*Papers and Originals***SOME UNSOLVED PROBLEMS OF CERVICAL SPONDYLOSIS***

BY

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I need hardly say how great an honour I consider it to be invited to deliver a lecture associated with the name of Sir William Gowers—a name so well known, and so distinguished, that nothing I could say would add to its lustre. Gowers was a great clinician who took all neurology for his province and set out his experience in books which are of lasting value, because in medicine true observation remains true, however our interpretation of it may develop. So Gowers is still a mine of clinical wisdom, and before I begin my lecture on cervical spondylosis let us look it up in Gowers. You will not find the name there, because it had not then been invented, and the pathology of the intervertebral disk had not been studied. Nevertheless, Gowers (1899) recognized the condition. Under the heading "vertebral exostoses" he said: "Exostoses sometimes grow from the bodies of the vertebrae into the spinal cord, and may compress the cord or nerves. They are, however, extremely rare. The symptoms may be those of slow compression of the cord, or of irritation, expressed chiefly by pain. . . . Their chief characteristic is extreme chronicity. . . . Exostoses constitute a more promising field for the surgeon than other kinds of vertebral tumour. Many of them are so placed that their removal is feasible. If situated in front of the cord, the division of some nerve roots, at least in the dorsal region, might permit access to the growth." Vertebral exostoses were then regarded as arising from the spine without distinction between the vertebral bodies and the intervertebral disks.

Gowers also recognized arthritis of the spine as a cause of nervous disease, but he mentioned only rheumatoid arthritis. "In chronic rheumatoid arthritis," he wrote, "the intervertebral articulations are sometimes involved. The symptoms are local pain, tenderness, and limitation of movement, which may go on to absolute fixation, especially in the cervical region, if ankylosis occurs. . . . The cord is scarcely ever compressed, but the narrowing of the foramina may damage the nerve roots. The articular processes, it will be remembered, form one side of the intervertebral foramina, which are thus encroached on by any enlargement of the bones. The nerves may thus be compressed or irritated, and radiating pains are produced, sometimes even a descending neuritis." We cannot be sure whether he distinguished rheumatoid arthritis from other forms of arthritis of the cervical spine, since he mentioned no others, but he said, "it must be remembered that the vertebral affection sometimes exists alone, and it may cause symptoms during many years, often when their nature is unsuspected."

I have chosen this subject because I thought that I could best pay tribute to a great clinician by discussing a clinical problem. Fifteen years ago we knew little more about the neurological effects of cervical spondylosis than Gowers did when he first published his textbook seventy-five years ago. Since then our knowledge has

steadily increased, first with regard to the effect of the disease upon the spinal cord and nerve roots, and more recently concerning its possible influence upon the cerebral circulation through the vertebral arteries. The fact that cervical spondylosis is an extremely common disorder after middle age often raises diagnostic problems when one has to decide whether, or to what extent, the spondylosis present is responsible for the symptoms of which the patient complains. But even when there is good reason to think so, it is not always clear how it produces them, and, without knowing that, treatment must be empirical rather than rational. So the scope of this lecture will naturally extend beyond cervical spondylosis and include a review of the pathogenesis of some symptoms which are common in the latter part of life.

Incidence of Cervical Spondylosis

How common is cervical spondylosis in this country? Professor Kellgren and Dr. J. S. Lawrence have kindly allowed me to quote from a large radiological study carried out in Leigh, a small Lancashire industrial town, and Wensleydale, a hill farming area in Yorkshire. Lawrence and his colleagues investigated the radiological evidence of cervical disk degeneration in over 2,000 of the combined Leigh and Wensleydale populations, where, incidentally, the prevalence appeared to be much the same in the two areas. I have abstracted, and present in Tables I and II, some items from their more detailed findings.

