Dr Bergmann: What I was saying was that in the early 1950's and up until quite recently people were prepared to include the affective disorders under a unitary concept of psychosis in old age, such as you are suggesting we should keep for the organic disorders. I believe that the unitary concept of psychosis inside both the organic and the functional groups has been harmful to the psychiatric care of the aged.

Dr Dawe (*Caterham*): Would you give us your diagnostic criteria for arteriosclerotic dementia and would you indicate how we should tackle it as a clinical problem? Does the administration of cyclandelate or isoxsuprine, for example, help the condition or not?

Dr Bergmann: Blessed's work, which has perhaps been the most accurate prospective neuropathologically checked study, showed that there was about an 80 per cent chance of being right if you diagnosed arteriosclerotic dementia in those cases with a history of stroke, localized neurological signs plus any two of the following—hypertension, a variability in course (as opposed to senile psychosis which tends to become steadily worse), epileptic fits, the preservation of insight and personality, and the presence of emotional incontinence. These are the criteria for diagnosing it, and I think a reasonable case has been made for recognizing this as a group. As to the management, I agree entirely with Dr Rao who says that the minor and asymptomatic insults to the brain that may come from associated physical disease are of the greatest importance. Conditions that in non-brain-damaged people you might consider insignificant and not worth treating, such as the secondary infection of an upper respiratory tract infection, might well require antibiotics for a person with an early arteriosclerotic psychosis who is still existing at home. I believe that vasodilators are no good at all, I think it is very deceptive to look at a disease with a fluctuant and remittant course, give your favourite pill, and claim to have done a great deal of good in the last 10 or 12 cases. Probably you are also treating a urinary infection, heart failure, or hypertension at the same time. I see no convincing evidence of the value of vasodilators. And as Dr Jacobs at the Whittington Hospital once said, 'ischaemic tissue contains some of the best vasodilators acting at the point where they are needed'.

Big and little strokes

Dr J. C. Brocklehurst, M.D., M.R.C.P. (Ed.), M.R.C.P. (Glasg.) (*consultant physician*)

Since stroke is primarily a disease of blood vessels a moment's thought must be given to the blood supply of the brain, and the effects of ageing upon it. The internal carotid arteries are uncomplicated, dividing within the cranium into anterior and middle cerebral arteries. The other pair of arteries concerned—the vertebrals—are however more complex, and their course tortuous. They arise from the subclavian arteries, pass back at the root of the neck to enter the transverse foramina of the sixth cervical vertebra, and then pass upward through the foramina in the vertebrae above this. They are thus particularly exposed to pathological changes affecting the upper spine. In old age intervertebral discs become dehydrated and squashed—and vertebral bodies porotic and collapsed. The vertebral arteries may thus become tortuous—and liable to kink on flexion or extension of the neck.

The vertebral arteries pass through the foramen magnum and join together to form the basilar artery, overlying the pons. This supplies blood to the hind-brain and cerebellum. The basilar artery finally divides into the two posterior cerebral arteries.

The anterior cerebral arteries are joined by the anterior communicating artery: the middle and posterior cerebral arteries by the posterior communicating artery. Thus is formed the Circle of Willis.

Age changes affecting arteries are loss of muscle fibres, increase in fibrosis, and thinning of the media and adventitia. The most important abnormal feature in age, however, is atheroma—the underlying abnormality in most big and little strokes.

One aspect of ageing only recently described is the formation of cerebral micro-aneurysms, in the presence of age and hypertension. Cole and Yates demonstrated them —also showing the small haemorrhages often associated with them. Here may be the basis of some of our little strokes.

Big strokes

Classically, strokes have been divided into those caused by cerebral thrombosis, cerebral haemorrhage and cerebral embolus, and this still describes accurately the pathological basis. The only addition that may be required is that of cerebral ischaemia occurring as a result of narrowing of blood vessels without actual thrombus formation. However, we do not really know whether or not this is an important cause of cerebral infarction. Nowadays, as the title of this contribution suggests, the emphasis is less on the pathogenesis and more on the variety of clinical presentation of strokes. This approach which has been advocated, particularly by Marshall, was perhaps necessary as a corrective to the previous rather rigid categorization. It does take into account that strokes may present in many different ways even when the underlying pathology is the same.

Marshall divides big strokes into the following two categories:

1. Stroke in evolution.
2. Completed stroke.

The *stroke in evolution* or the developing stroke may come on over a period of one to two days. The clinical presentation is the familiar gradual progression of increasing sensory, motor and perceptive deficit until the full-blown picture of hemiplegia, hemi-anaesthesia, dysphasia, apraxia, or any combination of these has developed.

Stroke in evolution may be a cerebral infarct with underlying thrombosis which is gradually extending, or with developing oedema. It may be due to cerebral haemorrhage in which a slow seepage of blood develops as it were in fissures between the axones, or it may be due to cerebral embolus with retrograde extension of thrombus over a period of a day or two.

