HANDBOOK OF CLINICAL NEUROLOGY

P. J. VINKEN AND G. W. BRUYN

12

VASCULAR DISEASES OF THE NERVOUS SYSTEM

PART II

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Edited by

P. J. VINKEN and G. W. BRUYN

NORTH-HOLLAND PUBLISHING COMPANY · AMSTERDAM
AMERICAN ELSEVIER PUBLISHING CO., INC. · NEW YORK

Library of Congress Catalog Card Number: 68-8297

ISBN for the complete set of 30 volumes: 0 7204 7200 8

ISBN North-Holland for this volume: 0 7204 7212 1

ISBN American Elsevier: 0 444 10061 X

284 illustrations, 50 tables

PUBLISHED BY:

NORTH-HOLLAND PUBLISHING COMPANY · AMSTERDAM

SOLE DISTRIBUTORS FOR THE U.S.A. AND CANADA:

AMERICAN ELSEVIER PUBLISHING COMPANY, INC.

52 VANDERBILT AVENUE

NEW YORK, N.Y. 10017

PRINTED IN GERMANY

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VOLUME 12

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Foreword to volumes 11 and 12

'On a l'âge de ses artères'. The contents of these two volumes on vascular diseases of the brain and spinal cord reaffirm the truth of this maxim, first formulated by Cazalis in 1893 [Debore-Achard, Manuel de Médecine I (1893) p. 378]. At a time when cancer research, cardiac ailments and heart transplantation are monopolising the various publicity and news media (and thus the consequent financial assistance), the less news-worthy, but more prolonged misery and loss of dignity due to 'aging of the arteries' is often only fully appreciated by the neurologist. Since cerebrovascular disease is more prevalent amongst an already vulnerable section of society, namely the aged, the clinical neurologist is indeed faced with a gargantuan task when called upon to treat, help and comfort an aphasic, hemiplegic patient who is often already at the end of his life span. The concepts of treatment and diagnosis currently employed in this weighty task form the basis of these two volumes.

Among the provinces of the body supplied by the cardiovascular system, the brain occupies an unique position. In spite of its most economical use of energy, which should thus give it some immunity to a possible impairment in blood supply, the brain cannot in fact survive a serious delay or interruption without sustaining considerable damage to both 'function and form'. This vulnerability to interception of the blood supply results from the metabolic structure of the brain which, in contradistinction to muscle and liver for example, is geared in such a way as to be devoid of substrate reservoirs. Damage thus sustained cannot be repaired as the nerve cell possesses no regenerative or reproductive powers, these capabilities having been relinquished in favour of attaining the ultimate specialisation, differentiation and development required to evolve into what might be termed the 'peer' of all cells. The immense problems in treatment thus created by this evolutional loss and further complicated by the nerve cell's dependence on the glial cells, which feed and sustain it, perhaps partly explain why apoplexia cerebri has been rather neglected by neurological investigators for long periods of time. Only in the last two decades has fresh impetus been given to the study of this disease with the advent of serial angiography, cerebral blood-flow studies, the discovery of chemical compounds aimed at interference with the arteriosclerotic process and deranged coagulation, and methods of surgical intervention in cases of aneurysms, hematomas and vascular occlusion.

The contents of these two volumes on vascular diseases of the brain and spinal cord have been so designed and arranged as to make them complete in their own right,

providing the clinician with a full coverage of both normal and morbid anatomy, epidemiology, physiology and pathophysiology, diagnosis and differential diagnosis, disease entities and syndromes, and conservative and surgical treatment. A few recently-defined and well-documented diseases (e.g., Moyamoya disease, so strikingly prevalent in the Japanese and probably due to perinatal cerebrovascular occlusion) and fibromuscular dysplasia have also been included. Contrarily, some syndromes such as carotid occlusion following blunt head or neck injury, and multiple microthrombosis in infancy, have not been allotted separate chapters.

