CEREBROVASCULAR DISORDERS:
A CLINICAL AND RESEARCH CLASSIFICATION

WORLD HEALTH ORGANIZATION
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The text presented in this publication has been adapted by the World Health Organization Collaborating Centres for Research and Training in the Neurosciences (see Appendix III) from the report of an ad hoc Committee on Cerebrovascular Diseases of the National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health, Bethesda, MD, USA.

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Coordinators of the present adaptation: Dr Murray Goldstein and Dr C. L. Bolis.
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CLASSIFICATION OF CEREBROVASCULAR DISORDERS

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INTRODUCTION AND OUTLINE

INTRODUCTION

The purpose of this classification is (1) to set out types of cerebrovascular disease in such a way that the resulting classification will be of value to clinicians, neurologists, epidemiologists, pathologists, specialists in physical medicine, and workers in other clinical and research disciplines; (2) to be of practical value by defining the terms so they can be employed interchangeably by clinicians and investigators in all parts of the world; and (3) to set down the salient diagnostic criteria for the different disorders in an organized way.

This classification of cerebrovascular disorders should not be confused with that appearing in the International Classification of Diseases (ICD). Rubrics 430-438 of the latter classification are intended for quite different purposes; namely, the statistical presentation of frequencies of cause of death, admission to hospital, etc., and the diagnostic indexing of hospital records.

Knowledge of the cerebrovascular disorders has been considerably expanded. It continues to be evident that, for such a complex set of clinical-pathophysiological phenomena, a standard reference language and set of definitions should be used or the literature of investigation will be impossible to interpret. For example: Does the term "transient ischaemic attack" include diffuse ischaemia (syncope) or should it be specific for episodes of focal ischaemia? Follow-up data about the frequency of stroke in patients with the former are entirely different from the corresponding data on patients with the latter. It is recognized that some of the statements on the following pages are tentative and, as understanding of mechanisms and causes increases, may need to be changed.

The term "cerebrovascular" denotes those disorders (1) in which an area of brain is transiently or permanently affected by ischaemia or bleeding, or (2) in which one or more brain blood vessels are primarily involved in a pathological process, or (3) a combination of (1) and (2). The first includes such phenomena as decreased arterial perfusion pressure in the brain, the temporary redistribution of blood in "hemiplegic migraine", etc.; the pathological process includes abnormalities of the blood vessel wall, vessel stenosis or occlusion by thrombus or embolus, and altered permeability to plasma and blood cells.

The term "cerebrovascular" is now well established as covering the range of disorders just mentioned. "Cerebrovascular" and "cerebral" are used in the original Latin sense, referring to the whole brain and not merely to the hemispheres of the forebrain.

This volume is the product of an international collaborative effort. It is hoped that its users will find it of service and that they will help WHO to improve it by offering their comments and suggestions.

OUTLINE OF THE CLASSIFICATION

Part I.  Clinical Stage

A.  Asymptomatic
B.  Focal cerebral dysfunction
   1.  Transient ischaemic and vascular attacks
       (TIA and TVA)
   2.  Actively changing neurological deficit
       (progressing stroke)
   3.  Prolonged neurological deficit
       (completed stroke)
C.  General cerebral dysfunction
   1.  Transient
   2.  Prolonged
      a.  Acute onset
      b.  Gradual progression

Part II.  Clinical Phenomena (history, physical examination, laboratory examination, roentgen examination, other)

A.  History
   1.  Demographic
   2.  Family history
   3.  Past history
   4.  Present illness
      a.  Transient attacks
      b.  Actively changing neurological deficit
      c.  Prolonged neurological deficit
B.  Physical examination
   1.  General
   2.  Neurological
   3.  Vascular (largely neurovascular)
C.  Laboratory examination
   1.  Urinalysis (specify abnormality)
   2.  Blood (specify abnormality)
   3.  Cerebrospinal fluid (specify abnormality)
   4.  Electrocardiography (specify abnormality)
   5.  Electroencephalography (specify abnormality)
   6.  Echoencephalography (specify abnormality)
   7.  Brain scan (specify abnormality)
D.  Roentgen examination
   1.  Chest
   2.  Head
   3.  Angiography (cranial)
E. Special procedures
   1. Computerized axial tomography
   2. Cerebral blood flow
   3. Retinal circulation time
   4. Thermography and thermometry
   5. Retinal photography
   6. Tilt-table study
   7. Phonocardiography
   8. Electronystagmography
   9. Doppler blood flow estimation
  10. Ocular plethysmography
  11. Cranial impedance plethysmography
F. Explanation of clinical phenomena
   A. History
   B. Physical examination
   C. Laboratory examination
   D. Roentgen examination
   E. Special procedures

Part III. Status of patient (performance and placement)

Performance
   Activities of daily living
   Avocational activities
   Occupation

Placement
   Patient performance and placement
   A. Performance
      Class I. No significant impairment
      Class II. Mildly impaired
      Class III. Moderately impaired
      Class IV. Severely impaired
   B. Placement
      Class A. No limitation
      Class B. Mild limitation
      Class C. Moderate limitation
      Class D. Severe limitation

Part IV. Anatomy

A. Blood vessels
   1. Arteries
   2. Arterial collateral circulation
   3. Arterial anomalies
   4. Veins
B. Brain and spinal cord
   1. Meninges
   2. Brain
   3. Spinal cord
Part V. Pathophysiological mechanisms

A. Primary abnormalities of cerebral circulation (specify whether transient or persistent)
   1. Thrombosis
   2. Embolism
   3. Haemorrhage (specify – See Part IV. Anatomy and Part VI. Pathology)
   4. Compression
   5. Vasospasm
   6. Direction of flow
   7. Alteration in rate and/or volume
   8. Dissection of arterial wall
   9. Associated with arteriography

B. Abnormalities of general circulation (specify whether transient or persistent)
   1. Hypotension (specify cause)
   2. Hypertension

C. Alterations in blood (specify type)

D. Alterations of metabolic demand (specify type)

E. Possible predisposing factors
   1. Hypertensive disease
   2. Diabetes mellitus
   3. Cardiac disease
   4. Hyperlipidaemia
   5-11. Others

F. Unknown

Part VI. Pathology

A. Pathological alterations in vessels
   1. Arteries
      a. Congenital, developmental, and inherited lesions
      b. Inflammatory lesions (arteritides)
      c. Trauma and physical agents
      d. Arterial lesions due to blood dyscrasias
      e. Arterial lesions associated with metabolic abnormalities
         (including familial hypercholesterolaemia, diabetes mellitus, etc.)
      f. Arterial lesions associated with drug toxicity, drug
         idiosyncrasy, and unknown drug effects
      g. Arterial embolism due to cardiac disease and diseases
         of extracerebral vessels
      h. Arterial lesions associated with neoplastic disease
      i. Arterial lesions due to unknown causes (including
         atherosclerosis)
      j. Arterial lesions associated with hypertension
2. Veins (specify)
3. Capillaries (specify)
4. Combined arterial, venous, and capillary abnormalities
   a. Congenital, developmental, and inherited lesions
B. Pathological alterations in brain
   1. Infarction (pale, haemorrhagic, and mixed)
      a. Without vessel stenosis or occlusion
      b. With arterial stenosis or occlusion associated
         with pathological alterations in arteries (VI. A.1.)
   2. Haemorrhage
      a. Without identification of vessel type
      b. Of arterial origin (see pathological alterations in
         arteries (VI. A.1.))
      c. Of venous origin (specify)
      d. With capillary lesions
      e. With combined arterial, venous, and capillary lesions
CLASSIFICATION OF CEREBROVASCULAR DISORDERS*

BASIS OF CLASSIFICATION

It is no longer possible to cover the whole range of clinical and research needs with a classification limited to pathology. From a review of previous classifications, it was apparent that a different approach would be required in order to classify: (a) the temporal profile of development of a cerebrovascular abnormality; (b) such transient pathophysiological mechanisms as vasospasm, changes in cardiac rhythm, systemic hypotension, hypercapnia, etc.; (c) anatomical parameters of the blood vessel system or of the brain and spinal cord; (d) neurological clinical phenomena (history, physical examination, laboratory results, etc.); and (e) the performance capabilities in relation to the placement requirements of the patient when changing care status, from hospital to home, nursing home, etc. The classification has been developed to promote communication among clinicians and scientists and as a basis for national and international collaborative efforts. The introduction of new methods of differential diagnosis suggests the need for revision at a later date.

* This classification has been prepared by the WHO Collaborating Centres for Research and Training in Neurosciences (see Appendix III) on the basis of a report by Clark H. Millikan, M.D., Professor of Neurology, Mayo Medical School, Rochester, MN., and Chairman, U.S. National Institute of Neurological and Communicative Disorders and Stroke, ad hoc Committee on Cerebrovascular Diseases, and the following members of the Committee: Raymond B. Bauer, M.D., Professor of Neurology, Wayne State University School of Medicine, Detroit, MI.; John Goldschmidt, M.D., Professor of Physical Medicine and Rehabilitation, Jefferson Medical College, Philadelphia, PA.; Murray Goldstein, D.O., Associate Director, National Institute of Neurological and Communicative Disorders and Stroke, Bethesda, MD.; Albert Heyman, M.D., Professor of Neurology, Duke University School of Medicine, Durham, NC.; John Stirling Meyer, M.D., Professor of Neurology, Baylor College of Medicine, Houston, TX.; John Moossy, M.D., Professor of Neuropathology, University of Pittsburgh School of Medicine, Pittsburgh, PA.; Lester Mount, M.D., Professor of Neurosurgery, College of Physicians and Surgeons, Columbia University, New York, NY.; Robert C. Siekert, M.D., Professor of Neurology, Mayo Medical School, Rochester, MN.; Reuel Stallones, M.D., Professor of Public Health, University of Texas School of Public Health, Houston, TX.; James F. Toole, M.D., Professor of Neurology, Bowman Gray School of Medicine, Winston-Salem, NC.

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The classification is divided into six parts: Part I. Clinical stage; Part II. Clinical phenomena; Part III. Status of patient (performance and placement); Part IV. Anatomy (blood vessels, brain); Part V. Pathophysiological mechanisms; and Part VI. Pathology (blood vessels, brain). This division makes it possible to classify any cerebrovascular problem at any point in time and also to fulfill the requirements of individuals with special interests or needs.

As in clinical medicine, sequential reclassification in Part I (Clinical Stage) will often be necessary as a patient's status proceeds through progressing cerebral ischaemia to completed cerebral infarction. Part II (Clinical phenomena) may be only partially completed in many instances, and Part III (Status of patient) may not be useful until well into the clinical course of the disorder. The idea of thinking through the entire classification is paramount, since it provides an orderly framework permitting the busy physician to proceed from the clinical stage to pathophysiological mechanisms, anatomical locus of the process, pathology, clinical phenomena (including neurovascular examination and laboratory evaluation), and the patient's capability status with or without need for special placement. In certain instances, a process or disease has had to be listed under more than one major category, i.e. thrombosis and embolism under "Pathophysiological mechanisms" as well as "Pathology".

Vascular disorders affecting the brain have a variety of components:

1. The basic pathophysiological process, which consists of decreased perfusion pressure because of a cardiac or systemic circulatory problem, abnormalities of the blood (polycythaeemia, etc.) or abnormalities directly impairing the vessel's transport of blood (atherosclerosis, embolism, thrombosis, arteritis, etc.), singly or in a variety of combinations. (The metabolic substrates for normal brain function are not included.)

2. Pathophysiological changes in brain parenchymal metabolism (commonly focal), which may be of short duration and reversible as in a transient focal cerebral ischaemic attack, may be infarction if impaired circulation persists, or may be haemorrhage if a vessel ruptures.

3. The neurological abnormality that results from the focal deficit in brain metabolism. The temporal profile of the neurological abnormality varies greatly—from a brief event (transient ischaemic attack or TIA) to permanent severe brain damage (completed stroke) producing hemiplegia, etc., or death.

An attempt has been made to make the Classification, in whole or in part, of service to the various disciplines dealing with clinical care, training, or research. To assist the user, the more common disorders are underlined.
PART I. CLINICAL STAGE

The primary purpose of this section is to provide a framework for the description of a patient's current status with reference to the temporal profile of the disease, without regard to other factors such as etiology, pathology, neural deficit, and the like, all of these being described in separate sections.

As the condition of the patient may be changing, sometimes rapidly, a static description presents certain problems particularly as regards the exact time of categorization. In general, a patient is placed in one of the categories at the point when he is studied in sufficient detail to permit a reasonably certain clinical diagnosis. Because of the continuing evolution of the deficit, it is likely that a patient will fall into different categories at different times; in such instances the time of categorization must be stated. It is possible, also, that some deficits will remain stable while other deficits evolve; in such instances, appropriate comments are necessary. By using more than one category, an accurate description of a progressing temporal profile can be built (e.g. a patient who has TIAs followed by a completed stroke with a permanent residual neurological deficit, and subsequently has TIAs again, would be classified as I.B.1., I.B.3., and later I.B.1.).

A. Asymptomatic

This category covers individuals without cerebral symptoms for whom there is potentially important evidence of a predisposition to future cerebrovascular disease. Such evidence is listed in detail in Part V. Some of the factors in this evidence have been termed the "stroke-prone profile".

B. Focal cerebral dysfunction

This category covers focal brain dysfunction regardless of the nature of the vascular pathology (e.g. ischaemic disease, intracranial haemorrhage, arteritis).

1. Transient ischaemic and vascular attacks (TIA and TVA)

These are episodes of temporary and focal cerebral dysfunction of vascular origin; rapid in onset (no symptoms to maximum symptoms in less than 5 minutes and usually in less than a minute); and variable in duration, commonly lasting from 2 to 15 minutes but occasionally lasting as long as a day (24 hours). The resolution or disappearance of each episode is swift (ordinarily a few minutes at most). A prolonged attack may take longer to clear up. An attack leaves no persistent neurological deficit. It is common practice to define these events

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1 Migraine syndrome. The aura of migraine (e.g. scintillating scotoma) is commonly attributed to focal cerebral ischaemia owing to vasoconstriction. In some instances more severe neurological deficits precede the headache; these may include hemiparesis, dysphasia, and homonymous hemianopia. In the great majority of instances these deficits are transitory, rarely persisting long enough to produce a cerebral infarct. The temporal profile may be classified under "Clinical stage", while the word "migraine" is found under V.A.5.b.
as being related either to the carotid arterial system or to the vertebrobasilar arterial system, depending upon the clinical locus in the customary distribution of one or the other of these arterial systems. There may be only one attack or there may be multiple attacks at varying intervals. There are unusual instances that fall outside this standard definition. This definition has been formulated in an arbitrary way in an attempt to provide a common basis for grouping TIA and TVA patients. The components of TIA and TVA are coded under II. Clinical phenomena, A. History, 4. Present illness, a. Transient attacks.

The typical history for a transient attack in the carotid arterial system is one of swift onset (no symptoms to maximum symptoms in less than 5 minutes, usually less than 2 minutes) and consists of:

(1) motor defect (weakness, paralysis, poor use, or clumsiness of one extremity or of both extremities on the same side); and/or

(2) sensory defect (numbness, including loss of sensation or paraesthesias involving one or both extremities on the same side); and/or

(3) aphasia (speech and/or language disturbance which may be global or only a minor defect and may or may not include difficulty in reading, writing, or performing calculations); and/or

(4) loss of vision in one eye or in part of one eye when vision in both eyes was intact (amaurosis fugax); and/or

(5) homonymous hemianopia.

These clinical phenomena generally represent a decrease or absence of function. When there is a sensory event it is commonly described as coming on all at once, i.e. without a buildup.

The typical history of a transient attack in the vertebrobasilar arterial system is one of swift onset (no symptoms to maximum symptoms in less than 5 minutes, usually less than 2 minutes) and consists of:

(1) motor defect (weakness, clumsiness, or paralysis of any combination of extremities up to quadriplegia, sometimes changing from one side to another in different attacks); and/or

(2) sensory defect (numbness, including loss of sensation or paraesthesias in any combination of extremities, including all four or involving both sides of the face or mouth. Frequently bilateral, the distribution may change from side to side in different attacks); and/or

(3) loss of vision, complete or partial in both homonymous fields (bilateral homonymous hemianopia); and/or

(4) homonymous hemianopia; and/or
(5) ataxia, imbalance, unsteadiness, or disequilibrium not associated with vertigo; and/or

(6) an episode of vertigo (with or without nausea and vomiting), diplopia, dysphagia, or dysarthria is not to be considered as a transient attack when any of these symptoms occurs alone. Such episodes should be considered as TIs or TVAs only when occurring in combination with (1), (2), (3), or (4) above.

These clinical phenomena generally represent a decrease or absence of function. At times, the motor, sensory, or visual defect constituting the content of a vertebrobasilar attack will be unilateral. It becomes difficult in such instances to distinguish whether the locus of ischaemia is in the carotid arterial system or in the vertebrobasilar arterial system. The above list does not include "drop attacks". Fainting (syncope) is frequently confused with a "drop attack", so the latter should be included in the vertebrobasilar profile only when the patient's description of the "drop attack" is absolutely clear. The variety of manifestations included in the vertebrobasilar profile makes the potential pattern of symptoms considerably more variable and complex than it is for the carotid system.

Except in those relatively few instances in which the physician is with the patient at the time of the attack, the diagnosis of transient attacks (TIA or TVA) rests on the history of the attacks, the skill with which the history is taken, and the interpretation of the history. The criteria for making the diagnosis will vary depending on whether a physician is working with an individual patient or whether the purpose is the screening of a population for transient attacks. A problem is created, as in much medical diagnosis, because of the relative weight or significance of some historical phenomena compared to other phenomena. The symptom "numbness" (mentioned above) is an example. If the question is "Have you ever had a numb hand?", the answer from most adults will be "Yes". This question is almost completely non-selective (non-diagnostic) and must be followed by a series of questions to establish the meaning and significance of the "numbness". In contrast, another phenomenon, when present, is simple and relatively much more significant than "numbness"; i.e. "Have you ever had painless blindness in one eye which came on very quickly (seconds) and lasted only a few minutes (5 to 20)?" is a question which, if answered by "Yes", is reasonably specific. Another similar but less specific question is "Have you ever had an attack of severe weakness of one side of the body (arm and leg) of sudden onset and lasting from 5 to 20 minutes?". Another is "Have you ever suddenly lost the ability to speak (for 5 to 20 minutes) or to understand the speech of others?". These examples illustrate the importance of understanding that different questions may be of relatively different complexity and importance; this is particularly true when different geographical areas are involved.

