and on August 27, 1956, a biopsy and tracheotomy were performed. The microscopical appearances of the biopsy tissue were those of a squamous-cell carcinoma which has responded completely to radiotherapy. He remains fairly well, but still wears a tracheotomy tube.

Case 5

The patient, a married woman of 67 years, had suffered from rheumatoid arthritis for 31 years. For about a year she had been subject to mild attacks of asthmatic bronchitis. She was referred to us by Dr. H. F. West, of the Rheumatism Research Unit, Sheffield, because of an inspiratory stridor and maxillary sinusitis. There had been no local pain in the jaws or larynx and no dysphagia. Laryngeal examination showed a symmetrical appearance. The vocal gap was 3 mm., and did not vary with respiration, but could be closed on phonation. Under general anaesthesia the arytenoid cartilages were completely lax. Antral lavage was carried out.

The degree of stridor and respiratory embarrassment is at the moment mild, and tracheotomy is not required.

Discussion

Although it is well recognized that in any polyarthritic condition the process may affect the cricoarytenoid joint (Ellis, 1951 ; De Vido and Ancetti, 1952), until recently there have been scant references in the literature. The appearance in the last few years of several reports of laryngeal involvement in rheumatoid arthritis indicates renewed interest in the subject and suggests that the condition is not such a rarity. It will be seen from the Table that these cases have a similar pattern, with the onset of respiratory obstruction in the sixth or seventh decades, following after the onset of rheumatoid arthritis by many years, when the joint condition was either quiescent or only mildly active. The period of increasing respiratory obstruction varied from a few months to two years. The four cases reported by Montgomery *et al.* (1955) showed a similar age incidence for both the rheumatoid arthritis and the laryngeal symptoms. These workers discussed the difficulties of the differential diagnosis of immobility of the vocal cord. They attached most importance to the history, painful spatula test, and inspiratory bowing of the cords in making a diagnosis of arthritis rather than of laryngeal palsy, but even these can be inconclusive. Only the ultimate demonstration of typical rheumatoid changes in the joints or the finding of degeneration in the recurrent laryngeal nerves makes the diagnosis certain.

Table Showing Age of Onset of Rheumatoid Arthritis and of Respiratory Obstruction

Case No.	Age of Onset	
	Of Rheumatoid Arthritis	Of Respiratory Obstruction
1	57	62
2	30	54
3	31	55
4	34	63
5	36	66

Similar difficulty in the differential diagnosis was encountered by Saunders (1956) and Copeman (1957). The latter reported three cases of rheumatoid arthritis developing recurrent attacks of hoarseness late in the course of the illness. As recurrent paralysis of peripheral nerves in association with exacerbations of rheumatoid arthritis has not been recorded, it was considered more probable that arthritis of the cricoarytenoid joints was the explanation. Tracheotomy was not necessary and no histological confirmation was possible. The most convincing case is that of Pearson (1957), in which post-mortem histological examination showed villous proliferation of the synovial membrane of the cricoarytenoid joint. No mention was made of the laryngeal muscles and nerves.

Baker and Bywaters (1957) recorded an unusual case of laryngeal stridor which was considered to be due to rheumatoid involvement of the cricoarytenoid joint. There were several features in this case suggestive of systemic lupus erythematosus, in which peripheral-nerve palsy sometimes occurs on a vascular basis (Heptinstall and Sowry, 1952). Despite vascular lesions elsewhere causing nail-fold thrombosis, a local arthritis in the laryngeal joint was favoured as a diagnosis even though the laryngeal picture resembled that of a bilateral abductor paralysis. No microscopical confirmation was possible, and tracheotomy was avoided by the use of delta-cortisone. Even this therapeutic response to cortisone cannot be relied upon as a confirmation of the diagnosis of rheumatoid arthritis of the cricoarytenoid joints, as in one of the cases of Montgomery *et al.* the laryngeal condition developed during cortisone treatment, and in another case the symptoms became worse.

Hart and Mackenzie (1955) described the case of a man of 45 with a 15-year history of rheumatoid arthritis who had an attack of dyspnoea followed by stridor necessitating tracheotomy. The cause of the stridor was thought to be abductor palsy, although this could not be explained except possibly on the basis of deep *x*-ray therapy for thyrotoxicosis. He died four years later, and at necropsy there was microscopical evidence of fibrosis in the region of the vocal cords. Unfortunately, no reference to the state of the cricoarytenoid joints or of the recurrent laryngeal nerves was made.