Discovery of an ultimate therapy to provide relief for the millions of sufferers from cerebrospinovascular (occlusive) disease, can, with a measure of almost absolute certainty, be expected to come from both a breakthrough in the biochemical elucidation of arteriosclerosis and the aging process of nerve and mesenchymal cells, and a final prophylaxis of these diseases. The main problems to be tackled remain: the pathogenesis of arteriosclerotic occlusion of major cerebral vessels, of congophile and amyloid angiopathy of the small cerebral vessels, and of the aging of neurones and neuroglia. Once these problems are solved, Man may enjoy a fully active and productive existence even up to an advanced age. Until that happy day arrives, however, the present generation can only be offered long-term rehabilitative treatment following a correct diagnosis; it is the sincere hope of the Editors that these two volumes may in some way both assist the present-day attack on cerebrospinovascular diseases and may contribute, in however small a way, to the eventual defeat of this disease.

During the final preparation of this volume in April 1971 the sad news reached us of the demise of Professor Raymond Garcin. The loss of his distinguished, humane and brilliantly spirited presence is deeply felt by us. From the very beginning of the Handbook of Clinical Neurology, Professor Garcin actively cooperated (volume 1, chapters 11 and 12) and advised in the countless discussions between us. His huge experience, reflecting the traditionally high standard of French Neurology he himself so extensively contributed to, unerringly led him to point out optimal solutions for numerous problems encountered in the realisation of this Handbook.

The hazy transparency of our skies revealed to him the essential secret of the 17th century Dutch landscape-painters; the lively luminescence of his mind revealed to us the secret of the 20th century French neurologists. The happiness and honour to have had him with us so many, though too few, years strengthens us to continue our endeavours of also shaping this Handbook of Clinical Neurology as a tribute to this 'neurologiste chevronné'.

P. J. V.
G. W. B.

Acknowledgement

Several illustrations and diagrams in this volume have been obtained from other publications. Some of the original figures have been slightly modified. In all cases reference is made to the original publications in the figure caption. The full sources can be found in the reference lists at the end of each chapter. The permission for the reproduction of this material is gratefully acknowledged.

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Contents

A survey of occlusive disease of the vertebrobasilar arterial system

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Vertebrobasilar ischemia is one of the commonest manifestations of cerebrovascular disease and, at the same time, one of the most baffling to the clinician. Its transient nature (for in this section we are not dealing with ischaemia of such severity and persistence as to lead to infarction), its protean symptomatology and the variety of conditions with which it may be confused, makes diagnosis and management extremely difficult. On the other hand, it offers a challenge to the physician interested in cerebrovascular disease for it provides a number of dynamic situations, study of which will help to elucidate the many unsolved problems of cerebrovascular disease.

Awareness of the importance of transient disturbances in patients with cerebrovascular disease was first aroused by Denny-Brown who suggested that episodic insufficiency in the circle of Willis may cause recurrent episodes of paralysis. Fisher and Cameron (1953) stressed the frequency with which these transient episodes preceded a completed stroke and Alajouanine et al. (1960) discussed their relationship to occlusive vascular disease. Millikan and Siekert (1955) paid special attention to those occurring in the vertebrobasilar territory the symptomatology of which was reviewed by Williams and Wilson (1962) and the natural history by Marshall (1964).

Anatomical considerations

The anatomical features of the vertebrobasilar arterial system have been fully treated in another chapter of this volume; here it is only necessary to emphasize a few points which are of importance in the clinical situation. The first of these is the origin of the vertebral arteries from the subclavian arteries, a fact which makes changes in subclavian flow important when considering vertebral flow, as seen in the subclavian steal syndrome. The second is the frequent disparity in size between the two vertebral arteries; this may, on occasion, be of such degree that one is dealing in effect not with paired vessels, but with a single artery. The third point is the juxtaposition of the vertebral arteries and the cervical intervertebral discs, coupled with the proclivity of the latter to degenerate, extrude and provoke osteophyte formation with impingement of the latter upon the artery.

These anatomical points cannot be considered in isolation but must be related to the facts about the available collateral circulation, a subject which has also been dealt with elsewhere in this volume. The obvious source of collateral supply to the vertebrobasilar system is from the carotid arteries via the circle of Willis. This involves the posterior communicating arteries which may be absent or congenitally narrowed or constricted by

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atherosclerosis. Congenital defects, as Alpers et al. (1959) have shown are present in about 23 per cent of normal brains for they found that in 78 out of 350 cases one posterior communicating artery had an external diameter of 1 mm or less. In these circumstances the circle of Willis cannot adequately fulfill its function as a source of collateral flow. On the other hand, when the posterior communicating artery is of normal calibre and healthy, its potential for hypertrophy is tremendous as may sometimes be demonstrated when the internal carotid artery has been ligated, the posterior communicating expanding to carry blood from the basilar to the middle and anterior cerebral arteries on that side.

