CEREBROVASCULAR DISEASES:
PREVENTION, TREATMENT, AND
REHABILITATION

Report of a WHO Meeting
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CEREBROVASCULAR DISEASES:
PREVENTION, TREATMENT, AND
REHABILITATION

Report of a WHO Meeting

A meeting on cerebrovascular diseases was convened by WHO in Monaco from 25 to 29 May 1970. The meeting was opened by the representative of the Monaco Government, Mr Marquet, Director of the Health and Welfare Campaign. Dr L. Lataillade, Director of Health Services, WHO Regional Office for Europe, welcomed the participants on behalf of both the Director-General and the Director of the Regional Office for Europe.

The purposes of the meeting were as follows:

(1) to assess and evaluate the cerebrovascular disease problem in different areas, particularly in Europe;

(2) to stimulate the application of present knowledge in the treatment of cerebrovascular disease and the rehabilitation of patients;

(3) to review the resources necessary for the prevention of cerebrovascular disease and to recommend appropriate measures; and

(4) to emphasize the value of prevention and to promote community programmes directed thereat.

1. INTRODUCTION

WHO's programme in cardiovascular diseases is based on assessment of the present and future public health importance of these conditions. Its objective is to promote and assist prevention and control of the major cardiovascular diseases by applying available knowledge in heart disease control programmes, by promoting, co-ordinating, and conducting research on etiology, pathogenic mechanisms, and prevention, and by promoting communication and training in the field of cardiovascular diseases. Several meetings of expert committees and scientific groups created a basis for present programmes, which concentrate mainly on atherosclerosis and ischaemic heart disease, cardiomyopathies, and rheumatic fever and rheumatic heart disease.

Research on atherosclerosis and ischaemic heart disease has been given top priority, and studies promoted and co-ordinated by the Organization in
several parts of the world aim at advancing understanding of the etiology as well as at testing the value of preventive and control measures. In Europe, an intensive long-term programme initiated in 1968 includes several projects dealing with the assessment of the extent of ischaemic heart disease in the community, evaluation of methods of treatment, rehabilitation, and a long-term follow-up of patients with acute myocardial infarction. An intensive training programme for health personnel and education of the population are integral parts of the pilot programme, which should eventually expand to provide effective community control of cardiovascular diseases.

WHO programmes are described in more detail in *International work in cardiovascular diseases* (1969).1

Among the cardiovascular diseases, cerebrovascular diseases are the second most common cause of death. In 1964, plans were drawn up by WHO to choose places and methods for collecting, preparing, and processing post-mortem data on the causes and location of cerebral haemorrhage and ischaemia. Subsequently, prospective studies were initiated to determine the frequency of the main cerebrovascular lesions in populations with different frequencies of arterial hypertension and atherosclerotic heart disease. A pilot study began in 1968 in Fukuoka and Tokyo (Japan), Moscow, Riga, and Ryazan (USSR), and Prague (Czechoslovakia).

2. SIZE AND NATURE OF THE PROBLEM

2.1 Terms of reference

Cerebrovascular diseases are diseases of the central nervous system (the brain and spinal cord) of vascular origin. The term covers a wide range of clinical manifestations, varying from subarachnoid haemorrhage resulting from a rupture of berry aneurysm on the one hand to arteriosclerotic parkinsonism and dementia on the other. In the present report, only diseases of the brain that are manifested as cerebrovascular accident ("stroke") are discussed. The common denominator of all forms of stroke is the sudden onset of a focal neurological deficit due to a local disturbance in blood supply to the brain. The word "sudden" here refers to a period of time ranging from a few seconds to several hours or even, on very rare occasions, several days.

Whatever the basic pathological changes in the arterial system, e.g., congenital malformation, inflammation, and other acute or chronic diseases, disturbance of the brain circulation is caused by three morphological abnormalities, i.e., stenosis, occlusion, or rupture of the arteries.

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1 This and many other terms used in this report are defined in the glossary (Annex 3).
Dysfunction of the brain manifests itself by various neurological symptoms that are related to the extent and site of the area involved, to the rapidity with which the pathological process develops, and to the underlying condition. Cerebral arteries are interconnected by anastomoses, and arterial obstruction is not always followed by clinical symptoms.

2.2 Mortality statistics

Under the heading of cerebrovascular disease, the *International Classification of Diseases* (1965 revision) lists the following conditions:

- 430  (330) Subarachnoid haemorrhage
- 431  (331) Cerebral haemorrhage
- 432  Occlusion of pre-cerebral arteries
- 433  (332) Cerebral thrombosis
- 434  Cerebral embolism
- 435  (333) Transient cerebral ischaemia
- 436  Acute but ill-defined cerebrovascular disease
- 437  (334) Generalized ischaemic cerebrovascular disease
- 438  Other and ill-defined cerebrovascular disease

The association with arterial hypertension may be shown by using a fourth digit:
- .8 With hypertension (benign)
- .9 Without mention of hypertension

In spite of their known shortcomings, national mortality statistics give the broadest view of the problem.

According to the *World Health Statistics Annual* (1966), cerebrovascular diseases appeared within the top 10 causes of death in 54 out of 57 countries (or parts of countries) where statistics were available and ranked among the three leading causes of death in 40 countries. They accounted for 11.3% of the total deaths in these 57 countries, and were surpassed only by ischaemic heart disease and cancer of all sites. In 1966, cerebrovascular disease accounted for 12.5% of the total deaths in the countries of Europe, North America, and Oceania, and 8.2% in Africa, Asia, and Latin America.

In the majority of countries where cerebrovascular disease was among the top three causes of death, it accounted for roughly 20-30% of all deaths from cardiovascular disease in men and 30-40% in women.

The number of deaths from this cause is small below the age of 25, but increases rapidly with age, by roughly three times every 10 years, in the majority of countries (see accompanying graph, p. 8).

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* Corresponding rubrics from the 1955 revision of the ICD are shown in parentheses.
Death rates in the 55–64-year age group, for both sexes, are shown in Table 1. The highest rates of over 3 per thousand per year were reported from Japan, Trinidad and Tobago, and China (Taiwan); the lowest rates, less than 0.9 per thousand per year, were reported from Iceland, Sweden,
<table>
<thead>
<tr>
<th>Countries, in descending order of death rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0+</td>
</tr>
<tr>
<td>Japan</td>
</tr>
</tbody>
</table>
the Netherlands, Switzerland, the Philippines, Poland, the Dominican Republic, and Thailand. Death rates for other age groups follow a similar pattern in these countries.

Validated local data have confirmed that a large difference in mortality really exists. In the urban mortality studies of the Pan American Health Organization,¹ death certificates of all persons who died between the ages of 15 and 74 from 1962 to 1964 in 12 cities (mostly in the American continent) were reviewed and the causes of death re-examined. High mortality rates from cerebrovascular disease were reported in Ribeirão Preto, Brazil (0.81 per 1000) and in Santiago, Chile (0.65 per 1000), while low rates were found in Guatemala City (0.31 per 1000), and in San Francisco, USA, and Caracas, Venezuela (0.38 per 1000).

Although cerebrovascular diseases usually cause death among older people, more than half of the subjects who died in 1966 from cerebrovascular accidents in Mauritius, the Philippines, Singapore, the Dominican Republic, Guatemala, Jordan, the United Arab Republic, and Thailand were under 65.⁴

Differences in mortality rates have been found not only between countries but also between different areas within countries. In the USA, mortality from cerebrovascular disease among white persons is three times higher in the southern states along the eastern seaboard than in the south-western and Rocky Mountain states.⁶ In Japan, mortality is highest in the northern part of Honshu Island and gradually decreases towards the south-west of the island.⁷-⁸ Regional differences have also been reported in European countries.⁹

High mortality and morbidity from cerebrovascular diseases in negroes in the USA have been reported ⁶,¹⁰ The Japanese and Chinese also have very high mortality from this cause, both in absolute figures and in relation to ischaemic heart disease.

Jews who emigrated to Israel from Afro-Asian countries were found to have lower death rates from ischaemic heart disease, but slightly higher cerebrovascular disease mortality, than immigrants from Europe and America; this applied to both sexes and was particularly marked in young people.¹¹ Japanese immigrants to the USA have ischaemic heart disease more commonly than cerebrovascular lesions.¹² The data indicate that environmental and other factors are more significant than genetic influence; better understanding of these environmental, cultural, and behavioural factors is needed.

2.3 Trends in mortality

According to the World Health Statistics Report,¹³ the number of deaths from cerebrovascular accidents increased in most countries during the
period from 1955 to 1964. Predictably, the rate of increase was highest in
countries where the population growth is large, e.g., Hong Kong, Singapore,
Thailand, and Venezuela. Only a few European countries, e.g., Belgium
and Bulgaria, reported a similar increase. In most other countries of Europe,
North America, and Oceania, the rate of increase was rather small. The
number of deaths fell slightly in Sweden and Guatemala. Changes in the
age structure of the population might be largely responsible for these changes,
although some of the increase could be accounted for by changes in certifi-
cation procedures and greater accuracy in diagnosis.

2.4 Morbidity data

2.4.1 Prevalence

Information on this point is limited. Studies in the USA showed that
the prevalence of cerebrovascular accidents was 2.6 per 1000 for the total
population of Baltimore, and 21 per 1000 for persons aged over 65.14
In Hiroshima, Japan, the prevalence of all cerebrovascular accidents
among persons over 40 years of age was found to be 7.9 per 1000; for men
it was 12.6 per 1000 and for women 4.7 per 1000.15 Transient ischaemic
attacks were rare.

2.4.2 Incidence

The extent of cerebrovascular disease is best expressed in terms of the
number of new cases occurring in a unit of time (incidence), but this informa-
tion is lacking for most parts of the world. Reports from some areas are
presented in Table 2.

Although wide variation was noted between the study areas, the average
incidence rate was roughly 0.2-0.5% per year in middle-aged white popula-
tions in the USA.14,16 A mid-Missouri survey showed that negro men and
women had a higher incidence rate up to the age of 74 than the white
population.10 In South Carolina the figure was four times higher than in
Denver for men aged 45 to 54.

The incidence of transient cerebral ischaemia was reported as 1.1 per 1000
per year in a community of retired people in the USA.17

The incidence of cerebrovascular disease in the Uppsala area of Sweden18
was of the same order as that in a low-incidence area of the USA.

