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PRACTICAL NEUROLOGY

Little Strokes

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The term "little stroke" is commonly applied to a brief neurological episode in which the focal nature of the signs and their mode of onset and regression suggest a vascular basis, and in which clinical recovery in the first attack is usually complete or nearly so. The phrase has no scientific merit, but at least it makes no assumption about the pathogenesis of the attack. In this it has the advantage over terms such as cerebral thrombosis, which is rarely substantiated at post mortem, and thrombo-embolism, which is usually a shrewd guess inspired by associated physical signs.

The importance of a little stroke is twofold. Firstly, while the qualifying adjective may be a comfort to both the physician and the patient, the episode may be a precursor of extensive damage to the central nervous system with either a fatal outcome or with subsequent lifelong disabilities. In selected cases treatment may avoid this. Secondly, such an episode may be closely simulated by conditions which, though rare, are eminently treatable. Both reasons are sufficient in themselves for these episodes to merit more attention than they commonly receive.

The purpose of this article is not to reiterate the well-known problems of pathogenesis of cerebral vascular disease, but to examine the clinical problem of the analysis of such an episode. It is assumed that obvious causes of cerebral emboli such as rheumatic heart disease and recent myocardial infarction have been excluded by the clinical findings.

History

The traditional teaching that the temporal sequence of events is the hallmark of a vascular episode of the central nervous system bears repetition. Once this is fully appreciated the minutiae of neuro-anatomy in relation to diagnosis assume less importance. Distinctions have been drawn between the sudden onset of an episode in some patients and the rather slow or stepwise progression which may be observed in others. It is probably more important to distinguish between attacks which last between 5 and 30 minutes and those which continue for several hours.

Very brief attacks are commonly referred to in the literature as transient cerebral ischaemia, and classically they are repeated at intervals; these intervals may vary from days to weeks or months. The overall frequency may vary from two or three to several score. Their recognition is useful, since present knowledge indicates that many are caused by platelet emboli which are capable of traversing the retinal and cerebral circulations without permanent damage to the structures supplied. The history of development of this concept and the pathological evidence is fully reviewed by Gunning *et al.*¹ Appre-

ciation of this mechanism may very well influence subsequent management and investigation.

The attacks, which continue for several hours, are the classic stroke in miniature, and it must be very rare for them to be repeated at the high frequency sometimes seen in transient cerebral ischaemia. Since both types of episode are usually followed by a complete recovery the first step in analysis lies with the information to be gained from the history, and from this certain problems emerge. A description of a clear-cut hemiparesis or hemiplegia requires no comment. In the carotid circulation, which supplies the territory of the retinal and the anterior and middle cerebral arteries, there are two areas of potential diagnostic difficulty, and these relate to episodes involving the sensory cortex and the retina.

An episode of sensory disturbance due to a vascular lesion in the parietal lobe may cause difficulty, since cortical sensory impairment may approximate roughly to peripheral nerve or dermatome distribution. This, in its turn, may initially suggest lesions of structures far removed from the true site of the lesion. However, if such episodes recur repetitively, then, in the proper context, the suspicion of a vascular disturbance of the sensory cortex may be entertained.

When visual disturbances occur during variations in the retinal circulation there may be some historical difficulties due to the fact that patients are often apt to ignore visual disturbance, and consequently it is essential to inquire routinely for such symptoms. The description of an episode lasting 5 to 10 minutes of a sudden visual impairment in one eye which ascends or descends "like a curtain," sometimes stopping short at the horizontal midline and then regressing, sometimes going on to total blindness, is diagnostic of transient failure of retinal circulation. Total recovery of vision is the rule in the first few attacks. There are variations on this theme, but the essence is the monocular quality of the episode. Such a history in the presence of signs of extracranial vascular disease—which are discussed later—indicates with some certainty that these episodes are due to platelet micro-emboli. Differential diagnoses such as migraine are usually no problem with an adequate history.

Vertebrobasilar Syndromes

The problem of diagnosis of a short-lived vascular episode in the vertebral circulation and its terminal branch, the basilar artery, stems from the complexity of the anatomy of the mid-brain, pons, and medulla. The problem is compounded by the fact that no fewer than 20 syndromes have been described in relation to vascular lesions in this area.