A less obvious source of collateral supply is via the deep cervical branch of the costocervical trunk which arises from the subclavian artery distal to the origin of the vertebral artery. The deep cervical artery can develop an extensive network of connexions with the segmental branches of the vertebral arteries. In addition collaterals may develop between the occipital branch of the external carotid artery and muscular branches of the vertebral, and between the two vertebral arteries via their intraspinal branches.

These various points have been stressed because, unless they are fully appreciated, the apparent vagaries of vertebrobasilar ischaemia cannot be understood. There are no simple rules of thumb to be applied here. The consequences of stenosis of the origin of the vertebral artery by atheroma in the subclavian are greatly affected by whether or not the atheroma also involves the origin of the costocervical trunk. Again involvement of the vertebral and the costocervical trunk may be of no consequence if the vertebral was congenitally small and never contributed a significant proportion to the basilar blood flow. The variety of possibilities makes each case of vertebrobasilar ischaemia a study in itself. The existence of a particular anatomical arrangement or the presence of a certain kind of lesion may be quite irrelevant in one case yet of critical importance in another.

Transient ischaemic attack

Before discussing the causes of ischaemic attacks in the vertebrobasilar territory, it seems best to define the concept underlying the use of this term and to outline the associated symptomatology.

Transient ischaemic attacks in the cerebrovascular system as a whole are important for two reasons. Firstly, they often herald the advent of a more serious cerebrovascular episode. Secondly, their transient nature indicates that dynamic factors are at work, modification of which may prevent the development of a stroke. A transient ischaemic attack may be defined as a short-lived and focal disturbance of cerebral function arising as a result of ischaemia and occurring in a patient suffering from degenerative cerebrovascular disease. How long a disturbance may last, yet still be called transient, is entirely a matter of arbitrary decision. Episodes to which the label transient ischaemic attack is commonly attached, usually do not persist for more than a few minutes though in some instances the disturbance may last for an hour or two. In some studies an upper limit of 24 hours has been set, though it is not envisaged that the symptoms will trouble the patient for that length of time, but rather that residual evidence of the attacks, such as a trace of nystagmus or an equivocal plantar response, may be found during that period. Twenty-four hours has certain advantages as an upper limit for it is rare for signs which persist beyond that time to disappear entirely.

The concept underlying the clinical term transient ischaemic attack is that of a period of ischaemia insufficient in duration and severity to cause infarction. The complete restoration of function is essential to the definition. Because of this it has been tacitly assumed that pathological examination would fail to reveal any evidence of a lesion, but it must be recognized that small areas of infarction may occur without producing persistent clinical deficits; a lesion must have a minimum size, which varies according to its site, before persistent clinical manifestations can be expected. Even more important is recognition of the fact that a very small haemorrhage may give rise to nothing more than a transient ischaemic attack. Despite these ambiguities the term transient ischaemic attack is useful in clinical practice.

Besides being short-lived, transient ischaemic attacks are also focal in character. Clearly, an

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ischaemia which is transient need not be focal; thus cerebral ischaemia which is diffuse and transient will produce a syncopal attack and not a focal disturbance. This type of diffuse disturbance is not usually included under the clinical term transient ischaemic attack, an essential feature of which is the focal nature of the symptomatology. There are, therefore, two essential features in a transient ischaemic attack, firstly its transient nature and secondly its focal distribution.

Transient ischaemic attacks may occur in the territory of any of the cerebral arteries but there are good reasons for distinguishing between those occurring in the carotid tree and those which involve structures supplied by the vertebrobasilar arteries. The present chapter is confined to a consideration of the latter.

THE CLINICAL PICTURE

The clinical features of transient ischaemic attacks occurring in the vertebrobasilar territory are extremely varied. This is because the part of the brain supplied by these vessels, viz. the brain stem, part of the temporal lobes and the occipital poles, contain anatomical structures subserving a multiplicity of functions. A simple, practical approach is to consider the disturbances which may be produced by vertebrobasilar ischaemia from the more distal to the more proximal part of the arterial tree.