Thus, apart from the case described by Pearson (1957) in which the cricoarytenoid joint was sectioned and the arthritis confirmed, the diagnosis in the other cases is accepted as the most likely on clinical grounds, although the difficulty in differential diagnosis is acknowledged. In none of the cases reported were the laryngeal nerves and muscles studied. The death of two of the patients in our series of five enabled a complete examination of the laryngeal joints, muscles, and nerves to be made together with a neuropathological study of the central connexions with the brain-stem and the descending pathways from the cortex.

In Case 1 the cricoarytenoid joint was still freely mobile, although its synovial sheath was thickened and showed chronic inflammation. The cricothyroid joint and the subhyoid bursa showed similar chronic inflammation. The inflammatory reaction was non-specific and not typically rheumatoid, although this may have been responsible. As there were scattered foci of chronic inflammatory cells in the subcutaneous tissue of the neck there was evidence of additional chronic infection from the tracheotomy. The

laryngeal muscles showed neural atrophy and there were severe degenerative changes in the superior and recurrent laryngeal nerves and vagus. No indication as to the cause of this neuropathy was seen in the numerous sections examined. Such causes of nerve degeneration as vitamin deficiency, virus disease (Critchley, 1946), toxic effects from heavy metals, and amyloid (Missen and Taylor, 1956) were excluded. Thyroid disease was absent, and no tumour or disease process could be found in the mediastinum, thoracic inlet, neck, or nasopharynx. The brain-stem was normal, but the cerebrum showed several small scattered infarcts of varying age due to arteriosclerosis. As death had resulted from coronary thrombosis the possibility of ischaemic neuropathy was considered, but no evidence to support this could be found.

It is claimed that rheumatoid arthritis can produce both a polymyositis and neural atrophy due to pressure palsy on nerves by the diseased joint (Adams *et al.*, 1953). No evidence of polymyositis was found in the laryngeal muscles, while the vagus and its branches are sufficiently distant from joints likely to be affected by rheumatoid arthritis that a pressure palsy is not possible. No focal lesions in the nerves or muscles as described in rheumatoid arthritis by Freund *et al.* (1942), Steiner *et al.* (1946), and Cruickshank (1952) were found. The cause of the neuropathy in this case remains obscure (Ellis, 1946; Huppler *et al.*, 1956).

In Case 3 the cricoarytenoid joints were healthy, but the muscles again showed neural atrophy which had resulted from ischaemic neuropathy. Gross obliterative endarteritis of the vasa nervorum was found. A variety of vascular changes have been described in rheumatoid arthritis (Sokoloff *et al.*, 1951; Cruickshank, 1954; Ball, 1954; Bywaters, 1957). These ranged from intimal proliferation to a severe necrotic arteritis resembling polyarteritis nodosa. Vessels in the heart, muscles, and nerve sheaths were most frequently involved. The changes in the vasa nervorum could be of rheumatoid origin, while the severe calcification might have resulted from metastatic calcification in necrotic tissue from the bones of limbs kept fairly immobile by the joint disease or possibly as a superimposed arteriosclerotic effect. The leg ulceration and gangrene of the toes were regarded as further indication of generalized vascular disease of rheumatoid origin (Bywaters, 1957; Allison and Bettley, 1957). Systemic lupus erythematosus was considered as a possible diagnosis, but could not be substantiated.

Thus, although in cases of rheumatoid arthritis in which stridor develops involvement of the cricoarytenoid joint may be responsible, it is not the only factor, and nerve degeneration can be the cause. That this may be of ischaemic origin related to a "rheumatoid" arteritis of the vasa nervorum fits in with the wider concept of "rheumatoid disease" (Ellman and Ball, 1948; Cruickshank, 1957) as both a pathological and clinical entity, rather than "rheumatoid arthritis," in cases presenting unusual or more diffuse features.

Summary

The difficulty of distinguishing between an arthritis of the cricoarytenoid joints leading to stridor and a bilateral abductor paralysis of the vocal cords is emphasized. Clinical examination may not provide sufficient evidence, and in the final analysis microscopical examination of the joints, muscles, and nerves is needed.

Five patients suffering from advanced rheumatoid arthritis are described, each of whom developed laryngeal stridor, and in four instances tracheotomy became necessary. Two of the patients died, and post-mortem examination revealed indisputable evidence of nerve degeneration and mobile cricoarytenoid joints, even though chronic inflammation of the joints was present in one case. In the second case the microscopical changes in the nerves appeared to be due to ischaemic neuritis resulting from a rheumatoid type of arteritis of the vasa nervorum.