Williams and Wilson,² in an analysis of a personal series of 65 cases, have shown that the clinical problems are not as complex as the literature would suggest. In their account of

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the frequency of presenting symptoms they emphasize that vertigo was complained of in 48% of the patients (Table I).

TABLE I

Symptoms	No.	Percentage
Vertigo	32	48
Visual hallucinations	7	10
Drop attacks or weakness	7	10
Visceral sensations	5	8
Visual field defects	4	6
Diplopia	3	5
Headaches	2	3

Presenting symptoms in vertebrobasilar insufficiency from Williams and Wilson².

Visual symptoms (10%) and drop attacks (10%) were the next most frequent. This association of vertigo and visual disturbance is a useful clinical guide, since it indicates with certainty a vascular cause which could arise only from the vertebrobasilar circulation. Disordered consciousness, ataxia, and hemiplegic symptoms may all be symptoms of vascular disturbance within the territory of the vertebrobasilar circulation.

In the face of symptomatology which is typical of brain stem ischaemia it is essential to determine whether or not the symptoms are precipitated by head movement. If this is so, then cervical spondylosis is commonly present, and intermittent compression of the vertebral artery may be deduced. The importance of recognizing this condition is that the management is simple and the prognosis benign. A short period in a cervical collar is all that is usually required if the symptoms are frequent enough to merit treatment.

Once an adequate history has been obtained by paying due attention to the essential temporal sequence of events, the diagnosis of impairment of blood supply to a focal area within the central nervous system is relatively easy. The diagnosis of the presumptive cause is based on other information derived from subsequent examination and investigation. However, one must acknowledge that in many cases it is the advanced age of the patient which represents the sole supporting evidence for a diagnosis of vascular disease affecting the cerebral blood supply.

Finally, no history of a presumptive vascular episode within the central nervous system is complete without detailed inquiry about the occurrence of obvious colour changes preceding the attack, evidence of impairment of consciousness, and the history of vascular lesions elsewhere.

Examination

General examination may reveal xanthelasmic patches over the eyelids, suggesting a disturbance of cholesterol metabolism. Typical papillary lesions round the elbows and knees together with a brown pigmentation of the palmar creases will immediately suggest the presence of familial hypercholesterolaemia. A florid complexion may suggest but rarely indicate primary polycythaemia. If a significant degree of hypertension is found during or immediately following an acute episode it is essential to take further steps in order to determine sensible management.

Attention is then concentrated on the clinical evidence for extracranial vascular disease, and by this is implied stenosis or occlusion of the subclavian, the innominate, or the carotid arteries. A comparison of the radial pulses by palpation and ideally a comparison of the blood pressures of both arms may show a significant difference in the two limbs, which immediately suggests subclavian arterial disease. Auscultation of the cervical region, listening for the typical stenotic bruit over the subclavian in the supraclavicular fossa, over the origins of the internal carotid arteries, and over the orbit should be done. If the bruit is very prominent a thrill may be palpated over the appropriate vessels. Palpation of the neck vessels contributes very little to the examination. Uncommonly one can distinguish the residual cord of a totally occluded common

carotid artery, but differences in pulsation between the two carotid arteries are usually an artifact and due to the artery being medially displaced.

Hypertension

The presence of a significantly raised diastolic blood pressure during or immediately following an episode of cerebral ischaemia is a common clinical observation. This is particularly true in an episode involving the vertebrobasilar circulation, when the diastolic blood pressure may reach alarming heights and then return to normal within hours or days. If the blood pressure is significantly raised it is essential to repeat the observation after a period of rest in bed. If the diastolic blood pressure remains elevated it immediately raises the problem of the diagnosis of the intracranial episode and the subsequent management.

The diagnostic problem may be briefly stated. It is now recognized that in many patients it is not possible to differentiate between a focal intracerebral haemorrhage and a lesion due to an occlusion of the arteries supplying that territory. It is certain that such focal haemorrhagic lesions may simulate the "little stroke." The rediscovery of the Charcot-Bouchard aneurysm by Ross Russell,³ and the subsequent confirmation of this work by Cole and Yates,⁴ has added a further dimension to the analysis of the little stroke. While the evidence may not be absolutely complete, it seems probable that microaneurysms of the cerebral circulation, which are a direct result of hypertension, may be capable of mimicking exactly a small stroke due to occlusive vascular disease and possibly also of being responsible for very much shorter episodes. The implication of this is that such episodes, in the presence of hypertension, may be an absolute indication for the treatment of the hypertension.

