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Part I

Introduction

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1

Recent trends in the epidemiology of stroke

Stroke like other illnesses has a dynamic evolution as a result of increasing knowledge and availability of healthcare around the world. In the 1970s, much of the epidemiological literature documented the declining incidence of stroke from 1945 to 1974. The annual incidence of stroke for both men and women between 1945 and 1949 was 190 per 100,000 population in the Rochester, Minnesota area. In 1970–1974, the rate had dropped to 104 per 100,000 population. During this time, the rates had dropped in all age categories, but were particularly dramatic in the very old. For example, in 1964, the average annual rates for those 55 to 59 years and those 80 years or older were 209 and 2932 per 100,000 persons respectively or a ratio of 1:14. By 1974, the average annual rates for these age groups had declined to 205 and 1287 per 100,000 or a ratio of 1:6. For both men and women the annual incidence rates increased with increasing age from 55 through 80 (Garraway *et al.* 1979).

In Japan, the incidence of cerebral infarction declined by 34% and that of cerebral hemorrhage by 29% between the periods 1961–1966 and 1972–1976 (Ueda *et al.* 1981). This decline was thought to be primarily the result of improved control of hypertension. Beginning in approximately 1980, however, the annual incidence of stroke appeared to increase. In Sweden, there was a 38% increase in the incidence in stroke among women from 1975–1978 to 1983–1985 (Terent 1989). This increasing incidence of stroke has continued throughout the 1990s. Pessah-Rasmussen *et al.* (2003) examined the incidence of stroke in the city of Malmö, Sweden between 1989 and 1998. During this period, the age standardized incidence was 647 per 100,000 person years for men and 400 per 100,000 person years for women. The annual increase, however, was 3.1% in men and 2.9% in women. Among men, that were 50–59, 60–69, and 70–79 years of age, the age standardized stroke incidence increased annually by 4.8%, 4.5%, and 2.1%, respectively. Among women, the corresponding values were 7.3%, 1.8%, and 2.8% (Fig. 1.1(a, b)).

Community based longitudinal studies performed in Minnesota (Brown *et al.* 1996) and Massachusetts (Derby *et al.* 2000), however, found that stroke incidence rates were unchanged in the USA during the past two decades. Nevertheless, in the

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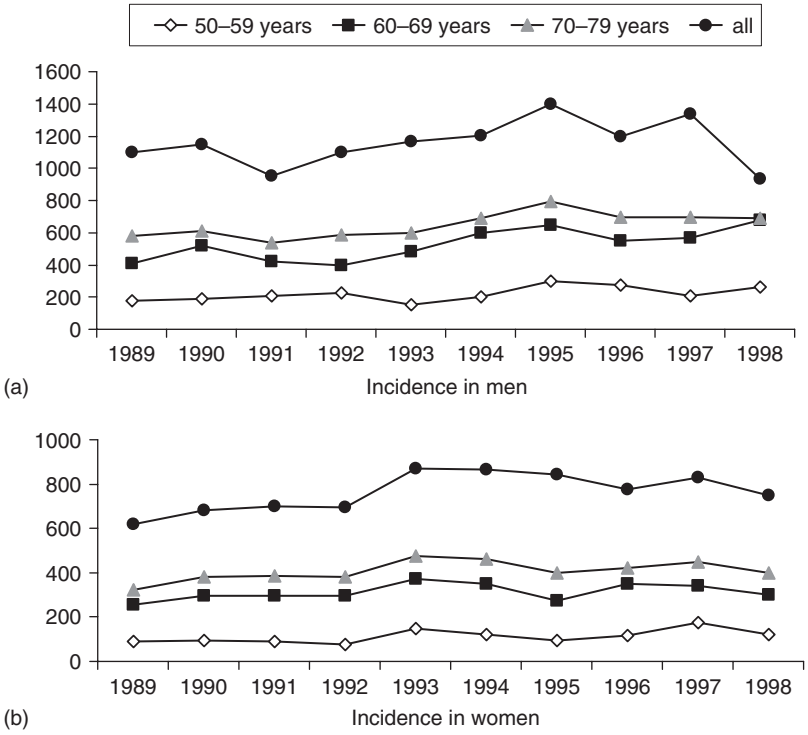

Figure 1.1 (a) Time trends in stroke incidence in Malmö, Sweden, 1989–1998, in men; (b) time trends in stroke incidence in Malmö, Sweden, 1989–1998, in women (reprinted with permission from Pessah-Rasmussen *et al.* 2003).