Studies carried out in Japan showed a higher incidence than in the USA
for both men and women. The highest incidence rate, 10.8 per 1000 among
persons aged over 40, was reported from Akita district, and a low rate,
3.4 per 1000, was reported from Osaka in the west of Japan.19 The inci-
dence in Hiroshima was 7.4 per 1000 for men and 4.1 per 1000 for women over the
age of 30.15
<table>
<thead>
<tr>
<th>Place</th>
<th>Date of study</th>
<th>Total number of new cases</th>
<th>Incidence per 1000 population per year</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>35-44 45-54 55-64 65-74 75-84 85+</td>
<td>35-44 45-54 55-64 65-74 75-84 85+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Average incidence rate</td>
<td>Average incidence rate</td>
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<tr>
<td>Japan a</td>
<td>1964-68</td>
<td>332</td>
<td>0.39 3.99 13.11 21.49 27.69 40.00</td>
<td>2.85 (9.40)</td>
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<tr>
<td>Akita</td>
<td></td>
<td>173</td>
<td>0.12 1.31 5.05 13.81 25.00 33.23</td>
<td>1.60 (4.20)</td>
</tr>
<tr>
<td>Osaka</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>USA a</td>
<td>1957-58</td>
<td>191</td>
<td>0.82 2.40 5.70 c 1.94 b</td>
<td>0.70 1.72 4.30 b</td>
</tr>
<tr>
<td>Denver</td>
<td>354</td>
<td></td>
<td>2.64 6.13 15.78 c 5.32 b</td>
<td>0.46 3.21 8.72 b</td>
</tr>
<tr>
<td>Georgia</td>
<td>792</td>
<td></td>
<td>1.18 3.29 6.92 c 2.90 b</td>
<td>0.83 2.11 4.32 c</td>
</tr>
<tr>
<td>Miami</td>
<td>1959-61</td>
<td>477</td>
<td>2.70 7.03 12.24 c 5.33 b</td>
<td>0.49 1.66 6.42 c</td>
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<tr>
<td>S. Carolina</td>
<td>705</td>
<td></td>
<td></td>
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<tr>
<td>Middlesex, Conn.</td>
<td>1957-58</td>
<td>191</td>
<td>1.2 4.8 11.9 24.3 35.3 2.2</td>
<td>0.2 1.0 3.5 7.2</td>
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<tr>
<td>Missouri</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>White</td>
<td>1964-65</td>
<td>163</td>
<td>0.3 3.9 7.4 23.3 32.2 0</td>
<td>0 0 2.6 4.6</td>
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<tr>
<td>Negro</td>
<td>26</td>
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<td>10.3 7.5 27.7 30.5 63.3 0</td>
<td>0 7.5 0 19.4</td>
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<tr>
<td>Denmark d</td>
<td>1940-52</td>
<td>769</td>
<td>0.4 2.9 8.0 20.5 48.5 62.8 10.3$ e</td>
<td>0.4 1.6 6.8 19.4 48.7 64.0</td>
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<tr>
<td>Japan a</td>
<td>1958-64</td>
<td>129</td>
<td>0.2 2.0 5.3 15.9 34.9 7.4 e</td>
<td>0.1 0.8 4.2 6.0</td>
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<tr>
<td>Hiroshima</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Sweden d</td>
<td>1964</td>
<td>2719</td>
<td>0.21 0.65 2.14 5.43 13.32 19.81 2.11 e</td>
<td>0.23 0.71 2.14 5.67 13.88 20.46 2.44</td>
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</table>

*a* Prospective, population-based data.  
*b* Incidence rate for population aged 45-69.  
*c* Incidence rate for population aged 60-69.  
*d* Retrospective, hospital data.  
*e* Incidence rate for population aged over 30.
2.5 Frequency of types of stroke

Different diagnostic criteria and differences in data collection may prevent direct comparison, but some data on the frequency of different types are presented for information in Tables 3 and 4. These data are based either on community studies or on data from hospitals admitting patients without selection.

Although there are substantial differences in frequency from place to place, cerebral thrombosis is usually the most frequent form of stroke encountered in clinical studies, followed by haemorrhage. Subarachnoid haemorrhage and cerebral embolism come next as regards both mortality and morbidity, but usually account for less than 10\% of all cerebrovascular accidents.

2.6 Prognosis

2.6.1 Transient cerebral ischaemia as a precursor of stroke

It is reported that 16–28\% of patients with transient cerebral ischaemia and followed for an average of 3–4 years had a single attack only.\textsuperscript{20, 21, 22} About half of the patients had another attack in the first year, but no further recurrences during the observation period; over one-third of patients in one series of investigations had more than 5 attacks.\textsuperscript{21}

In one study stroke developed in about 2\% of patients with transient cerebral ischaemia within a 4-year follow-up period,\textsuperscript{20} while in other studies about 15\% developed stroke within the same period.\textsuperscript{17, 21, 22, 23} Only a single attack of ischaemia was experienced in one-quarter to half of patients with later cerebral infarction.\textsuperscript{17, 20, 21}

About 4–5\% of patients with transient cerebral ischaemia died within a year.\textsuperscript{20, 21, 22} Myocardial infarction was the cause of death in half of them, and cerebral infarction in only 12\%.\textsuperscript{21}

These findings indicate that transient cerebral ischaemia is a warning of atherosclerotic cardiovascular disease.

2.6.2 Mortality from stroke

About one-quarter of patients die within 24 hours\textsuperscript{54, 25, 26, 27} and nearly a half in 2–3 weeks. Initial mortality from subarachnoid haemorrhage and cerebral haemorrhage is higher than from cerebral infarction. In Middlesex County, Connecticut, USA, for example, 82\% died within one month after stroke from cerebral haemorrhage, but only 42\% after cerebral infarction.\textsuperscript{14} Improved care, particularly the prevention of infective complications, has helped to reduce the number of deaths occurring later.

Nearly half the patients who survive acute attacks of cerebral infarction die within 4–5 years.\textsuperscript{16, 28, 29, 30, 31} The commonest cause of death is recurrent
<table>
<thead>
<tr>
<th>Place</th>
<th>Date of study</th>
<th>Age group</th>
<th>Subarachnoid haemorrhage</th>
<th>Cerebral haemorrhage</th>
<th>Cerebral thrombosis</th>
<th>Cerebral embolism</th>
<th>Other and ill-defined categories</th>
<th>Total</th>
<th>Total number of cases</th>
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<tr>
<td>Ribeirão Prêto (Brazil)</td>
<td>1965-66</td>
<td>15-74</td>
<td>2.1</td>
<td>51.9</td>
<td>37.2</td>
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<td>Bristol (England)</td>
<td>1965-66</td>
<td>15-74</td>
<td>8.0</td>
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<td>San Francisco (USA)</td>
<td>1965-66</td>
<td>15-74</td>
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<td>36.2</td>
<td>33.6</td>
<td>9.9</td>
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<td>9.6</td>
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<td>6.1</td>
<td>73.3</td>
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<td>15-74</td>
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<td>23.9</td>
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<td>Uppsala (Sweden)</td>
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<td>60.5</td>
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<td>7.8</td>
<td>4.3</td>
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<td>100.0</td>
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<td>1964-65</td>
<td>15-74</td>
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<td>59.5</td>
<td>31.9</td>
<td>100.0</td>
<td>100.0</td>
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<td>15-74</td>
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<td>134</td>
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<td></td>
</tr>
</tbody>
</table>

* Retrospective population-based data; weighted figures.  
+ Prospective hospital data.

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# Retrospective hospital data.  
# Prospective population-based data.  
# Cerebral thrombosis or embolism.
<table>
<thead>
<tr>
<th>Place</th>
<th>Date of study</th>
<th>Age group</th>
<th>Place</th>
<th>Date of study</th>
<th>Age group</th>
<th>Relative frequency (%)</th>
<th>Total number of cases</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bombay (India) a</td>
<td>1954-62</td>
<td>All</td>
<td>Zerifin (Israel) c</td>
<td>1955-66</td>
<td>30+</td>
<td>3.0</td>
<td>127</td>
<td>108</td>
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<tr>
<td></td>
<td></td>
<td>ages</td>
<td>Hiroshima (Japan) d</td>
<td>1961-66</td>
<td>40+</td>
<td>6.1</td>
<td>132</td>
<td>103</td>
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<td></td>
<td></td>
<td></td>
<td>Hsinayama (Japan) d</td>
<td>1962-67</td>
<td>11+</td>
<td>13.6</td>
<td>100.0</td>
<td>104</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Lagos (Nigeria) c</td>
<td>1964</td>
<td>All</td>
<td>6.4</td>
<td>205</td>
<td>105</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Uppsala (Sweden) c</td>
<td>1952-54</td>
<td>7.5</td>
<td>46.8</td>
<td>2719</td>
<td>18</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Middlesex (England) c</td>
<td>1955-57</td>
<td>Not stated</td>
<td>5.7</td>
<td>195.0</td>
<td>426</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Framingham (USA) d</td>
<td>1949-61</td>
<td>30-62</td>
<td>18.0</td>
<td>552</td>
<td>506</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Middlesex (USA) d</td>
<td>1957-58</td>
<td>45+</td>
<td>35.6</td>
<td>191</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>USA d</td>
<td>1957-58</td>
<td>40-69</td>
<td>7.4</td>
<td>461</td>
<td>14</td>
</tr>
<tr>
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<td>'B' areas f</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

a Prospective hospital data.  
b Cerebral thrombosis or embolism.  
c Retrospective hospital data.  
d Prospective population-based data.  
e Areas with low incidence of cerebrovascular disease, i.e., Denver, Kansas, Miami.  
f Areas with high incidence of cerebrovascular disease, i.e., Georgia, North and South Carolina.
cerebrovascular accident, followed by myocardial infarction, congestive heart failure, and pneumonia. Patients with untreated subarachnoid haemorrhage have a poorer prognosis: 35% of survivors from the acute phase die within a year of the onset, and only a quarter of those who survive for a year are still alive after 5 years.  

2.6.3 Recurrence

The recurrence rate of cerebral infarction was found to vary between one-quarter and one-third within the 2–5 years following acute stroke. Embolism tends to recur as long as the source of emboli exists, and two-thirds of the recurrences occurred within one year of the initial attack. Subsequent attack results in similar mortality to initial cerebral infarction. In subarachnoid haemorrhage, about 80% of recurrences are within 6 weeks, concentrated largely within the first 2 weeks, and cause greater mortality than the initial attack.

2.6.4 Disability after stroke

The available data vary greatly, mainly because of the way cases are selected and the lack of a standard assessment of disability. About one-fifth to one-half of stroke patients can eventually resume independent daily life activities, but one-quarter to two-thirds of them are permanently disabled. Maximum functional improvement was obtained within one month of the stroke in one-third of the cases, and in most other cases only a little more functional recovery is expected after 6 months.

2.7 Conclusions

Available information suggests the wide distribution of cerebrovascular diseases everywhere in middle-aged and old people. Case fatality is high and disability after an attack is common.

A prevention programme is urgently needed and facilities for care and treatment should be improved, depending on the size of the problem and the available resources. Information on the size of the cerebrovascular disease problem could be improved by international agreement on terminology, classification, and diagnostic criteria, and by uniform procedures for collecting data on the extent and nature of morbidity from cerebrovascular disease.
3. CLASSIFICATION AND DIAGNOSIS OF CEREBROVASCULAR DISEASES

3.1 Classification

The classification presented here is intended to be used primarily by epidemiologists, public health authorities, and general medical practitioners in a wide variety of circumstances throughout the world. With this in view, the selection of items has been limited to those that appear most important at the present time and for which assessment is feasible.

The primary classification proposed is based partly on the nature of the pathological changes in the brain and partly upon the clinical stage of the disease.