There is little doubt that the dangers of treating hypertension in these circumstances has been relatively overemphasized. An increasingly good case can be made out for the fact that significant hypertension should be treated in patients who present with evidence of a markedly raised diastolic blood pressure and restricted cerebral vascular episodes.^{5 6}

Cardiac Lesions

With an adequate history, suspicion that an underlying circulatory cause is responsible for the transient focal disturbance in the central nervous system will already have been entertained. Particularly will this be so if there is a clear history of pallor before the attack, blurring of vision, or any of the usual concomitants of a transient fall in cardiac output. It is usually suggested that the focal flavour of the attacks in these patients indicates pre-existing stenosis of the vessels supplying that particular territory.

Once a cardiac lesion is suspected there are three unrelated diagnostic problems. They are the hypersensitive carotid sinus, episodes of transient cardiac standstill, and lesions in the heart which may act as sources of emboli. The first is easy to recognize, the second and third are difficult, if not at times impossible.

Hypersensitivity of the carotid sinus is rare but important, since it is a treatable condition. If the history is suggestive of a transient fall in blood pressure or an episode of cardiac standstill preceding the neurological episode then it is essential to test the carotid sinus reflex. This is done first of all with the patient lying flat. While auscultating the heart, gentle stimulation is carried out of first one and then the other carotid sinus. The stimulation must be light, since it is surprisingly easy to occlude the carotid vessel; auscultation is essential, since cardiac standstill must be recognized immediately and stimulation of the sinus ceased. If a positive reflex is obtained

then it should be repeated with the patient in the sitting position. The purpose of this is that if transient neurological symptoms are to be attributed to a hypersensitive carotid sinus reflex, then the symptom complex complained of by the patient must be reproduced during the response of the hypersensitive carotid sinus. It should be appreciated that in the elderly and in patients receiving digitalis the carotid sinus reflex may be hypersensitive and yet bear no relationship to any presenting clinical symptomatology.

Intermittent cardiac standstill as a possible cause of a brief neurological episode is difficult, if not impossible, to diagnose at times, since the electrocardiograph and the pulse rate between attacks may be entirely normal. The characteristic flushing of the face following an episode may be highly suggestive of a Stokes-Adams attack. Gilchrist⁷ reviewed the problem and indicated from his wide experience how many patients have attended medical and neurological clinics with a variety of diagnoses before the diagnosis of heart block could be established. The following case history outlines the problem:

A 62-year-old man presented with episodes of transient vertigo associated with tingling in the limbs. The attacks were short-lived and their quality suggested a vascular basis. There were no associated physical signs to indicate a possible underlying cause. They continued at intervals over the next five years at a frequency of two to three times a year and were no great disability. One month before the patient's death there was a sudden increase in the frequency of symptoms. The patient was seen again at this time, and a pulse rate of 30 was noted with recurring episodes of cardiac standstill. In each of these episodes symptoms of brain stem ischaemia developed and passed off with the restoration of normal cardiac rhythm. The patient died within a month of developing complete heart block.

The third difficult situation is the heart as a source of emboli where there are no obvious physical signs suggesting such a diagnosis. Obvious causes of cerebral emboli, such as long-standing fibrillation associated with rheumatic heart disease, have been implicitly excluded from consideration in this context. There are, however, other circumstances in which the heart can be an unsuspected cause of recurring cerebral emboli. One difficulty is that endomural thrombus from cardiac infarction may continue to be a source of embolic material for some months after an episode of cardiac infarction; a history in recent months of substernal pain or cardiac infarction may be a useful indicator. Another difficulty is bacterial endocarditis, which presents a particularly difficult diagnostic problem in the elderly and is worth bearing in mind should there be any unusual features in the whole clinical picture. Thrombotic endocarditis, which sometimes develops in the presence of a systemic neoplasm, is a similar diagnostic problem. Finally there are the extremely rare examples of myxoma in the left atrium or ventricle. The observation of emboli elsewhere than in the central nervous system is an immediate indication that emboli from the heart may be responsible for the neurological episode.