The diagnosis of stroke in evolution requires exclusion of a rapidly growing cerebral tumour; subdural haematoma must also be excluded. Treatment must be expectant until the stroke is complete. The only important indication for therapy during a stroke in evolution is malignant hypertension, in which case the blood pressure should be brought under control.

The *completed stroke* is one in which maximal neural damage occurs within minutes, and certainly within an hour of onset. The pathogenesis again may be due to haemorrhage, intracerebral, subarachnoid or subdural; or infarction due to thrombosis or embolism or possibly arterial stenosis.

Intracerebral haemorrhage is generally associated with hypertension. It is commoner in the fifth and sixth decades than in the seventh and eighth. Subarachnoid haemorrhage is uncommon in old age but does occur. There is frequently a third nerve lesion and 20 per cent of patients with subarachnoid haemorrhage have a hemiplegia. A subdural haematoma is generally associated with some impairment of consciousness and often with one dilated pupil. Although atheroma with associated thrombosis generally underlies cerebral infarction, this is not always found. It has been assumed

that stenosis leading to diminution of blood flow and consequent local cerebral ischaemia may result in an infarct.

Investigation of the big stroke

All patients who are admitted to hospital with either an evolving or a completed stroke should have a lumbar puncture. This is necessary, not only to try and obtain an accurate diagnosis, but also for later reference possibly affecting therapy at a future time. It is clearly impracticable however to send patients into hospital particularly for this purpose, and many patients with big strokes can and must be managed at home. It is important that all such patients should have their blood examined within a week or two of the stroke since anaemia is common in elderly people and its presence may affect recovery and polycythaemia occurs occasionally in the elderly and may itself be the cause of a cerebral thrombosis. If polycythaemia is discovered it requires further investigation and treatment.

Lumbar puncture will distinguish the majority of cerebral haemorrhages since about 80 per cent show blood in the cerebrospinal fluid and the pressure may also be raised. The isolated finding of protein must raise suspicion of cerebral tumour. Further investigations which are occasionally needed in hospital are x-ray of the skull, to show shift of a calcified pineal gland with either cerebral tumour or haematoma; cerebral angiography in subarachnoid haemorrhage and sometimes in subdural haematoma; also for the latter, exploratory burr holes by the neurosurgeon.

Little strokes

The little stroke is characterized by transient features such as impairment of consciousness, motor or sensory loss, dysphasia, apraxia or confusion. There may be rapid recovery within a matter of minutes or a few hours. Perhaps the commonest causes of little strokes in old people are transient ischaemic attacks. The clinical presentation of such attacks will depend on whether the aetiological basis lies within the carotid blood supply or the vertebrobasilar blood supply to the brain. If the former, there may be eye symptoms including unilateral blindness, hemiparesis, dysphasia or confusion. If the vertebrobasilar arteries are at fault, the presentation may be with giddiness, nausea or vomiting, drop attacks, hemiparesis, eye symptoms or dysarthria.

The transient ischaemic attack may be triggered off by emboli emanating from a thrombus in the common carotid artery, by sudden drop of blood pressure or by the effects of cervical spondylosis and disc degeneration which by diminishing the height of the cervical spine cause the vertebral arteries to become tortuous and thereby particularly liable to kinking on certain neck movements. Also the rare but frequently mentioned syndrome of subclavian steal in which narrowing of the subclavian artery proximal to the origin of the vertebral artery occurs. This leads to impairment of blood supply to the arm, and when the arm is used a reversed flow down the vertebral artery may occur. Thus the arm receives blood at the expense of the Circle of Willis and particularly of the hindbrain.

In the treatment of transient ischaemic attacks Marshall suggests the use of anticoagulants. In old age however there are added dangers to the use of anticoagulants, particularly in patients who are very liable to fall, and most geriatricians rule them out in the treatment of transient ischaemic attacks in the elderly. Unfortunately the therapeutic possibilities which remain are very limited. A cervical collar will sometimes prevent kinking of the vertebral arteries by preventing the patient from flexing or extending his neck, and may thereby diminish the incidence particularly of drop attacks. If postural hypotension is a factor, it may be useful to bind the legs with crepe bandages before the patient gets up in the morning. If this is successful, elastic stockings can be substituted for the crepe bandages. It is important to warn the patient and his family

about general precautions which they ought to take. No patient known to have had a transient ischaemic attack should go up and down stairs unaccompanied; fires should be guarded and other sensible precautions taken. Stemetil is often helpful in diminishing the nausea or vomiting associated with vertebrobasilar artery insufficiency.

Transient ischaemic attacks which include hemiparesis have a worse prognosis than those in which the symptoms are largely of hind-brain type, and a completed stroke may be the eventual outcome.

The differential diagnosis of little strokes is most important since there are many other causes of transient loss of consciousness in old people. These include epilepsy, Stokes Adam's attacks, hypertensive crisis, carotid sinus sensitivity, myocardial infarction and cerebral tumour.