A. Pathological changes in the brain

1. Subarachnoid haemorrhage: a haemorrhage originating in the subarachnoid space.
2. Intracerebral haemorrhage: a haemorrhage originating in the brain parenchyma.
3. Cerebral ischaemic necrosis: death of brain parenchyma due to lack of blood supply, however caused. This includes infarction, both haemorrhagic and anaemic.

B. Clinical stage

Transient cerebral ischaemia. This is a focal neurological deficit commonly lasting for some minutes but never longer than 24 hours, leaving no residual deficit, occurring in patients with vascular disease, and frequently showing a tendency to recur. The premonitory symptoms of a migraine attack are not included in this category.

A proposal for a detailed classification of cerebrovascular diseases is reproduced as Annex 2.

Certain associated conditions should also be sought. The following list has been designed without prejudice to etiological possibilities in order to discover conditions that may be associated with strokes.

1. Arterial hypertension
2. Ischaemic heart disease
3. Atrial fibrillation
4. Diabetes mellitus
5. Abnormalities of blood lipids
6. Smoking
7. Obesity

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a For further information see the report of the WHO Expert Committee on Arterial Hypertension and Ischaemic Heart Disease (Wld Hlt Org. techn. Rep. Ser., 1962, No. 231).
b For further information see the report of the WHO Committee on Diabetes Mellitus (Wld Hlt Org. techn. Rep. Ser., 1965, No. 310).
c For further information see reference 107.
3.2 Diagnostic procedure

The clinical signs and symptoms shown in Table 5 are helpful in distinguishing the three primary categories of cerebrovascular disease.

<table>
<thead>
<tr>
<th>Signs and symptoms</th>
<th>Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Subarachnoid haemorrhage</td>
</tr>
<tr>
<td>Preceding transient ischaemic attacks</td>
<td>None</td>
</tr>
<tr>
<td>Onset</td>
<td>Abrupt (within 1 or 2 min)</td>
</tr>
<tr>
<td>Headache</td>
<td>Very severe and instantaneous</td>
</tr>
<tr>
<td>Vomiting at onset</td>
<td>Frequent</td>
</tr>
<tr>
<td>Hypertension a</td>
<td>Not usually</td>
</tr>
<tr>
<td>Consciousness</td>
<td>May be lost momentarily</td>
</tr>
<tr>
<td>Neck stiffness</td>
<td>Invariably present</td>
</tr>
<tr>
<td>Weakness of one side of body</td>
<td>Not at onset</td>
</tr>
<tr>
<td>Deviation of the eyes</td>
<td>Never at onset</td>
</tr>
<tr>
<td>Speech disturbances</td>
<td>Very rare</td>
</tr>
<tr>
<td>Cerebrospinal fluid (early)</td>
<td>Invariably blood-stained</td>
</tr>
<tr>
<td>Subhyaloid haemorrhage (by ophthalmoscopy)</td>
<td>May be present</td>
</tr>
</tbody>
</table>

a The readings obtained at onset may not reflect the usual level of blood pressure and records of antecedent readings should be obtained if possible.

3.3 Examination of the patient with cerebrovascular disease

Present techniques of investigation permit full and detailed examination of a patient with cerebrovascular disease. The extent of the examination will vary according to the needs of the individual patient and the facilities available. It will not be possible to apply the schedule described below in every case, but items in sections 1, 2, and 3 should be considered as a minimum requirement.
1. **History**
   Special reference should be made to previous cerebrovascular episodes, especially transient cerebral ischaemia, cardiac disease, and peripheral vascular disease. The history of vascular disease in the family should also be recorded.

2. **Physical examination**
   Particular attention should be paid to neurological status, the presence or absence of bruits over the arteries and over the skull, the presence or absence of arterial pulses, cardiac rate and rhythm, blood pressure in both upper extremities and sometimes in lower extremities, the presence of xanthomata, and the findings at ophthalmoscopy.

3. **Laboratory examination**
   (a) **Urine analysis.** The following examinations should be performed:
       - Specific gravity
       - Protein content
       - Glucose content
       - Microscopy
   
   (b) **Blood analysis**
       - Test for syphilis
       - Haemoglobin
       - Red cell count
       - White cell count
       - Differential white cell count
       - Haematocrit
       - Lipids (at least cholesterol)
       - Sedimentation rate
       - Glucose content
       - Creatinine level

   (c) **Electrocardiogram**

   (d) **Radiological examination of chest and head**

4. **Cerebral angiography**
   This essential diagnostic tool should be available in all systems for care of patients with stroke. It is plainly necessary as a diagnostic instrument in younger people, but could also be important in the elderly (e.g., detection of a subdural haematoma).

   There is as yet no universal agreement on the indications for cerebral angiography, although most doctors would agree on certain contra-indications, such as advanced age and poor general condition. At present, however, angiography is the best method of discovering those lesions that are potentially amenable to surgical treatment, and should be employed for this purpose. Angiography is therefore indicated in:

   - Subarachnoid haemorrhage
   - Suspected subdural or epidural haematoma
   - Arteriovenous aneurysm
   - Cerebral neoplasms presenting as stroke
   - Suspected intracerebral haemorrhage that might require surgical intervention
   - Suspected extracranial thrombo-atherosclerotic disease in which surgery appears to be indicated. An angiographic evaluation of co-existing intracranial vascular disease should always be made before surgical intervention.
5. Examination of cerebrospinal fluid

When the condition of the patient allows, and if there is no clear evidence of a rise in intracranial pressure, a sufficient amount of cerebrospinal fluid should be drawn off to permit estimation of cells and protein content and a test for syphilis.

6. Other procedures that may be of value

(a) Echoencephalography 39—particularly within the first 24 hours after an ictus
(b) Electroencephalography 40
(c) Isotope brain scanning 41, 42

7. Other procedures currently being developed

(a) Measurements of regional cerebral blood flow 43
(b) Detection of circulatory disturbances, including extracranial occlusion, by the use of gamma-recording devices 44

4. CARE OF STROKE PATIENTS

4.1 Introduction

It is important to distinguish between the acute and chronic phases of stroke. The participants agreed that the term "acute phase" should refer to the first 3 weeks after the onset of stroke.31 The special features of impaired autoregulation of cerebral circulation during the first few days following the attack should be noted. The chronic phase covers the period after 3 weeks from onset. Attention should also be paid to the phenomenon of transient cerebral ischaemia and its special features, which may give warning of a later acute major attack.17, 21, 22, 23

Acute stroke should be equated with the medical or surgical emergency, and requires an optimum care system. Rehabilitation should be instituted from the outset of the attack, and it is important to avoid a passive attitude towards therapy.

In every patient with acute stroke, an accurate diagnosis should be made without delay. This can best be done in a hospital, where diagnostic facilities and intensive care as well as the necessary monitoring techniques are available. Before rational treatment can be instituted the type and cause of the stroke must be defined, i.e., correct diagnosis and related etiological factors must be established. It is of the utmost importance to know when intracranial haemorrhage is present, and whether it is intracerebral or extracerebral (i.e., subdural or extradural in location). Likewise, if cerebral ischaemic necrosis is present it is necessary to determine whether or not it is due to thrombosis or stenosis or emboli in intracranial or extracranial vessels.

Another important consideration when cerebral ischaemia is present is whether this results from a systemic haemodynamic crisis such as myocardial infarction or from insufficiency of cardiac output due to other disorders of
the heart (i.e., complete heart block and other dysrhythmias, shock due to toxaemia and other causes, hypoglycaemia, etc.) or due to changes in the composition of the blood (anaemia, polycythaemia, dehydration, electrolyte imbalance, etc.).

For rational organization of the diagnosis, treatment, and prognostic evaluation of the patient, co-ordinated and comprehensive care, ideally concentrated in a "stroke centre", should be available in all major hospitals.

The personnel providing care should include a neurologist, a general physician, a cardiologist, a rehabilitation specialist (doctor), a radiologist, a neurosurgeon, and a physiotherapist. When mental or emotional symptoms are present, psychiatric help and advice should be available to the medical team. The team outlined here is considered optimum, but its composition will vary from place to place in accordance with local policies and the interests of individual team members.

In particular circumstances, first-aid stations with equipment and trained personnel may be useful. The first-aid personnel are responsible for the emergency care of the patient at his home and during transport to the hospital, particularly for control of the respiratory and circulatory functions (see section 4.2).

4.2 Acute phase

4.2.1 General care

During the first few days many patients are comatose or stuporous and therefore need careful supervision, including maintenance of an adequate airway, control of fluid and electrolyte balance, and urinary catheterization. Some patients, especially the elderly, will already have a urinary infection; others will acquire one as a consequence of catheterization. This must be adequately treated. The catheter should be withdrawn as soon as possible.

Adequate ventilation and the avoidance of airway obstruction are particularly important in the care of comatose or stuporous patients. Hypercapnia leads to raised intracranial pressure. Maintaining adequate ventilation is life-saving and death may occur if it is neglected, even though the stroke may not have primarily involved the respiratory centre. Primary involvement of the respiratory centre may result in hyper- or hypoventilation and in alteration of the blood pH and electrolyte content. Measurement of water and electrolyte balance and of arterial blood gas serves as a useful guide.

4.2.2 Improvement of blood circulation in the brain

4.2.2.1 Principles. Special therapeutic measures are directed towards improving blood circulation to the ischaemic brain area, reducing brain swelling, and preventing thrombus formation. The perfusion rate of any
tissue, including the brain, is determined by the driving blood pressure (i.e., arteriovenous pressure difference) and the vascular resistance. The vascular resistance in the brain is determined by the diameter and length of the vessels, the intracranial pressure, and the viscosity of the blood. The therapy used must reduce one or more of the factors in the ischaemic area of the brain that lead to an increased resistance.

4.2.2.2 Blood pressure. The blood pressure must be maintained at an optimum level. If it is too low, i.e., systolic pressure below 90 mm of mercury, intravenous infusion of a hypertensive drug and/or replacement or restoration of an adequate blood volume should be considered. Known hypertensive subjects may require higher blood pressure levels (e.g., systolic pressure 160 mm Hg). When in doubt, monitoring of central venous pressure, arterial blood pressure, and ECG may be helpful.

In patients with extreme hypertension accompanying the stroke, cautious hypotensive therapy should be instituted (e.g., saline drugs, reserpine, or methyldopa) in order to prevent a hypertensive crisis (hypertensive encephalopathy, hypertensive haemorrhage, etc.). It is recommended that the blood pressure be lowered cautiously to approximately 170/100 mm Hg under careful clinical observation. In order to prevent excessive decrease of blood pressure, ganglionic blocking agents should be avoided. Hypotensive therapy may be dangerous and contraindicated in some atherosclerotic patients with high systolic and low diastolic blood pressure.

4.2.2.3 Vasodilator substances. There is disagreement at present as to whether available cerebral vasodilator substances are of therapeutic benefit in acute stroke. Treatment of stroke with drugs causing dilatation of peripheral vessels may decrease blood pressure and hence decrease cerebral blood flow, because of dilatation of the extracerebral vessels.