Hypoglycaemia

The quite unrelated condition of hypoglycaemia may from time to time simulate a small stroke. Again it is postulated that stenosis of a local vessel may focus the maximal effect of hypoglycaemia in a certain area of the brain. There are two problems. The first relates to the treated diabetic and the second is the occurrence of the spontaneous hypoglycaemia.

Two case examples will suffice to demonstrate that admittedly unusual clinical variants on the theme of hypoglycaemia may simulate a brief focal vascular lesion.

A lady of 70 had been under treatment for diabetes for 10 years. She and her relatives had noted that, following a change in her insulin dosage, on waking she was occasionally unable to move her arms and legs and unable to speak because of immobility of her jaw. The state she described was suggestive of that seen occasionally in

basilar artery occlusion, which bears a superficial relationship to the state of akinetic mutism. Observation of an attack in the early hours on waking established that she was in fact quadriplegic, with a palsy of the jaw, palate, and tongue. Her blood sugar at this time was below 35 mg./100 ml. Adjustment of the insulin dosage to prevent early morning hypoglycaemia was all that was required to control her symptoms.

Another patient demonstrates the problems sometimes posed by the diagnosis of spontaneous hypoglycaemia.

The patient, a middle-aged, moderately obese woman, was referred with transient episodes of vertigo, diplopia and tingling round the mouth, which occurred several times a month. There were no known precipitating factors. Although the majority were short-lived, lasting for 20 minutes or so, she did volunteer that after some of these attacks she occasionally had episodes of minor mental confusion for two to three hours. The significance of this particular point was not appreciated in the history. After inappropriate investigations the symptoms were considered typical of hind brain ischaemia. At subsequent follow-up examination two months later she was advised to reduce her weight by avoiding carbohydrates in her diet. The patient took this advice literally and within four days she was admitted in coma to another hospital with a blood sugar of under 20 mg./100 ml. An islet cell tumour was removed from the pancreas and she has subsequently remained symptom-free.

There are more sophisticated methods of making a diagnosis of islet cell adenoma, but this experience underlined the importance of paying due attention to patients with presumed vascular lesions where mental confusion is a notable symptom.

Miscellaneous Conditions

Under this heading are included a variety of conditions which may produce transient circulatory disturbances of the central nervous system. In every patient the Wassermann reaction should be done and diabetes mellitus excluded. Thereafter there are a number of conditions which, though rare, may be treatable and should be considered in the analysis.

In the elderly it is always necessary to exclude the possibility of giant cell arteritis. A history of pain over the temporal artery or less commonly in the occipital region, or a history of pain in the jaw while eating may be suggestive. The occurrence of an intracranial vascular lesion in this disease without any of these associated features would be very unusual. The most important investigation is the sedimentation rate, which is usually elevated. Early diagnosis is a matter of urgency because of the necessity for prescribing steroids, not only with the object of relieving pain—which treatment will achieve in a short space of time—but also to avoid the complication of retinal artery occlusion and subsequent total blindness.

In youth and middle age the diagnostic possibilities cover a wider field. In a recent review of the problem Levine and Swanson⁸ examined the many possibilities. Oral contraceptives are receiving considerable attention at the moment in the young female and are unlikely to be overlooked: myeloproliferative disorders, haemoglobinopathies, thrombocytosis, and dysproteinaemias are representative of some haematological disorders; lupus erythematosus, polyarteritis nodosa and the rarer forms of arteritis, such as Takashay's syndrome, may also be considered. The list is lengthy and diverse, which emphasizes the need for full-scale investigations in this group of patients.

Management

The possibility that the effects of degenerative cerebral vascular disease may be affected by any therapeutic measure has prompted an enthusiastic attack by surgeons on the extracranial circulation and attempts by physicians to alter the prognosis by the use of anticoagulants. Subsequent reports have produced two possible sources of confusion. The first is that the concentration of attention on the late effects of atheroma may obscure the fact that the "little stroke" is a clinical syndrome

which requires careful definition before any decision on management can be made. The second is that therapeutic trials on hospital patients are in the main reporting a highly selected group who may represent only the fringe of the problem as it relates to the general practitioner.