The matter of the relative significance of different symptoms is important in the vertebrobasilar system, as in the carotid system. For instance, if asked the question, "Have you ever had any dizziness?", almost all adults will answer "Yes". This question is almost completely non-selective (non-diagnostic) and if answered affirmatively must be followed by a series of direct and accessory questions to establish the meaning and significance of the original phenomenon of
"dizziness". A diagnosis of a TIA or TVA in the vertebrobasilar system should not be made on the basis of a history of a few minutes of vertigo, if this is the only symptom. This is emphasized since vertigo is the most common symptom in the vertebrobasilar system; however, a diagnosis of vertebrobasilar TIA or TVA is made only when there is an additional and concurrent symptom or symptoms.

In some instances, patients with transient carotid system attacks may have physical signs of appropriate arterial disease. These include diminished pulsation in the carotid artery, a bruit over the carotid artery or eye, emboli in the retinal vessels, or other signs of ischaemic retinopathy and relative hypotension in the retinal artery as measured with the ophthalmodynamometer; however, these are only signs of arterial disease and may be present in the absence of a history of transient attacks. In certain instances, bruits signifying compromise of flow in the innominate artery, either subclavian artery, or at the origin of either vertebral artery may be present; however, the absence or presence of such sounds does not weigh heavily in the diagnosis of vertebrobasilar transient attacks since the diagnosis is dependent upon the history of the attack, not upon morphological evidence of change in patterns of blood flow.

Certain symptoms may appear in transient attacks in either arterial system. The most important of these are:

1. Dysarthria, if it occurs alone; and
2. Homonymous hemianopia, if it occurs alone.

The occurrence of certain symptoms in solitary fashion constitutes an "uncertain TIA". An attack that consists solely of each of the following symptoms should be categorized as an uncertain TIA:

1. Vertigo alone
2. Dysarthria alone
3. Dysphagia alone
4. Diplopia alone

For the sake of clarity, the following symptoms, transient or prolonged, are not to be considered as primary for TIA or TVA:

1. Unconsciousness, including syncope.
2. Tonic and/or clonic activity.
3. Onset of a sensory defect.
4. Vertigo alone.
5. Dysphagia alone.
(6) Dysarthria alone.

(7) Incontinence of bowel or bladder.

(8) Dizziness or "wooziness" alone.

(9) Loss of vision associated with alteration of consciousness.

(10) Focal symptoms associated with migraine.

(11) Scintillating scotomata.

(12) Confusion alone.

(13) Amnesia alone.

The differential diagnosis of TIAs and TVAs must exclude "hemiplegic" migraine, focal convulsive events (often due to neoplasm and producing either sensory or motor phenomena), Menière's disorder, sensory phenomena associated with hyperventilation, and finally any unknown mechanism. The differentiation of "hemiplegic" migraine is a semantic and practical problem. In instances where the aura of migraine is associated with a definitely focal neurological event, the latter may well be the result of transient focal cerebral ischaemia but the implications are not the same as in the case of the usual TIA. To establish a diagnosis of migraine, there should ordinarily be a positive family history, characteristic unilateral headache with nausea and sometimes vomiting, and onset of the attacks several decades ahead of the age at which TIAs commonly begin. Very careful history-taking usually differentiates the transient focal events associated with brain neoplasm from TIAs, and this is also true of the other items in the differential diagnosis.

Many factors might be included to describe the characteristics of the change or rate of change in each individual attack or the pattern in a number of attacks. These factors are dealt with in I.B.2.

2. Actively changing neurological deficit (progressing stroke)

This category comprises patients in whom the amount of neurological deficit is actively changing during the period of observation (specify duration from time of onset). The deficit may be getting more severe or less severe.

a. Improving.

b. Worsening (also known as "progressing stroke" or "stroke-in-evolution").

The latter (b) represents the common situation in which focal ischaemia is worsening and the process of infarction beginning or expanding. (In exceptional instances, bleeding of slow onset may produce a similar temporal profile.) In some cases the focal pathology is in the characteristic territory supplied by the carotid arterial system; in others, the supply is through the vertebrobasilar arterial system.
Because of the evolution of the deficit, it is likely that patients will fall into different categories at different times. For instance, a patient seen at 10 hrs with mild weakness of the left upper extremity (history of onset at 9 hrs the same day), re-examined at 11 hrs and found to have paralysis of that extremity, would be classified under "progressing stroke"; however, if the deficit had disappeared at 11 hrs, the category would be "transient ischaemic attack".

A description of the neurological findings most often characteristic of a focal deficit in specific arterial territories will be found under II.B.2.

3. **Prolonged neurological deficit (completed stroke)**

This category comprises patients with a relatively stable neurological deficit. During the period of observation for categorization (specify duration from time of onset), little or no change occurs in the deficit.

a. Duration more than 24 hours but less than 3 weeks (sometimes referred to as a **reversible neurological deficit** or RND).

b. Duration more than 3 weeks - often permanent (commonly known as "completed stroke").

**Thrombotic infarction**

Thrombotic infarction is divided into those instances in which there is infarction in the characteristic territory supplied by the carotid arterial system and those in which the supply is through the vertebrobasilar arterial system. The infarctions occurring in the carotid distribution often come on rather abruptly (in a matter of some minutes up to a very few hours). The neurological deficit may become maximum during sleep and may, therefore, be maximum when first discovered or may require several hours to a day to evolve. In a significant percentage of cases, there will have been warning TIA's. While there may be some head discomfort, violent headache is rare. There is relative preservation of consciousness, and at times there may be rapid improvement. The cerebrospinal fluid (CSF) commonly remains clear and there is often associated evidence of atherosclerosis elsewhere in the coronary and peripheral vessels. The clinician searches for the presence of disorders commonly associated with atherosclerosis (hypertension, diabetes mellitus, gout, various forms of heart disease, and xanthomatosis). The neurological examination reveals weakness or numbness limited to one side of the body. If the dominant hemisphere is involved, there may be aphasia or, more commonly, a dysphasia that varies as far as its precise content is concerned. It is interesting to note that in this type of "stroke" the visual field defects will generally be homonymous, and it is rare for there to be simultaneous evidence of complete unilateral retinal ischaemia and focal brain ischaemia on the same side. Looking at the temporal profile of the disorder, the clinician must attempt to decide whether the cerebral infarction is still progressing, as evidenced by an increasing neurological deficit, or whether the "stroke" is essentially completed, meaning that
there is no further progression of the neurological deficit. This decision can be made only be relating the history of the preceding few minutes or hours to the existing findings or by picking up the temporal profile at the point where the patient is first seen and carefully re-examining the situation every few minutes or hours.

As in TIAs there may be, with the overt evidence of a focal neurological defect, signs that suggest general or local vascular disease. Hypertension and various forms of cardiac abnormality are related to the former, while bruits over the carotid bifurcation, retinal emboli, other evidence of ischaemic retinopathy, and a unilateral decrease of retinal artery pressure, demonstrated by the use of the opthalmodynamometer, exemplify the latter. The differentiation of thrombosis in the vertebrobasilar system from the intracerebral haemorrhage is not difficult, but the differential diagnosis of thrombosis in the carotid system and intracerebral haemorrhage presents a significant problem. Table I lists the principal differences.

Table I. - Differences between clinical picture of cerebral infarction and that of intracerebral haemorrhage.

<table>
<thead>
<tr>
<th>Infarct</th>
<th>Intracerebral haemorrhage</th>
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<tr>
<td>Often TIA ¹</td>
<td>No TIA ¹</td>
</tr>
<tr>
<td>Often onset at rest</td>
<td>Onset during activity</td>
</tr>
<tr>
<td>Minimal cranial discomfort (often none)</td>
<td>Headache (sometimes severe)</td>
</tr>
<tr>
<td>Focal neurological deficit without change in consciousness. Often paralysis of a function with normal mentation</td>
<td>Rapidly changing neurological deficit including state of consciousness alternating to coma</td>
</tr>
<tr>
<td>Moderate HBP (occasionally normotensive)</td>
<td>Severe HBP ² (occasionally moderate hypertension)</td>
</tr>
<tr>
<td>Clear CSF</td>
<td>Bloody CSF</td>
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¹ TIA = transient ischaemic attack.
² HBP = high blood pressure
³ CSF = cerebrospinal fluid


**Intracerebral haemorrhage**

The absence of a history of TIAs is mentioned first since, if a history of such episodes is obtained, the diagnosis of intracerebral haemorrhage is almost eliminated from differential consideration. Symptoms associated with intracerebral haemorrhage generally come on during activity and, if the patient is sufficiently conscious to report his symptoms, the existence of head discomfort (sometimes described as very severe headache) is commonly mentioned. There is a rapid evolution of focal neurological phenomena over many minutes or a few hours, hemiplegia being the most frequently observed neurological deficit. This rapid evolution, unfortunately, progresses to involvement of consciousness going on quickly to coma. Discrete paralysis of a function with normal mentation is rare. Usually the blood pressure is markedly elevated; in infrequent instances there may be only moderate hypertension. In 75% to 85% of patients with intracerebral haemorrhage, there will be grossly bloody CSF.

**Cerebral infarction secondary to embolic arterial occlusion (cardiac source)**

It is now important to make the distinction between an embolus that comes from a relatively distant source, such as the heart or lungs, and the embolus or emboli that originate in an artery to the brain such as the carotid, vertebral, or basilar arteries.

The most characteristic component of the clinical picture of cerebral infarction secondary to embolic arterial occlusion (cardiac source) is the extraordinarily sudden development of focal neurological symptoms and signs. The patient is often struck down within seconds or at most a few minutes, i.e. there is a very swift progression to maximum neurological deficit. Frequently there is no complaint of pain and a relative preservation of normal consciousness. There is an absence of history of antecedent TIAs, ordinarily a clear CSF, and, generally, a discrete and clearly focal neurological constellation of physical abnormalities. Most important to the diagnosis is the demonstration of a source of emboli, usually in the heart. This "source" commonly consists of a cardiac arrhythmia, valvular heart disease, or myocardial infarction. In a minority of instances, the basic pathological condition may be subacute bacterial endocarditis. The search for a source of emboli must be accompanied by a search for evidence of recent embolism in other organs: spleen, kidney, lungs or extremities. The CSF is usually clear. Rapid improvement may take place.

**Cerebral infarction secondary to embolic arterial occlusion (emboli from an intra-arterial source)**

When emboli consist of thrombus that has formed on an atherosclerotic plaque or of material from an ulcerated atherosclerotic plaque, the source is called "intra-arterial". The temporal profile of the clinical events range from quite swift (many seconds to a few minutes) to several hours. Particularly in the carotid system, a history of previous attacks of amaurosis fugax, with or without transient focal cerebral ischaemic attacks, favours an embolic cause, while a loud, long systolic or systolic-diastolic carotid bifurcation bruit, cholesterol retinal emboli, or ipsilateral decrease in retinal arterial pressure is important evidence of emboli of carotid origin.
"Primary" Subarachnoid Haemorrhage

This term refers to a situation in which initially the bleeding occurs directly into the subarachnoid space, and a distinction is therefore made between this disorder and the condition occurring when an intracerebral haemorrhage breaks either into the subarachnoid space or into a ventricle.

The characteristic clinical picture of "primary" subarachnoid haemorrhage begins with the extraordinarily sudden onset of severe headache. The suddenness of onset and the severity of the pain are usually dramatic. The degree of suddenness ranges from a second or two of time to a minute, and the headache is immediately so intense that it alters the pattern of the patient's activity. In some instances, there is an immediate or almost immediate disturbance of consciousness (including unconsciousness with recovery of consciousness in a few minutes). Often there is an absence or poverty of definite focal neurological signs. The somewhat frequent exception to this is the appearance of a partial oculomotor nerve palsy. In a few minutes there is commonly nausea and maybe vomiting. The patient may complain of a stiff neck or an extension of the cranial discomfort into the posterior cervical region. On physical examination, particularly if the patient is not in extremis, there is a stiff neck on forward bending and there may be Kernig's or Brudzinski's signs indicating meningeal irritation. Subhyaloid (pre-retinal) haemorrhages may be detected within a few minutes of the onset of the symptoms. Mandatory to the diagnosis is the presence of gross bleeding into the CSF. The clinical diagnosis cannot be substantiated without this finding. The precise source of the bleeding and the genesis of the defect in the arterial wall are often not predicted accurately on the basis of clinical examination. Subsequent arteriography has demonstrated that the most common defect is the rupture of a saccular aneurysm, but in some situations there is no arteriographic evidence of any abnormality. Rarely, a neoplasm may be the source of bleeding, but with the whole clinical picture the differential diagnosis can be made with a high degree of accuracy. A history of previous convulsive phenomena, a patient in the second, third, or fourth decades of life, a cranial bruit, and particularly a characteristic calcification which appears in X-rays of the head are indicative of the probable existence of an arteriovenous malformation.

Intracranial haemorrhage from a vascular malformation

While subarachnoid haemorrhage resulting from a defect in the wall of an intracranial vascular malformation and the onset of symptoms may be sudden, there is seldom the dramatic swiftness and severity commonly associated with subarachnoid haemorrhage from other sources.

As mentioned above, there is generally a history of preceding convulsive phenomena and in some instances focal cerebral symptoms. When there is subarachnoid bleeding with a mild focal neurological deficit and gross blood in the CSF in an individual in the second, third, or fourth decades, the rupture of an intracranial arteriovenous malformation should be suspected. In 20–25% of instances, a cranial bruit is present. Some subhyaloid (pre-retinal) haemorrhages may develop, and the detection of retinal angiomas is particularly important since vascular malformations may occur in multiple sites (the retina as well as the brain). X-rays of the head may show calcification, which sometimes is characteristic evidence for the presence of an arteriovenous abnormality.
As implied above, the CSF contains gross blood which is not due to a "mechanically bloody cerebrospinal fluid tap" and blood may be observed in the spinal fluid secondary to an intracranial or intraspinal pathological focus.

Extradural and Subdural Haemorrhage

Intracranial bleeding due to head trauma is not ordinarily thought of as a "stroke". However, it is included here because of the frequency with which the question of diagnosis of traumatic intracerebral haemorrhage arises in instances where the history is inadequate or where the patient has fallen and injured himself at the time of onset of the "stroke". Acute extradural and subdural haemorrhage must always be considered in the patient who has had any kind of head injury or an abrupt ictus under known circumstances, whether the spinal fluid is bloody or clear.

It is particularly important for acute intracerebellar haemorrhage to be considered in the differential diagnosis when there is sudden onset of a severe focal cerebellar deficit with progression, which often includes nausea and vomiting, cranial nerve palsies, drowsiness, and subsequent further changes in consciousness. Appropriate neurosurgical treatment may save the patient’s life.

Chronic subdural haemorrhage may occur without a history of trauma, and the clinical picture of headache with some drowsiness, mental confusion, and occasionally mild focal neurological deficit, especially in the elderly, may be incorrectly diagnosed as cerebral infarction. Acute extradural haemorrhage, acute subdural haemorrhage, and chronic subdural haematoma must be constantly considered; there should be no hesitation about placing diagnostic burr holes in instances where subdural bleeding cannot be appropriately excluded.

Other causes of intracranial haemorrhage

Haematological disorders that may give rise to intracranial bleeding include leukaemia, aplastic anaemia, and thrombocytopenic purpura. Any portion of the brain may be involved and the lesions are frequently multiple. When cerebral haemorrhage occurs, there is often evidence of previous abnormal bleeding elsewhere in the body (skin, mucous membrane, kidney, or bowel). Rarely, an intracerebral haemorrhage may be associated with chronic liver disease, and intracranial bleeding is a rare complication of anticoagulant therapy.

Brain stem haemorrhage secondary to herniation of a portion of the temporal lobe through the tentorial notch is an all too frequent complication of an expanding supratentorial lesion such as a neoplasm, an abscess, primary intracerebral haemorrhage, or massive cerebral infarction. This type of haemorrhage is in the midbrain and pons and constitutes and serious complication of temporal lobe herniation, almost always resulting in irreversible coma and death. These haemorrhages are named after Duret, who was one of the first workers to observe and describe them.
Haemorrhage into primary and secondary brain tumours is an uncommon complication of these lesions. In rare instances, the first clinical phenomenon produced by the basic pathological lesion will be the sudden occurrence of a focal neurological deficit. In these unusual instances, the clinical picture is that of a stroke. In some situations, the clue that the bleeding is secondary and not primary is provided either by the known presence of neoplastic lesions or by a history of the gradual onset of a focal neurological lesion preceding the sudden change occasioned by the haemorrhage.

Septic embolism from the lesions of acute or subacute bacterial endocarditis may produce cerebral infarction with a considerable amount of haemorrhagic reaction in the infarct. A mycotic aneurysm, resulting from local inflammation and destruction of the wall of an artery at the site of arrested septic material, may rupture and give rise to severe bleeding. At autopsy, no aneurysmal dilatation may be found, only the edge of the torn vessel. Mycotic aneurysms are generally at the bifurcation of small vessels, close to or in the subarachnoid space.

**Hypertensive Encephalopathy**

This term is specifically reserved for a syndrome in which there is a stereotyped sequence of events of serious import and dramatic development. This syndrome occurs in persons with moderate or severe hypertension and is characterized by an increase in the severity of the hypertension over a few hours and severe progressive headache often associated with nausea and/or vomiting proceeding to alterations of consciousness (apathy progressing to coma), and convulsions. There is generally severe hypertensive retinopathy and there may or may not be evidence of various degrees of the renal involvement so frequently associated with hypertension. The CSF pressure is increased but the fluid is usually normal otherwise. This is the classical basic syndrome of hypertensive encephalopathy.

To these events and physical signs may occasionally be added a history of the development of focal neurological signs, these signs being present on examination. It is emphasized that the addition of these focal neurological signs (for the primary diagnosis of hypertensive encephalopathy to be seriously considered) must be in the context of the syndrome described above. Otherwise, the clinician will repeatedly make a diagnosis (in the absence of the basic syndrome) of hypertensive encephalopathy when the disorder is simply some other type of "stroke".

**Vascular malformations and developmental abnormalities: aneurysm**

The principal effect of saccular or berry aneurysms is the production of subarachnoid haemorrhage and brain damage due to rupture of the lesion.
The different varieties of aneurysm - fusiform, diffuse, globular, etc. - are part of the enlargement of the entire circumference of the artery. Fusiform aneurysms are tortuous, relatively circumscribed dilatations most commonly involving the basilar artery or in the internal carotid arteries within or near the cavernous sinus. The distinction between fusiform aneurysms and diffuse aneurysms is probably of little importance. Globular aneurysms are a group in which there is marked spherical dilatation, with the parent vessel coming in at one side and leaving from the other side of the lesion. Any of these aneurysms may produce symptoms by exerting pressure on neighbouring structures or being the site of thrombosis.