USA, there are approximately 600,000 new or recurrent strokes occurring each year and, among those who survive, according to the American Heart Association, 70% are left with sufficient disability to limit vocational capacity.

Explanations for the increasing incidence of stroke found in several studies may be related to decreasing mortality from ischemic heart disease, thus theoretically increasing the population at risk for stroke. Other possible reasons for the increasing stroke incidence may be worsening of the cardiovascular risk factors. For example, treatment of hypertension continues to be a challenge in daily practice with only a 50% success rate in treated patients in the USA (Weber 1998). In addition, the increasing number of immigrants to all of the industrialized countries of the West may have contributed to the increasing incidence of stroke.

This increasing incidence of stroke in some countries, which has lasted for approximately two decades, however, has been accompanied by a decline in stroke mortality. Howard *et al.* (2001) analyzed mortality data in the USA between 1968 and 1996. Jefferson County in Alabama, for example, declined from 600 deaths due to stroke per 100,000 population in 1968 to 280 in 1996. Queens County in New York

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City declined from 350 per 100,000 in 1965 to 100 in 1996. In Malmö, Sweden, (Pessah-Rasmussen *et al.* 2003) found that although the mortality rates among men were unchanged from 1989 to 1998 among women there was a significant decline from 12.3% in 1989 to 2.0% in 1998 (odds ratio (OR): 0.89; 95% confidence interval (CI): 0.8–0.95). The decrease in stroke mortality appears to be related to declines in case fatality in the first few weeks after stroke onset. The changes in case fatality within the first 2 weeks following stroke may be related to changes in disease severity or to changes in stroke care. For instance, a large change in fatality was noted in the Malmö study to be related to the opening of a specialized stroke unit. Furthermore, the same factors that affect stroke incidence may also affect stroke severity.

The continuing decline in stroke mortality would be expected to influence the prevalence of stroke survivors in the community. Muntner *et al.* (2002) reported that in the USA, age-, race-, and sex-adjusted prevalence of stroke increased from 1.41% in 1971–1975 to 1.87% in 1988–1994. This represented an average increase of 7.5% for each 5-year period during this time. The prevalence of stroke among US population 60–74 years old increased from 4.2% to 5.2% during the study period. In the USA, the number of stroke survivors was 1.5 million in 1973, 2.0 million in 1978 and 2.4 million in 1991. At the present time the American Heart Association estimates that the prevalence of stroke in the USA is 4 million.

Men with Japanese heritage participating in the Honolulu heart study were compared to non-Japanese heritage men participating in the Framingham, Massachusetts study for risk factors associated with stroke (Rodriguez *et al.* 2002). In both cohorts, hemorrhagic and thromboembolic stroke were consistently elevated in the presence of hypertension and cigarette smoking. In addition, diabetes and body mass index increased the risk of thromboembolic stroke in both samples, while diabetes increased the risk of hemorrhagic stroke only in the Framingham study. Alcohol intake and low total cholesterol were associated with hemorrhagic stroke in Honolulu but not in Framingham. Similarly, the finding from the World Health Organization monitoring of trends and determinants of cardiovascular disease (WHO-Monica project) found that among 35–64 year old people in 15 populations across 9 countries, that systolic blood pressure showed a strong association with stroke trends in women but not in men. In women, 38% of the variation in stroke event trends was explained by changes in systolic blood pressure. However, combining trends in systolic blood pressure, daily cigarette smoking, serum cholesterol, and body mass index explained only a small fraction of the variation in stroke event trends for the entire population (Tolonen *et al.* 2002). These differences from studies that have found an association of stroke with the usual factors of cardiovascular disease may be due to the fact that the Monica study examined only patients up to age 65 and did not differentiate between first episode of stroke and recurrent strokes.