The problems of definition have already been discussed. In summary in every patient it is essential to exclude active neurosyphilis, associated diabetes, and blood dyscrasias. These require trivial investigations for the patient. In the elderly it is essential to exclude giant cell arteritis; in the young and middle-aged the diagnostic field is so wide that hospital investigation is probably indicated in the majority of cases. The presence of sustained hypertension demands a critical appraisal of the subsequent management.

After these possibilities have been eliminated it must be accepted that the late effects of atheroma will be the underlying cause in the majority of patients of any age. The possibility that the advice implicit in hospital reports published in the literature on the management of these patients may be only marginal to the total problem of cerebral vascular disease is worth examining. For example, if stenosis of the extracranial blood supply were the sole criteria for operation, the available evidence from two hospital studies would suggest that only 15 to 20% of all patients suffering from vascular disease would be candidates for surgery.^{9 10} There is also the possibility that the general practitioner may be seeing a very different aspect of cerebral vascular disease compared to that seen in hospital. In a survey in general practice in Stoke-on-Trent¹¹ the mean age of onset of cerebral vascular disease was 65 years, nearly a decade older than the age of patients usually reported in hospital series. The incidence rate per 100,000 per year for all cases of cerebral vascular disease was 536, and of these 183 per 100,000 would be classified as having sustained a little stroke. If an unquestionably effective treatment free from complications and suitable for this age group became available this would be a considerable addition to the clinical load of every active practitioner.

Scepticism on the effectiveness of any method of treatment to counteract the late effects of degenerative vascular disease is understandable and desirable in the absence of properly controlled trials. There is one circumstance where the clinical presentation may suggest an urgent need for referring the patient to a unit experienced in the handling of the problem. Ideally the patient is in the middle or late 50s, physically well preserved, with no significant vascular disease elsewhere. The

presenting complaints of repeated transient episodes in the carotid or vertebrobasilar circulation are associated with a bruit in the neck over the appropriate major vessel. Subsequent contrast radiology demonstrates a clear-cut stenosis of this vessel, and subsequent surgical treatment results in removal of an ulcerating atheromatous plaque with its obvious potential for producing massive emboli or platelet micro-emboli. If surgery is precluded for whatever reason there is some evidence that the use of anticoagulants in these circumstances may be of value.

It must, however, be emphasized that the opportunities for such energetic therapeutic measures are rare, and the management of these patients still demands common-sense advice coupled with a determination not to miss the limited opportunities for treatment that may emerge from a careful assessment of the cause of the neurological episode.

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ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Analgesics in Injury

Q.—What is the analgesic of choice for acute pain in accident cases when continuing observation is necessary to establish whether there is also cranial injury and a subdural haemorrhage?

A.—There is little new to offer in the answer to this question, but before seriously considering giving an analgesic drug it is necessary to be sure that the patient is in pain and not just frightened. In general, the more severely injured a person is the less troubled he is by pain. Those who suffer most are usually the victims of solitary injuries that do not in any way cloud consciousness and do not therefore impose any restriction on the choice of drug.

In my experience there has rarely been any serious clash between kindness and safety in the use of analgesic drugs, but the fact that more or less unconscious patients have at times received full doses of morphine, for example, suggests that some confusion exists. It is perhaps still necessary to state that morphine in particular has no place in the treatment of injured persons when they are not in pain or faced with a painful experience. It is an analgesic drug, and should be used only when a person is suffering more or less severe pain. It acts most rapidly—and briefly—when given by intravenous injection. This has the advantage that it allows erring on the side of safety by giving fairly small doses, which can be repeated as frequently as symptoms demand.

The two most effective analgesic drugs are still morphine and pethidine, and the fact that authoritative opinion has found little to choose between them does not prevent most doctors from regarding one more favourably than the other.

Pigeon-toe

Q.—What can be done to correct the deformity of pigeon-toe?

A.—Pes varus does not need any treatment unless the deformity is fixed—that is to say, the foot cannot be put into a neutral position by the examiner or the child's own muscles. The majority are a reflexion of medial-rotation deformity of the tibia or the neck