**Trigeminal encephaloangiomatosis (Sturge-Weber-Dimitri disease)**

A localized degeneration of the cerebral cortex is found in association with a port-wine stain (capillary angioma) in the distribution of the first division of the trigeminal nerve in the face. The cerebral cortex adjacent to the angioma often contains calcium and produces a double contour opacity on an X-ray of the head. These cerebral lesions may be associated with mental retardation, convulsive disorders, and focal neurological deficits, although the lesions seldom bleed.

**Congenital abnormalities in the anatomical pattern of cerebral arteries**

Many variations in the relative size of the vessels making up the circle of Willis have been described. Deviations from the "normal pattern" are present in almost half of the specimens which have been examined. These deviations are probably only of clinical importance when vessels are either absent or of threadlike size. Rarely, one or both internal carotid arteries are missing; occasionally one is only of rudimentary size. Carotid-basilar anastomosis via the cavernous sinus rarely occurs.

**Inflammatory disease of arteries**

Varieties of metabolic encephalopathy or cerebral infarction are produced by the arteritides.

**Infections and Infestations**

Meningovascular syphilis is now a relatively uncommon cause of stroke. Neurosyphilis produces a chronic meningitis, and the blood vessels of the brain and spinal cord, lying within the meninges, become involved. If the arteritic change involves the intima, thrombosis may result, producing cerebral infarction. The infarcts are characteristically small in size. If there has not been previous antiluetic therapy, the CSF will invariably show an increased cell count and an elevation of the protein level. False-positive tests for syphilis in the CSF are virtually non-existent.
Pyogenic meningitis (influenza, staphylococcus, pneumococcus) and tuberculous meningitis occasionally cause cerebral arteritis and thrombosis. Generally, the primary diagnosis will already have been made; the initial clinical abnormality is rarely the sudden onset of a focal brain lesion.

Rare instances of arteritis may occur with typhus, schistosomiasis mansoni, mucormycosis, malaria, and trichinosis. Papillary and arterial changes and perivascular inflammatory cells may be present in the nervous system in typhus and other rickettsial diseases. The abnormal chemistry underlying the confusional psychoses, convulsions, and coma that may occur with these lesions is not understood. Schistosomiasis mansoni infection may be associated with the occlusion of small arteries and multiple cerebral infarcts. In rare instances, diabetes mellitus may be complicated by mucormycosis and occlusion of the internal carotid artery.

A clinical state called "cerebral malaria" may occur with malaria of the malignant or falciparum variety. There is acute onset of hyperpyrexia, convulsions, somnolence deepening to coma, and often death. If the patient lives, there may be focal brain involvement with hemiparesis, aphasia, etc. The cerebral infarction is caused by the occlusion of cerebral capillaries and arterioles by masses of red blood cells.

Arteritides of undetermined cause

Cranial arteritis (temporal arteritis) rarely causes the sudden onset of a focal brain lesion. However, the differential diagnosis of this disorder is extremely important since there is effective treatment (steroids) which will prevent the unilateral or bilateral blindness that occurs in more than one-third of cases. In this disorder the branches, mainly of the external carotid artery (especially the temporal arteries), are involved by subacute inflammation which may lead to thrombosis. There is generally some kind of cranial or scalp discomfort, together with lassitude and malaise, and without treatment there is almost always a marked elevation of the erythrocyte sedimentation rate. In some instances, the syndrome of amaurosis fugax has been caused by cranial arteritis.

Systemic lupus erythematosus produces arteritic changes in the central nervous system (also at times the peripheral nervous system) which may cause a form of metabolic encephalopathy characterized by delirium, confusional states, and convulsions with drowsiness, sometimes progressing to coma. In rare instances, the effective inflammatory pathology appears to be limited to one area so that a cerebral infarct occurs. More commonly, micro infarcts and sometimes petechial haemorrhages are multiple and widely scattered. In other instances where there is severe kidney involvement, intracerebral haemorrhage or hypertensive encephalopathy may occur. Very rarely there is embolic occlusion of large and small brain arteries secondary to verrucous endocarditis (Libman-Sacks).
Rheumatic arteritis is an uncertain entity. In acute rheumatic fever or in chronic endocarditis of rheumatic origin, brain embolism may take place. Lupus erythematous and rheumatic fever are closely interrelated, and it may be that lesions thought to be due to the latter are actually due to the former.

Polyarteritis nodosa (panarteritis) directly involves the cerebral arteries in less than 10% of cases. The neurological picture produced is sometimes, though rarely, that of the acute onset of cerebral infarction. There is more likely to be a more general cerebral syndrome in which headache, confusional reactions, and convulsions are present. The coexistence of mononeuritis multiplex may be of great assistance in establishing a clinical diagnosis. Hypertension is commonly present and if there is renal involvement it may become so severe as to lead to hypertensive intracerebral haemorrhage or hypertensive encephalopathy.

Idiopathic granulomatous arteritis (Takayasu's disease) of the aorta and its major branches, including the common and internal carotid arteries, occurs from time to time in all countries, although it was originally described in Japan. It is frequently referred to as the "pulseless disease" owing to the occlusion of carotid and limb arteries that occurs. Along with various types of emboli and angioma, this disorder should be considered in the differential diagnosis of unexplained stroke in young adults.

Moyamoya (a Japanese expression for something hazy like a wisp of fog drifting in the air) disease is a rare condition, which is characterized in its fully developed form by an angiographical picture showing occlusion or stenosis at the carotid bifurcation with a netlike cluster of vessels intracranially on the same side. In children, the disorder is commonly characterized by attacks of paroxysmal hemiplegia, while in the adult the onset may be sudden with seizures and subarachnoid haemorrhage.

Thromboangiitis obliterans (Winiwarter and Buerger) is not included; its existence as an entity is uncertain.

**Dural sinus and cerebral venous thromboses.**

In everyday practice, intracranial phlebothrombosis and thrombophlebitis are uncommon and do not often cause confusion in the differential diagnosis of stroke. With inflammation in the structures involved, blood may be dammed back into small venules and capillaries, obstructing venous drainage so that local brain ischaemia occurs, sometimes followed by cerebral oedema and haemorrhage infarction. Focal convulsive events may occur.

Vein and sinus thromboses were much more common in the pre-antibiotic era when they were secondary to pyogenic infections of the mastoid, paranasal sinuses, or face. The inflammatory process sometimes travels to the larger veins directly or it may come about through the production of a local osteomyelitis, or thrombophlebitis of small diploic vessels which carry the infection intracranially.
Thrombosis of cerebral veins may be seen shortly after childbirth or a surgical operation. In the former instance, it may be due to hypercoagulability of the blood (hyperfibrinogenaemia) or increased blood platelet count.

The manifestations greatly depend upon the site and severity of the cerebral pathological process. Convulsions and/or hemiparesis may occur; the CSF may be bloody. Weakness involving only a leg or an arm (sparing the face) is probably due to a lesion adjacent to the sagittal sinus, the common site of infarction, if occlusion of the sagittal sinus has taken place. If the sagittal sinus is occluded, another clinical picture is one of bilateral neurological signs. With this and/or occlusion of the transverse sinuses, there may be an increase in intracranial pressure which can be associated with headache, choked discs, and visual obsurations. The changes in the CSF are variable, from a mild increase in the white blood cell count and rise in the pressure of modest degree to bloody CSF under high pressure if extensive haemorrhagic infarction has taken place. In those instances where the venous thrombosis is secondary to a purulent meningitis, the characteristic CSF findings of the latter will be present.

The clinical neurological picture - arterial patterns

The clinical neurological picture is determined by the site of the brain damage (infarction or haemorrhage). It is now common practice to divide the brain blood supply into two major categories: (1) the carotid system, and (2) the vertebrobasilar system.

Internal carotid artery. Occlusion of the internal carotid artery in the neck does not produce any characteristic clinical picture. In the presence of adequate intracranial collateral circulation, such occlusion may produce no symptoms or signs. At the other end of the spectrum, in instances where the collateral circulation is faulty, there may be infarction of a major portion of the ipsilateral hemisphere with contralateral hemiplegia, hemianesthesia, homonymous hemianopia, and aphasia (if the dominant hemisphere is involved). Stupor may ensue quickly, particularly if there is brain swelling with compression of the brain stem. With occlusion of the internal carotid artery in the neck, there is very seldom a simultaneous onset of permanent blindness in the ipsilateral eye together with the contralateral hemiplegia, hemianesthesia, and aphasia. Likewise, occlusion of the internal carotid artery in the neck is seldom a direct cause of permanent ipsilateral blindness (without hemispheric signs). Much more common than the very severe syndrome outlined above is the situation in which there is atherosclerotic occlusive disease in the internal carotid artery in the neck and an associated distal arterial occlusive event which produced only a fragment of the neurological defect that would occur if all the territory supplied by the carotid artery were infarcted. Thus, the neurological picture may range from a monoparesis to hemiparesis with or without a homonymous defect in vision, a variety of impairments of speech and language, different types of agnosia, and a whole
range of partial to full sensory abnormalities. A cervical internal carotid lesion is particularly to be suspected as a pathogenetic source of a sudden onset hemispheric neurological defect when there have been antecedent characteristic TIAs (particularly amaurosis fugax) or a long systolic or systolic-diastolic bruit at the take-off of the ipsilateral internal carotid artery, ipsilateral decrease in retinal artery pressure, or cholesterol or fibrin-platelet emboli in the appropriate eye.

**Middle cerebral artery.** The most significant clinical subdivision of the internal carotid artery system is the middle cerebral artery (MCA) or what might be more appropriately called the middle cerebral arterial system. Occlusion of the first portion of the MCA is almost always associated with the production of a neurological defect. Such a lesion is more distal in the total carotid arterial system and occurs at a site where the chance of collateral supply via the circle of Willis is no longer present. Occlusion of the artery is said characteristically to produce a contralateral hemiplegia, hemihypaesthesia or hemianesthesia, homonymous hemianopia, and aphasia if the defect is in the dominant hemisphere. However, occlusive disease in the middle cerebral arterial system more commonly produces a portion or fragment of this total picture, the variations in the neurological findings providing a broad spectrum of abnormalities. If the infarct is very large, there is more likely to be brain oedema with brain stem compression and stupor a few hours after the onset. Depending on the pattern of collateral blood supply and the actual artery or arteries occluded in the middle cerebral system, there will be variations such as: hemiplegia to mild paresis of one side of the face alone, mild weakness of one upper extremity alone, etc. If the focal impairment of blood supply is in the posterior portion of the middle cerebral system, there is more likely to be homonymous hemianopia or a partial homonymous visual field defect, aphasia, or dysphasia. If penetrating branches of the MCA are the only vessels occluded, the defect may be only a motor one, or if arteries to sensory cortex are the only ones involved a cortical-type hypaesthesia will result. It must be emphasized that there is a wide spectrum of neurological abnormalities (including many variations on the parietal lobe syndrome) that may occur with occlusive disease in the middle cerebral arterial system.

**Anterior cerebral artery.** Occlusive events in the main stem of the anterior cerebral artery or its various branches are also associated with the production of a variety of clinical events. No solitary syndrome has been delineated as characteristic of occlusion; this is because of the variety of patterns assumed by the branches of the vessels and particularly the variety of patterns of collateral supply. When there is paralysis or severe weakness of the opposite lower extremity with mild or no involvement of the opposite arm, a lesion in the distribution of the anterior cerebral artery is likely. Mental change, often subtle and mild but sometimes severe enough to be called dementia, dyspraxia, or apraxia of the use of an extremity or in walking, grasping, and sucking reflexes, and problems of maintaining continence of bowel and bladder may be associated with infarction of the brain in this distribution.
Vertebrobasilar arterial system. The vertebrobasilar system supplies blood to the medulla, pons, cerebellum, mesencephalon, thalamus, occipital lobes, and even portions of the temporal-occipital and parieto-occipital junctions. The most common defects include abnormalities of motor function: weakness, clumsiness, or paralysis of any combination of extremities up to quadriplegia with appropriate pyramidal tract signs combined with unilateral, or sometimes bilateral, cranial nerve palsies, in particular oculomotor defects or signs of trigeminal nerve or facial nerve involvement. A so-called "crossed" defect (motor or sensory on one side of the face and the opposite side of the body) is evidence of a brain stem lesion until there is proof to the contrary. Involvement of sensory function is common and this may be in any combination of the extremities, including all four, or in both sides of the face and mouth. If the occipital lobes are the site of ischaemia, there will be loss of vision, complete or partial in both homonymous fields (bilateral homonymous hemianopia). Ataxia, imbalance, unsteadiness, or disequilibrium not necessarily associated with vertigo may occur because of defects of the labyrinthine or cerebellar systems. Vertigo, notably in such conditions as the lateral medullary syndrome, is a very common complaint and, when produced by brain stem infarction, is usually associated with one of several types of nystagmus. Dysphagia and/or dysarthria may occur in combination with any of the abnormal neurological signs already mentioned. The most important evidence that the ischaemia is located in the brain stem is bilaterality of sensory or motor abnormalities coupled with definite evidence of cranial nerve (III to IX) involvement. Impairment of consciousness early in the course of events is unusual. If nystagmus alone is noted, it may be impossible to decide whether the lesion is in the vestibular-cerebellar system or in a peripheral portion of the vestibular system. However, if, in addition to the nystagmus, there is impaired ocular rotation or some evidence of an upper motor neuron defect in one or more extremities or some abnormality such as dysarthria or dysphagia, the anatomical site of the ischaemia is identified as the brain stem. The neurological syndrome in acute occlusive disease of the vertebrobasilar system develops over a period of hours or a few days, frequently progressing to spasmodic or stepwise fashion. The successive steps may occur over a period of 72-96 hours, and there is therefore, the risk that the progression may be prematurely presumed to be completed.

Posterior cerebral artery. As has already been mentioned in connexion with other main cerebral arteries, the posterior cerebral artery supply actually constitutes a subdivision of the vertebrobasilar system. The neurological abnormality produced by occlusive disease in this system depends on the site of the occlusion or stenosis and on the effectiveness of the available collateral flow. If the occlusion is distal, a homonymous hemianopia or even quadrantanopia may result. There may be variations in the position of the "watershed" between the posterior portion of the middle cerebral system supply and the anterior portion of the posterior cerebral supply so that a lesion in the posterior cerebral artery on the dominant side may produce dyslexia, dyscalculia, and a variety of speech and language abnormalities, while on the non-dominant side elements of a parietal lobe syndrome defect may occur. If the occlusion is more proximal in the posterior
cerebral arterial system, there may be a contralateral hemiparesis (cerebral peduncle), third nerve palsy (oculomotor), or a contralateral thalamic syndrome (posterior-thalamus). Cortical blindness and varieties of visual-verbal agnosia may be produced if both occipital lobes are affected because of impaired blood flow to the territories of both posterior cerebral arteries. The patient may be unaware that vision is affected or may have strange variations in behaviour.

Vertebral artery. Although it is now common practice to use the phrase "vertebrobasilar system", it has been demonstrated that occlusion of a vertebral artery at or near the origin of the posterior inferior cerebellar artery will produce a lateral medullary syndrome (Wallenberg's syndrome) which is characterized by the sudden onset of severe rotational vertigo, nausea and vomiting, dysphagia, ipsilateral cerebellar ataxia, ipsilateral Horner's syndrome, and involvement of pain and temperature sense on the ipsilateral face with contralateral loss or impairment of sensation for pain and temperature on the extremities and trunk. When there is severe stenosis or occlusion of a subclavian artery, there may be reversal of flow in the ipsilateral vertebral artery. This has not been consistently associated with any neurological symptoms or signs. When one vertebral artery is very small and the other unusually large, occlusion of the principal vertebral artery may be associated with symptoms and signs usually occurring with main stem basilar artery occlusive events.

C. General cerebral dysfunction

This category is concerned with general cerebral ischaemia, which results from reduction in blood supply to the brain, yet is applicable also to lesions from other causes. The presence or absence of disease of the cerebral arteries is not implied.

1. Transient. The classic example of a transient episode of general cerebral ischaemia is simple fainting. It is possible that loss of consciousness may result from ischaemia of focal areas of the brain, areas whose integrity is required for maintenance of consciousness; where it is possible to decide that such episodes are in fact focal, the patients should be categorized under I.B.

2. Prolonged. The acute onset of generalized brain ischaemia is most commonly a result of cardiac arrest or of something causing a great decrease in cardiac output. Consciousness may be lost or impaired for the remainder of the patient's life. If the decrease in blood supply is of very short duration, there may be permanent damage to higher intellectual function. A gradually progressive onset is uncommon in cerebrovascular disease per se; it is more likely to be associated with some change in the blood, i.e., pernicious anaemia, azotaemia, hepatic disease, etc.

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1 As far as is known, chronic dementia of the elderly without evidence (clinical or pathological) of neurological deficits of vascular origin is rarely caused by cerebral atherosclerosis alone. Although the patients are frequently diagnosed as having "arteriosclerotic dementia", "cerebral atherosclerosis with dementia", or "chronic brain syndrome with cerebral atherosclerosis", their conditions are often confused with other toxic metabolic or degenerative diseases (e.g., Alzheimer's disease or senile dementia). A patient with focal neurological deficit of vascular origin e.g., hemiparesis plus a defect in mentation, should be classified under I.B.
PART II. CLINICAL PHENOMENA

(HISTORY, PHYSICAL EXAMINATION, LABORATORY EXAMINATION, ROENTGEN EXAMINATION, OTHER)

Part II (Clinical phenomena) is designed to permit classification or codification of material from the patient's history, physical examination, and laboratory examination, and from a number of special procedures including angiography and cerebral blood flow determinations. The physician caring for an individual patient will find this section valuable as a "check list" or reminder of symptoms, physical signs, and many laboratory and special procedures. Epidemiologists, statisticians, and clinical investigators will find Part II of assistance in designing protocols and forms for the systematic recording of clinical phenomena.

In subdivision A, "History", the categories "Demographic", "Family history", and "Past history", are self-explanatory.

In using category 4, "Present illness", it is important to refer to the definitions in Part I (Clinical stage) covering such categories as "Transient attacks (TIA)" and "Actively changing neurological deficit (progressing stroke)". The definition of TIA includes comments on certain individual symptoms (e.g., vertigo) which in themselves are not to be designated as TIA. Although the arterial system involved is classified under Part IV (Anatomy), the anatomical site of TIA, etc., is also included in Part II to facilitate the use of the Classification.

The first two categories of subdivision B. "Physical examination" are traditional ones "General" and "Neurological". However, the third category "Vascular" includes the material permitting the results of the neurological (mainly neurovascular) examination to be classified.