Disturbance in the visual fields

Firstly, there may be loss of vision in part or all of the visual field. The patient may experience a sudden loss of vision in the whole of both visual fields. It may be abrupt in onset, giving rise to an almost immediate blackness, or it may develop over the course of a few seconds, greying of vision preceding the loss. The episode persists for only a few minutes, rarely more than 3 or 4, and restoration of vision is usually gradual, a period of grey or blurred vision preceding total recovery. This type of disturbance depends upon reduction of flow through both posterior cerebral arteries simultaneously.

Another manifestation which occurs when the posterior cerebral artery on one side only is involved is the development of a hemianopia. Altitudinal defects may also occur due to ischaemia of selective parts of the posterior end of the optic radiations. Yet another type of disturbance is the loss of peripheral vision in both fields with some degree of preservation of the central part. This is attributed to that part of the occipital cortex concerned with macular vision being anastomotically supplied from the middle cerebral artery. Because patients experiencing these visual upsets are by definition suffering from cerebrovascular disease, this anastomotic circulation is often inadequate, hence the more usual picture of loss of vision in the entire field.

On occasion the ischaemia of the occipital cortex may be less severe, so that instead of there being loss of vision positive symptoms develop; thus the patient may see flashing or coloured lights in part or all of the visual fields. This phenomenon may occur alone, or it may be experienced as a preliminary phase in the development of visual loss. Sometimes the flashing lights may continue for a considerable period of time, even for some days, longer than is the case when loss of vision occurs.

Amnesia and confusional episodes

The next type of disturbance to be considered is that arising from ischaemia of the tentorial aspect of the temporal lobe, which is supplied by branches from the posterior cerebral artery. The most striking clinical picture is that referred to as global amnesia. In this syndrome a patient loses his memory for current events for a period of some hours. During this time he may undertake activities, even of a complex nature, in a manner which gives no clue to an observer of there being anything wrong. Subsequently, however, it becomes apparent that the patient has no recollection of what he was doing during that time. Thus one patient presided at a meeting and conducted the business impeccably, yet some time after its conclusion asked his secretary whether it was time for him to go to the meeting. It then became apparent that he had no recollection of anything that had passed. Global amnesia has on several occasions been encountered when an elderly gent-

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e man on vacation has taken an unaccustomed swim in a cold sea.

Another clinical picture which may arise from ischaemia in this territory is a confusional episode in which the patient becomes disorientated, especially for time and place. This picture contrasts markedly with that of global amnesia in which orientation and behaviour appear completely normal during the attack.

Diplopia

Descending into the brain stem, the next structure which may be involved is the complex of the third and sixth nerve nuclei and the medial longitudinal fasciculus. Ischaemia in this region causes diplopia which is a common manifestation of vertebrobasilar ischaemia. The transient weakness of the ocular muscles may produce nystagmus which may be seen by the observer. Alternatively, nystagmus may be due to associated ischaemia of the vestibular neurones and their connexions.

Disturbance in the face

Disturbance of sensation in the face is a common manifestation of vertebrobasilar ischaemia. It is usually bilateral and commonly circumoral in distribution and consists of pins and needles or numbness; in the case of the latter symptom the patient usually recognizes it as being like the sensation he experienced when he had a nerve block for a dental extraction.

Facial weakness may occur but is more commonly encountered in transient ischaemic attacks in the carotid territory. When it is a sign of vertebrobasilar ischaemia, it is usually lower motor neurone in type and may be unilateral or bilateral.

Vertigo

Vertigo is a particularly difficult symptom to assess. This is partly because it is difficult to know what the patient means when he complains of 'dizziness' or 'giddiness'. Is it true vertigo, that is the subjective sensation of movement in space of which he is complaining, or is it blurring of vision, darkening of vision, a faint feeling or some other presyncopal symptom about which he speaks? Even if it is possible to establish with reasonable certainty that the patient is experiencing true vertigo, there remain the problems of its site of origin and its cause.