These tables show a steady increase in both the prevalence and the severity of cervical spondylosis with age, and that, though the incidence of disk degeneration

TABLE I.—*Prevalence of Radiological Evidence of Disk Degeneration in the Cervical Spine in Combined Leigh and Wensleydale Populations. (By Courtesy of Professor Kellgren and Dr. J. S. Lawrence)*

Age	Total X-rayed	No Disk Degeneration	% of Total X-rayed	Grades 1-4 in 4 or More Disks. % of Total X-rayed
Males				
15-24	165	155	94	0
25-34	176	147	84	0
35-44	183	114	62	0
45-54	233	76	33	2
55-64	139	35	25	12
65-74	93	8	9	19
75+	47	3	7	23
Females				
15-24	170	162	95	0
25-34	172	154	89	0
35-44	213	156	73	0
45-54	224	88	35	3
55-64	174	39	22	18
65-74	137	21	15	13
75+	57	11	2	12

TABLE II.—*Results in Patients Aged 55 and Over*

Sex	Total X-rayed	No Disk Lesion	% with Disk Lesion
Males ..	279	46	83.5
Females ..	368	71	80.7

*The Gowers Memorial Lecture delivered on November 9, 1962.

is about the same in men and women, women tend to be less severely affected than men. In both sexes some degree of cervical disk degeneration is present in about 80% of all persons over the age of 55. We do not know if the incidence of cervical spondylosis in London is as high as in Lancashire and Yorkshire, but it would certainly be safe to say that a large majority of our patients over the age of 55 present radiological evidence of it.

But to say that a patient has radiological evidence of cervical spondylosis is to say no more than that some degenerative change is present at some point in the cervical spine. The change may not produce any neurological symptoms. If the neck has been x-rayed it is presumably because cervical spondylosis is suspected as the cause of the symptoms. I shall begin, therefore, by considering how to get the most information out of the x-ray films.

Radiology of Cervical Spondylosis

Obviously x-ray films need to be as complete as possible, and more information is needed than can be obtained from routine lateral, antero-posterior, and oblique views of the neck. Lateral views on flexion and extension, as well as in the erect position, show the degree of movement, where it is taking place, and whether abnormal movement is occurring. Views on lateral flexion are sometimes helpful. The apophysial joints have tended to be neglected, partly because perhaps films need to be taken at a special angle to show them well. Dr. Allan Young has devised a method which gives on two films two views of most of the apophysial joints. Disk degeneration and apophysial arthritis do not always occur at the same level, and concentration on the disk lesions may lead to relative neglect of the apophysial lesions, which may from a clinical standpoint be the more important.

Antero-posterior views of the articulations between the second and third vertebrae are difficult to obtain, but a useful view of these can often be got through the mouth in edentulous patients. Finally, tomography often provides useful additional information. If a really complete investigation with plain x-ray films is carried out, and considered in relation to the clinical picture, it is rarely necessary to resort to myelography, except when surgery is contemplated. Moreover, though angiography alone can show what is happening to the vertebral arteries, we can often infer something about that from adequate plain x-ray films.

For the purpose of this lecture I have reviewed a hundred consecutive patients with x-ray evidence of cervical spondylosis, in whom either I thought the cervical spondylosis was wholly, or in part, responsible for their symptoms, or this question presented a diagnostic problem. It will be obvious that my figures give no information about cervical spondylosis in general, not even about the relative frequency with which it produces particular clinical pictures. All they show is how often individual syndromes occurred in the experience of one neurologist particularly interested in the subject. Table III shows the presenting symptom or syndrome.

The total is more than 100 because some patients complained of a combination of symptoms or

syndromes. I do not propose to discuss now either brachial radiculitis or myelopathy, which have been very fully described before. I need only mention acute lesions of the spinal cord occurring in the absence of trauma in the cervical region in patients with cervical spondylosis. These were all incomplete lesions from which some recovery occurred. Two patients were hypertensive, and the lesions were thought to be vascular.

Headache

Headache is one of the commonest presenting symptoms of cervical spondylosis in this series of cases, and cervical spondylosis is a common and important cause of headache beginning in middle life and later. Spondylotic headache presents such distinctive features that when once these are familiar it is easy to diagnose. It is always occipital on one or both sides, and a headache which spares the occipital region is unlikely to be due to spondylosis. It is often described as spreading to, or independently involving, either the forehead or the eye on the same side, when it is unilateral. Much less frequently the intervening vertical region is also affected. It is not uncommon for the pain to be said to start in the back of the neck and spread up to the occipital region and to the eye, and in a proportion of cases it is associated with pain in the arm on the same side, the two pains tending to wax and wane together. The pain tends to be of a nagging and wearing character, but not throbbing or bursting like a headache of vascular origin or one associated with increased intracranial pressure. In long-standing cases it tends to be continuous, but in the earlier stages it is paroxysmal, and may then be related to the posture of the head, and sometimes made worse by active or passive movement of the neck. Some patients say it is present when they awaken in the morning but passes off an hour or two after getting up. Sometimes straining and coughing make it momentarily worse. It is most exceptional for the headache to be associated with any objective change in sensibility of the scalp in the regions in which it is felt.