Subdivision C "Laboratory examination" covers many standard types of tests. The relative importance of many of these tests is considered in the appropriate sections. Certain arbitrary decisions are necessary, e.g., placing "Echo encephalography" and "Brain scan" in this subdivision rather than subdivision E "Special procedures".

Because of the importance of cranial angiography, a special subdivision D "Roentgen examination" is provided. Indications for angiography are discussed in the appropriate part of subdivision F "Explanations of clinical phenomena".

Subdivision E "Special procedures" provides space for listing various procedures that are usually exclusive to centres performing special clinical investigations of cerebrovascular disease.

Computerized axial tomography (computerized tomography, computed tomography) is included and is clearly an important non-invasive technique for the differential diagnosis of cerebral infarction, intracerebral haemorrhage, and intracranial neoplasm.
A. History
   1. Demographic
      a. Sex
      b. Age (specify day, month and year of birth)
      c. Race, ethnic origin, and other related information
      d. Occupation and/or socioeconomic status
      e. Education
      f. Environment (urban or rural)
   2. Family history
      a. Cerebrovascular disease
      b. Hypertension
      c. Diabetes
      d. Coronary artery disease
      e. Other vascular disease
      f. Other
   3. Past history
      a. Hypertension
      b. Vascular disease
         1) Migraine
         2) Raynaud's
         3) Arteriosclerosis obliterans
         4) Noninfectious arteritis (specify type)
         5) Familial hypercholesterolaemia
         6) Syphilis
         7) Other (specify)
      c. Diabetes
      d. Heart disease
         1) Coronary
         2) Congenital
         3) Rheumatic
         4) Hypertensive
         5) Other (specify)
      e. Haematological disorders (specify)
      f. Metabolic disease (specify)
      g. Chronic alcoholism
      h. Obesity
      i. Smoking habits
      j. Drugs (specify)
      k. Pregnancy
      l. Head injury
      m. Cerebrovascular event(s) (specify according to definition in Part I -
do not include data thought to be dynamically related to patient's
current problem).
      n. Other neurological disease
      o. Other past illness (specify)
4. Present illness
   a. Transient attacks (transient ischaemic or vascular attacks) (see I.B.1.)
      1) Number (specify)
      2) Date of first attack
      3) Date of most recent attack
      4) Frequency
         a. Becoming more frequent
         b. Becoming less frequent
         c. No change
      5) Duration (describe)
      6) Precipitating factors
         a. Position of body (describe)
         b. Change of position of head, neck, or arm (describe)
         c. Exercise (describe)
         d. Hypotension
         e. Cardiac arrhythmia
         f. Emotion
         g. Other (specify)
      7) Content of typical transient attack (symptoms)
         The following list of symptoms is not meant to be inclusive of all
         neurological phenomena. The list may be used as a framework or check
         list. The content of a typical attack or of several attacks may need
         to be described in narrative form and the sequence of occurrence of
         several symptoms and their severity may need to be indicated (specify
         site, type, and degree). These items will be applicable for all
         subsequent categories of clinical stage.
         a. Impaired consciousness
         b. Confusion
         c. Amnesia
         d. Visual disturbance
            (1) Unilateral
            (2) Bilateral
            (3) Homonymous
            (4) Diplopia
            (5) Variable
         e. Vertigo or dizziness
         f. Dysarthria
         g. Dysphasia - aphasia
         h. Auditory disturbance(s)
         i. Motor dysfunction
            (1) Mono
            (2) Hemi
            (3) Other
         j. Dysphagia
         k. Sensory disturbance
            (1) Mono
            (2) Hemi
            (3) Other
         l. Impaired equilibrium
         m. Headache
         n. Head noise (bruit, etc)
         o. Impaired bladder function
         p. Impaired bowel function
q. Convulsions
   (1) Focal
   (2) Generalized
r. Other
8) Arterial system
   a. Carotid
   b. Vertebrobasilar
   c. Mixed
d. Uncertain
b. Actively changing neurological deficit (progressing stroke)
   1) Time of onset (specify date and hour)
   2) Duration of change (progression) (specify time)
   3) Predisposing factors (See V.E.)
   4) Precipitating factors (See II.A.4.a.6.)
   5) Symptoms (See II.A.4.a.7.)
   6) Arterial system
c. Prolonged neurological deficit (reversible ischaemic neurological deficit
   RIND) and completed stroke
   Please make distinction between RIND and long course as defined in I.B.3.
   1) Time of onset (specify date and number of minutes or hours from first
      symptom to maximum neurological deficit)
   2) Precipitating factors (See II.A.4.a.6.)
   3) Symptoms (See II.A.4.a.7.)
   4) Arterial system (See IV)
      a. Carotid
      b. Vertebrobasilar
c. Mixed
d. Uncertain
B. Physical Examination
1. General
   a. Temperature
b. Respiration
   1) Rate
   2) Rhythm
   3) Airway (tongue, secretion, cough, ventilation)
c. Pulse
   1) Rate
   2) Rhythm
d. Blood pressure Left_______  Right_______
   Postural change (specify)
e. Cardiac status (describe if abnormal)
f. Bowel status (describe if abnormal)
g. Bladder status (describe if abnormal)
2. Neurological
   a. State of consciousness (It is recognized that there are many ways of
      describing this item. It is important that the user of this classification
      construct a standard system of description of state of consciousness, such
      as:)
      1) Normal
      2) Semi-stupor (lethargy) - response slowed)
3) Stupor (appropriate response to verbal stimulus)
4) Deep stupor (purposeful response to noxious stimulus)
5) Semicoma (nonspecific response to pain)
6) Coma (above reflexes present - no response to pain)
7) Deep coma (absent DTRs, pupillary reactions, and corneal reflexes - no response to pain)

b. Mental function
1) Normal
2) Impairment of intellect
   a. Memory
   b. Recall
   c. Calculation
   d. Judgment
   e. Orientation
   f. Other
3) Speech and language
   a. Dysarthria
   b. Dysphasia (describe; "aphasia" is often used to describe a speech defect which would more correctly be termed "dysphasia")
   c. Agnosia
      (1) Visual
      (2) Auditory
   d. Dyspraxia
   e. Dysgraphia
   f. Dyslexia
   g. Other (palilalia, etc.)
4) Disorders of emotion (affect) (describe)
5) Distortion of thought content (describe)

c. Cranial nerves and related functions
1) Visual acuity
2) Optic fundus - ischaemic retinopathy (See II.B.3.d.6., etc.)
3) Visual fields
4) Pupillary reactions
5) Ocular movements
   a. Muscle weakness (describe)
   b. Nystagmus
   c. Gaze impairment
   d. Internuclear ophthalmoplegia
   e. Optokinetic responses
6) Sensations on face including corneal reflex
7) Muscles of mastication including jaw jerk
8) Muscles of facial expression
   a. Supranuclear
   b. Nuclear and peripheral
9) Auditory function
10) Vestibular function
11) Pharyngeal and laryngeal muscles including gag reflex
12) Sternomastoid and trapezius muscles
13) Tongue muscles including alternate motion rate (AMR)
14) Miscellaneous
   a. Horner's
   b. Pseudobulbar palsy
   c. Palatal myoclonus
   d. Singultus
15) Other
d. Sensation (exclusive of special senses) (describe)
   1) Vibratory
   2) Two-point discrimination
   3) Sense of position
   4) Pain
   5) Light touch
   6) Temperature
   7) Stereognosis
   8) Deep pressure
   9) Double simultaneous stimulation
10) Figure writing (graphaesthesia)
11) Localization of touch
12) Texture, appreciation of weight, etc.
e. Reflexes
   1) Muscle stretch
      a. Biceps
      b. Triceps
      c. Brachioradialis
      d. Hoffman
      e. Knee
      f. Hamstring
      g. Ankle
      h. Other
   2) Cutaneous
      a. Abdominal
      b. Other
   3) Miscellaneous
      a. Plantar (Babinski)
      b. Grasp
      c. Snout
      d. Sucking
      e. Other
f. Motor function
   1) Gait (describe)
   2) Posture (describe)
   3) Strength (grade each muscle if appropriate)
      a. Normal
      b. Mild impairment of strength (fair). Full range of motion against resistance
      c. Moderate impairment of strength (poor). Full range of motion against gravity.
      d. Severe impairment of strength (combine polio classification "trace" and "poor") Full range of motion with gravity eliminated
      e. No strength
4) Coordination (limb)
   a. Ataxia (faulty synergistic action throughout movement)
   b. Dysmetria (faulty in measuring to the object)
   c. Rebound phenomena
5) Praxis and alternate motion rate (AMR) (describe)
6) Adventitious movements (describe)
7) Other phenomena disturbing motor function (describe)
   a. Contractures
   b. Muscle tone
8) Evaluation of the function of groups of muscles
   a. Upper extremity (patient sitting)
      (1) Normal (4+) (No impairment of function)
      (2) Good (3+) (Mild impairment of function) Normal except
          for skilled acts and endurance
      (3) Fair (2+) (Moderate impairment of function) Shoulder
          abduction and extension of elbow + grasp and release of
          hand + supination-pronation of forearm through full range
          of motion but with impaired speed, coordination and strength
      (4) Poor (1+) (Severe impairment of function) Has shoulder
          abduction and extension but not through full range of motion
      (5) Bad (0) (No function)
   b. Lower extremity
      (1) Normal
      (2) Mild impairment of function
          a. Knee extension normal
          b. Hip and knee flexion good
          c. Dorsiflexion of ankle poor
          d. Walks well - lacks full skill and endurance
      (3) Moderate impairment of function
          a. Knee extension present
          b. Knee and hip flexion poor
          c. Dorsiflexion of ankle out
          d. Walks - much trouble
      (4) Severe impairment of function
          a. Some range of motion (see above)
          b. Cannot walk
      (5) No function
   c. Trunk
3. Vascular (largely neurovascular)
   a. Inspection (specify abnormality)
   b. Palpation (specify abnormalities; tenderness, symmetry,
      thrill, etc.)
      (1) Pulses (specify grade: 0 = absent to 4 = normal)
          a. Carotid
          b. Temporal
          c. Brachial
          d. Radial
e. Femoral
g. Popliteal
h. Dorsal pedis
i. Other (specify)
(2) Carotid compression test

1 The value of carotid compression tests relative to the information gained weighed against the risk to the patient limits the use of the test to laboratories where careful monitoring of the patient (with ECG, EEG, etc.) is possible.

c. Auscultation - bruits (See subdivision F "Explanations")
(1) Location (specify)
(2) Grade 1 to 6 (1 = barely discernible with stethoscope,
6 = audible without stethoscope)
(3) Pitch (high, medium, low)
(4) Quality (rough, soft, smooth, etc.)
(5) Duration (ss = short systolic, ls = long systolic,
sd = systolic-diastolic, c = continuous)

d. Ophthalmoscopy (See subdivision F "Explanations")
(1) Retinal embolus
   a. Cholesterol
d. Calcific
e. Other
(2) Retinal haemorrhage (specify site)
(3) Hypertensive arterial changes
(4) Cotton-wool patches
(5) Microaneurysms
(6) Ischaemic retinopathy
(7) Papilloedema
(8) Retinal oedema
(9) Other (specify)

e. Ophthalmodynamometry (record as listed) (See subdivision F "Explanations")
(1) Position of patient
   a. Sitting
   b. Lying
   c. Standing
(2) Value (systolic/diastolic)
   a. Right
   b. Left
f. Neurological signs produced by position
   (1) Head turning
   (2) Standing up
   (3) Sitting up

g. Veins (specify pertinent abnormalities)

C. Laboratory examination
1. Urinalysis (specify abnormality)
2. Blood
   Haemoglobin, red blood count, white blood count, haematocrit,
   viscosity, volume, cholesterol, lipids, serum enzymes, sugar,
   blood urea nitrogen, uric acid, creatinine, sedimentation rate,
   coagulation studies, platelet count, total thyroxine, serological
   test for syphilis, L.E. cell preparation.
3. Cerebrospinal fluid (specify abnormality)
4. Electrocardiography (specify abnormality)
5. Electroencephalography (specify abnormality)
6. Echoencephalography (specify abnormality)
7. Brain scan
   a. Static (specify isotope, specify abnormality)
   b. Rapid serial scintigraphy (gamma camera)

D. Roentgen examination
1. Chest
   a. Normal
   b. Abnormal (specify)
2. Head
   a. Normal
   b. Abnormal (specify)
3. Angiography (cranial)
   a. Anaesthesia
      (1) General
      (2) Local
   b. Media (specify)
   c. Site(s) of injection (specify)
      (1) Catheter (specify type)
      (2) Pump (type)
   d. Vessels visualized
      (1) Extracranial (specify)
      (2) Intracranial (specify)
   e. Abnormalities
      (1) Extracranial (specify)
      (2) Intracranial (specify)
   f. Complications
      (1) Extracranial
         a. Cardiac
         b. Airway
         c. Hypotension
         d. Bleeding
         e. Other
E. Special procedures
1. Computerized axial tomography (computerized tomography, computed
tomography) (See subdivision F "Explanations")
2. Cerebral blood flow (specify method) (list results) (describe
complications)
3. Retinal circulation time (specify method and results) (fluorescein)
4. Thermography and thermometry (specify method and results)
5. Retinal photography (specify method and results)
6. Tilt-table study (specify methods and results)
7. Phonocardiography (specify methods and results)
8. Electronystagmography
9. Doppler blood flow estimation (specify method, site, results)
10. Ocular plethysmography (specify method and results)
11. Cranial impedance plethysmography (rheoencephalography) (specify
method and results)
12. Other

F. Explanations of clinical phenomena

A. History

The family history may contain significant information with a bearing on
hypertension, diabetes, and cardiac disease. The past history of the patient
may contain relevant material about these and other conditions, including
previous stroke.

In reviewing the history of a current illness, the physician must seek
to elicit answers that are relevant (directly or by inference) to the different
sections of this classification. If the complaint has been transient, the
circumstances of onset, including activity being performed, physical position
of the patient, quickness of development of symptoms, and the duration as well
as the types of complaints must be indicated. The history may contain important
information concerning the pathophysiological mechanism, i.e., whether the
source of emboli is cardiac or intracarotid if there has been amaurosis fugax.
A history of rapid onset and progression of a focal neurological deficit, with
headache, against a background of untreated hypertension may point the way
to a diagnosis of intracerebral haemorrhage. Statistically, antecedent TIAS
strongly favour a diagnosis of cerebral infarction rather than one of cerebral
haemorrhage. These are but a few examples of how history-taking should be
designed to bring out information concerning the clinical stage, pathophysiological
mechanisms, anatomy, and pathology.
8. **Physical examination**

1. **General**

   In the general physical examination the emphasis is on detecting evidence of any pathology in the cardiovascular system — evidence that may of great importance, such as severe arterial hypotension with a cardiac arrhythmia, or of minimal importance, such as a blood pressure level of 20/12 kPa (150/90 mmHg). Attention should be given to heart rate, rhythm and size, blood pressure in both upper extremities, heart sounds, peripheral pulses and detection of congestive failure including dyspnoea, venous engorgement, hepatomegaly, ascites, pedal oedema, and pulmonary congestion or oedema.

   Skin lesions such as ecchymosis and petechia may suggest processes producing similar lesions in the brain. Skin tumours as well as masses in other sites may be at the origin of intracranial metastases.

2. **Neurological**

   The neurological examination and the interpretation of the neurological findings often make it possible to diagnose the site of the nervous system lesion or lesions. Certain combinations of neurological signs almost establish a diagnosis, e.g., lateral medullary syndrome, bilateral homonymous hemianopia, etc. Often the accurate inspection of a patient with "confusion" will reveal aphasia, and the physician then knows that the neuropathology is focal rather than diffuse. The reader is referred to one or more of the many textbooks that describe the neurological examination.

3. **Vascular (largely neurovascular)**

   To place special emphasis on the importance of certain abnormal physical signs, certain portions of the examination have been grouped together under this term. These include (a) inspection of vessels, (b) palpation (including carotid sinus massage and carotid compression), (c) auscultation at cervical and cranial sites, (d) ophthalmoscopy (including inspection of the retina for emboli, cotton-wool patches, vascular occlusions, haemorrhage and ischaemic retinopathy), and (e) ophthalmodynamometry.

   (a) **Inspection of vessels.**

   Cranial arteritis is unusual as a cause of stroke. However, accurate diagnosis is vital to correct treatment, and significant arterial change may be detected by thoughtful viewing of superficial temporal arteries, coupled with palpation.
(b) Palpation

Palpation of cervical-cerebral vessels should be done gently. Minor differences in pulse between sides are difficult to interpret, and it may be impossible to distinguish a pulse coming from the first portion of the internal carotid artery or from the external carotid artery. Patients suspected of having atherosclerosis of cervical vessels may have ulcerated plaques or early thrombus forming; in either case manipulation of the arteries could dislodge emboli. Similarly, carotid compression tests and carotid massage are inherently dangerous for patients with cervical-cerebrovascular disease. The important dangers include dislodgement of emboli, temporary decrease in carotid flow, and significant changes in cardiac rhythm. If the "sick sinus" syndrome is suspected, massage should be performed only with continuous ECG, EEG, and blood pressure monitoring and with personnel and equipment for dealing with a cardiac emergency available.

(c) Auscultation

Auscultation of the cervical vessels often provides important evidence concerning the pattern of blood flow. A bell-type stethoscope is most easily applied in the supraclavicular fossa and over the eyes without using physical pressure, which may produce artifactual noise. The bell of the stethoscope is first placed over the aortic arch and then moved (1 cm or less at a time) superiorly. This progressive movement of the stethoscope is necessary to distinguish transmitted cardiac sounds from sounds arising in the innominate, subclavian, common carotid, or internal carotid arteries. A neutral position (patient sitting or lying prone with the head straight) is less likely to create sounds difficult to interpret than is a variety of twisted neck positions. If respiratory (tracheal) sounds obscure auscultation, the patient is requested to "stop" breathing for a few seconds, then to "start" breathing. Bruits should be graded for loudness, the scale being 1 (least) to 6 (loudest); i.e., 1/6 is barely audible, 6/6 is the loudest. The timing (systolic, diastolic, systolic-diastolic), duration (short, medium, long) and quality (rough, soft, smooth, etc.) should be described. A bruit of 1/6 loudness is of little significance, while one of 2/6 to 3/6 loudness, long systolic-diastolic duration, and timing of fairly high pitch over the origin of the internal carotid artery means high-grade carotid stenosis until it is proved otherwise. A soft (1/6 to 2/6) diastolic sound varying with slight change in neck position, is commonly an unimportant venous hum. Soft, sometimes almost continuous cervical bruits are fairly common in children and ordinarily do not indicate the presence of significant pathology. By carefully recording descriptions of bruits and correlating them with arteriographical and other findings, the examiner will quickly learn to interpret such sounds correctly.