Some authors insist that the experience of a sensation of rotation is necessary for the diagnosis of vertigo. This view ignores the fact that many patients with undoubted brain stem vascular disease experience sensations other than rotation. The vestibular complex is concerned not only with movement through space but also with position in space and either or both may be disturbed. Thus patients often speak of a sensation of the ground moving or being tilted, of themselves being pushed to one side, of falling, or of being in a boat. In this article the word vertigo will be used to indicate any subjective sensation of movement in space whether rotatory or not.

Even when due allowance has been made for the fact that vertigo is often attributed to ischaemia without sufficient justification, it remains the commonest manifestation of vertebrobasilar ischaemia. It may occur as an isolated symptom, its vascular origin finding proof in the fact that some cases go on to develop a brain stem infarct within 4 to 6 weeks. More often it is accompanied by other symptoms and signs which may give a clue as to its site of origin and its aetiology. In assessing associated symptoms distinction must be made between those which may be consequent on the vertigo itself and those which indicate involvement of brain stem structures other than the vestibular neurones. Thus nausea, vomiting, diarrhoea, sweating, feeling faint and an inability to stand may all be the result of the vertigo and cannot be used as a necessary indication of brain stem involvement. On the other hand, dysarthria, numbness and paraesthesiae in the face, hemiparesis, hemianaesthesia and diplopia clearly indicate that the brain stem is the site of the disturbance. If, in addition, there is loss of vision, the vertebrobasilar origin of the brain stem disturbance is clearly established for it is only via their common arterial supply that the occipital cortex and the brain stem can be linked.

Whilst the presence of associated symptoms and signs may indicate that the lesion is vascular, their absence does not, as has already been men-

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tioned, exclude this possibility. Apart from the fact that the vestibular neuronal complex appears to be the structure in the brain stem which is most sensitive to ischaemia, it must also be remembered that the labyrinth itself is supplied from the vertebrobasilar system. It receives its blood supply via the internal auditary artery which usually takes origin from the anterior inferior cerebellar artery but may arise directly from the basilar. Reduction of flow through the internal auditary artery may give rise to vertigo with or without cochlear symptoms.

The difficulties in the way of assessment of the significance of vertigo are reflected in the fact that whereas other symptoms occurring in transient ischaemic attacks correlate well with the site of the lesion causing a subsequent stroke, vertigo does not. Marshall (1964) found that in some cases in which vertigo was the only symptom in a transient ischaemic attack, the subsequent completed stroke was in the left carotid territory.

Whilst vertigo is the commonest symptom encountered in vertebrobasilar transient ischaemic attacks, tinnitus or transient deafness is probably the rarest. Occasionally, however, a patient will describe an attack being heralded by noise or whistling in the ears.

Dysphagia and dysarthria

Infarction of the brain stem can certainly be followed by dysphagia, sufficient at times to require tube-feeding for a long period and even to necessitate tracheotomy, yet dysphagia is an uncommon symptom in transient ischaemic attacks. This is presumably because they do not usually persist long enough for the patient to have the opportunity of testing his ability to swallow.

Dysarthria on the other hand, is frequently encountered and may, when the transient ischaemic attack follows the taking of alcohol, be wrongly attributed to intoxication. It may be confused with dysphasia and lead to the ischaemia being mistakenly sited as occurring in the carotid instead of the vertebrobasilar territory.

Ataxia, hemiplegia and hemianaesthesia

Ataxia is a common manifestation of vertebrobasilar transient ischaemic attacks being usually bilateral and affecting stance and gait more than functions involving use of the arms. It is sometimes difficult to tell whether the ataxia complained of is due to a cerebellar disturbance or whether it is a concommitant of vertigo; at times both components may in fact be present.

A hemiparesis may also occur due to involvement of the pyramidal tract. Most characteristically it involves the arm and leg but spares the face. There is frequently bilateral involvement in the one attack, but alternation of the hemiparesis from one side to the other in different attacks may be seen. Even when the patient complains of weakness on one side only, examination during or shortly after an episode may reveal unequivocal signs such as an extensor plantar reflex on the opposite side also.

Similarly, sensory disturbance down one or other or both sides of the body may occur, consisting of pins and needles or numbness; pain however is a very rare symptom.