How is the headache of cervical spondylosis produced? The most likely way to find the answer to this question would seem to be to try to discover whether there are any x-ray changes in the cervical spine which are present in those patients suffering from headache but absent in those without. This investigation, however, is hampered by the fact that the routine plain x-ray films of the cervical spine usually taken often provide very inadequate information about the state of the apophysial joints. So far as the intervertebral disks are concerned headache may be associated with a disk lesion at any level, but for various reasons it is unlikely that the disk lesion in itself is the cause of the pain. Such comparative x-ray studies as I have been able to make between patients with and without headache show that the distinctive features of those with headache are a predominant involvement of the upper cervical spine and of the apophysial joints.

A systematic study of the apophysial joints in all cases, however, is necessary to draw any more precise conclusions. If we may assume that headache is the result of apophysial arthritis in the upper cervical spine, how does that cause it? It seems most unlikely that it is due to pressure on posterior nerve roots, since the objective signs of this are lacking, and that would hardly explain pain in the forehead and eye. It might be referred pain, either from the joints themselves or from surrounding tissues, including muscles. Kovács (1955),

TABLE III.—*Presenting Symptom or Syndrome*

Brachial radiculitis ..	..	32	Vertebro-basilar ischaemia ..	5
Headache ..	..	28	Attacks of unconsciousness ..	4
Vertigo ..	..	17	Drop attacks ..	3
Myelopathy ..	..	13	Acute lesion of spinal cord ..	3
Pain in neck ..	..	13		

who has made a special x-ray study of the cervical apophysial joints, believes that the headache is due to pressure upon the vertebral artery and accompanying sympathetic nerves by the superior articular process. However, there is no evidence in favour of this view, and the fact, which he mentions, that in many cases flexion combined with traction on the head gives immediate relief to the headache is equally, if not more, consistent with its origin in the joints themselves. This common symptom is still an unsolved problem.

Cervical Spondylosis and Vertebro-basilar Ischaemia

The observations of Hutchinson and Yates (1956) drew attention not only to the effects of narrowing, or occlusion, of the vertebral arteries upon the cerebral circulation as a whole, but also to the contribution which cervical spondylosis might make to impairment of the blood flow through one or both vertebral arteries. As they point out (Hutchinson and Yates, 1961), "normal vessels are well able to accommodate for changes in their course," and it is the presence of atheroma, which has a similar age-incidence to cervical spondylosis, which makes the displacement of the vertebral arteries by osteophytes potentially serious. Hutchinson and Yates (1961) mention particularly in this connexion osteophytes projecting from the tips of the intervertebral joints—that is, the neurocentral joints—which displace the vertebral arteries both anteriorly and laterally, but Kovács (1955) observes that the bone in the region of the apophysial joint may be indented by the vertebral artery. Osteophytes from this joint are particularly likely to distort the vertebral artery between the second and third cervical vertebrae because the vessel is there moving somewhat laterally to reach the transverse process of the atlas.

There seems no doubt, then, that cervical spondylosis can intensify the symptoms of vertebro-basilar ischaemia due to atheroma. A striking example of this is provided by a patient who after being put into a collar for his severe cervical myelopathy developed symptoms of cerebellar deficiency. In the collar his neck was more extended than without it, and this posture presumably impaired the circulation through his vertebral arteries.

Apart from narrowing of these vessels, does a thrombus ever become detached from the artery in the neck and carried into some part of its intracranial distribution, as suggested by Sheehan, Bauer, and Meyer (1960)? This question is raised by two cases in this series, and one additional one.

Case 1.—A man aged 52 had persistent pain in the back of the neck on both sides, which, he said, "clicked" on turning. Four weeks after the onset of the pain he had a sudden cerebellar lesion on the right side. X-ray examination of his neck showed severe extensive spondylosis.

Case 2.—A man aged 62, who also had an acute lesion of the cerebellum, showed the following x-ray changes. In the lateral view the C 5/6 and C 6/7 disks were narrowed, but there was also a severe apophysial arthritis at C 2/3 and C 3/4, well shown in the antero-posterior view, and better still in the antero-posterior view through the mouth. The vertebral artery has to pass these joints on its way to the atlas.