If the patient's history suggests the presence of a neoplasm or arteriovenous malformation, auscultation over the cranial vault and orbits should be performed. When there is a complaint of a rhythmic head noise, particular attention should be paid to auscultation of the locus of the sound. It may be necessary to wet the
patient's hair to eliminate artifactual noise. Auscultation of the orbit is performed by instructing the patient to close the eyes, placing the bell of the stethoscope over the eye and having the patient open the eyes to eliminate artifactual muscle sounds. Soft bruits over the cranial vault of children are of little importance. Loud bruits may be caused by angiomas, arteriovenous shunt and, rarely, brain neoplasms. A continuous, almost machinery-like murmur or bruit over the orbit is most commonly caused by a carotid cavernous arteriovenous shunt. Noises heard over the orbit have been of little help in establishing the site and severity of lesions of the internal carotid artery.

(d) Ophthalmoscopy

Ophthalmoscopy provides an opportunity for the direct inspection of small blood vessels that are a direct continuation of the internal carotid arterial system. In office and hospital practice relatively little use is made of this simple, safe method of acquiring important data concerning the cervical-cerebral portion of the circulation. The retina should be inspected for arterial or venous occlusion, emboli (cholesterol, platelet-fibrin, calcific, mixed, foreign body), haemorrhages, cotton-wool patches, venous stasis, microaneurysms, changes associated with arterial hypertension, papilloedema, and ischaemic retinopathy.

1. Retinal emboli

In the last two decades the importance of detecting (with the ophthalmoscope) a retinal embolus or emboli has been demonstrated. The most common emboli are made up of cholesterol crystals. These appear as shiny orange-yellow plaques, often situated at the bifurcation of retinal arterioles. The plaque may appear to be wider than the arteriole; the outer dimension of the column of red blood cells is seen, rather than the wall of the arteriole. Pressure on the eye often changes the position of the embolus slightly - the material may appear to glint or change shade, a characteristic sometimes referred to as a heliographic reflection. The blood flow in the arteriole is often seemingly unimpeded by these bright orange-yellow plaques. The emboli may move distally and often disappear in a few days. The presence of one or more cholesterol retinal emboli indicates that there is or has been ulcerated atheromatous carotid (internal) lesion, unless there is proof to the contrary.

Another important type of embolus in retinal vessels consists of gray-white material, thought to be made up of blood platelets and fibrin. These emboli may be long and may be seen to move through an arteriole, but are commonly stationary; pressing on the eye does not move them, and there is no heliographic reflection. Blood does not appear to flow past these emboli; there may be infarction of the retina. Special studies show that some of these emboli have a high lipid content. In many instances the source of these emboli is an atheromatous lesion at the origin of the internal carotid artery. Particles of calcium are another type of retinal embolus. These are white, generally short, and stationary. Calcium emboli commonly come from heart valve lesions.
Septic emboli, talc and cornstarch emboli, and others may be seen in the retina but are less common than those described above.

(e) Ophthalmodynamometry.

Ophthalmodynamometry is a procedure for measuring the arterial systolic and diastolic pressures in the main retinal branch or branches of the ophthalmic artery. The convex foot-plate of the instrument is applied to the conjunctiva over the insertion of the lateral rectus muscles in a horizontal manner so that the instrument points directly toward the opposite eye. When measurements are being made in the patient's right eye, the instrument is held in the observer's left hand and the ophthalmoscope is held in the right hand. To measure pressure in the left retinal artery, the observer holds the ophthalmodynamometer in the right hand and the ophthalmoscope in the left. When the instrument is in position, the observer must bring the central artery on the disc into focus through the ophthalmoscope. The instrument then is pressed gradually against the eye to raise the intraocular pressure sufficiently to exceed the diastolic level of the blood pressure in the retinal artery. The diastolic pressure is that level which produces the first collapsing pulsation of the artery. At this point a finger is applied to the brake on the instrument and the reading is taken from the scale. The ophthalmodynamometer is reapplied, and several more readings are taken to ensure accuracy. The systolic pressure is obtained by increasing the force of application of the instrument still further. The visible arterial pulsation gradually diminishes as the pressure increases and, when pulsation ceases, the reading on the instrument is the systolic blood pressure.

Ophthalmodynamometry is ordinarily useless unless the arterial pressures are measured in both eyes. It cannot be performed unless the patient is cooperative. It is helpful to instil a mydriatic; this should not be done if there is glaucoma. The test should not be done soon after cataract extraction, recent retinal detachment, etc.

The clinical significance of the retinal arterial pressures is dependent on comparing the values in the two eyes. A difference of 15% to 20% is almost always a sign of stenosis or occlusion of the internal carotid artery ipsilateral to the lower pressure. The arterial pressure may be equal and/or normal in the presence of unilateral carotid stenosis or occlusion because of the development of collateral blood supply. Immediately following acute occlusion of an internal carotid artery the ipsilateral retinal arterial pressure drops. Return of the pressure to that of the contralateral eye depends on the speed with which collateral circulation develops. A marked decrease in the retinal arterial pressure (brachial arterial pressure remaining normal) when the patient moves from the supine position to the upright position (ocular orthostatism) is important evidence of carotid occlusive disease.
A suction ophthalmodynamometer requiring a source of electric power is available. It is safe and highly accurate, but awkward to use.

(f) Neurological signs produced by position.

If the patient describes phenomena that are precipitated by certain positions of the head and/or torso, it is wise to try carefully to reproduce the symptoms and simultaneously search for physical signs of dysfunction. Thus, extension of the neck may be associated, in the patient's history, with the onset of "dizziness"; have the patient reproduce the motion and observe for nystagmus. The history provides a clue to the existence of orthostatic hypotension. There are a variety of such phenomena.

C. Laboratory examination

A number of laboratory tests are helpful in the diagnosis and management of cerebrovascular disease, but none of them can approach in general usefulness a careful history and physical examination.

1. Urinalysis

a. Sugar.

In normal circumstances, there is no glucose in the urine. This gives a crude but very useful screening test for abnormal glucose metabolism.

b. Protein

Normally, on qualitative evaluation, there is no protein in the urine. This affords a crude but valuable way of screening either for abnormal substances coming through the kidney or for abnormal kidney function.

c. Sediment microscopy

This examination may give valuable information about such things as the presence of vasculitis and inflammatory disease of the kidney as well as being a crude way of screening renal function in patients with hypertensive vascular disease.

d. Other

A wide variety of tests for different substances including vanilmandelic acid, porphobilinogen, homocystinuria (cyanide-nitroprusside test), aldosterone, lead, metanephrines, etc., may be done in selected instances but are only rarely of help in the management of a stroke patient.
2. Blood

a. Test for syphilis, such as the VDRL test, etc.

In the 1970s, meningovascular lues, or luetic vasculitis, is not a common cause of stroke; however, a blood screening test for syphilis should still be included in the laboratory profile.

b. Red blood count, white blood count, blood haemoglobin, haematoctrit, and differential blood count.

These tests frequently give valuable information concerning the presence of haemoconcentration secondary to dehydration, the presence of polycythaemia vera which in turn may be associated with thrombosis or with haemorrhage, and other forms of polycythaemia. The differential blood count may suggest the existence or beginning of an inflammatory process, and the peripheral blood film provides the opportunity to estimate the number and types of platelets.

c. Erthrocyte sedimentation rate (ESR)

While it is relatively uncommon for a form of vasculitis (temporal arteritis, giant cell arteritis, lupus erythematosus, polyarteritis nodosa, etc.) to be a direct cause of stroke, it may be of tremendous etiological importance and the diagnosis may be immediately suggested by the marked elevation of the ESR which is so commonly present in acute vascular (collagen) disorders such as those referred to.

d. Fasting blood sugar or casual blood sugar test.

This is an excellent screening test for the detection of diabetes mellitus and, in certain patients who are acutely ill, hypoglycaemia of a variety of origins may be significant.

e. Creatinine and urea tests.

Either is a reasonably good screening test for renal disease, especially when coupled with urinalysis. If either creatinine or urea is normal in the blood and urinalysis is normal, significant renal disease is generally not present.

f. Cholesterol - triglycerides

Slight or moderate elevations of cholesterol and/or triglycerides are probably of little significance at the time a stroke actually occurs. However, elevated blood cholesterol has been identified as a likely risk factor for occlusive cerebrovascular disease in persons under 50 years of age. Elevation of triglycerides has been indicated as a risk factor in the development of coronary heart disease. Thus, one or both substances may be important to the prevention of further focal cerebral ischaemia in a person with TIAs or in a patient who has had a completed cerebral infarct. When there is marked elevation of the values, as in familial hyperlipidaemia, the risk of cerebral infarction is definitely increased.
g. Prothrombin time test.

In certain selected instances anticoagulant therapy may be initiated early in the course of occlusive cerebrovascular disease; in such instances it is important to have the results of this test for "baseline" purposes.

h. Uric acid

Mild to moderate hyperuricaemia is of little direct importance in the practical management of an acute progressing stroke or even a completed stroke. However, hyperuricaemia has been associated with atherosclerosis of the coronary, peripheral, and cerebral arteries and may, therefore, be somewhat distantly related to the profile of "risk factors for stroke" and worth considering for this reason.

i. Other.

This includes numerous tests that may be important in the care of selected patients but are not essential for all patients thought to have some form of cerebrovascular disease. These include: acid-base balance, bilirubin, brom-sulfalein dye retention, calcium, catecholamine, chloride, haemoglobin genotyping, clot retraction, whole blood coagulation time, plasma coagulation time, partial thromboplastin time, plasma fibrinogen, euglobulin lysis time, serum osmolality, arterial oxygen saturation, oxygen tension (air), potassium, protein electrophoresis, sodium, free and/or total thyroxine, enzymes, partial pressure of carbon dioxide, and pH. It may be that tests of platelet adhesiveness and ability to aggregate may become clinically important.

3. Cerebrospinal fluid

This examination should be performed when the clinician has a serious problem in establishing the differential diagnosis of the intracranial pathology - bleeding, focal ischaemia, or inflammatory disease. The fluid is ordinarily obtained by lumbar puncture and the examination should not be considered routine. The amount of fluid withdrawn depends upon the question to be asked; 1 ml will be adequate to demonstrate gross bleeding, while a much greater amount will be needed to determine the etiology of a meningitis that is increasing the number of lymphocytes in the fluid. In some instances the clinician may be reluctant to do a lumbar puncture even though there is no papilloedema, because of clinical evidence of increased intracranial pressure with the apparent brain pathology limited to one hemisphere. If there is a macroscopic amount of blood in the CSF, it is not possible (without analysis of the history and a neurological examination) to know whether the bleeding comes from a primary subarachnoid site or from an intracerebral source. The observations made depend on the clinical situation.
a. Pressure.

Information about the CSF pressure is of only relative value in the differential diagnosis. The Queckenstedt manoeuvre (compression of the jugular veins) should not be performed for the differential diagnosis of intracranial lesions. In certain instances the examiner will not wish to withdraw enough fluid even to measure the pressure in the usual manometer.

b. Colour.

Normally the CSF is crystal clear and colourless. If there is gross blood and the intensity of colour decreases after a few drops of fluid appear, the bleeding is due to trauma to a blood vessel. If the fluid is xanthochromic immediately after it is withdrawn or within a very few minutes, the xanthochromia has been produced by something other than a traumatic lumbar puncture. A trace of xanthochromia may be quickly detected by putting 1 cm³ or less of CSF in a Wassermann tube and comparing the appearance with that of a tube containing water. The line of vision should be directed down the length of the tube, and the background should be white. The colour may be produced by high CSF protein content or icterus but is commonly due to intracranial bleeding (rarely to intraspinal bleeding). If the CSF has a "ground-glass" appearance, there are generally more than 400 white blood cells present per ml of fluid.

c. Cells.

The CSF should be examined for red blood cells and white blood cells. In certain instances yeast, neoplastic cells, and bacteria may be detected. It should be noted that in the usual setting in which CSF examination is performed in a patient strongly suspected of having cerebrovascular disease, the appearance and the microscopic examination of the fluid give the answer to the question most commonly asked "Has there been bleeding into the subarachnoid space?".

d. Protein.

Modest elevation of the CSF protein is common in patients with various categories of cerebrovascular disease and is not of particular assistance in establishing an accurate differential diagnosis. In this general diagnostic situation, the electrophoretic fractionation of the proteins is only rarely of assistance in differential diagnosis.

e. Glucose.

Unless there is massive subarachnoid haemorrhage, CSF glucose level is not commonly altered by various categories of cerebrovascular disease. When attention is to be paid to the glucose determination, the blood and CSF glucose levels should be drawn simultaneously to determine whether the normal ratio (CSF glucose approximately two thirds of the blood glucose) is present.
f. Test for syphilis.

A positive test for syphilis in the CSF is highly significant; either such a result is a laboratory error or the chances are very great that the patient has luetic involvement of the nervous system, active or inactive. Although meningo-vascular syphilis is less common now as a cause of stroke than it was a few decades ago, this etiology continues to occur.

g. Enzymes.

Glutamic oxalacetic transaminase (GOT), lactic dehydrogenase (LDH), and creatine phosphokinase (CPK) in the CSF have been studied but are not significantly helpful in the diagnosis of various categories of cerebrovascular disease or in distinguishing between cerebrovascular disease and brain neoplasms. Thus, tests for these enzymes are not usually performed.

h. Gases and pH.

CSF pH and PO2 tests are not normally done as part of the differential diagnostic studies or for the clinical management of the patient. They have been done for investigative purposes.

4. Electrocardiogram

Because of the fully documented active interrelationship between various forms of cardiac pathology, including disturbances of rhythm, and cerebral ischaemia (both diffuse and focal), an electrocardiogram should be obtained in virtually all patients suspected of any category of cerebrovascular disease. If there is no history of any cardiovascular symptoms or pathology, if heart size, sounds, and rhythm are normal for clinical examination, and if the blood pressure is normal, active cardiac pathology is unlikely. However, in rare instances, a "silent" myocardial infarct may be present and there may be some change in the cardiac conducting mechanisms which the clinician cannot detect by physical examination. If the electrocardiogram is normal, it is unlikely that there is any intermittent disturbance of rhythm that might play a role in the pathogenesis of either diffuse or focal cerebral ischaemic events. Possible exceptions to this may occur in rare instances of "sick sinus syndrome". If there is a history suggesting some transitory change in cardiac rhythm, an abnormal cardiac physical finding, or a certain change in the electrocardiogram (an example being left ventricular hypertrophy with a right bundle branch block), it is often important to obtain long-duration electrocardiographical recordings by the use of equipment such as the Holter monitor.
5. Electroencephalogram

In the usual stroke patient (typical TIAs, most instances of progressing stroke, and almost all cases of completed stroke), the electroencephalogram adds little significant information and is not necessary. In vertebrobasilar disease the electroencephalogram usually shows no focal abnormality. It has been said that serial electroencephalograms may very well accurately portray the favourable or unfavourable progression of the brain lesion in stroke. However, the clinician can almost always get the same information by spending three or four minutes with the patient one or more times a day.

In selected instances an electroencephalogram may reveal multiple focal abnormalities, thus giving potential evidence of the presence of multiple metastatic lesions.

During carotid arterial surgery and cardiac surgery, the electroencephalogram is of value in monitoring brain function, but only if the personnel are knowledgeable about the effect of anaesthesia on the electroencephalogram. In rare instances when the clinician is unable to establish a differential diagnosis by the procedures described in the preceding pages, the combination of electroencephalography and echoencephalography may be helpful in the first few hours following the onset of the focal neurological abnormality. If, within 36 hours of the onset, the electroencephalogram shows a focal abnormality and the echoencephalogram reveals a shift of 2 mm or more of the midline away from the side of the EEG focus, the statistical chances are that the lesion is an intracerebral haemorrhage or other expanding mass.

In certain instances, the electroencephalographical recordings may be of aid in evaluating the stage of "irreversible coma". Criteria for "brain death" have been outlined by, among others, the ad hoc Committee of the American Electroencephalographic Society on Criteria for the Determination of Cerebral Death.

In medical centres where computerized axial tomography (e.g. EMI scanner, ACTA scanner, etc.) is available, there is very little need for EEG in the diagnosis of stroke patients.

6. Echoencephalography

Physicians or trained technicians can perform echoencephalography equally well. A shift of midline structures can be determined with an accuracy of 1 to 2 mm; a shift of 3 mm or more is ordinarily considered abnormal. Although the test is safe, it is seldom of significant help in establishing a differential diagnosis, particularly if the clinician has been willing to obtain a careful history and do the examinations recommended in the preceding pages. A shift
of the midline structures may be produced by cerebral infarction with oedema, intracerebral haemorrhage, subdural haematoma, brain abscess, brain neoplasms, and brain atrophy. As mentioned in the paragraphs about electroencephalography, a shift of midline structures of more than 2 mm within 36 hours of the onset of presumed cerebrovascular symptoms and signs suggests that the lesion is an intracerebral haemorrhage or other expanding mass. However, a shift developing after 36 hours from the time of onset may be due to oedema associated with infarction.

In medical centres where computerized axial tomography (e.g., EMI scanner, ACTA scanner, etc.) is available, there is very little need for echoencephalography other than in the evaluation of a patient in the emergency room.

Composite B-mode ultrasonography using radar techniques is being developed. The cervical portion of the carotid arteries has been scanned by several investigators but the method is still not feasible for clinical use.

Doppler techniques, including the use of a Doppler flowmeter, are under research and may be developed for clinical use.

7. Isotope brain scan.

The static brain scan has become an established procedure for the detection of intracranial neoplasms. However, in medical centres where computerized axial tomography (e.g., EMI scanner, ACTA scanner, etc.) is available, there is now very little need for the static brain scan in the differential diagnosis of focal brain lesions. Non-neoplastic lesions, such as subdural haematomas, abscesses, cerebral infarcts, or intracerebral haemorrhages, may at times produce focal abnormalities in the static brain scan. Brain scanning equipment is now widely available; however, it is unfortunate that in many instances clinical personnel competent to use and evaluate the scans for neurological differential diagnosis are not available. The length of time elapsing between the injection of the radionucelotide and the scanning is of paramount importance. Arteriovenous malformations are most commonly detected if the examination is done within 30 minutes of the injection, while cerebral infarcts are more likely to be detected if the scanning is delayed until 2 - 4 hours after the injection of the radioactive material. Cerebral infarcts are usually not identifiable by brain scanning until the third to eighth day following the occurrence of the lesion, and if a very definite focal abnormality is found within the first 24 hours after onset, brain neoplasm should be suspected.