Drop attacks

We come next to drop attacks which are a puzzling phenomenon often attributed to vertebrobasilar ischaemia. Drop attacks are typical and unmistakable; the patient suddenly without warning or provocation falls to the ground. He is certain he did not lose consciousness and is usually able to rise immediately. The question as to whether consciousness is lost or not is a difficult one. On the one hand we know that patients can lose consciousness momentarily without being aware of it, as occurs in petit mal. On the other hand, such brief interruptions of consciousness do not cause the patient to fall. Current theory attributes drop attacks to brief ischaemic episodes involving that part of the brain stem reticular formation which projects to the spinal motoneurones. The sudden removal of background tonus results in the antigravity muscles giving way and the patient falling to the ground. Whilst the theory is attractive, proof is lacking and though drop attacks commonly occur in the age groups in which there is a high incidence of cerebrovascular disease and some patients have associated symptoms of vertebrobasilar ischaemia,

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many patients experience these attacks over months or years without ever developing other manifestations of vascular disease. The occurrence of drop attacks alone does not permit a confident diagnosis of vertebrobasilar ischaemia to be made.

Headache

Finally we come to the symptom of headache, the significance of which may readily be overlooked. Focal ischaemia provokes dilatation of collateral vessels with the object of increasing the blood supply to the threatened territory. This dilatation may produce pain which, in the case of collaterals to the vertebrobasilar territory, is commonly referred to the occipital region and back of the neck. When it is the posterior communicating artery that is mainly involved, the pain is situated deep below the region of the temple. The pain is throbbing in character, may be quite severe and may persist for some time after the ischaemic symptoms have subsided. When the symptoms of the transient ischaemic attack are visual, the fact they are followed by headache often leads to the mistaken diagnosis of migraine. It should be remembered however that migraine rarely appears for the first time at the age at which transient ischaemic attacks associated with atherosclerosis are becoming manifest.

Some indication of the frequency with which these various symptoms are encountered is given by Marshall (1964). In a series of 76 cases of transient ischaemic attacks in the vertebrobasilar territory he found that vertigo occurred in 48, visual field disturbance in 22, drop attacks in 16, dysarthria in 11, hemisensory symptoms in 9, hemiparesis in 8, diplopia in 7, monoparesis in 4 and facial paraesthesiae in 2.

The large number of symptoms which have been described as occurring in vertebrobasilar ischaemia may give the impression that this condition does not give rise to a typical clinical picture. This is far from being the case for though many symptoms may be encountered, some occur more frequently than others so that typical patterns can be discerned. As already mentioned, vertigo is the commonest symptom met with and may occur alone, but typically is found in combination with dysarthria and numbness or paraesthesiae in the face. This could be called the lower brain stem syndrome. Another pattern is the association of vertigo with diplopia and disturbance of the visual fields, an upper brain stem syndrome. A third clinical picture is that of one or other brain stem nuclear disturbance occurring in combination with simultaneous or alternating hemiplegias or hemianaesthesia and ataxia.

PROGNOSIS

Transient ischaemic attacks by definition do not cause death or persisting disability; prognosis is, therefore, confined to a consideration of the frequency with which they are followed by a completed stroke. To ascertain this fact is not easy for it is of little help to see how often patients presenting with a completed stroke give a history of preceding transient ischaemic attacks. Nor is it completely satisfactory to follow a series of patients presenting at hospital with a transient ischaemic attack for it may be only the more alarming manifestations which lead to referral to hospital. Acheson and Hutchinson (1964) overcame these difficulties by securing the collaboration of family doctors in a defined geographical area, who notified them of all patients with transient ischaemic attacks who were then followed. Eighteen out of 44 patients with vertebrobasilar transient ischaemic attacks developed a completed stroke, the average interval from the transient ischaemic attack to the stroke being 13 months.

The risks of a completed stroke are therefore considerable and there is some indication as to which patients are most at risk. Thus Marshall (1964) found that patients with hemiplegia, hemianaesthesia and ataxia more frequently developed a brain stem infarction than did those whose transient ischaemic attacks were manifest only by vertigo, dysarthria, diplopia and drop attacks. The appearance of one of the former group of symptoms therefore demands urgent action. In the remaining cases, however, transient ischaemic attacks may continue to occur with varying frequency more or less indefinitely without the patient developing a completed stroke. This type of vertebrobasilar transient ischaemic attack con-

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