The occurrence of brain-stem infarction after manipulation of the neck is well established, and two cases, one of them fatal, have recently been reported by Smith and Estridge (1962). Case 3 is presumably an example of this.

Case 3.—A woman aged 68 complained for a year of stiffness of the neck, with pain extending up the back of

her head. She was treated with short-wave diathermy and neck traction. Two weeks later she was referred for a neurological opinion because she had suddenly developed left homonymous hemianopia. X-ray examination of her neck showed severe spondylosis involving especially the upper apophysial joints, particularly well shown in the antero-posterior tomograms. There seems little doubt that the treatment of the neck caused obstruction of the right posterior cerebral artery, and this may have been due to detachment of a thrombus at a site of compression of one vertebral artery by an osteophyte.

To accidental trauma to the neck we must now therefore add therapeutic manipulation as a cause of damage to the nerve roots, spinal cord, or, by way of the circulation, the brain. But not only may a vascular accident follow therapeutic manipulation, it may also occur when the neck has been manipulated by the surgeon or anaesthetist in a variety of surgical procedures in elderly patients, who are often not known to have cervical spondylosis.

Anatomical and Physiological Effects of Head Movement

Seventeen patients complained of giddiness as a presenting symptom, and 10 of these noted that it was brought on by head movement, usually rotation or extension, rarely flexion. The anatomical and physiological effects of head movement are complex in normal people, and still more so in those patients with cervical spondylosis who also suffer from atheroma.

Effects of Head Movement on the Cerebral Circulation

The vertebral arteries are usually unequal in size. Gowers pointed out that the left is usually larger than the right, and the latter may be very small, so that the basilar artery is practically a continuation of the left vertebral artery only. This has been confirmed by Stopford (1916) and by Hutchinson and Yates (1956). Its importance lies in the fact that when one vertebral artery is very small compression of the other may cause disproportionately severe effects. The effects of head movement on the blood flow through the vertebral arteries have been studied by a number of workers both in the cadaver and by vertebral angiography (de Kleyn, 1939; Toole and Tucker, 1960; Tatlow and Bammer, 1957; Maslowski, 1960; Bauer, Sheehan, and Meyer, 1961; Bauer, Wechsler, and Meyer, 1961). To summarize their observations: (1) Even in normal individuals rotation of the head to one side may temporarily impair, or even abolish, the blood flow through the atlanto-axial segment of the *contralateral* vertebral artery. Since the vertebral artery passes through the transverse process of the atlas (Fig. 1) it has to move with it, and since it is relatively fixed where it enters the foramen magnum rotation of the head to the opposite side, carrying the atlas with it, puts traction on the artery and actually pulls it out of its canal in the cervical spine (Fig. 2).

When a person has cervical spondylosis, rotation of the head may also diminish the blood flow through the *ipsilateral* vertebral artery in its course through the cervical spine. Angiography shows that a remarkable degree of tortuosity may occur in the vertebral artery in its course through the cervical spine; but this is not surprising, for one effect of intervertebral disk degeneration is to shorten the cervical spine, and since the vertebral artery remains at its original length it must become tortuous, or even kinked, as a result. Moreover,

atheroma increases the liability of the vessel to become tortuous and also narrowed, and hence obstructed by head rotation. So in the presence of cervical spondylosis, and still more if the patient is atheromatous, head rotation to one side may impair the circulation through both vertebral arteries. Whether symptoms occur then may depend upon the patency of the internal carotid arteries.

Though symptoms are often produced by extension of the neck I have been unable to find any angiographic studies of the effects, and anatomical studies of the effects of head movement suggest that the obstruction is not in the atlanto-axial segment.

Obviously some torsion of the brain-stem must occur when the head is rotated, and it is difficult to estimate the effects of that, but when the circulation is already impaired by vertebro-basilar ischaemia it might be a contributory factor in causing temporary additional failure of the circulation.

It has been shown by several workers (Boldrey, Maass, and Miller, 1956; Hardesty, Roberts, Toole, and Royster, 1960; Bauer, Sheehan, and Meyer, 1961) that head rotation may lead to reduction of blood flow in the contralateral internal carotid artery by compression of this vessel against the lateral process of the atlas, deep to which it can be seen passing in Fig. 1, and it has also been shown that kinking and stenosis of the cervical portion of a tortuous internal carotid artery may be produced by head rotation on either the ipsilateral or the contralateral side. Focal symptoms of disturbance of one cerebral hemisphere on head rotation are uncommon, but Professor Clifford Wilson has a patient

who had a right hemiplegia as the result of cerebrovascular disease, and who found that he could not talk to anybody on his right, because when he turned his head to that side he lost the power of speech; but he had no similar difficulty in talking to somebody on his left.