Forms of therapy that require further investigation are (a) the intermittent use of 5% CO₂ plus oxygen and (b) hyperventilation. Some workers consider that intermittent 5% CO₂ plus oxygen will increase total cerebral blood flow and thereby increase collateral blood flow to the ischaemic areas. The overall increase in cerebral blood flow in acute stroke may be of additional benefit, as has been shown with regional blood flow measurements, but generally blood flow is decreased in the acute stages of cerebral infarction.

The opposing view is that during the first 3 days following cerebral infarction there is regional loss of autoregulation in the infarcted areas.⁸

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⁸ Autoregulation is defined here as the property of the brain to keep its blood flow constant despite changes in blood pressure. In addition, the normal cerebral vessels are able to dilate and constrict in response to changes in the gases and metabolites of the blood (e.g., PCO₂, PO₂, pH). This may be called metabolic vasomotor control. Although autoregulation and metabolic vasomotor control are commonly impaired together in cerebral infarction, it is also possible for one mechanism to be disordered without the other.
Hence 5% CO₂ inhalation may shunt or "steal" blood from the ischaemic zone into the "normal" part of the brain. As a consequence, therapy with passive hyperventilation is being investigated, the working hypothesis being that vasoconstriction in "normal" parts of the brain resulting from hyperventilation will shunt blood into the infarcted area.

*Stellate ganglion blockade* is a form of treatment not recommended, since it does not lead to an increase of cerebral blood flow and clinical trials have shown no improvement in clinical status.

4.2.2.4 *Hyperosmotic solutions.* Reduction of intracranial pressure is an important factor in acute stroke. This pressure is increased in stroke patients, largely because of oedema. Drugs that reduce intracranial pressure by their hyperosmotic action, such as 20% mannitol, 40% sorbitol, and glycerol, require further investigation to define their action in cerebral haemodynamics at various stages of cerebral ischaemic necrosis. Urea solution should be used with caution since it may increase blood pressure; the effect on cerebrospinal fluid pressure is of short duration. Potent saluretics given intravenously may reduce intracranial pressure by causing diuresis.

Dextran of molecular weight below 60,000 may also be considered in the therapy of non-haemorrhagic ischaemic necrosis. It should not be given to patients with haemorrhagic infarction or intracranial haemorrhage or with other potential sites of bleeding, since the drug has an anticoagulant action. In patients with left heart failure the drug may produce acute pulmonary oedema.

After intravenous infusion of 500 ml of dextran over a period of one hour cerebral blood flow is increased, mainly because blood viscosity is reduced by haemodilution. Prolonged use of dextran in doses of 500 ml every 12 hours appears to reduce mortality and neurological deficit because of the reduction of blood viscosity and possibly also because of inhibition of platelet aggregation and reduction of brain swelling.

4.2.2.5 *Adrenal corticoids.* There is little evidence that adrenal corticoids reduce brain swelling in acute cerebral infarction.

4.2.2.6 *Treatment of associated cardiac disorders.* Cerebral blood flow is dependent on cardiac output. Cardiac insufficiency, which often passes unrecognized in patients with stroke, should be actively treated with digitalis and other widely accepted measures. It should be borne in mind that acute myocardial infarction is commonly associated with stroke and should be treated.

Heart block and other cardiac problems should be treated according to the cardiological indications (e.g., pacemaker, cardioversion, appropriate drugs).
4.2.3 Anticoagulant treatment

In some patients with transient cerebral ischaemia, prolonged and strictly controlled anticoagulant treatment may delay the occurrence of major strokes.\textsuperscript{33,56,57} In cases of chronic auricular fibrillation, anticoagulant treatment may prevent cerebral emboli.\textsuperscript{58}

In the acute phase after a completed stroke, the cerebrovascular pathological process cannot be influenced by the administration of anticoagulants, but when a severe motor handicap exists anticoagulant treatment has been shown to reduce the incidence of venous thrombosis and pulmonary embolism.

In the anticoagulant treatment of patients with cerebrovascular disease, special attention should be paid to contraindications, particularly arterial hypertension, because of the risk of intracranial haemorrhage.

4.2.4 Other therapeutic considerations

Hypothermic therapy. Another therapeutic principle in the acute stroke patient is to treat fever, as in fever the cerebral demand for oxygen is increased. The temperature is usually lowered by drugs; in severe hyperthermia external cooling may be added. The validity of this principle has been proved in experimental animals, but not up to now in clinical trials.\textsuperscript{59}

Hyperbaric oxygenation. The oxygen tension within the brain can be raised by hyperbaric oxygenation, and a few reports indicate a short but definite clinical effect.\textsuperscript{60}

Other symptomatic treatment. Anticonvulsive medication should be undertaken in patients with seizures; these occur in about 10% of subjects with cerebral embolism. In subjects with vertebrobasilar insufficiency, vertigo may be controlled with antivertiginous drugs. In patients with cerebral haemorrhage and subarachnoid haemorrhage, drugs such as pethidine may be useful for controlling headache and the chlorpromazine type of drug may be used for sedation.

4.2.5 Rehabilitation

The aim of treatment of any patient is to preserve not simply life, but independent life. Success should be measured not by the number of lives saved but by the number of patients able to return to the community. The entire treatment team should be aware of this principle.

From the very first day, even if the patient is still unconscious, prevention of contractures is just as important as prevention of bedsores. Correct positioning of the paralysed extremities should be combined with gentle, passive movements of every joint, by doctor, nurse, or physiotherapist, at least 3 times a day.

If the patient is conscious, active exercises of the non-paralysed limbs should begin. A grave problem at this stage may be sensory deprivation
by immobility and lost contact. In contrast with subjects with many other conditions, the patient with stroke is often depressed and usually lacks the will to recover. It is therefore very important that the therapeutic team should establish early and continuing contact. At the same time, support for the family, including explanation and reassurance, is a necessary component of successful rehabilitation. This can be reinforced by any sign of physical recovery. In this sense, the physical rehabilitation is complementary to psychological improvement.

The patient is taught to move his paralysed limbs with the help of his non-paralysed arm and leg. It has been found that these exercises have less influence on heart rate and blood pressure than most nursing procedures. Usually this is possible within a few days of his regaining consciousness.

As soon as the patient's general condition allows, he sits on the side of the bed with support and, if this succeeds, on a chair. When sitting on a chair, he is taught more passive exercises with the arm. When the patient is used to the sitting position he should learn to stand up and bring his weight on to his paralysed leg. A leg that is trained in weight-bearing from the beginning will not develop spastic contractures. Training in self-care begins at this stage. The doctor should be present the first time exercises in standing are performed.

When this stage is reached the patient can go to the gymnasium, where exercises are done under medical supervision; they should be short but repeated very often. The gymnasium should be the social centre of the stroke department. Stress should be placed on the training of non-affected limbs.

Training to walk should now begin, with the support of a physiotherapist and in the presence of the doctor. The patient is taught the use of a stick.

Training in the activities of daily living should be an essential part of the daily programme. It is necessary for the patient to walk in a street with traffic and learn to use a normal staircase before returning home.

To summarize, the patient goes through the following phases of physical rehabilitation:

*Unconscious*:
- Passive movements

*Conscious*:
- Passive and active movements when sitting up in bed
- Sitting on a chair
- Training in self-care
- Standing
- Exercises in the gymnasium
- Walking, activities of daily living
- Climbing staircases
- Going out
- Regaining confidence in walking in traffic (preferably in the presence of a doctor)
4.2.6 Surgical treatment

The importance of surgical treatment needs further evaluation, and the success of such treatment depends on appropriate selection of patients. Patients with acute cerebrovascular disease should be admitted to a department with angiographic facilities as soon as possible after the onset of symptoms.

Treatment is indicated to prevent recurrences in patients with transient cerebral ischaemia, rupture of saccular aneurysm, or arteriovenous aneurysm. Evacuation of haematoma is indicated in a minority of cases with intracerebral haemorrhage, usually at the stabilized and liquefied stage.

4.2.6.1 Accessible stenotic lesions of the carotid artery and of the proximal portion of the subclavian artery. Reconstructive surgery of carotid stenosis has good and long-lasting results with acceptable operative mortality. Besides improving the cerebral collateral circulation, it reduces the risk of embolism from untreated atheromatous lesions and provides prophylaxis against further stroke.

It is indicated in transient cerebral ischaemia with associated carotid stenosis; in stenosis of the carotid artery with mild neurological deficit, if another major operation is planned; in stenosis of the proximal portion of the subclavian artery. Opinion differs as to whether surgery is indicated in cases of acute thrombotic occlusion.

Contraindications to surgery are poor general condition, severe concomitant diseases, advanced cerebral arteriosclerosis, and severe neurological deficit.

4.2.6.2 Spontaneous intracerebral haemorrhage. Opinions differ as to the usefulness of surgery. Some neurosurgeons postpone operation until stabilization has taken place; others advocate immediate surgical therapy, while a third group prefers conservative treatment.

In cases of localized haemorrhage (haematoma) it is necessary to draw a sharp distinction between a typical hypertensive haematoma localized in basal ganglia and an atypical (usually normotensive) haematoma localized in white matter. They differ etiologically, clinically, angiographically, and prognostically. A third type may be hypertensive haemorrhage, affecting white matter more than basal ganglia. The prognosis for the first type of haematoma is poor and surgical treatment is seldom indicated. Patients with normotensive haematomas in the white matter have a better prognosis following surgery than the other two groups. Where there are signs that the haemorrhage is progressing into the white matter, an operation is indicated.

Intracerebellar haematomas are rare, but may require urgent evacuation.

4.2.6.3 Ruptured intracranial aneurysm. There is some variability in the expected course of events according to the site of aneurysm.
The danger of rebleeding is greatest for all patients during the first 5 weeks.

Surgical treatment (direct attack on the aneurysm by occlusion of its neck, wrapping of its sack, or proximal ligation of the feeding artery) is the only way of preventing rebleeding, but the attendant risks of mortality and morbidity are considerable. The most important factor determining the result of surgery is the condition of the patient subsequent to rupture of the aneurysm. An absolute indication for surgery in the acute stage is a life-threatening haematoma or an acute hydrocephalus.

Operative risk immediately after the initial bleeding tends to be high. A delayed procedure is safer, but its prophylactic effect is smaller. In any individual case, therefore, it is important to determine when the prophylactic gain and the operative risk are optimally balanced.

Coma, grave neurological deficit, and mental changes caused by widespread vasospasm, whether anticipated clinically or diagnosed by arteriography, are a contraindication for surgical therapy. At the present time there is no effective method of treating intracranial arterial spasm. The operation in such cases should be postponed for 14–21 days.

A sharp age limit should not be imposed in considering surgical treatment. The evaluation of the general condition of the patient is more important.

4.2.6.4 Arteriovenous aneurysm. An imperative indication for surgery is an intracerebral haematoma with serious neurological deficit or increased intracranial pressure. In other cases the number of previous bleedings, the extent of arteriovenous shunt, the degree of chronic circulatory insufficiency, and increasing neurological disability of the surrounding brain must be taken into consideration.77, 78

The present state of neurosurgery permits the removal in most instances of large lesions, even those localized in functionally important cortical areas, without grave consequences.79, 80, 81

4.3 Chronic phase

Bronchopneumonia and thromboembolic complications used to be prevalent, but since early mobilization has become the rule these complications are less common in those patients who have a reasonable chance of surviving their brain disease.