Cerebral infarcts of about 2.5 cm or less in diameter do not show in a brain scan and, therefore, go undetected by this test. It is well established that a single abnormal brain scan confirms the existence of a focal brain lesion but gives no significant differential information concerning the pathology of the lesion.
Although this special procedure is safe, it is seldom necessary in making a differential diagnosis, is unduly expensive, and so far has not in any way replaced angiography.

Likewise, dynamic rapid serial scintigraphy using a gamma camera, thus giving a serial display of the images, provides much less precise information of clinical significance than is available through angiography. For instance, it is rarely possible with this technique to determine whether a carotid artery is occluded or whether there is a severe degree of stenosis. Likewise, modest amounts of stenosis are generally missed and ulceration of an atherosclerotic plaque in the internal carotid artery in the neck cannot be detected. Large and small vessel occlusive disease occurring intracranially is commonly missed by this method.

D. Roentgen examination

1. Radiographs of the chest

Standard radiographs of the chest give valuable information concerning heart size and configuration, aortic pathology, and pulmonary pathology, including infection and neoplasia.

2. Radiographs of the head

Plain radiographs of the skull give significantly positive diagnostic information in relatively few patients with clinically typical occlusive cerebrovascular disease - cerebral infarction. However, it has become standard practice to obtain such films. Occasionally evidence of cranial trauma or an intracranial mass lesion may be detected. Calcification of the carotid artery in the region of the sella is very common but does not give significant statistical evidence concerning the presence or absence of occlusive disease of a carotid artery at the place where the calcification is noted.

3. Angiography (cranial)

An important special procedure is cervical-cerebral angiography. There are numerous angiographical techniques for visualizing cervical-cerebral vasculature. The most important thing however, is the skill of the personnel and their total familiarity with the method they are using. It is now fully apparent that in almost every case in which angiography is indicated, the entire cervical-cerebral circulation should be shown on technically first-rate films. It is still the practice in some institutions to get films that show only a portion of the cervical part of the circulation, and in some instances the films of the intracranial vasculature are technically so poor that no significant information is available from them. Films of all portions of the cervical-cerebral circulation are necessary in order to be certain of the primary diagnosis as well as to ascertain whether a second or even more lesions may be present. If any kind of surgical intervention is being planned, it is important for the surgeon to know the state of the collateral circulation, and this can be obtained or evaluated only if fine films of all portions of the cervical-cerebral circulation are available.
Indications for angiography are:

(1) Differential diagnosis of the brain pathology. Even with careful attention to all the items listed under history, general examination, neurological examination, neurovascular examination, and additional tests, there still remain about 5% of patients for whom the diagnosis is uncertain. In such instances, cervical-cerebral angiography is the best method of making a distinction between vascular occlusive disease, an intracerebral expanding mass such as haemorrhage, abscess, or brain tumour, cerebral infarction, and subdural haematoma, as well as demonstrating aneurysms and arteriovenous malformations.

(2) Transient focal ischaemic attacks - particularly in the carotid system. In such instances cervical-cerebral angiography should be performed if there are one or more of the following: amaurosis fugax, bruit over the beginning of the internal carotid artery, retinal emboli, unilateral decrease in retinal artery pressure or ischaemic retinopathy. If none of these are present, the likelihood of finding a lesion accessible to the surgeon is very small.

(3) Selected instances of vertebrobasilar TIAs. In some situations, it may be difficult to make a clinical distinction between the carotid and the vertebrobasilar system. If the TIAs are characteristic of those coming from the vertebrobasilar system, there is little point in doing extensive angiography.

(4) Very early progressing stroke or very frequent TIAs in the carotid system with, as a part of the history, amaurosis, an appropriate bruit, retinal emboli, etc.

(5) Many patients with subarachnoid haemorrhage and some patients with intracerebral haemorrhage.

An uncertain indication is a long systolic or systolic-diastolic loud internal carotid artery bruit in patients scheduled for major general surgery. If there is prolonged hypotension or very severe blood loss, the carotid stenosis may decrease blood supply to a focal region of the brain to a critical level of ischaemia. Recent observations suggest that such patients do not have an increased risk of stroke; arteriography is therefore, not necessary.

Angiography is contraindicated in advanced forms of systemic disease. Acute myocardial infarction, allergy to contrast media, etc. must be considered as contraindications or relative contraindications to cervical-cerebral angiography. If the cerebrovascular disease has produced a situation where there is cardio-respiratory depression, coma, or extraordinarily severe neurological deficit, angiography is not indicated.
E. Special procedures

1. Computerized soft tissue tomography (CT, EMI scanner, ACTA scanner, etc.)

Computerized tomography equipment employs a narrow beam of X-rays to scan a patient's head in a series of "slices". The rays pass through the head and are detected by two sensing devices which always point towards the X-ray source. Both the X-ray tube and the detector scan across the patient's head linearly, and multiple readings of X-ray transmissions through the head are made during each traverse in the EMI scanner. At the end of each scan, the system is rotated 180° and the process is repeated. This continues for 180 scans when 28000 readings will have been taken. These readings are then processed in a minicomputer, which calculates the absorption values of the material within the "slice" from the 28000 simultaneous equations. From these calculations a three-dimensional picture or matrix is built which in essence displays differential X-ray "densities" inside the head in a way never before possible. The matrix for each "brain slice" is displayed on a cathode ray viewing unit, and a numerical print-out is also produced. The former is photographed with an automatic-development camera.

The system is about 100 times more sensitive than conventional X-ray systems and enables small variations in tissue density to be differentiated. The skin area irradiated is confined to a narrow band around the edge of the slice and the dosage is approximately equivalent to that for a conventional X-ray picture.

The cerebral ventricles are accurately shown and there is good display of the subarachnoid spaces. Brain atrophy or any abnormality that alters ventricle size, configuration, or position can be detected.

Extravasated blood has a much greater density than brain. A small focal haemorrhage is easily visible, and blood in the ventricles is easily detected.

Non-haemorrhagic cerebral infarcts can be seen; within a few hours of onset, oedema around the infarct may be apparent. A very haemorrhagic infarct may be difficult to differentiate from a haemorrhage; the latter often has a rounded shape.

Brain tumours can usually be distinguished from cerebral infarcts or intracerebral haemorrhage.

Circulating blood is not displayed; intracranial aneurysms and arteriovenous malformations will not be seen unless they contain clotted blood or calcium.

Subdural haematomas may be difficult to show, particularly when they are bilateral and do not produce shift of the ventricles.

It is apparent that computerized tomography will revolutionize the differential diagnosis of intracranial lesions.
2. Cerebral blood flow measurements

A variety of methods for measuring cerebral blood flow have been devised in the last 25 years. None of the currently used methods are accurate, reproducible, and non-invasive. The methods have been used primarily in research and have provided information about cerebrovascular physiology. The most accurate techniques involve catheterization of the internal carotid artery and/or jugular vein with the attendant risks. At the present time, these methods are not significantly helpful in clinical practice.

By numerous invasive techniques, jugular venous oxygen tension, jugular venous lactate concentration, and arterial-jugular venous oxygen differences may be measured. The data provided have occasioned much discussion but are not of significant value in the clinical care of patients with stroke.

3. Retinal circulation time

Ten per cent fluorescein is injected into an antecubital vein, and the time elapsing between the injection and the arrival of the dye in each retina is determined by ophthalmoscopic examination, using special filters. The test is cumbersome (three people are needed—one to observe each retina and one to inject the fluorescein) and does not give enough unique information to make it worthwhile. The development of appropriate devices for simultaneously photographing the retina might make the test more practical.

4. Thermography and thermometry

Thermography (as relating to stroke) measures the temperature (by recording infra-red emissions from the skin) particularly over the mesial supraorbital area of the forehead which is supplied by terminal branches of the ophthalmic artery originating from the internal carotid artery. Sometimes occlusion or very severe stenosis of the internal carotid artery is associated with reduced skin temperature in this particular area. The equipment is expensive and the test takes several minutes. Because of the expense and general cumbersomeness of the equipment, the test has not become popular.

Thermistor recordings of temperature from various points across the forehead give information similar to that obtained by thermography. This testing technique has not been of enough practical value, particularly in replacing other methods, to be useful.

A variety of thermochromic liquid crystals, and paints which detect small changes in temperature have been investigated but have not become useful practically because difficulties in their application outweigh the value of the information obtained.

5. Retinal photography

It is possible to photograph the retina in colour and show the changes of vessel occlusion, retinal infarction, hypertensive disease, diabetes, haemorrhage, and the various types of emboli. However, the procedure is so cumbersome and expensive that it is not practical for screening or for the diagnosis of relatively large numbers of patients.
6. **Tilt-table study**

Lowering of the effective arterial perfusion pressure to the brain by tilting the patient to an upright position on a tilt-table has been suggested as a way of inducing transient focal cerebral ischaemic attacks. Because it is ineffective in almost all instances, as well as requiring special equipment, the procedure is seldom used.

7. **Phonocrianiography**

The recording and/or transmission of bruits is possible, but the current equipment and methods do not permit the satisfactory application of this method on a large scale.

8. **Electronystagmography**

This is an electrical technique for recording nystagmus and other eye movements. It is possible to record nystagmus when the patient's eyes are closed. The test is, therefore, of value in recording caloric nystagmus, which may be absent when the eyes are open but present when the eyes are closed. However, so far no significant differential information about cerebrovascular disease has been obtained by this technique. Further study of electronystagmography may produce methods that will make it of some clinical value.

9. **Doppler blood flow estimation**

Continuous sonic energy has been used in an attempt (non-invasive) to map the morphology of extracranial arterial blood flow, i.e., carotids, supraorbital vessels, etc. So far the method has not produced satisfactorily accurate results. Further refinement of the equipment may make this technique a useful one.

10. **Ocular plethysmography (pulse propagation measurements)**

The technique involves carrying light into the skin of the face; changes in the intensity of light scattered back from the skin are converted by a photocell to voltage for polygraphical recording during each cardiac cycle. By recording the electrocardiogram simultaneously, the interval between electrical ventricular systole (the R-wave) and the arrival of the opacity pulse wave at the site being monitored can be measured. It is possible that further development of the equipment may produce a useful technique for the screening of patients with carotid occlusive disease.

11. **Cranial impedance plethysmography (rheencephalography)**

This is a term commonly, though incorrectly applied to measurement of the impedance of the head to the passage of an electrical current applied externally. Although this technique has been under study for over a decade, it does not appear to be of significant assistance in the evaluation of stroke.
PART III. STATUS OF PATIENT
(PERFORMANCE AND PLACEMENT)

At many points in the natural history of stroke, it is desirable to estimate the performance ability of the patient. A classification of performance ability and placement potential is presented in this section. Such an assessment serves as a significant guide to the course the disease process has taken, the management effort required, and the selection of a life situation to which the patient can appropriately return. Ability of the patient to perform satisfying and productive activity in a supportive environment is a resultant of many intrinsic and extrinsic factors. The intrinsic factors may include such items as the degree of general and focal cerebral damage, the residual neurological function, the integration of adaptive and substitute patterns of function, the pre-existing level of development of intellect and skill, and developed attitudes and emotions. The extrinsic factors are even more varied and numerous and include such influences as the nature of the physical environment and living arrangements, the degree of family involvement and support, interpersonal relationships, the social and economic resources, and even the attitudes of the family, friends, potential employers, and the community at large. Prescriptions for continuing management within and outside the health care facility must be based on information derived in detail from all available clinical and social sources. The classification of performance and placement provides a broad outline of what life-tasks the patient is able to do and how he will be able to live.

Performance

In the context of this classification, performance is defined as the execution of set tasks. The design of a task can be based on formal testing or can be variably determined by the environmental or situational circumstances of the life-setting. The nature of the task determines the action to be taken by the patient. The patient must be able to recognize the demands made on him by the task, and then to carry out the selected action successfully. Because the wide range of life situations makes for a great variety of task requirements, this classification is based only on descriptions of performance generally recognized to be of key significance in the care of the stroke patient. It is further recognized that performance in a test setting varies according to test conditions and social situations and may not necessarily predict performance in other social contexts, e.g., in the house, at work, or in the community at large. These considerations colour but do not negate the value of classification by test and observation.

For the purposes of this classification, three descriptors of life situations are selected as indices of performance status. These are: (a) activities of daily living (ADL), (b) avocational activities, and (c) occupational activities.
Activities of daily living (ADL) - A battery of tasks has been designed to provide an index of ability to perform self-sustaining activities that meet personal requirements in daily life. These essential activities are relatively independent of geographical locale, physical environment, or social situation. They include such common activities as dressing, washing, eating, and travelling from place to place; they involve fundamental body and limb movements, sitting balance, changes in body position, standing, reaching grasping, holding, and the like.

Avocational activities are those active pursuits of living not directly related to one's primary life work from which the individual gains significant participatory, creative, or productive satisfaction. These are not essential requirements of everyday living but have value in bringing purpose and fulfilment to the individual, his family, and his circle of friends. They involve multifaceted behaviour patterns and at times employ complex hierarchies of physical and mental activity. Avocational activities include such pursuits as hobbies, travel, recreation, socializing, and the like.

Occupation is the primary life work to which the individual devotes the major portion of his time, skill, energy, thought, and effort, deriving from it role and status, physical and mental satisfaction, and economic support. In addition to recognized modes of remunerative employment, occupation includes homemaking, being a student, retirement, and other primary life pursuits. In this classification all such pursuits engaged in at the time of onset of stroke are considered the primary occupation.

Placement

The goal of care is the ultimate return of the recovered patient to the optimal physical, mental, social, vocational, and economic condition consistent with his remaining performance abilities and requirements for continued health maintenance. Suitable physical environment, living arrangements, and social, economic, and health care support must be selected. Medical status, performance ability, and conditions in available environmental settings are variables that determine the placement status. Among the critical considerations leading to optimal placement are the suitability of the architecture and surrounding environment, the kind and frequency of personal supervision and direction required, and the degree of continuing medical-nursing care required. Possibilities for placement vary widely but may include placement in full competitive employment, placement in sheltered or selected work situations, resumption of homemaker duties, retirement from the work market, independent living in a personal residence with full responsibility for its management, home care with family supervision, home care with outside assistance, domiciliary care, convalescent care, custodial care, or continued rehabilitation centre or hospital care.
Patient performance and placement

A. Performance

Class I. No significant impairment - Patient is fully independent in activities of daily living (ADL), pursues usual avocational activities, and returns to previous living site and occupation without any changes.

Class II. Mildly impaired - Patient is semidependent (requiring some assistance) in activities of daily living, and/or subject to a slight restriction of avocational activities, and/or able to return to previous occupation with some changes.

Class III. Moderately impaired - Patient is semidependent (requiring assistance in the form of lifting) in activities of daily living, and/or subject to considerable restriction of avocational activities, and/or unable to return to previous occupation, having to seek selective occupation.

Class IV. Severely impaired - Patient is fully dependent in the conduct of activities of daily living, and/or unable to participate in avocational activities, and/or unable to carry out any occupation.

B. Placement

Class A. No limitation.

Class B. Mild limitation - requires occasional supervision, and/or modified environment, and/or occasional medical care.

Class C. Moderate limitation - requires much supervision, and/or physical assistance or outside help, and/or regularly available medical care.

Class D. Severe limitation - requires constant or nearly constant attendance and/or immediately available medical-nursing care.
PART IV. ANATOMY

Anatomical structures are considered in two major subdivisions: (A) blood vessels (arteries and arterial anastomoses important for collateral circulation, arterial anomalies, arterioles, veins, venules, and capillaries); and (B) central neural parenchyma (brain or spinal areas to which blood is supplied, or from which it is drained; peripheral and autonomic nervous systems are not included).

The nomenclature of Nomina Anatomica (NA) (1966) and the Standard Nomenclature of Diseases and Operations (SN) are used as much as possible. More detailed anatomy of some vessels and neural structures can be found in NA, e.g., thalamus, hypothalamus, brain stem, cerebellum, cranial nerves, spinal cord. SN lists left or right side only for certain structures. When these are of importance, but no specifically designated, the lower-case letters "l." and "r." will be used in this classification, indicating left and right, respectively. The lower-case letter "a." is used as an abbreviation for artery, "aa." for arteries, "v." for vein, "vv." for veins, and "s." for venous sinus.

Although conventional anatomical subdivisions are used as much as possible, there are some exceptions due to practical considerations, e.g., the subclavian artery is divided into three parts, which are different from the three or four parts of some anatomy texts. Not all known branches of a specific artery are included but only those considered important in cerebrovascular and spinovascular disease or those known to have important anastomoses. A strict order of arterial origin is not always followed, e.g., in the case of branches of the anterior cerebral artery. Arterial anastomoses are not separately listed but may be described by indicating specific arterial systems and branches involved.

Under "Brain and spinal cord", major subdivisions of "Hemisphere" and "Brain stem" have been introduced, as these anatomical terms are commonly used by clinicians and investigators working with cerebrovascular disease.