Another factor which may influence the cerebral circulation on head-turning is carotid sinus compression, a point to which attention has been drawn by Bauer, Wechsler, and Meyer (1961).

Role of the Labyrinth

Another possible source of symptoms on head rotation is the labyrinth. The ordinary method of testing for positional vertigo by passive rotation of the head, with the patient in a supine posture, does not discriminate between the effects of labyrinth displacement and the circulatory effects of rotation of the neck. The latter can be eliminated only by rotating the whole patient while maintaining the position of the head on the shoulders constant.

Most of the patients in the present series who suffered from vertigo resembled those described by Hallpike (1962) and whose vertigo was attributed to basilar insufficiency. "The vertigo," he wrote, "may occur in attacks which are usually of short duration, but it may take the form of prolonged bouts of unsteadiness aggravated by head movements. It may be precipitated by head position and is then recognized as the vascular variety of the benign paroxysmal type of positional nystagmus." Either rotation of the head or extension

FIG. 1.—Dissection to show relations of the left vertebral artery: head facing straight forwards. The artery is passing through the transverse process of the atlas vertebra. The internal carotid artery is also seen passing in front of the transverse process. 1, Vertebral artery. 2, Transverse process of atlas. 3, Vertebral artery. 4, Internal carotid artery.

FIG. 2.—The same with the head passively rotated to the right. The vertebral artery is carried forward with the transverse process of the atlas and the angle between the portions above and below the vertebra becomes acute. Cinematography shows that the lowest portion (to which the marker points) has been drawn up out of its canal in the lower cervical vertebrae. 1, Vertebral artery. 2, Transverse process of atlas. 3, Vertebral artery. 4, Internal carotid artery.

may precipitate such vertigo, but less often flexion. Before we consider its pathogenesis further we must discuss an associated symptom, the drop attack.

Drop Attacks

Three patients in my series of 100 suffered from drop attacks, which may be defined as sudden falls due to involuntary loss of the normal extensor posture of the lower limbs while standing, and not necessarily associated with loss or impairment of consciousness, though the fall may lead to concussion, in which case the patient may have a retrograde amnesia for the fall itself. It will be seen that I accept Kremer's (1958) ascription of the drop attack to interference with the mechanism for the maintenance of postural tone, and I also agree with him that this usually occurs at the level of the brain-stem. In the three cases he quoted the lesion was such as to produce compression, and he concluded that as the condition was reversible it was probable that the compression acted on the vascular supply. I have a similar case in my series.

Case 4.—The patient, a man of 62, suffered from both giddiness on head-turning and drop attacks. X-ray examination of his cervical spine showed a Klippel-Feil abnormality, a defect in the laminal arch of the atlas, and cervical spondylosis. He also had, however, an acoustic neuroma.

In my experience drop attacks are often associated with evidence of vertebro-basilar ischaemia, the vascular factor being, as Kremer says, the important one. However, the following case raises an interesting question.

Case 5.—A man aged 61 had suffered for three years from attacks of giddiness associated with progressive deafness and tinnitus in the right ear, which were considered to be due to Ménière's syndrome. He found during this period that looking up would make him dizzy. For six months he had suffered from drop attacks, of which he had had seven. He had one while playing golf, and though he fell and broke his glasses he was able to finish the round. Beyond slight unsteadiness on standing in Romberg's test he showed no evidence of organic nervous disease, but his neck movements were limited and x-ray examination showed cervical spondylosis from the fourth to the seventh cervical vertebrae, with involvement of the neurocentral joints and, at the highest level, of the apophysial joints, but without posterior osteophytes. It was thought probable that his cervical spondylosis was interfering with his vertebral-artery circulation and a collar was prescribed. A year later he had had no further attacks.

The fact that drop attacks and vertigo on head-turning may coexist in the same patient, and that drop attacks may be precipitated by head movement, suggests that the two symptoms may have a common basis in some cases.