Post-hemiplegic epilepsy

The question of epilepsy in particular needs further consideration. Fine has demonstrated that post-hemiplegic epilepsy is not uncommon in elderly people. It is generally undiagnosed but if treated with appropriate anticonvulsants it can be entirely controlled. Post-hemiplegic epilepsy occurs in patients suffering from cerebrovascular disease, and the attacks may be motor, in which case they may be followed by further transient paresis (Todd's palsy). Episodic pain or episodic incontinence of urine are two other manifestations of hemiplegic epilepsy, and the presence of either of these symptoms for which other causes cannot be found requires a therapeutic trial with phenytoin sodium.

Barriers to recovery

To conclude this discussion of big and little strokes in old age, something must be said about barriers to recovery. Geriatricians have perforce become particularly interested in the rehabilitation of stroke patients, and this has led to a much greater awareness of the many factors involved in recovery. Adams of Belfast has propounded the concept of barriers to recovery from stroke. His classification should always be borne in mind when clinicians are faced with stroke patients who appear to be making no progress.

Perhaps the most obvious barriers to recovery are impairment of sight and impairment of hearing. Visual defects are not always easily corrected in old people, but it is most important to recognize the presence of homonymous hemianopia. This is a very common accompaniment of hemiparesis and when it is recognized that a patient is completely blind on one side, the practical application of this knowledge as far as therapists are concerned needs no emphasis. Impaired hearing may be a peripheral defect possibly due to no more than wax in the ears. Deafness when present has often been diagnosed previously, and there is every likelihood that the patient has been supplied with a hearing aid which he chooses not to use, or which he has left behind. This always necessitates an enquiry, and many discarded hearing aids are found to be perfectly satisfactory if their use is insisted upon. Auditory agnosia may be the cause of apparent loss of hearing. Words are heard but not understood.

Impaired sensation is common in hemiplegic patients. To test for it, the paralysed arm should be held in the air, and the patient with his eyes closed asked to get hold of his bad thumb with his good hand. More advanced levels of sensory impairment include that involving the body image in which there may be marked neglect of one half of the body, and of one half of objects with which the patient is involved. Such a person may read only half of the headline in the newspaper and take his food from only half of his plate. Anosognosia is present when the patient is completely unaware of disease affecting the limb, or else denies that the paralysed limb belongs to him at all.

Other barriers to recovery are those due to intellectual impairment such as defects

in memory, orientation and concentration. Similarly perseveration often indicates the presence of a severe intellectual barrier to recovery. Apraxia also is a barrier and a bad prognostic sign. Depression is common in all people with stroke and may form a great impediment to co-operation with therapists, and to recovery. It may well be treated by antidepressant drugs. Dysphasia also is a considerable barrier and perhaps the greatest value of speech therapy in stroke is in the speech therapist's ability to overcome the barrier rather than in her ability to add significantly to the vocabulary of the dysphasic patient.

The existence and recognition of any of these barriers to recovery is very important since some may be removed and others give prognostic signs of great value.

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Discussion

Dr Gancz (*Bexley*): The term 'little stroke' was invented by Professor Alvarez and from his description in his book I could not quite make up my mind whether he was telling me to treat a person with little strokes or big strokes at home or in hospital. I would like to hear from Dr Brocklehurst what he actually thinks: Where should a patient with a stroke be treated and how should treatment be phased?

Dr Brocklehurst: This depends on many different factors, but mainly on the home and the availability of hospital beds. No patient, particularly an old person, should be admitted to hospital if it can be avoided. Clearly if there is some doubt as to the diagnosis, the patient should be admitted for investigation. If nursing is impracticable at home or if the patient has malignant hypertension, then again he should be admitted to hospital. In the absence of these things, by and large it is probably impracticable to admit all patients to hospital. Many people can be adequately dealt with at home, perhaps attending at the day hospital if they require physical rehabilitation, as many of them do. Apart from the diagnostic procedures, the possibilities of therapy are so limited that there is no great advantage in having the patient in hospital. If adequate nursing is available, if district nurses can have more instruction in the early treatment of stroke, if physiotherapists can start the early treatment of stroke at home and teach the relatives what to do, many patients can be managed at home.

Dr Wollner: When, if at all, is there an indication for carotid endarterectomy after transient strokes due to carotid stenosis?

Dr Brocklehurst: I am no expert on this, I am afraid, but a few years ago I heard a lecture at the British Geriatrics Society by Professor Irvine who was a great advocate of vascular surgery for all the things I have been describing. Ever since I have been looking hard for patients to send to him and have only found two, neither of whom was operated on. I think vascular surgery is rather going out of fashion now, but no doubt there is a scientific basis for surgery in some of these cases in old age.

***Question*:** Could you say something about hypertensive encephalopathy or hypertensive cerebral attacks? Where does it fit in, what is the pathology, should there be any immediate first-aid treatment and if so, what?

Dr Brocklehurst: Middle-aged hypertensives are particularly prone to hypertensive encephalopathy with transient loss of consciousness; older people may have confusional states associated with it and may develop a variety of symptoms, associated with a transient rise in