It is now well-established that the tendency for contracture has to be counteracted from the first day. Prophylaxis against the shoulder-hand syndrome involves a range of passive movements of the arm and physical therapy of the arm and hand; prevention of contracture of the ankle joint calls for dorsiflexion of the foot and physical therapy of the leg and foot. The need for prevention of decubitus ulcers in all unconscious patients must, of course, be remembered.
4.3.1 Persisting signs and handicaps

Initially, a certain proportion of patients are comatose without any symptoms of focal cerebral damage, and many of them die without regaining consciousness. In patients with focal neurological symptoms, some symptoms disappear during the early period, and about 15% of all patients will be symptom-free within a few hours, days, or weeks. The spectrum of symptoms will therefore vary.

The following signs and symptoms are the most prevalent:

1. *Hemiplegia, hemiparesis.* This constitutes the main somatoneurological disorder in about 90% of patients. In the early stages the paralysed muscles are usually flaccid, but later a combination of rigidity and spasticity develops. In those whose general medical condition is satisfactory, the ability to stand and walk will be regained even if a severe paresis of the leg persists (stability due to stretch reflex). On the other hand, a persisting paralysis of the hand will—irrespective of the reflex hypertonus—result in a useless hand.

2. *Hemihypaesthesia.* This symptom is sometimes dominant but in about 50% of cases is combined with hemiparesis and adds to the handicap, owing to lack of feedback control of movements, which interferes with physical training.

3. *Hemianopia.* This occurs in 10–15% of cases, and adds to the handicap by making reading and writing more difficult.

4. *Aphasia.* This symptom appears in about one-third to one-quarter of cases, usually in combination with right-sided hemiplegia. It is a more severe handicap than the hemiplegia in most cases and therefore requires particular attention.

5. *Pseudobulbar paresis.* Due to lesions in bilateral central structures of the brain, this symptom appears predominantly in old persons who have previously had a cerebrovascular lesion on the opposite side to the present acute one. The dysarthria makes speech difficult and sometimes even unintelligible. In some cases dysphagia and/or uncontrollable weeping may also occur.

6. *Dementia.* From the rehabilitation point of view this is the most severe of all syndromes. It occurs after extensive brain lesions (often due to repeated cerebrovascular accidents), or as independent disease, or both. The term "dementia" should be used when impairment of intellectual functions seems to be irreversible.

7. *Syndrome of asthenic reactions (insufficiency) and emotional lability.* This is one of the most common syndromes of the brain lesion and com-

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* Much of the information in this section is taken from reference 82 and from a personal communication from T. Broman (1970).
prises several of the symptoms, e.g., inattention and reduced concentration, energy, and drive; increased irritability; and readiness to weep. Some patients display dissatisfaction, in others a tendency to depression supervenes. These symptoms often fluctuate; they significantly impede the rehabilitation process.

(8) Depression. This symptom is usually not manifest in the early stages after a stroke. Instead the patients are often emotionally flat and may even react with euphoria. Later, when they have recovered from their initially lowered level of consciousness, they become aware of their handicap and understand its significance for their future. Then the tendency for mental depression increases.

(9) Other mental symptoms—lack of initiative, amnestic syndromes, and changes of personality. These symptoms are often combined with those mentioned above but may also appear more or less independently. Lack of initiative and apathy seem to be most frequent in cases with a lesion of the central (particularly diencephalic) structures. Korsakoff's syndrome appears when the limbic system has been damaged, which among stroke patients is sometimes seen in connexion with frontal lobe lesions due to haemorrhage from a saccular aneurysm. Very often personality traits become accentuated after cerebrovascular accidents.

(10) Other brain symptoms. Due to the location of the lesion any brain function may be defective, and a great range of symptoms may appear. From a practical point of view it is important to recognize whether the patient shows difficulties in spatial orientation or in own-body recognition, or whether symptoms of apraxia occur.

(11) Epilepsy. Epilepsy or epileptic seizures are a rather common complication in cerebrovascular disease, and are more frequently found following haemorrhage than infarction. Whether anti-epileptic therapy should be introduced as a prophylactic measure in all patients with brain lesions due to haemorrhage, or in those who show particular EEG changes, is an open question.

(12) Other common functional defects. As many patients are old, they may already have acquired other diseases or other manifestations of cardiovascular disease. For example, in a study of aphasic cases after stroke about 40% were found to have arterial hypertension, about 30% cardiac disease (angina pectoris, infarction, or vascular heart disease), about 5% intermittent claudication, about 7% cancer, about 10% deafness, and about 7% amblyopia. Furthermore, some patients suffer from obesity, severe osteoarthritis, chronic bronchitis, and emphysema.

A "complete" handicap diagnosis usually reveals that the patient is suffering from a complex spectrum of functional disturbances. This is important for deciding the prognosis and the goal for rehabilitation in
individual cases. A proper rehabilitation programme also requires an understanding of the significance of the different functional deficiencies.

4.3.2 Social situation

In planning the rehabilitation programme, it is always necessary to obtain sufficient knowledge of the patient's social situation and interests. Important points on which information is needed include:

1. family situation, attitude of the family members to the patient;
2. home situation, with technical data about the premises and their facilities;
3. economic situation;
4. former interests of the patient, his hobbies, and his working capacity;
5. information about the patient's education, profession, and working conditions where the prognosis seems good enough to warrant the expectation that he will regain work capacity; this actually occurs in only a small number of patients;
6. information about some special features, e.g., possession of motor car (or driving licence), boat, summer residence, garden, etc.

4.3.3 Rehabilitation

General aspects of the rehabilitation programme have been outlined in section 4.2.5. Matters that relate specifically to the chronic phase of the disease are discussed below.

4.3.3.1 Duration of rehabilitation. In order to permit effective use of personnel, the rehabilitation programme must sooner or later be terminated, even if a severe handicap remains. The length of the training period should be decided by the team, although the final responsibility for this decision should rest with the rehabilitation doctor. The rehabilitation goal is normally reached within 6–12 months, but there are exceptions. Sometimes effective training cannot even be started until 6–12 months after the stroke.

If lost functions cannot be regained, it may be possible to compensate for them. Rehabilitation should always be based upon remaining functions, not upon lost ones. Normalization is not the main goal.

4.3.3.2 Speech therapy in aphasia. Speech training should be carried out by everyone in the rehabilitation team. In the early stages of the disease the first aim is to establish emotional contact with the patient. As patients are often incapable of receiving systematic training, verbal activation has to be limited to practical situations during their daily care and mobilization. Later, when they are able to concentrate without tiring rapidly, the speech therapist should start systematic training exercises.
It is usually advisable to start with short exercises and to repeat them often, if possible 2-3 times a day. Later, patients are as a rule able to undertake special tasks on their own, on the advice of the speech therapist. The training programme should be modified and developed during the treatment period, which should generally last for several weeks or months (seldom more than half a year).

4.3.3.3 The rehabilitation team. Rehabilitation requires teamwork. It should be directed by the rehabilitation doctor, who should have training in neurology. He should be prepared to call upon the internist when indicated. The physiotherapist, occupational therapist, social worker, clinical psychologist, speech therapist, and nurses are all important members of the team. Every member should know about the principal medical, psychological, and social problems of the patient, the prognosis, and the aim of rehabilitation, and they should all co-operate in the planning of the treatment programme. Adequate information must also be given to one or more members of the family, whose co-operation is usually of cardinal importance in deciding when the patient will be able to leave the hospital.

The patient himself is the centre of the team. No rehabilitation is possible without his active co-operation. He should be given realistic but hopeful information about his future. He must be stimulated and persuaded to adopt a positive attitude to training. This is easier if his interests and hobbies are known and if he can be provided with a general therapeutic environment; other patients can often be of great help in this respect. The training programme should always be arranged in stages, so that the patient can experience success and does not become discouraged.

The physiotherapist and the occupational therapist are complementary partners in mobilization and activation therapy. In a broad sense, the physiotherapist concentrates on systematic training of separate motor functions, on prophylactic measures against contracture, on walking exercise, and on improving the general physical status, while the occupational therapist concentrates during the early stage on training in the activities of daily living and adapts different technical aids for that purpose. Training with the aid of mechanical devices is helpful. Later, the occupational training may be directed to household work, handicrafts, writing exercises, etc. Before discharge, the occupational therapist should normally visit the patient’s home in order to plan adequately for his activities of daily living.

4.3.3.4 Problems of organization. The organization of the care and rehabilitation of stroke patients depends fundamentally upon the way patients are selected. From a social and humanitarian point of view the goal must be to take care of every single patient in an optimum manner. For this a variety of institutions must be available. Initially, what is chiefly needed is diagnosis and immediate hospital care. At this stage patients should be treated in a hospital for acute diseases, in the medical, neuro-
logical, and neurosurgical departments, and early rehabilitation should be started. In the second stage, which usually begins a few days or weeks after the stroke, the patient who needs further hospital care should be transferred either to a rehabilitation department or to a hospital for long-term care. As the rehabilitation department must have highly developed resources and is therefore more expensive, the transfer of patients to such units must be restricted. On the other hand, hospitals for long-term treatment should also have resources for rehabilitation, even if their goals cannot usually be as high as those of rehabilitation departments.

After a treatment period of weeks or months, patients in a rehabilitation department can usually be discharged to their homes and receive further treatment as out-patients; adequate transport is required for this. A temporary stay in a day-hospital, a night-hospital, or a half-way house may facilitate early and smooth adaptation to home life. Patients admitted to a hospital for long-term treatment are often so severely disabled that they have to stay there for the rest of their lives; but some will improve and can eventually—after some months or years—be discharged to their homes. Much depends upon the family and the home situation. For some of these patients care and supervision are often needed during the day, either as a home service or in special establishments for the daytime care of severely handicapped patients. Elderly people with only a moderate handicap do not necessarily need hospital or nursing-home care and can be best accommodated in a special “protected residence” in the community.

Finally, it is important to emphasize that, in the evaluation and comparison of the results of rehabilitation of disabled stroke patients, the procedure used for the selection of patients should be carefully described.

5. PREVENTION OF CEREBROVASCULAR DISEASES

In affluent societies stroke patients probably occupy more hospital and nursing-home beds and make more use of social welfare services than cancer patients and injured persons together. Because of the mental and physical crippling caused by stroke, no single measure could so improve the quality of life in old people as the prevention of this condition. Nevertheless, recent advances in prevention are not being applied by health services.

A major cause of stroke is arterial hypertension. Recent prospective, controlled therapeutic trials in several countries have demonstrated that the incidence of stroke is significantly reduced by controlling moderate and severe hypertension (diastolic pressure averaging 105 mm Hg or higher during 3 or more visits). In a study in which male patients with diastolic pressure averaging 105–114 mm Hg were followed up for 1–5 years, the incidence of stroke was 11% in the group receiving no drugs but only 1% in the group receiving antihypertensive drugs. In another study, similar
beneficial results were obtained in female patients. In addition to stroke, other complications of hypertension such as heart and kidney failure were similarly reduced. Overall, the risk of male hypertensives developing a serious complication over a 5-year period was reduced by treatment from 55% to 18%. These results were obtained with drugs that have few adverse effects and can readily be administered by physicians in general practice.