Pathogenesis of Vertigo and Drop Attacks Provoked by Head Movement

How, then, does head movement produce vertigo and drop attacks? Head movement can impair the circulation, and it seems highly probable that in some cases both vertigo and drop attacks are circulatory in origin. It has been suggested that vertebro-basilar ischaemia may impair the blood supply to the labyrinths, but deafness is no essential part of this picture, so the vertigo is more probably due to ischaemia of the brain-stem or cerebellum. Even when deafness is present as well as vertigo it does not necessarily incriminate the labyrinth, for Hallpike (1962) has suggested that in such

cases deafness may be central in origin. But there is, I believe, a group of patients in whom the pathogenesis of the drop attacks is different. In these there is no evidence of vertebro-basilar ischaemia, and it has been suggested that the attacks are a form of epilepsy. If so, the same symptom may be produced in different ways, the postural mechanism being put out of action either by ischaemia or in some other way not fully understood.

Attacks of Unconsciousness

Four patients in the series suffered from attacks of unconsciousness, and all four presented interesting features.

Case 6.—A man aged 55 had attacks of vertigo for 12 years, sometimes brought on by head movement or change of posture. He had lost consciousness only in the first two, in one of which he fell downstairs. He had severe cervical spondylosis, and his electroencephalogram showed a slight to moderate dysrhythmia, consistent with cerebrovascular disease.

Case 7.—Another patient, a man of 61, for some months noticed that he was dizzy on raising his head when lying down and getting up in the morning. Then, without warning, he had an attack of loss of consciousness while driving his car. He had severe cervical spondylosis, and on flexion of the neck—the movement which made him dizzy—the body of C 3 moved forwards on C 4, which must obviously narrow the canals of the vertebral arteries. His E.E.G. showed a low-voltage dysrhythmia compatible with epilepsy.

These two cases show that there is a spectrum of symptoms extending from vertigo through drop attacks to attacks of unconsciousness which are probably all symptoms of vertebro-basilar ischaemia temporarily caused, or exacerbated, by head movements in the presence of cervical spondylosis. I will not now speculate about the precise cause of the attacks of loss of consciousness, but only suggest that when these occur in the elderly we should not assume that the cause must always be found above the tentorium.

Precipitants of Cardiac Asystole in Bundle-branch Block

Heart-block has been recognized for so long as a cause of attacks of unconsciousness that it may be thought that there is nothing new to say about it. But two of my patients, both of whom had cervical spondylosis, presented diagnostic difficulties and raised some questions of physiological interest.

Case 8.—A man aged 64 had had eight attacks of loss of consciousness during the previous three years. The first occurred while he was running across the street, the second when he bent down to pat his dog, and the third after turning over on to his back while swimming. Two occurred when he turned suddenly to the left in bed in order to reach the telephone, and another in bed when he turned to the right to put out the light. There was no convulsion. For 18 months he had complained of dizziness on bending, and he had also had some occipital headache and pain in the left shoulder. The only abnormality in his nervous system was that his right plantar reflex was extensor. X-ray examination of his cervical spine showed spondylotic changes extending from the second to seventh vertebrae. An electrocardiogram showed a left bundle-branch block. Before I saw him he had had both carotid and vertebral angiograms, which were negative, and it had been thought that his attacks were not due to his heart condition, but were epileptic. To investigate this, with the collaboration of Dr. Last, the patient had an E.E.G., in the course of which his head was turned to either side, both when he was sitting and lying, and carotid

compression was applied when his head was turned and also when his neck was extended. The resting E.E.G. record was somewhat asymmetrical, but turning and extending the head, with and without carotid compression, produced no change, and no symptoms.

Case 9.—A man aged 58 had had his first attack two and a half years before I saw him. This also took place when he was crossing a road, and some months later he had another while water-skiing. A later one occurred when he was playing golf, and yet another while he was swimming. He had had five in all, again without convulsion. Like the patient in Case 8 he had some physical signs of organic nervous disease, showing nystagmus on lateral fixation to right and left, relative increase in the tendon reflexes on the right side, and a right extensor plantar reflex. He, too, was known to have a left bundle-branch block, but the presence of the abnormal physical signs in his nervous system again suggested a cerebral origin for the attacks, and before I saw him he had been very fully investigated from that point of view with negative results. Plain x-ray films of his neck showed disk narrowing with osteophyte formation between C 6 and 7, and both vertebral and carotid angiography were normal. This patient had several more attacks, in one of which he happened to be seen by his doctor, who found that his pulse rate was 16.