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A. Blood vessels

1. Arteries
   a. Ascending aorta
   b. Aortic arch
      (1) Brachiocephalic a. (innominate a.)
      (2) Common carotid a. (specify r. or l.)
      a) External carotid a. (specify r. or l.)
         1) Superior thyroid a.
         2) Lingual a.
         3) Facial a. (external maxillary)
         4) Occipital a.
            (a) Meningeal branch
         5) Posterior auricular a.
         6) Ascending pharyngeal a.
            (a) Posterior meningeal aa.
         7) Superficial temporal a.
         8) Maxillary a.
            (a) Middle meningeal a.
            (b) Infra-orbital a.
      b) Internal carotid a. (specify r. or l.)
         1) Carotid sinus portion (Part I)
            (from bifurcation to end of bulbous dilatation)
         2) Cervical portion (Part II)
            (from end of sinus portion to entry in petrous bone)
         3) Petrous portion (Part III)
            (from entry in petrous bone to emergence from petrous bone)
            (a) Caroticotympanic aa.
            (b) Other inconstant branches
         4) Cavernous sinus portion (Part IV)
            (from emergence in petrous bone through cavernous sinus course)
            (a) Hypophyseal aa.
            (b) Semilunar aa.
            (c) Anterior meningeal a.
         5) Intracranial portion (Part V)
            (from emergence from cavernous sinus to trifurcation;
            also termed intradural portion, supraclinoid portion, etc.)
            (a) Ophthalmic a.
               (1) Central retinal a.
                  a. Nasal branches
                  b. Temporal branches
               (2) Ciliary aa.
               (3) Lacrimal a.
               (4) Supraorbital a.
               (5) Ethmoidal aa.
               (6) Medial palpebral aa.
               (7) Frontal aa.
               (8) Dorsal nasal a.
(b) Posterior communicating a.
(c) Anterior choroidal a.
(d) Anterior cerebral a.
   (1) Anterior communicating a.
   (2) Anteromedial (perforating) aa.
   (3) Medial striate a. or aa. (recurrent a. etc.)
   (4) Orbital a.
   (5) Frontopolar aa.
   (6) Callosomarginal a.
   (7) Pericallosal a.
(e) Middle cerebral a.
   (1) Lenticulostriate aa.
   (2) Orbitofrontal aa.
   (3) Anterior temporal a. or aa.
   (4) Ascending frontal aa.
      a. Pre-Rolandic aa.
      b. Rolandic aa.
   (5) Ascending parietal a. (post-Rolandic a.)
      a. Posterior parietal a. (including angular a.)
   (6) Parietotemporal aa.
   (7) Posterior temporal aa.
(3) Subclavian a. (specify r. or l.)
   a) Vertebral a.
      1) Proximal portion (Part I)
         (origin to entry in transverse foramen of sixth cervical
          vertebra or, in some cases, a lower or higher vertebra)
      2) Intra-osseous portion (Part II)
         (entry in C6 transverse foramen to emergence from C2
          transverse foramen)
         (a) Spinal aa. (specify segments).
         (b) Muscular aa.
      3) Distal extracranial portion (Part III)
         (emergence from C2 transverse foramen, course around the
          posterior aspect of the atlas (C1) to point of entry in
          the dura)
         (a) Muscular aa.
      4) Intracranial portion (Part IV)
         (entry in dura to juncture with basilar a.)
         (a) Anterior spinal a.
         (b) Posterior inferior cerebellar a.
            (1) Medullary aa.
            (2) Medial branch
            (3) Lateral branch
         (c) Posterior meningeal a.
         (d) Posterior spinal a.
b) **Basilar a.**
   1) Anterior inferior cerebellar a.
   2) Internal auditory a.
   3) Pontine aa.
   4) Superior cerebellar a.
   5) Posterior cerebral a.
      (a) Posteromedial (perforating) aa.
      (b) Posterior choroidal aa.
      (c) Posterolateral a. (thalamic, thalamogeniculate aa.)
      (d) Anterior temporal a.
      (e) Posterior temporal a. (temporo-occipital)
      (f) Parieto-occipital a.
      (g) Calcarine a.

c) **Thyrocervical trunk**
   1) Thyroid branches
   2) Cervical branches
d) **Internal mammary a.**

c. **Thoracic aorta**
   (1) Intercostal aa.
      a) Spinal aa. (specify segments)
   (2) Subcostal aa.
      a) Spinal aa. (specify segments)
         (anterior spinal artery listed above)
d. **Abdominal aorta**
   (1) Lumbar aa.
      a) Spinal aa. (specify segments)
         (anterior spinal artery listed above)

2. **Arterial collateral circulation**
   a. Extracranial-extracranial
      (specify by arterial branches involved)
b. Extracranial-intracranial
      (specify by arterial branches involved)
c. Intracranial-intracranial
   (1) Circle of Willis (specify arterial branches)
   (2) Leptomeningeal anastomoses of anterior, middle, and posterior
cerebral aa. and superior, anterior inferior, and posterior
der inferior cerebellar aa. (see above)
d. **Persisting embryonic arteries**
   (1) Otic a.
   (2) Trigeminal a. (carotid-basilar anastomosis)
   (3) Hypoglossal a.

3. **Arterial anomalies**
   a. **Aortic arch**
      (1) Common origin of left common carotid a. and brachiocephalic a.
      (2) Origin of left common carotid a. from brachiocephalic a.
      (3) Origin of left vertebral a. from aortic arch
      (4) Origin of right subclavian a. distal to left subclavian a.
         (aberrant right subclavian a.)
      (5) Other
b. Circle of Willis
   (1) Posterior communicating aa.
      a) Bilaterally small, hypoplastic, or atretic
      b) Unilaterally small, hypoplastic, or atretic (specify side)
   (2) Anterior communicating a.
      a) Absent
      b) Bifid or multiple and small
   (3) Posterior cerebral a. origin from internal carotid a. (specify if r., l., or bilateral)
   (4) Other

c. Leptomeningeal branches of major cerebral aa.

4. Veins
   a. Internal cerebral vv.
      (1) Septal vv.
      (2) Choroidal vv.
      (3) Thalamostriate (striothalamic) vv.
      (4) Internal cerebral vv.
      (5) Great cerebral v. (v. of Galen)
   b. External cerebral vv.
      (1) Superior cerebral vv.
      (2) Superficial middle cerebral v. (superficial Sylvian)
         a) Superior anastomotic v. (Trolard)
         b) Inferior anastomotic v. (Labbé)
      (3) Inferior cerebral vv.
         a) Basal v. (Rosenthal)
         b) Deep middle cerebral v. (deep Sylvian v.)
         c) Anterior cerebral vv.
         d) Striate vv.
   c. Cerebellar vv.
      (1) Superior cerebellar vv.
      (2) Inferior cerebellar vv.
   d. Dural venous sinuses
      (1) Superior sagittal s.
      (2) Inferior sagittal s.
      (3) Transverse s.
      (4) Sigmoid s.
      (5) Straight s.
      (6) Occipital s.
      (7) Confluence of the sinuses (torcular Herophili)
      (8) Cavernous s.
         a) Sphenoparietal s.
         b) Superior ophthalmic v.
         c) Inferior ophthalmic v.
      (9) Inter cavernous s. (circular s.)
      (10) Basilar plexus
      (11) Superior petrosal s.
      (12) Inferior petrosal s.
         a) Internal auditory v.
e. Emissary veins
   (1) Mastoid emissary v.
   (2) Parietal emissary v.
   (3) Plexus of hypoglossal canal
   (4) Condyloid emissary v.
   (5) Plexus of the foramen ovale
   (6) Plexus of internal carotid vv.
   (7) Occipital emissary v.

f. Diploic veins
   (1) Frontal
   (2) Anterior temporal
   (3) Posterior temporal
   (4) Occipital

g. Internal jugular v.
h. External jugular v.
i. Subclavian v.
j. Brachiocephalic (innominate) v.
k. Superior vena cava

l. Spinal vv.
   (1) Anterior spinal v.
      a) Sulcal vv.
   (2) Posterior spinal vv.
   (3) Vertebral venous plexuses

m. Inferior vena cava

B. Brain and spinal cord

1. Meninges
   a. Cranial dura mater (specify site according to lobes of brain or cranial bony fossae)
      (1) Epidural space, cerebral
      (2) Subdural space, cerebral
   b. Spinal dura mater
      (1) Epidural space, spinal
      (2) Subdural space, spinal
   c. Leptomeninges (arachnoid and pia mater)
      (1) Subarachnoid space, cerebral
      (2) Subarachnoid space, spinal

2. Brain
   a. Hemisphere (specify r. or l.)
      (1) Frontal lobe
         a) Frontal pole
         b) Precentral gyrus
         c) Superior frontal gyrus
         d) Middle frontal gyrus
         e) Inferior frontal gyrus
            (1) Opercular portion
            (2) Triangular portion
            (3) Orbital portion
         f) Orbital (orbitofrontal) gyri
         g) Gyrus rectus
(2) Temporal lobe  
   a) Temporal pole  
   b) Superior temporal gyrus  
      (1) Transverse gyri (Heschl)  
   c) Middle temporal gyrus  
   d) Inferior temporal gyrus

(3) Parietal lobe  
   a) Postcentral gyrus  
   b) Superior parietal lobule  
   c) Inferior parietal lobule  
   d) Supramarginal gyrus  
   e) Angular gyrus

(4) Occipital lobe  
   a) Occipital pole  
   b) Lateral occipital gyri  
   c) Calcarine gyri (area striata)  
   d) Cuneus

(5) Central white matter (specify by lobe)

(6) Internal capsule  
   a) Anterior limb  
   b) Genu  
   c) Posterior limb

(7) Thalamus

(8) Inferior and medial hemisphere surfaces  
   a) Hippocampal gyrus  
   b) Corpus callosum  
   c) Other (specify)

(9) Corpus striatum  
   a) Caudate nucleus  
   b) Lentiform nucleus  
      (1) Putamen  
      (2) Globus pallidus

(10) Hypothalamus

(11) Insula

(12) Other (specify)

b. Brain Stem  
   (1) Midbrain  
      a) Tectum  
      b) Tegmentum  
      c) Cerebral peduncle

   (2) Pons  
      a) Tegmentum  
      b) Basis

   (3) Medulla oblongata  
      a) Dorsal  
      b) Ventral
c. Cerebellum
   (1) Vermis
   (2) Cerebellar hemisphere
   (3) Cerebellar nuclei
   (4) Superior cerebellar peduncle
   (5) Middle cerebellar peduncle
   (6) Inferior cerebellar peduncle

d. Cranial nerves
   (1) Olfactory n. (I)
   (2) Optic n. (II)
      a) Optic disk
      b) Prechiasmatic portion
      c) Optic chiasm
      d) Optic tract
   (3) Oculomotor n. (III)
   (4) Trochlear n. (IV)
   (5) Trigeminal n. (V)
      a) Ophthalmic n.
      b) Maxillary n.
      c) Mandibular n.
      d) Gasserian ganglion
   (6) Abducens n. (VI)
   (7) Facial n. (VII)
   (8) Acoustic n. (VIII) (vestibulocochlear n.)
      a) Cochlear division
      b) Vestibular division (labyrinthine division)
   (9) Glossopharyngeal n. (IX)
   (10) Vagus n. (X)
   (11) Accessory n. (XI) (spinal accessory n.)
   (12) Hypoglossal n. (XII)

e. Cerebral ventricles
   (1) Lateral ventricle (specify r. or l.)
   (2) Third ventricle
      a) Choroid plexus
   (3) Cerebral aqueduct (Sylvius)
   (4) Fourth ventricle.

3. Spinal cord
   a. Gray matter (specify level)
   b. White matter (specify level)
   c. Dorsal roots (specify level)
   d. Ventral roots (specify level)
PART V. PATHOPHYSIOLOGICAL MECHANISMS

Clinicians have continually to consider mechanisms as they watch events unfold in the course of an illness. If the mechanisms (pathogenesis) of a focal abnormality of blood supply to the brain can be determined, corrective measures can often be instituted. There are processes that produce no specific tissue change which can ultimately be identified by the pathologist. These include such common conditions as thrombosis with lysis, embolism with fragmentation, vasospasm, hypotension, abnormalities or cardiac rhythm, and many others. Their consideration is vital in outlining the mechanisms in a specific case. In certain instances, it may be necessary to list more than one mechanism, e.g., cerebral embolism from a cardiac source, hypotension, and myocardial infarction.

Subsection E - "Possible predisposing factors" - is included to record the role of these abnormalities as "risk factors".

As the pathologist works with the problem of classifying autopsy findings, he may find it helpful to be able to use this section on pathophysiological mechanisms in instances where the clinical record contains evidence of such factors as cholesterol retinal emboli and atrial fibrillation, while the autopsy findings do not indicate any cause for a haemorrhagic cerebral infarction. This section may stimulate a more detailed search for evidence of some causes of stroke.

A. Primary abnormalities of cerebral circulation (specify whether transient or persistent)
   1. Thrombosis
      a. Lysis
      b. Recannulation
      c. Collateral flow
   2. Embolism
      a. Intraluminal source
      b. Cardiac source
      c. Other source
   3. Haemorrhage (specify - see Part IV. Anatomy and Part VI. Pathology)
   4. Compression
      a. Change of position of head, neck, or arm
         (1) Osteoarthritis of cervical vertebrae
         (2) Atlas to axial joint
         (3) Fibrous bands
         (4) Kinks
         (5) Loops
         (6) Fracture
      b. Expanding mass
      c. External forces
         (1) Manipulation
         (2) Surgery
      d. Cerebral oedema
      e. Acceleration
5. **Vasospasm**
   a. Post-traumatic
   b. Migraine
   c. Post-intracranial haemorrhage (specify subarachnoid, intracerebral)
   d. Post-subarachnoid haemorrhage
   e. Hypertension (e.g., phaeochromocytoma, acute renal disease, eclampsia)
   f. Manipulation (e.g., surgery)
   g. Embolism
   h. Drugs (e.g., ergot derivatives, methysergide maleate, etc.)
   i. Other

6. **Direction of flow**
   a. Reversal
   b. Shunts

7. **Alteration in slow rate and/or volume** (as seen in serial arteriogram)
   (specify decrease or increase)
   a. Focal
   b. General

8. **Dissection of arterial wall** (specify artery)
   a. Transient disturbance of flow with subsequent restoration

9. **Associated with arteriography**

B. **Abnormalities of general circulation**

1. **Hypotension** - define
   (specify whether transient or persistent)
   a. Cardiac abnormalities
      (1) Abnormality of rate
      (2) Abnormality of rhythm
      (3) Conduction defects
      (4) Myocardial impairment
         a) Infarct
         b) Myocarditis
         c) Myocardial degenerative disease
      (5) Valvular disease including prosthesis
      (6) Pericardial disease and/or effusion
   b. Reflex
      (1) Carotid sinus hypersensitivity
      (2) Vasovagal
   c. Shock (specify cause)
   d. Endocrine (specify cause)
   e. Blood loss
      (1) Haemorrhage
      (2) Pooling (specify region)
   f. Orthostatic hypotension
   g. Valsalva's manoeuvre
   h. Neurological disease
      (1) Central nervous system (specify site)
      (2) Peripheral neuropathy
      (3) Autonomic nervous system
i. Iatrogenic
   (1) Medication
   (2) Post-sympathectomy
   (3) Post-reconstruction vascular surgery (specify site)
j. Undetermined
2. Hypertension (specify whether transient or persistent) See Appendix II for grading of retinal changes - Wagener & Keith classification
   a. Unknown cause, including essential hypertension
   b. Endocrine
   c. Medication
   d. Renal
   e. Emotional
   f. Physical exertion
   g. Toxaemia of pregnancy

C. Alterations in blood
1. Viscosity
   a. Dehydration
   b. Overhydration
   c. Other (specify)
2. Cellular constituents
   a. Erythrocytes
      (1) Anaemia
      (2) Polycythaemia
      (3) Haemoglobinopathy
         a) Sicklaemia
         b) Haemoglobin C
   b. Leukocytes
   c. Thrombocytes (e.g., thrombocytosis, thrombocytopenia)
3. Clotting defects
   a. Hypercoagulability (specify cause when known, including medication)
   b. Hypocoagulability (specify cause when known, including medication)
4. Proteins
   a. Macroglobulins (specify type if known)
   b. Cryoglobulins
   c. Hyperfibrinogenaemia
   d. Other
5. Lipids
   a. Cholesterol
   b. Triglycerides
   c. Lipoprotein
   d. Other
6. Glucose
   a. Hypoglycaemia
   b. Hyperglycaemia
7. Blood gases
   a. Oxygen
      (1) Hypoxia
         a) Hypoventilation
            (1) Musculoskeletal disease
            (2) Pulmonary disease
            (3) Neurogenic
         b) Environmental $O_2$ deficiency
(2) Hyperoxia
  b. Carbon dioxide
(1) Hypercapnia
   a) Hypoventilation
      (1) Musculoskeletal disease
      (2) Pulmonary disease
      (3) Neurogenic
   b) Environmental CO₂ excess
(2) Hypocapnia
   a) Hyperventilation
      (1) iatrogenic
      (2) Psychophysiological
      (3) Neurogenic
   c. Carbon monoxide
   d. Nitrogen
   e. Other gases
  8. Electrolytes
     a. Hyponatraemia
  9. Hydrogen ion-content
 10. Others including radiopaque material

D. Alterations of metabolic demand
  1. Thermal changes
     a. Hypothermia
     b. Hyperthermia
  2. Convulsion
  3. Medications (specify, e.g., barbiturates, etc.)

E. Possible predisposing factors
  1. Hypertensive disease
  2. Diabetes mellitus
  3. Cardiac disease
  4. Hyperlipidaemia
  5. Genetic
  6. Cigarette smoking
  7. Hyperuricaemia
  8. Obesity
  9. Drugs
 10. Endocrine (? hypothyroidism, etc.)
 11. Other

F. Unknown
PART VI. PATHOLOGY

Pathological alterations are considered in two major subdivisions: (A) blood vessels (arteries, arterioles, capillaries, venules, veins, and combined arterial, venous, and capillary lesions); and (B) neural parenchymatous lesions.

The etiological classification of the 1961 Standard Nomenclature of Diseases and Operations, (SN)\(^1\) and the 1958 Classification and Outline of Cerebrovascular Disease\(^2\) are used whenever possible. Some additions, deletions, and modifications have been made, but the main sections can be compared with the SN and the 1958 classification. In the section dealing with lesions of capillaries, the word "petechiae" is used since it connotes haemorrhages small enough to be recognized as presumably of capillary origin, although such haemorrhagic lesions may also be of arteriolar and venular origin.

The World Federation of Neurology code for grading atherosclerosis is included as an Appendix.