The magnitude of the problem in developed countries is illustrated by data on persons in Bergen, Norway, having casual diastolic blood pressures over 110 mm Hg (see Table 6).

<table>
<thead>
<tr>
<th></th>
<th>Number of persons per 1000 population, by age group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>30-39</td>
</tr>
<tr>
<td>Men</td>
<td>2</td>
</tr>
<tr>
<td>Women</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Problems involved in the application of current knowledge to the control of hypertension, and the consequent prevention of stroke, include (1) the identification of hypertensive subjects and (2) the provision of effective treatment. The obvious method of identification is through ordinary clinical practice. However, ad hoc surveys such as that in Atlanta, Ga (USA),\(^5\) have indicated that about half of all hypertensive subjects are not aware of their condition, which is not surprising since hypertension is usually asymptomatic until the occurrence of organic complications. This survey further indicated that only about half of the identified subjects were currently receiving treatment, and even among this group only about half were receiving adequate treatment. Thus, the problem involves not only case-finding but the whole of the organization of medical care, including the provision of facilities and the education of physicians and other health workers in optimum techniques for the delivery of effective treatment. Moreover, the education and support of patients is necessary so as to impress upon them the importance of life-long treatment in preventing future complications. The complementary method of case-finding is by community screening, though the search for unrecognized hypertension, particularly in middle-aged subjects, should not be delayed by the over-elaboration of multi-phase screening. The optimum age for such preventive therapeutic interventions in hypertension can be determined only by systematic long-term trials.

The control of hypertension and thereby of the incidence of stroke is therefore grossly unsatisfactory, and it is urgent that authorities initiate
model programmes on a community basis to determine the most practical methods (as regards acceptability and cost) for the delivery of effective treatment by local health services. In the first instance these model programmes would cover middle age, and would incidentally collect the basic data on the local prevalence of hypertension that is essential for planning full-scale programmes. Each country should speedily develop at least one model community programme of a comprehensive nature. The co-ordination of such programmes is an important task for WHO, and the procedures used should be sufficiently standardized to permit international comparison.

5.1 Mild and labile forms of hypertension

At present the situation regarding mild and labile hypertension, particularly in younger age groups, is unclear. Controlled trials have not yet been carried out in such subjects to determine the prophylactic value of treatment, and might be initiated on an international scale by WHO. Such trials are of course also relevant to the involved problems of ischaemic heart disease.

5.2 Prevention of secondary hypertension

Prevention of some forms of secondary hypertension, especially that due to poststreptococcal chronic glomerulonephritis, is now becoming possible. Effective treatment of renal and urinary tract infections and of toxemia of pregnancy may also reduce the incidence of secondary hypertension. Controlling the abuse of analgesic drugs will further reduce renal damage and possibly the subsequent high blood pressure. Although essential hypertension can usually be controlled, no practical methods for preventing its occurrence can at present be recommended.

5.3 Research into other possible pathogenic factors in stroke

Stroke, especially cerebral infarction, is often associated with atherosclerosis and thrombosis. There is a need for further research on this association, possibly utilizing facilities and personnel currently devoted to the etiology of ischaemic heart disease and the identification of groups susceptible to it. However, major modifications would be required to include older age groups and greater numbers of females in study populations. The established value of treatment of polycythemia vera in the prevention of stroke needs to be emphasized. Among possible risk factors to be investigated are blood lipids, coagulation factors, cigarette smoking, obesity, lack of exercise, and diabetes mellitus.
Present hormonal contraceptives are associated with cerebral thrombosis in a very small percentage of women and it has also been suggested that they are involved in the development of hypertension and lipid disorders.\textsuperscript{96, 97} Since the incidence of these complications is very low, the collation of long-term data on an international scale is an appropriate task for WHO.

It has been suggested that levels of salt ingestion,\textsuperscript{9, 98} hardness of drinking-water (possibly connected with trace metals)\textsuperscript{99, 100, 101} and other dietary factors may be involved in the pathogenesis of hypertension and cerebrovascular disease. Conclusive evidence might be obtained from epidemiological studies, including planned experiments, in contrasting local situations.

### 6. STROKE CONTROL PROGRAMME

The aim of a stroke control programme is to apply at community level efficient measures for the prevention of stroke and for the detection, treatment, and rehabilitation of stroke patients. Facilities for long-term follow-up of patients are essential. The education and training of health personnel and of the public form an integral part of the programme, which should be related to ongoing community health services. Built-in continuous evaluation of the efficiency of the programme is desirable.

The basic requirement for realistic planning of such control programmes in the community is reliable knowledge of the extent of the problem in the community concerned.

#### 6.1 Background information needed

Existing information on mortality and morbidity from cerebrovascular diseases is generally imperfect, and from some areas it is practically non-existent. This situation could be improved by seeking accurate information about the various forms of cerebrovascular accidents recorded on death certificates and in hospital records, and by \textit{ad hoc} population surveys.

In some countries existing mortality and morbidity recording systems are being used as a basis for obtaining information about cerebrovascular diseases. Such additional information might also be obtained from other countries at relatively little cost in terms of effort and money.

Another way of improving background information would be to select specific populations in different countries for intensive study. The methods of study would vary according to local situations. In developing countries surveys of the prevalence and incidence of cerebrovascular diseases could be undertaken by a special team in a clearly defined area and using local paramedical personnel. In developed countries, on the other hand, cerebrovascular disease registers\textsuperscript{102} could be compiled on the lines of myocardial
infarction registers, or existing myocardial infarction registers could be used. These cerebrovascular disease registers could serve as a basis for:

(1) better planning of health services, including control programmes in the community;
(2) comparative studies of various types of management;
(3) study of the natural history of the disease and research into its etiology;
(4) comparative national and international studies.

Information-gathering programmes intermediate between these two types might be preferable in some places.

In all types of study clearly defined terms should be used. Common protocols are also essential to facilitate international comparisons. Checklists of symptoms and signs relevant to cerebrovascular diseases, comparable to those developed for ischaemic heart disease and peripheral vascular disease, could be useful. Further work is needed to reach agreement on terminology and protocols (see section 3).

6.2 Control programmes in the community

Comprehensive community control programmes should cover:
(1) prevention;
(2) detection and diagnosis;
(3) treatment, including rehabilitation;
(4) follow-up;
(5) education and training; and
(6) evaluation.

Items (1), (2), and (3) have already been considered in this report.

Item (4). Follow-up

There is a need for complete follow-up of all patients who have had a cerebrovascular accident in the community under study so that the adequacy of long-term care can be monitored. This will also lead to a better understanding of the natural history of the different varieties of the condition. During follow-up, the opportunity should be taken to evaluate particular measures that might favourably influence the course of the disease. \(^{28, 87}\)

Item (5). Education and training

There is a need to educate the general public about the meaning of such terms as "stroke" and "cerebrovascular" accident and to emphasize why a fatalistic attitude is incorrect. The possibilities of prevention through
control of blood pressure, and the potential in many cases for useful recovery should be stressed. These attitudes should also be encouraged in doctors and other health personnel.

WHO might assist by making a list of films, pamphlets, and other educational materials on this subject that have been prepared in many countries. The list could be widely circulated so that people would know what material is already available. As in other fields, there is a need to try to evaluate health education of this sort.

The number of health personnel now available for the total management of stroke patients is insufficient. Even in the long-established discipline of physiotherapy there is a shortage. In the newer fields of speech and occupational therapy, in social work, and in sheltered workshops, the deficiencies in many areas are catastrophic. Increased facilities for training such personnel are needed everywhere.

The contributions to the care of stroke patients that can be made by lay organizations are also of cardinal importance. They include home nursing, meals on wheels, home aids, and special transport facilities.

Item (6). Evaluation
In view of the cost of modern health services, continuous surveillance of their efficiency and effectiveness is essential. 87

7. RECOMMENDATIONS

7.1 Prevention
Cerebral haemorrhage can often be prevented by controlling high blood pressure. The first priority therefore is to try to improve the efficiency of hypotensive treatment in patients recognized to have hypertension.

The feasibility of treating people who have been discovered by ad hoc screening to have high blood pressure needs investigation. Each country should initiate at least one model programme in the context of its local health services in order to develop practical methods for case-finding and for the delivery of adequate continuing treatment of moderate and severe hypertension.

In mild and labile hypertension, controlled trials should be undertaken to determine the value of antihypertensive treatment in the prevention of major complications. The participants in the meeting recognized the difficulties in defining the populations to be studied and in designing trials that would be feasible in asymptomatic subjects who need to be followed up for long periods of time. A study group should be established to define these problems more clearly.

The necessary steps should be taken to prevent the development of certain forms of secondary hypertension.
7.2 Control programme

Active comprehensive management, including physical and mental rehabilitation and speech therapy, should be available so far as practicable to all stroke patients.

The importance of comprehensive rehabilitation and, for some cases, of facilities for long-term partial and complete care, needs emphasis. The possible contribution of day hospitals to the management of stroke patients in different communities should be investigated. Adequate aftercare facilities, both institutional and domiciliary, should be provided in order that hospital beds may be utilized in the most rational manner. Clinical evaluation of the usefulness of mechanical devices designed to assist the disabled patient is needed.

Studies are needed in specific cases to compare the outcome of hospital and home-based management.

The feasibility of screening appropriate populations for transient cerebral ischaemia and of providing treatment should be investigated.

Complete follow-up studies are needed to elucidate further the natural history of cerebrovascular disease; these studies should include therapeutic evaluation.

Pilot studies using cerebrovascular disease registers (comparable to or associated with acute myocardial infarction registers) that permit the complete coverage of specified population groups should be carried out so that the above recommendations can be implemented.

7.3 Autopsy procedures

Well-defined and internationally standardized autopsy procedures are required.

7.4 Education

The number of professional and lay personnel available for the full rehabilitation of stroke patients is generally inadequate and needs to be increased.

Education about cerebrovascular diseases is needed at all levels of the community, and attempts should be made to evaluate different types of educational approach. WHO could help by preparing a catalogue of published material.

7.5 Communication

Mortality and morbidity statistics need to be improved. To improve mortality statistics, advantage should be taken of the project on improve-
ment of death certification undertaken by WHO. The best morbidity statistics are likely to be derived from cerebrovascular disease registers.

As knowledge advances and international co-operation develops, the need for a more extended classification will become apparent. The participants therefore recommend that WHO take steps to prepare such a classification for international use, and suggest that Annex 2 of this report would form an excellent basis.

7.6 Further research

Further research is needed on the identification of risk factors such as salt intake and hardness of water. International co-operative research projects are also recommended for the study of coagulation and thrombosis in relation to ethnic, dietary, cultural, occupational, and climatic factors.