Here, then, are two patients whose attacks of loss of consciousness in one case certainly, and in the other almost certainly, were due to paroxysmal heart-block. Although both patients were known to have bundle-branch block, their attacks had been thought to be cerebral in origin, since they both showed physical signs of organic nervous disease, which in Case 8 might have been due either to the cervical spondylosis or to vertebro-basilar ischaemia, and in Case 9, in view of the associated nystagmus, was probably due to vertebro-basilar ischaemia.

There is an odd similarity about the precipitating factors in both cases, since both patients had attacks while crossing a street and while swimming. One common factor is presumably exertion, but in the first case head-turning, when the patient was lying, on several occasions precipitated an attack, and both golf and swimming involve changes of head posture. In neither case did investigation suggest that the cervical spondylosis contributed to the attacks by interfering with the circulation through the vertebral arteries, but in neither case was the effect of head rotation on the vertebral angiogram observed.

Both of these patients were investigated by the late Dr. Paul Wood, who thought that in one case it was established, and in the other case practically certain, that the attacks were due to transitory complete heart-block produced by paroxysmal right bundle-branch block superimposed upon permanent left bundle-branch block, and he said it was well known that exertion could precipitate such attacks. He was unable to explain the effect of head-turning, which could hardly operate as a form of exertion. But he told me that he had produced a Stokes-Adams attack in a patient with a permanent right branch block by compressing the carotid sinus, which he thought must have caused left branch block. It was not a sinus arrest because the atria continued to beat normally.

I have one further observation which illustrates the complexity of these questions.

Case 10.—A man aged 59, a patient of Mr. Northfield's, for 15 months had had progressive symptoms of cervical myelopathy, and during that time had had three generalized convulsions. X-ray examination revealed severe cervical spondylosis C 5 to 7 and myelography showed a well-marked

indentation at these levels. Compression of the left carotid artery caused cardiac asystole for three seconds followed by bradycardia at 36/minute. The E.E.G. at that time showed irregular slow waves generalized over the left cerebral hemisphere and slightly also over the right. Compression of the right carotid sinus caused cardiac arrest for nine seconds followed by impaired consciousness with generalized high-voltage slow waves over both hemispheres on the E.E.G. An interim E.C.G. was physiological. An aortogram showed normal filling of both carotid arteries and of the right vertebral, but there was obstruction of the left vertebral artery opposite the fifth cervical vertebra.

Mr. Northfield performed two operations. On November 12, 1959, he exposed the intervertebral foramen between C 5 and C 6 on the left side and liberated the vertebral artery on its antero-lateral aspect. He observed that manipulation of the vertebral artery, as well as retraction of the carotid sheath, caused considerable disturbance of the pulse. On November 25 he performed cervical laminectomy to decompress the spinal cord.

The patient had a generalized convulsion on November 13 and another on December 28, 1959. The symptoms of the myelopathy have slowly improved and he wrote on November 30, 1962, that he had had no further attacks.

Here, then, was a patient with cervical spondylosis and myelopathy who suffered from epileptiform convulsions. Compression of either internal carotid artery caused cardiac asystole. The only radiologically demonstrable vascular lesion was compression of the left vertebral artery by a protruded intervertebral disk. Manipulation of this artery at operation also caused cardiac irregularity. Decompression of both the left vertebral artery and the spinal cord was followed within two months by cessation of the attacks, which had not recurred three years later.

This case illustrates the point made by Toole (1959) and by Bauer, Wechsler, and Meyer (1961) that vertebral-artery obstruction may contribute to the causation of the so-called "cerebral form" of carotid sinus sensitivity.

Paroxysmal heart-block should always be borne in mind as a cause of attacks of loss of consciousness after middle life, and such attacks may occur in cases of bundle-branch block. Such patients may well also have signs of organic nervous disease, which may misleadingly suggest a cerebral origin for the attacks. But how does head-turning precipitate heart-block in such cases? Dr. Wood's observation that carotid compression can produce paroxysmal complete heart-block in a patient with bundle-block suggests that head-turning may operate in this way. And Case 10 suggests that other factors, including possibly vertebral artery disease, may sensitize patients without organic heart disease to carotid sinus compression.