Although rheumatoid changes in the cricoarytenoid joints do occur, it is suggested that in some cases the stridor is due to abductor paralysis resulting from nerve degeneration.

We wish to thank Dr. H. F. West, who referred Case 5, for his helpful advice; Mr. J. H. Cobb for his interest and for allowing us access to his records on Case 3; Dr. A. J. N. Warrack for carrying out the two post-mortem examinations; and Professor D. H. Collins for his valued opinion and useful criticism.

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CERVICAL MYELOPATHY PRESENTING AS PERIPHERAL NEUROPATHY

BY

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[WITH SPECIAL PLATE]

Considerable attention has recently been given to cervical spondylosis and its neurological complications (Brain *et al.*, 1952; Clarke and Robinson, 1956; Bradshaw, 1957). The term "spondylosis" refers to the chronic degenerative changes in the cervical spine occasioned by disk degeneration, which is usually the result of senile changes, but which may appear in the presence of congenital bony anomalies or as a delayed sequel to injury. The acute cervical prolapsed intervertebral disk does not come within the scope of the term, and it must be rigorously excluded from considerations of this subject, especially therapeutic. "Cervical myelopathy" has been used to describe those cases in which the neurological complication of spondylosis is manifested principally by a spinal-cord lesion, but some degree of coexisting root involvement can rarely be entirely excluded.

It is well known that the clinical results of nervous-tissue compression by cervical spondylosis are varied, both in their presenting features and in the eventual clinical picture. Thus it is possible to observe syndromes which may closely simulate known diseases of the nervous system. The commonest of these are disseminated sclerosis, cervical-cord neoplasm, motor neurone disease, and syringomyelia; but differentiation on clinical grounds alone is often possible. The possibility of coincidental cervical spondylosis with a neurological

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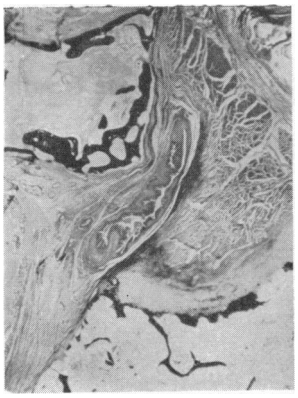


FIG. 1.—Longitudinal section of larynx (Case 1), showing thickened synovial membrane of cricoarytenoid joint. (×6.)

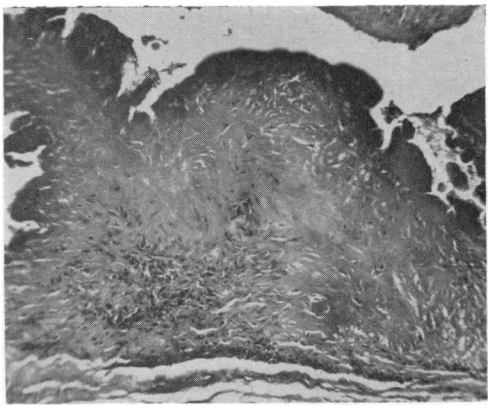


FIG. 2.—Higher power view of thickened synovial membrane of cricoarytenoid joint (Case 1), showing chronic inflammatory cells. (×98.)

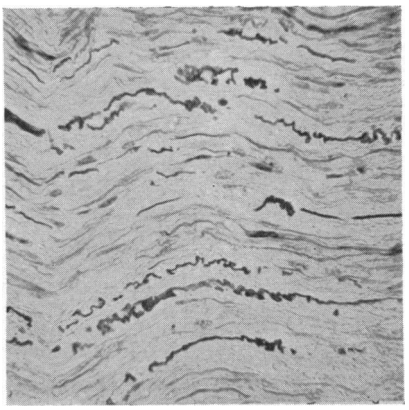


FIG. 3.—Lower part of left recurrent laryngeal nerve (Case 1), showing degenerate tortuous axis cylinders. (Silver stain. ×306.)

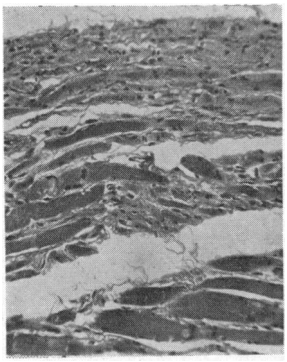


FIG. 4.—Longitudinal section of posterior cricoarytenoideus muscle (Case 3), showing neural atrophy. Bundles of atrophic fibres with increased numbers of nuclei are separated by larger intact fibres. (×86.)