A. Pathological alterations in vessels
   1. Arteries (and arterioles, when applicable)
      a. Congenital, developmental, and inherited lesions
         (1) Absence of artery (aplasia)
         (2) Hypoplasia of artery
         (3) Anomaly of artery (fetal form)
         (4) Anomaly of artery (unspecified)
         (5) Redundancy (loops), dilatation, elongation; congenital, of artery.
         (6) Aneurysms, congenital
            a) Ruptured aneurysm, congenital
         (7) Genetically determined defects in arteries
            a) Marfan's syndrome (arachnodactyly)
            b) Ehlers-Danlos syndrome
            c) Pseudoxanthoma elasticum
            d) Others


\(^2\)Neurology, 8:395-434 (1958)
b. Inflammatory lesions (arteritides)
   (1) Infectious arteritides
      a) Tuberculous arteritis
      b) Pyogenic arteritis
      c) Syphilitic arteritis
      d) Septic embolism
      e) Infectious arteritides of other types
      f) Complications of infectious arteritides
         (1) Thrombosis
         (2) Haemorrhage
         (3) Scar
         (4) Aneurysm (mycotic aneurysm)

   (2) Non-infectious arteritides
      a) Periarteritis nodosa
      b) Lupus erythematosus disseminatus
      c) Thrombotic microangiopathy (thrombotic thrombopenia, TTP)
      d) Rheumatic arteritis
      e) Cranial arteritis (temporal arteritis)
      f) Pulseless disease (Takayasu's disease)
      g) Non-infectious arteritides of other types (including
         allergic or hypersensitivity arteritis, etc.)
      h) Complications of non-infectious arteritides
         (1) Thrombosis
         (2) Haemorrhage
         (3) Scar
         (4) Aneurysm

c. Trauma and physical agents
   (1) Trauma to artery due to external forces, bony anomalies,
       fractures, dislocations, degenerative bone disease, fibrosis
      a) Local effects
         (1) Extramural haemorrhage
         (2) Intramural haemorrhage
            a) With dissection
            (3) Thrombosis
            (4) Stenosis
         b) Remote effects
            (1) Fat embolization
            (2) Bone marrow embolization
            (3) Air embolization

   (2) Trauma due to angiography
      a) Intramural haemorrhage
         (1) With dissection
      b) Extramural haemorrhage
      c) Rupture of atherosclerotic plaque
         (1) Atheromatous embolization
      d) Thrombosis
      e) Embolization due to foreign materials (cotton fibres, etc.)
(3) Trauma due to cardiac catheterization and other intra-arterial diagnostic and therapeutic procedures
   a) Intramural haemorrhage
   b) Rupture of atherosclerotic plaque
      (1) Atheromatous embolization
   c) Thrombosis
   d) Embolization due to foreign materials (cotton fibres, etc.)
(4) Trauma due to surgery
   a) Reconstructive and reparative arterial surgery
      (1) Embolization by thrombotic fragments
      (2) Atheromatous or tissue embolization
      (3) Embolization due to foreign materials (cotton fibres, etc.)
      (4) Thrombosis
      (5) Thrombosis in arterial anastomosis
      (6) Thrombosis in arterial grafts
         a. Natural grafts
         b. Synthetic grafts
      (7) Aneurysm
      (8) Stenosis
   b) Other surgical procedures
      (1) Occlusion (ligation, clamp, etc.)
      (2) Rupture or accidental division
      (3) Intramural haemorrhage
         a. With dissection
      (4) Rupture of atherosclerotic plaque
         a. Atheromatous embolization
      (5) Thrombosis
(5) Trauma of artery due to brain herniation
   (transventorial, subfalcial, foramen magnum, etc.)
   a) Thrombosis
   b) Haemorrhage
(6) Radiation effects
   a) Post-irradiation thrombosis
   b) Post-irradiation scar (fibrosis)
d. Arterial lesions due to blood dyscrasias
   (1) Thrombosis
      a) Polycythaemia vera
      b) Secondary polycythaemia (specify cause)
      c) Haemoglobinopathy (specify type)
      d) Other
   (2) Haemorrhage
      a) Leukaemia (specify type)
      b) Haemoglobinopathy (specify type)
      c) Hypoprothrombinaemia
      d) Other
e. Arterial lesions associated with metabolic abnormalities
   (1) Atherosclerosis (See VI.A.1.i.i.)
      a) Familial hypercholesterolaemia
      b) Other (including diabetes mellitus, hyperthyroidism, etc.)
f. Arterial lesions associated with drug toxicity, idiosyncrasy, and unknown effects
(1) Thrombosis
   a) Dehydration
(3) Calcification
   a) Hypoparathyroidism

(2) Haemorrhage
   a) Anticoagulants (specify)
   b) Sympathomimetic amines (specify)
   c) Heavy metals (arsenic, etc., specify)

(3) Calcification
   a) Hypervitaminosis D

g. Arterial embolism due to cardiac disease and diseases of extracerebral vessels
(1) Cardiac arrhythmias (specify basic disease)
(2) Valvular disease (endocarditis)
   a) Septic (specify)
   b) Aseptic (specify)
(3) Myocardial infarction
(4) Atherosclerosis plus thrombosis
(5) Ulcerated atheroma
(6) Paradoxical embolism
   a) Congenital heart disease
   b) Systemic venous thrombosis
      (1) Aseptic
      (2) Septic
(7) Pulmonary venous thrombosis

h. Arterial lesions associated with neoplastic disease
(1) Thrombosis associated with neoplasm (specify)
   a) Intracranial neoplasm (primary or secondary)
   b) Extracranial neoplasm
(2) Haemorrhage associated with neoplasm (specify)
   a) Intracranial neoplasm (primary or secondary)
   b) Extracranial neoplasm
(3) Embolism associated with neoplasm (specify)
   a) Intracranial (primary or secondary)
   b) Extracranial neoplasm

i. Arterial lesions due to unknown causes
(1) Atherosclerosis
   a) Atherosclerotic stenosis
      (1) Thrombosis
   b) Atherosclerotic occlusion
      (1) Thrombosis
   c) Ulceration of atherosclerotic plaque
      (1) Atheromatous embolization
   d) Atherosclerotic dilatation, ectasia, or aneurysm (fusiform)
      (1) With rupture

*Uncertain
e) Atherosclerotic haemorrhage
f) Intramural haemorrhage in plaque
g) Calcification of atherosclerotic plaque

(2) Möncheberg's sclerosis (calcific medial arteriosclerosis)
(3) Mineralization (ferrugination, calcification, siderocalcific change) in parenchymal arteries (and arterioles) of central nervous system (CNS)
   a) Non-familial
   b) Familial calcification ("of basal ganglia")
   c) Other
(4) Fibromuscular dysplasia
(5) Thromboangiitis obliterans (Buerger's disease)*
(6) Haemorrhagic dissection of arterial wall
   a) Cystic medial necrosis of aorta
   b) Dissection of other arteries
(7) Amyloid (congophilic) angiopathy

j. Arterial and arteriolar lesions associated with hypertension (specify type, etiology, or associated disease)
(1) Atherosclerosis (arteriosclerosis) (See VI.A.1.i.1)
(2) Hyaline medial degeneration with or without fibrinoid change
   a) With millary intracerebral aneurysm*
(3) Other degenerative changes (specify)

2. Veins (and venules, when applicable)
   a. Congenital, developmental, and inherited lesions
      (1) Anomaly of veins (fetal form)
      (2) Anomaly of veins (unspecified)
      (3) Aneurysm of veins, congenital (phlebectasia)
         a) Ruptured aneurysm, congenital
   b. Inflammatory lesions (phlebitides)
      (1) Infectious phlebitides
         a) Septic phlebitis
            (1) With thrombosis
         b) Tuberculous phlebitis
      (2) Non-infectious phlebitides
         a) Thrombotic microangiopathy (thrombotic thrombopenial, TTP)
   c. Trauma and physical agents
      (1) Trauma to vein due to external forces, anomalies, fractures, dislocations, degenerative bone disease, fibrosis.
         a) Local effects
            (1) Extramural haemorrhage
            (2) Intramural haemorrhage
               a. With dissection
            (3) Thrombosis
         b) Remote effects
            (1) Fat embolization
            (2) Bone marrow embolization
            (3) Air embolization

* Uncertain
(2) Trauma due to angiography
   a) Intramural haemorrhage
      (1) With dissection
   b) Extramural haemorrhage
   c) Thrombosis
      (1) With embolization
   d) Embolization due to foreign materials (cotton fibres, etc.)
(3) Trauma due to intravenous procedures, diagnostic and therapeutic
   a) Intramural haemorrhage
   b) Extramural haemorrhage
   c) Thrombosis
      (1) With embolization
   d) Embolization due to foreign materials (cotton fibres, etc.)
(4) Trauma due to surgery
   a) Surgery involving veins
      (1) Embolization by thrombotic fragments
      (2) Tissue embolization
      (3) Embolization due to foreign materials (cotton fibres, etc.)
      (4) Thrombosis
      (5) Thrombosis in venous anastomosis
      (6) Thrombosis in venous grafts
         a) Natural grafts
         b) Synthetic grafts
   b) Other surgical procedures
      (1) Occlusion (ligation, clamp, etc.)
      (2) Rupture or accidental division
      (3) Intramural haemorrhage
      (4) Thrombosis
         a) With embolization
(5) Trauma of veins due to brain herniation (transstentorial, subfalcial, foramen magnum, etc.)
   a) Thrombosis
   b) Haemorrhage
(6) Radiation effects
   a) Post-irradiation thrombosis
   b) Post-irradiation scar (fibrosis)
   d. Venous lesions due to blood dyscrasias
(1) Thrombosis
   a) Polycthaemia vera
   b) Secondary polychaemia (specify cause)
   c) Haemoglobinopathy (specify type)
   d) Other
(2) Haemorrhage
   a) Leukaemia (specify type)
   b) Haemoglobinopathy (specify type)
   c) Hypoprothrombinaemia
   d) Other
e. Venous lesions associated with metabolic abnormalities
   (1) Thrombosis
      a) Dehydration
   (2) Calcification
      a) Hypoparathyroidism
f. Venous lesions associated with drug toxicity, idiosyncrasy, and unknown effects.
   (1) Thrombosis
      a) Progestational agents*
      b) Others (specify)
   (2) Haemorrhage
      a) Anticoagulants (specify)
      b) Heavy metals (specify)
   (3) Calcification
      a) Hypervitaminosis D
g. Venous lesions associated with neoplastic disease
   (1) Thrombosis associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   (2) Haemorrhage associated with neoplasm
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
   (3) Embolism associated with neoplasm (specify)
      a) Intracranial neoplasm (primary or secondary)
      b) Extracranial neoplasm
h. Venous lesions due to unknown cause
   (1) Phlebosclerosis
   (2) Phlebolith
   (3) Mineralization (ferrugination, calcification, siderocalcific change) in parenchymal vein (and venules of CNS)
      a) Nonfamilial
      b) Familial calcification ("of basal ganglia")
      c) Other
   (4) Varix
3. Capillaries
   a. Inflammatory lesions
      (1) Infectious capillary purpura
   (2) Non-infectious capillary lesions
      a) Thrombotic microangiopathy (thrombotic thrombopenia, TTP)
      b) Allergic capillary purpura
b. Trauma and physical agents
   (1) External trauma
      a) Petechiae
   (2) Trauma by adjacent structures
      a) Petechiae
   (3) Remote effects of external trauma
      a) Petechiae due to fat embolization
      b) Petechiae due to bone marrow embolization
      c) Petechiae due to air embolization

* Uncertain
(4) Trauma due to angiography
   a) Petechiae due to radiopaque contrast medium
   b) Capillary thrombosis due to radiopaque contrast medium
   c) Petechiae due to atheromatous embolization
   d) Petechiae due to embolization by foreign material (cotton fibres, etc.)
(5) Trauma due to cardiac catheterization and other diagnostic and therapeutic intravascular procedures
   a) Petechiae due to atheromatous embolization
   b) Petechiae due to embolization by foreign materials (cotton fibres, etc.)
(6) Trauma due to surgery
   a) Petechiae
(7) Trauma of capillaries due to brain herniation (transventorial, subfalcial, foramen magnum, etc.)
   a) Petechiae
(8) Heat stroke (sunstroke)
   a) Petechiae with heat stroke
(9) Radiation effects
   a) Post-irradiation petechiae
   b) Post-irradiation thrombosis
   c) Post-irradiation fibrosis
c. Capillary lesions due to blood dyscrasias
(1) Thrombosis
   a) Polycythaemia vera
   b) Secondary polycythaemia (specify cause)
   c) Haemoglobinopathy (specify type)
   d) Other
(2) Petechiae
   a) Haemophilia
   b) Leukaemia (specify type)
   c) Haemoglobinopathy (specify type)
   d) Hypoprothrombinaemia
   e) Other
d. Capillary lesions associated with metabolic abnormalities
(1) Thrombosis
   a) Dehydration
(2) Petechiae (capillary purpura) due to metabolic disturbance (specify)
(3) Calcification
   a) Hypoparathyriodism
   b) Other
(4) Capillary proliferation
   a) Hyperoxia
   b) Hypoxia
e. Capillary lesions associated with drug toxicity, idiosyncrasy, and unknown effects.
(1) Thrombosis
   a) Progestational agents*
   b) Other (specify)

*Uncertain
(2) Petechiae
   a) Allergic capillary purpura (See V.C.2.a.3)
      (specify drug or chemical)
   b) Anticoagulants (specify)
   c) Sympathomimetic amines (specify)
   d) Heavy metals (arsenic, etc., specify)
(3) Calcification
   a) Hypervitaminosis D
f. Capillary lesions due to cardiac disease and diseases of extracerebral vessels
   (1) Petechiae with embolism (See V.A.2.c.)
g. Capillary lesions associated with neoplastic disease
   (1) Thrombosis associated with neoplasm (See V.A.8.)
   (2) Petechiae (haemorrhage) (See V.A.8.)
h. Capillary lesions due to unknown cause
   (1) Idiopathic brain purpura
   (2) Mineralization (ferrugination, calcification, siderocalcific change) in capillaries of CNS
      a) Nonfamilial
      b) Familial calcification ("of basal ganglia")
      c) Other
i. Capillary lesions associated with hypertension (specify type, etiology, or associated disease)
   (1) Hyalinization and fibrosis of capillary
4. Combined arterial, venous, and capillary abnormalities
   a. Congenital, developmental, and inherited lesions
      (1) Arteriovenous fistula, congenital
      (2) Arteriovenous fistula due to ruptured congenital aneurysm
      (3) Vascular malformations (hamartomas, angiomas)
         a) Telangiectasis
         b) Cavernous (venous) angioma (haemangioma)
         c) Arteriovenous angioma
      (4) Vascular malformations with the phakomatoses
         a) Lindau's disease
         b) Sturge-Weber's disease
         c) Others
   b. Inflammatory lesions
      (1) Infectious
         a) Arteriovenous aneurysm due to infection
   c. Trauma
      (1) Trauma due to external forces, bony anomalies, fractures, dislocations, degenerative bone disease, fibrosis.
         a) Arteriovenous fistula, traumatic.
   d. Metabolic abnormalities
      (1) Avitaminosis (specify type)
   e. Lesions due to unknown causes
      (1) Arteriovenous fistula due to ruptures
      (2) Arteriosclerotic aneurysm
B. Pathological alterations in brain

1. Infarction (pale, haemorrhagic, and mixed)
   a. Without vessel stenosis or occlusion
   b. With arterial stenosis or occlusion due to:
      (1) Congenital, developmental, and inherited lesions of arteries (See VI.A.1.a.)
      (2) Inflammatory lesions of arteries (See VI.A.1.b.)
      (3) Trauma and physical agents (See VI.A.1.c.)
      (4) Blood dyscrasias (See VI.A.1.d.)
      (5) Metabolic abnormalities (See VI.A.1.e.)
      (6) Drugs, etc. (See VI.A.1.f.)
      (7) Cardiac and extracerebral vessel disease (See VI.A.1.g.)
      (8) Neoplastic disease (See VI.A.1.h.)
      (9) Unknown causes (chiefly atherosclerosis) (See VI.A.1.i.)
      (10) Hypertension (See VI.A.1.j.)
      (11) Other
   c. With venous stenosis or occlusion due to:
      (1) Congenital, developmental, and inherited lesions of veins (See VI.A.2.a.)
      (2) Inflammatory lesions (See VI.A.2.b.)
      (3) Trauma and physical agents (See VI.A.2.c.)
      (4) Blood dyscrasias (See VI.A.2.d.)
      (5) Metabolic abnormalities (See VI.A.2.e.)
      (6) Drugs, etc. (See VI.A.2.f.)
      (7) Neoplastic disease (See VI.A.2.g.)
      (8) Unknown causes (See VI.A.2.h.)
   d. With capillary lesions (See VI.A.3.)
   e. With combined arterial, venous, and capillary lesions (See VI.A.4.)

2. Haemorrhage (intracranial, intracerebral, etc.)
   a. Without identification of vessel type
   b. Of arterial origin
      (list as under A "Pathological alterations in vessels", or use anatomical and etiological terms as follows)
      (1) Anatomical site
         a) Intracerebral
            (1) With hypertension
            (2) Without hypertension
         b) Subarachnoid
         c) Subdural
         d) Intraventricular
      (2) Etiology
         a) Congenital, developmental, and inherited lesions of arteries (See VI.A.1.a.)
         b) Inflammatory lesions of arteries (See VI.A.1.b.)
         c) Trauma and physical agents (See VI.A.1.c.)
         d) Blood dyscrasias (See VI.A.1.d.)
         e) Metabolic abnormalities (See VI.A.1.e.)
         f) Drugs, etc. (See VI.A.1.f.)
g) Cardiac and extracerebral vessel disease
   (See VI.A.1.g.)

h) Neoplastic disease (See VI.A.1.h.)

i) Unknown causes (See VI.A.1.i.)

j) Hypertension (See VI.A.1.j.)

k) Other

c. Of venous origin
   (1) Congenital, developmental, and inherited lesions of veins (See VI.A.2.a.)
   (2) Inflammatory lesions (See VI.A.2.b.)
   (3) Trauma and physical agents (See VI.A.2.c.)
   (4) Blood dyscrasias (See VI.A.2.d.)
   (5) Metabolic abnormalities (See VI.A.2.e.)
   (6) Drugs, etc. (See VI.A.f.)
   (7) Neoplastic disease (See VI.A.2.g.)
   (8) Unknown causes (See VI.A.2.h.)

d. With capillary lesions (See VI.A.3.)

e. With combined arterial, venous, and capillary lesions
   (See VI.A.4.)
APPENDIX I

THE WORLD FEDERATION OF NEUROLOGY CODE FOR GRADING ATHEROSCLEROSIS

Grade 1 +
Opacity involving only a small part of the vessel circumference. No lumen narrowing.

Grade 2 +
(A) A diffuse thin plaque that does not involve the entire vessel circumference, with minimal lumen narrowing.
(B) A small thick plaque that produces less than 25% lumen narrowing.

Grade 3 +
(A) A diffuse thin plaque involving the entire circumference of the vessel, with mild lumen narrowing.
(B) A localized thick plaque producing 25% to 50% lumen narrowing.

Grade 4 +
(A) A thick plaque involving the entire circumference of the vessel, with moderate or marked lumen narrowing (pipe-stem).
(B) A localized thick plaque resulting in more than 50% lumen narrowing.

1World Federation of Neurology (L. van Bogaert, ed.).
APPENDIX II

WAGENER & KEITH CLASSIFICATION OF RETINAL VASCULAR DISEASE

Group I: Retinal changes are minimal and consist of mild narrowing and mild sclerosis of arterioles.

Group II: Changes are somewhat more advanced; patient's general health is good.

Group III: Pronounced abnormality of small retinal vessels, and small artery may be obstructed; there are definitely localized narrowed areas in the retinal vessels, haemorrhages, and exudates. May be mild alternation of vision.

Group IV: Similar to Group III but with florid haemorrhages, retinal oedema, and sometimes swelling of the nerve head. Visual impairment is present, and neurological symptoms are common.

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1 Based on: Wagener, H.P. & Keith, N.M. Medicine, 18: 317-430 (1939)
APPENDIX III
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