It is sometimes said that clinical observation has little more to offer neurology, yet clinical observation continues to discover new syndromes and to reinterpret old symptoms, and I foresee great scope for it still. Moreover, epidemiology is opening up a new field for the clinical observer, and may well provide vital clues to the aetiology of some neurological disorders. Though biochemists and electrophysiologists can answer questions which the clinician can only ask, it is often only the clinician who can ask them. After all, techniques, whether clinical or instrumental, discover nothing by themselves, and medicine is, and always will be, more than a technology. The insight derived from the new techniques should fertilize clinical observation, and the neurologist of to-day may still take Gowers for his model.

Summary

Cervical spondylosis is present more often than not after the age of 55. Special points which may be brought out by x-ray examination of the cervical spine are discussed.

The clinical features of headache due to cervical spondylosis are described. The headache is associated with a predominant involvement of the upper cervical spine and of the apophysial joints, but precisely how it is produced is still unknown.

The contribution which cervical spondylosis may make to vertebro-basilar ischaemia is discussed. Acute vascular lesions of the cerebellum or brain-stem may occur, and a case is reported in which this happened after therapeutic manipulation of the cervical spine in a patient suffering from spondylosis.

The effects of head movement on the cerebral circulation are considered, including the production of vertigo and drop attacks.

The cases of four patients who suffered from attacks of unconsciousness are described. All had cervical spondylosis. Two in addition had bundle-branch block, and their attacks were attributed to paroxysmal complete heart-block. Some factors which appeared to precipitate such paroxysms are considered. In one patient in whom cardiac asystole could be produced by carotid-artery compression, narrowing of one vertebral artery appeared to be a contributory factor, and its surgical decompression, together with decompression of the spinal cord, was followed by cessation of the attacks.

I am grateful to Mr. Douglas Northfield for allowing me to report Case 10, and to Dr. Marcia Wilkinson for Case 3, to Professor R. J. Harrison for the dissection, and to Mr. Ruddick for the photographs.

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The Ministry of Agriculture, Fisheries, and Food says that the agreement to restrict the use of cereal seed dressings containing aldrin, dieldrin, and heptachlor led to a very marked reduction in the number of birds dying as a result of toxic seed dressings last year, and it reminds farmers and agricultural merchants that these dressings should not be used on any cereal seed to be sown this spring.

OBLITERATIVE PULMONARY HYPERTENSION AND THROMBO- EMBOLISM*

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DISCUSSION

Pulmonary Thrombosis and Thrombo-embolism

The occurrence of thrombosis in the larger branches of the pulmonary arteries has been well documented (Ball *et al.*, 1956). Thrombosis in these sites usually, but not always, follows embolism. Pre-existing pulmonary hypertension and pulmonary vascular disease, such as in mitral stenosis, particularly favour thrombosis *in situ*, but thrombosis may also occur in severely debilitated and cachectic patients who are dying of a generalized disease, and also in children with polycythaemia due to congenital heart disease. Pulmonary-artery thrombosis is not infrequently found as an unexpected lesion in patients dying from cardiac infarction or severe valvular heart disease. It may also occur in atrial septal defect (Canada *et al.*, 1953; Bedford *et al.*, 1957). A low cardiac output favours not only thrombosis in peripheral veins with subsequent embolism, but also secondary embolism from the thrombosed pulmonary vessels. Repeated haemoptysis, attacks of pleuritic pain, recurrent syncopal attacks, and acute dyspnoea without obvious cause followed by progressive right ventricular failure have also been reported (Ball *et al.*, 1956).

If thrombosis occurs gradually in a major pulmonary vessel, collateral channels may develop via the bronchial circulation so that pulmonary infarction does not necessarily occur. Nevertheless, correct diagnosis is often delayed and haemoptysis attributed to other lesions such as carcinoma of the lung. By the time that obvious radiological changes suggesting occlusion of major pulmonary vessels have occurred the disease is well advanced. The condition should be suspected in any patient who develops right ventricular failure or signs of pulmonary hypertension without known cause, or who has unexplained dyspnoea or fainting.

While the occurrence of massive thrombosis of the pulmonary arteries following embolism has been well documented in patients with other diseases, the occurrence of a similar syndrome in previously healthy young persons has not been frequently recognized. Ehrner *et al.* (1959) described three cases with the syndrome very similar to those in our first group.

The occurrence of periods of freedom from symptoms may give rise to a false feeling of security that the condition has resolved completely. This point may be important with regard to the incidence of pulmonary embolism, occurring after medical or surgical disease, or spontaneously in fit young people. It is usually assumed that such incidents are self-limiting and that

*Concluded from p. 711 of last week's issue.