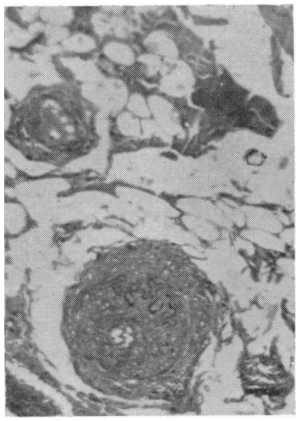


FIG. 5.—Vasa nervorum in left vagus (Case 3), showing severe intimal proliferation with reduction in size of lumen. (×60.)

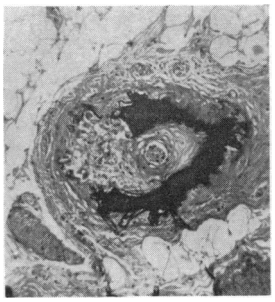


FIG. 6.—Calcification in intima and internal elastic lamina of artery supplying upper part of left recurrent laryngeal nerve (Case 3). Two minute channels are present in centre. (×56.)

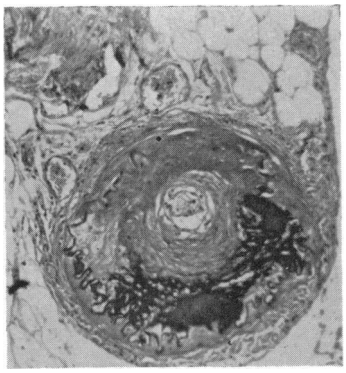


FIG. 7.—Similar calcification in vasa nervorum in lower part of left recurrent laryngeal nerve (Case 3). (×56.)

E. CLARKE: CERVICAL MYELOPATHY PRESENTING AS PERIPHERAL NEUROPATHY

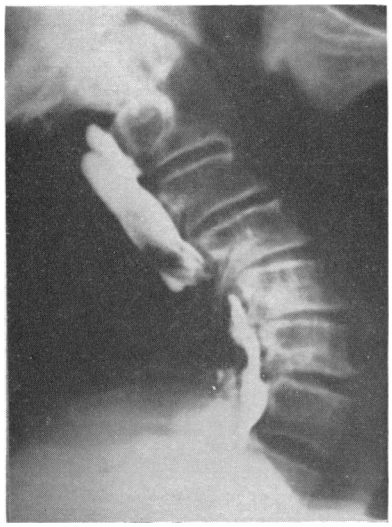


FIG. 1.—Case 1. Myelogram showing anterior and posterior filling defects in cervical region.

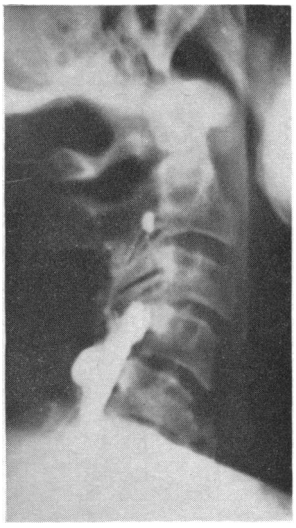
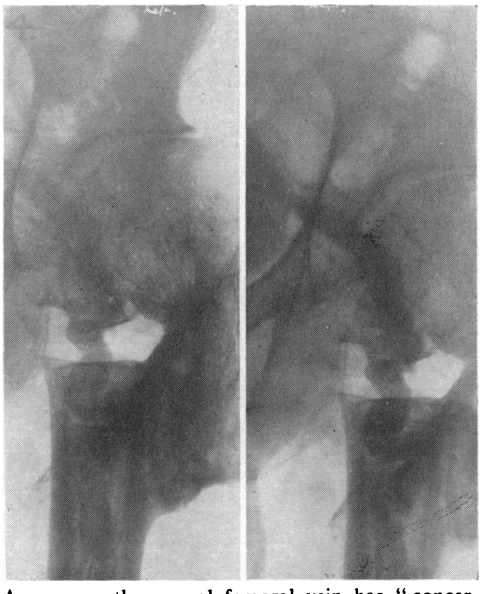


FIG. 2.—Case 2. Myelogram showing anterior and posterior filling defects in cervical region.

J. A. WILLIAMS: AN UNUSUAL SITE FOR GRAVITATIONAL ULCERS



An apparently normal femoral vein has "concertinaed" following a femoral osteotomy. There is 2 in. (5 cm.) of shortening.