The implications of family aggregations of berry aneurysms and other cerebrovascular diseases should be assessed.

WHO should collect information on an international scale on vascular thrombotic complications associated with the hormonal contraceptives.

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Annex 1

LIST OF PARTICIPANTS

Dr Z. Askanas, Director, 4th Medical Clinic, Institute of Cardiology, Warsaw, Poland
Dr C. Balan, No. 9 Hospital, Bucharest, Romania
Dr T. O. Dada, Senior Lecturer in Medicine and Consultant Neurologist, College of Medicine, Lagos University, Lagos, Nigeria
Dr G. Dean, Director, Medico-Social Research Board of Ireland, Dublin, Ireland
Dr E. Endre, Department of Medicine, Ullevaal Hospital, Oslo, Norway
Dr C. Fazio, Professor and Director, Ist Neuropsychiatric Clinic, University of Rome, Italy
Dr F. Fenech, Lecturer in Medicine, Royal University of Malta, and Physician, St Luke’s Hospital, St Vennera, Malta
Dr R. Fogelholm, Lecturer in Neurology, University of Helsinki, Finland
Dr Gillian R. Ford, Senior Medical Officer, Department of Health and Social Security, London, England (Rapporteur)
Dr E. D. Freis, Senior Medical Investigator, Veterans Administration Hospital, Washington, D.C., USA
Dr H. Garcia-Barrios, Chief Medical Officer, Division of Cardiovascular Diseases, Ministry of Health and Welfare, Caracas, Venezuela
Dr L. Geltners, Director, Assaf Haroof Government Hospital, Zariifin, and Head, Department of Internal Medicine, University of Tel Aviv Medical School, Tel Aviv, Israel
Dr U. Gottstein, Professor of Internal Medicine, 1st University Hospital, Faculty of Medicine, University of Kiel, Federal Republic of Germany
Dr T. V. B. Greitz, Professor and Head, Department of Neuroradiology, Karolinska Hospital, Stockholm, Sweden
Dr A. L. Gwee, Senior Consultant and Head, Medical Unit III, General Hospital, Singapore
Dr W. D. Hess, Dozent, University Clinic for Psychiatry and Neurology, Vienna, Austria
Dr M. Ivkov, Neurosurgeon, Neurosurgical Clinic of the Faculty of Medicine, Belgrade, Yugoslavia
Dr P. Kartin, Professor and Head, Neurological Clinic, Faculty of Medicine, Ljubljana, Yugoslavia
Dr S. Katsuki, Professor of Internal Medicine, and Head, 2nd Department of Internal Medicine, Kyushu University, Fukuoka City, Japan
Dr H. Kesteloot, Professor, Department of Cardiology, University Clinic of St Raphael, Leuven, Belgium
Dr M. Korsoren, Assistant Chief, Medical Department, Falun Hospital, Falun, Sweden
Dr Z. Kunc, Professor of Neurosurgery, and Head, Neurosurgical Clinic, Charles University, Prague, Czechoslovakia
Dr R. R. H. Lovell, Professor of Medicine, Royal Melbourne Hospital, Melbourne, Australia
REPORT OF A WHO MEETING

Dr. J. Marquardsen, Chief Neurologist, Frederiksberg Hospital, Copenhagen, Denmark
Dr. J. Marshall, Reader in Clinical Neurology, Institute of Neurology, National Hospital, Queen Square, London, England
Dr. H. Metz, Neurologist, Deich and St Thérèse Clinics, Luxembourg
Dr. J. S. Meyer, Professor and Chairman, Department of Neurology, Baylor College of Medicine, Texas Medical Center, Houston, Texas, USA
Dr. C. H. Millikan, Professor, Mayo Foundation of the University of Minnesota, Rochester, Minnesota, USA
Dr. J. N. Morris, Director, Social Medicine Research Unit, Medical Research Council, and Professor of Social Medicine, London School of Hygiene and Tropical Medicine, London, England
Dr. S. Pakarinen, Neurosurgeon, Neurosurgical Department, Helsinki University Central Hospital, Helsinki, Finland
Dr. J. J. Fastor, Chief, Cardiology Service, Princess Grace Hospital Centre, Monaco (Chairman)
Dr. Z. Poljaković, Neurologist and Head, Neuropsychiatric Dispensary, Trnje Public Health Centre, Zagreb, Yugoslavia
Dr. J. Richard, Chief, Cardiological Department, National Institute of Health and Medical Research, Boulogne-sur-Seine, France
Dr. H. A. Schreuder, Director, Department for Geriatry and Rehabilitation, General Hospital "De Zonnestraal", Hilversum, Netherlands
Dr. C. J. Schwartz, Professor, Department of Pathology, McMaster University, Hamilton, Ontario, Canada
Dr. I. Shigematsu, Chief, Department of Epidemiology, Institute of Public Health, Tokyo, Japan
Dr. E. Skinhøj, Professor of Neurology, and Chief, Neuromedical Department, Bispebjerg Hospital, Copenhagen, Denmark
Dr. E. V. Smidt, Professor of Neurology, and Director, Institute of Neurology, Academy of Medical Sciences of the USSR, Moscow, USSR
Dr. G. Tibrain, Medical Clinic I, Sahlgren’s Hospital, Gothenburg, Sweden (Rapporteur)
Dr. G. Verdunck, Professor and Chairman, Department of Internal Medicine, and Director, Department of Gerontology, Faculty of Medicine, State University of Ghent, Belgium

REPRESENTATIVES OF OTHER ORGANIZATIONS:

European Society of Cardiology, International Society of Cardiology:
Dr. P. Pich, Professor, Faculty of Medicine, Montpellier University, France

OBSERVERS:

Dr. C. Fumagalli, Professor, Institute of Gerontology and Geriatrics, University of Florence, Italy
Dr. P. Harmsen, Sahlgren’s Hospital, Gothenburg, Sweden
Dr. J.-P. Marc-Vergnes, Laboratory for the Haemodynamics and Metabolism of the Brain, Hôpital Purpan, Toulouse, France

SECRETARIAT:

Dr. Z. Fejfar, Chief, Cardiovascular Diseases, WHO, Geneva, Switzerland
Dr S. Hatano, Medical Officer, Cardiovascular Diseases, WHO, Geneva, Switzerland (Co-Secretary)
Dr L. Lataillede, Director of Health Services, WHO Regional Office for Europe, Copenhagen, Denmark
Dr A. May, Regional Officer for Mental Health, WHO Regional Office for Europe, Copenhagen, Denmark
Dr B. Ogun, Regional Adviser, WHO Regional Office for Africa, Brazzaville, People's Republic of the Congo
Dr Z. Pisa, Regional Officer for Chronic Diseases, WHO Regional Office for Europe, Copenhagen, Denmark (Co-Secretary)
Dr R. Strudwick, WHO Regional Office for South-East Asia, New Delhi, India
Annex 2

A SUGGESTED CLASSIFICATION OF CEREBROVASCULAR DISEASE *

I. Clinical stage
   A. Asymptomatic
   B. Focal cerebral dysfunction
      1. Transient attacks
      2. Actively changing neurological deficit
      3. Prolonged neurological deficit
   C. General cerebral dysfunction
      1. Transient
      2. Prolonged
         (a) Acute onset
         (b) Gradual progression

II. Pathophysiological mechanisms
   A. Abnormalities of cerebral circulation
      1. Thrombosis
      2. Embolism
      3. Compression
      4. Vasospasm
      5. Direction
      6. Alteration in rate
      7. Dissection of arterial wall
      8. Associated with arteriography
   B. Abnormalities of general circulation
   C. Haemorrhage
   D. Alteration in blood
   E. Alteration of metabolic demands

* Prepared by the Ad Hoc Committee on Classification and Outline of Cerebrovascular Diseases, established by the Advisory Council of the National Institute of Neurologic Disease and Stroke, National Institutes of Health, Bethesda, Md., USA.
F. Possibly predisposing factors
G. Unknown

III. Anatomy

A. Blood vessels

1. Arteries
   (a) common carotid artery
   (i) ophthalmic artery
   (ii) posterior communicating artery
   (iii) anterior cerebral artery
       anterior communicating artery
   (iv) middle cerebral artery
   (b) subclavian artery
       vertebral artery
   (c) basilar artery
       posterior cerebral artery

2. Arterial collateral circulation

3. Veins
   (a) dural sinuses

B. Brain and spinal cord

1. Meninges
   (a) epidural space
   (b) subdural space
   (c) subarachnoid space

2. Brain
   (a) frontal lobe
   (b) temporal lobe
   (c) parietal lobe
   (d) occipital lobe
   (e) central white matter
   (f) internal capsule
   (g) thalamus
   (h) midbrain
   (i) pons
   (j) medulla
   (k) cerebellum
(l) cranial nerves
(m) cerebral ventricles

3. Spinal cord

IV. Pathology

A. Pathological alterations in vessels

1. Arteries
   (a) congenital, developmental, and inherited lesions
       (i) congenital aneurysms
       (ii) congenital aneurysm, ruptured
   (b) Inflammatory lesions (arteritides)
       (i) infections
       (ii) non-infections
           cranial arteritis (temporal arteritis)
   (c) trauma and physical agents
       (i) trauma to artery due to external forces
       (ii) trauma due to angiography
       (iii) trauma due to catheterization and other intra-arterial procedures
       (iv) trauma due to surgery
   (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
       anticoagulants
   (g) arterial embolism due to cardiac disease and disease of extracerebral vessels
       (i) cardiac arrhythmias (specify basic disease)
       (ii) valvular disease
       (iii) myocardial infarction
   (h) arterial lesions associated with neoplastic disease
   (i) arterial lesions due to unknown causes
       (i) atherosclerosis
       (ii) atherosclerotic stenosis
       (iii) atherosclerotic occlusion
   (f) arterial lesions associated with hypertension

2. Veins

3. Capillaries
4. Combined arterial, venous, and capillary abnormalities
   (a) vascular malformations
   (b) lesions due to unknown causes

B. Pathological alterations in brain
   1. Infarction (pale, haemorrhagic, and mixed) encephalomalacia due to circulatory disturbance
      (a) without vessel stenosis or occlusion
      (b) with arterial stenosis or occlusion associated with: (list as under IV. A.1)
      (c) with venous stenosis or occlusion
      (d) with capillary lesions
      (e) with combined arterial, venous, and capillary lesions

   2. Haemorrhage
      (a) without identification of vessel type
      (b) of arterial origin (list as under IV.A.1)
      (c) of venous origin
      (d) with capillary lesions
      (e) with combined arterial, venous, and capillary lesions

Alternative classification, section IV.B

B. Pathologic alterations in brain
   1. Infarction (pale, haemorrhagic, and mixed) (encephalomalacia due to circulatory disturbance)
      (a) without vessel stenosis or occlusion
      (b) with arterial stenosis or occlusion associated with: (list as under IV.A.1)

   2. Haemorrhage
      (a) without identification of vessel type
      (b) of arterial origin (list as under IV.A.1, or use terms as follows)
         (i) intracerebral
             a. with hypertension
             b. without hypertension
         (ii) subarachnoid
         (iii) subdural
         (iv) epidural
         (v) intraventricular

V. Clinical phenomena (history, physical examination, laboratory examination, other)

A. Demographic

B. Family history

C. Past history
D. Present illness
   1. Transient attacks
   2. Actively changing neurological deficit (progressing stroke)
   3. Prolonged neurological deficit (reversible neurological deficit and completed stroke)

E. Physical examination
   1. General
   2. Neurological
   3. Vascular

F. Laboratory examination

G. Roentgen examination

H. Special procedures

VI. Performance and placement status

A. Performance
   Class I. Fully independent activities of daily living. Normal avocational activities. Able to return to previous living site and occupation without modification.
   Class II. Semi-dependent (requiring some assistance) in activities of daily living, and/or slight restriction of avocational activities, and/or able to return to previous occupation with some modification of the latter.
   Class III. Semi-dependent (requiring lifting assistance) in activities of daily living, and/or considerable restriction of avocational activities, and/or unable to return to previous occupation and must seek selective occupation.
   Class IV. Fully dependent activities of daily living, and/or no avocational activities, and/or no occupational capability.

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 helpers, and/or regularly available medical care.
   Class D. Severe limitation: requires constant or nearly constant attendance, and/or immediately available medical-nursing care.
Annex 3

GLOSSARY OF TERMS *

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tr>
<td>Acute but ill-defined cerebral vascular disease</td>
<td>An imprecise clinical diagnosis used to describe any acute attack of cerebral vascular disease when the underlying pathological change in the brain is not determined.</td>
</tr>
<tr>
<td>Apraxia</td>
<td>Inability to carry out a purposive movement, the nature of which is understood by the patient, without any severe motor paralysis, sensory loss, or ataxia. Any movement that is normally voluntarily initiated can be involved. Apraxia may be due to a loss of ideational concept of the action and/or loss of kinesthetic memory of the action.</td>
</tr>
<tr>
<td>Arteriosclerotic dementia</td>
<td>Mental deterioration caused by generalized arteriosclerosis of the brain.</td>
</tr>
<tr>
<td>Arteriovenous aneurysm, arteriovenous malformation</td>
<td>Direct connexion between artery and vein; this may be a developmental anomaly, or may be due to trauma or infection.</td>
</tr>
<tr>
<td>Autoregulation of cerebral circulation</td>
<td>See footnote, page 22.</td>
</tr>
<tr>
<td>Brain scan</td>
<td>Visual display showing the localization and concentration of gamma-ray-emitting radiisotope in the brain.</td>
</tr>
<tr>
<td>Cerebral embolism</td>
<td>Sudden blocking of a cerebral artery by a clot or other material brought to the point of blockage by the bloodstream. The term also refers to the resulting clinical disease.</td>
</tr>
<tr>
<td>Cerebral haemorrhage</td>
<td>See “Intracerebral haemorrhage”.</td>
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<tr>
<td>Cerebral infarction</td>
<td>Ischaemic necrosis of a part of the brain caused by obstruction of the perfusing artery by a thrombus or by emboli.</td>
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<tr>
<td>Cerebral thrombosis</td>
<td>Formation, development, or presence of thrombus in a cerebral artery, and the resultant clinical disease.</td>
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<tr>
<td>Cerebrovascular accident, cerebrovascular event</td>
<td>Terms used to cover different types of acute cerebrovascular disease, including transient cerebral ischaemia.</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>Disease due to impairment of circulation to and in the brain.</td>
</tr>
<tr>
<td>Cerebrovascular lesion</td>
<td>Pathological change in the brain caused by impaired circulation.</td>
</tr>
<tr>
<td>Completed stroke</td>
<td>Stage of a stroke when signs and symptoms are fully developed and stable for hours or days, subsequently tending to decrease.</td>
</tr>
<tr>
<td>Disability</td>
<td>Reduction in functional ability to lead a fruitful daily life; it is the result not only of mental and/or physical impairment but also of the individual's adjustment to this.*</td>
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* The definitions given in this glossary refer only to the sense in which the terms are used in this report and do not necessarily have more general application.

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<thead>
<tr>
<th>Term</th>
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<td>Echoencephalography</td>
<td>Record of reflected ultrasound pulses from intracranial structure.</td>
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<tr>
<td>Epidural haematoma</td>
<td>Accumulation of blood between the dura mater and cranium.</td>
</tr>
<tr>
<td>Generalized ischaemic</td>
<td>Chronic reduction of various mental functions and/or gradual development of neurological signs and symptoms commonly associated with acute cerebral thrombosis.</td>
</tr>
<tr>
<td>cerebrovascular disease</td>
<td></td>
</tr>
<tr>
<td>Half-way house</td>
<td>Accommodation provided for handicapped patients to allow them to make a gradual and smooth transition from hospital to home.</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>An increase in the volume of cerebrospinal fluid within the skull, due either to excess fluid compensating brain atrophy or to a disturbance of the formation, circulation, or absorption of the fluid.</td>
</tr>
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<td>Hypertensive encephalo-</td>
<td>An acute episode due to a sudden marked rise in arterial blood pressure and characterized by severe generalized headache, nausea, and vomiting, often with confusion, convolution, and stupor or coma. It is seen in patients with severe hypertension of any etiology, and is more common in renal hypertension or eclampsia.</td>
</tr>
<tr>
<td>pathy</td>
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<tr>
<td>Impairment</td>
<td>A permanent or transitory pathological condition resulting in a diminution of function.</td>
</tr>
<tr>
<td>Incidence</td>
<td>The number of new cases occurring in a given population during a given period of time.</td>
</tr>
<tr>
<td>Intracerebral haemorrhage</td>
<td>Haemorrhage originating from a vessel in the brain parenchyma. The term is used both for the pathological condition and for the resulting clinical disease. The condition is usually hypertensive in origin, but sometimes occurs as a result of ruptured arteriovenous aneurysm in the brain.</td>
</tr>
<tr>
<td>Korsakoff's syndrome</td>
<td>A symptom cluster, whose characteristic feature is a disturbance of attention and memory leading to disorientation of the patient in space and time. Memory of recent events is lost and the gap is filled by a confabulation, i.e., the invention of a purely imaginary past.</td>
</tr>
<tr>
<td>Limbic system</td>
<td>The part of the brain that gives an emotional colouring to sensory experience, and serves to maintain patterns of behaviour. This is a part of the brain that developed early in the evolution of vertebrates. The system includes the olfactory bulb, mamillary body of the hypothalamus, fornix, amygdala, hippocampus, and other neural connections.</td>
</tr>
<tr>
<td>Major attack, major stroke</td>
<td>Severe type of acute cerebrovascular disease, usually a cerebral haemorrhage or cerebral infarction.</td>
</tr>
</tbody>
</table>

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*b* For further discussion of the terms "incidence" and "prevalence" see Bull. *Wild Hlth Org.*, 1966, 35, 783.
Morbidity

The ratio of the total number of patients with a given disease to the total population. It is expressed either as prevalence at a given time or as incidence during a given period.

Mortality

The ratio of the total number of deaths from a given disease to the total population.

Multiphasic screening

Checking on the health or "risk" condition of large groups of people by using two or more screening tests.

Neurological deficit

An impairment of neurological function.

Night hospital

Hospital for night-time care only.

Parkinsonism

A disturbance of motor function characterized chiefly by slowing down and encroachment of emotional and voluntary movement, muscular rigidity, and tremor. This may be produced by a number of different pathological conditions and is usually ascribed to lesions involving the substantia nigra and/or corpus striatum.

Precerebral arteries

Common carotid, internal carotid, vertebral and basilar arteries.

Prevalence

The number of cases of a condition or disease in a defined population at a particular point in time ("point prevalence") or in a stated period of time ("period prevalence").

Progressing stroke

Acute stage of a stroke where focal neurological symptoms and signs are increasing in number and/or severity.

Protected residence

Residence provided for frail, aged people with handicaps who can be aided with minimal care and assistance.

Pseudobulbar paralysis

Paralysis or weakness of muscles pertaining to cranial nerves from the medulla oblongata (bulb), due not to lesion in the bulb but to cortical innervation of the nuclei in the bulb.

Rehabilitation

Combined and co-ordinated use of medical, social, educational and vocational measures for training or re-training the individual to the highest possible level of functional ability.

Rigidity

Muscular disorder characterized by a relatively constant resistance to passive movement of the muscles and with almost equal distribution to the flexors and extensors.

Screening

The presumptive identification of unrecognized disease or deficit by the application of tests, examinations, or other procedures which can be applied rapidly. Screening tests sort out apparently well persons who probably have a disease from those who do not.

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\(^a\) For further discussion of the terms "prevalence" and "incidence" see Bull. \(r\)ld Hlth Org., 1966, 35, 783.


**Shoulder-hand syndrome**  
Syndrome composed of pain, stiffness, and marked limitation of motion of the shoulder joint, shoulder girdle, hand, and arm.

**Spasm of cerebral arteries**  
1. Sudden and transitory contraction of cerebral arteries, an extensive spasm usually being accompanied by subarachnoid haemorrhage.  
2. Synonym for transient cerebral ischaemia, whose symptoms were supposed to be caused by focal functional contraction of a cerebral artery.

**Spasticity**  
Continuous contraction of muscles and increased resistance to passive movements, accompanied by exaggerated tendon reflex.

**“Steal” of blood**  
Shift of blood supply through abnormal anastomosis to an area other than that normally irrigated, thus resulting in inadequate supply of blood in the original site.

**Stellate ganglion block**  
Local injection of anaesthetizing drug into the ganglion stellatum.

**Stroke**  
A sudden onset of disturbance of focal brain function due to the blockage or rupture of blood vessels. In the wider sense, it denotes a sudden and severe attack, e.g., heat stroke, apoplectic stroke. Colloquially the term is used for a sudden onset of hemiplegia.

**Subarachnoid haemorrhage**  
Haemorrhage originating from the subarachnoid space. The major cause is rupture of a saccular aneurysm of arteries at the base of the brain or of an arteriovenous aneurysm at the brain surface.

**Subdural haematoma**  
Blood accumulation into the space between the dura mater and the arachnoid membrane of the brain.

**Subhyaloid haemorrhage**  
Bleeding into the subhyaloid space of the eye, which is connected to the subarachnoid space of the brain.

**Transient cerebral ischaemia, transient focal cerebral ischaemia, transient ischaemic attack**  
Sudden occurrence of usually repeated episodes of sensory or motor impairment, caused by temporary inadequacy of blood flow to a localized area of the brain and disappearing completely within 24 hours.

**Vertebrobasilar insufficiency**  
Reversible impairment of brain function caused by inadequate blood supply in the territory of the vertebral and basilar arteries. The parts of the brain involved are the medulla oblongata, pons, mid-brain and cerebellum. Ischaemic symptoms are bilateral sensory or motor disturbances (hemiplegia or hemianesthesia may also occur), blurred vision of both eyes, transient blindness, diplopia, vertigo, nystagmus, facial nerve paralysis, dysarthria, and dysphagia. These may occur singly or in combination, to various degrees.
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