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Just as stroke incidence rates have stopped declining and have been increasing in some studies, the stroke mortality rates are also expected to begin climbing in the next decade.

Elkins and Johnston (2003) estimated that the number of deaths would increase from 139,000 in 2002 to 275,000 in 2032 while the total US population would increase by only 27% in the same time period. These projected mortality rates, however, may be affected by continuing advances in stroke care. Although a detailed description of the advances in stroke care are beyond the scope of this text, the past decade has noted some dramatic improvements in the acute care of patients following stroke. The use of tissue plasminogen activator (TPA) as well as mechanical intravascular removal of thromboembolic infarcts has dramatically improved the outcome of patients who are able to receive treatment within 3 h following an acute stroke. Perhaps one of the most exciting advances in stroke research, however, has been the identification of neuronal regeneration after stroke (Liu *et al.* 1998; Arvidsson *et al.* 2002). Arvidsson *et al.* (2002) have shown that in a rat model of stroke, induced by temporary occlusion of the middle cerebral artery, stroke not only leads to a marked increase in the proliferation of neural progenitor cells in the subventricular zone but also to the migration of these recently regenerated neuroblasts into the damaged striata. Two weeks after the stroke, some of the migrating neuroblasts had differentiated into mature neurons and 5 weeks after the stroke, 42% of the population of cells had differentiated into the dominant type of striatal neuron. Thus, some of the new neurons had locally differentiated into the phenotype of neurons that had been destroyed in the ischemic striatum. None of the new cells, however, were found in damaged cerebral cortex and about 80% of the newly arrived neurons had died within 6 weeks after the stroke. Thus, only 0.2% of the dead striatal neurons were replaced through neurogenesis. Although it will be a challenge to create an environment within ischemic brain tissue to facilitate neuronal migration and differentiation, this newly discovered potential of neuronal regeneration after brain ischemia may represent an important new avenue to achieve functional brain reconstruction and recovery. Furthermore, pharmacological stimulation including antidepressant medications has been shown in animal models to induce neurogenesis. Neurogenesis has been reported primarily in two regions: the subventricular zone and the subgranular zone of the hippocampal dentate gyrus (Gage 2000). Thus, the findings that both cerebral ischemia and antidepressant medications lead to neurogenesis suggest an exciting new area for research and the potential for novel treatments of poststroke physical and cognitive impairments as well as emotional disorders.

In summary, stroke continues to represent one of the major public health problems in the world. It is the third leading cause of death in the industrialized countries of the world and the leading cause of disability and long-term functional

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impairment. Stroke is the second leading cause of nursing home placement, second only to dementia. The amount of healthcare resources devoted to the care of patients with stroke is obviously enormous. Recent advances in the acute care of stroke has led to increased survival but also to increased prevalence of long-term disability. There is a long way to go in eliminating or treating the impairments associated with stroke and advances in pharmacological treatment as well as neurogenesis may provide a whole new avenue for advancement in the care of patients with stroke. This book will be devoted to the assessment and care of the emotional disorders associated with stroke and their impact on long-term survival and recovery. Through mechanisms of neurotransmitter replacement, neuroreceptor regeneration and neurogenesis, we may be on the verge of a whole new era in the treatment of patients suffering from the neuropsychiatric consequences of stroke.

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2

Historical perspective

A historical perspective of the neuropsychiatric disorders associated with stroke must logically begin with a discussion of the word "stroke." Although many clinicians do not like the term "stroke," it has become so ingrained in both the public and professional literature that it will likely remain the primary word used to describe this cerebrovascular disorder.

One of the problems with the word "stroke" is that it has multiple meanings. A stroke of luck or a tennis stroke have very different meanings from the catastrophic medical disorder. Originally the term was used in medicine to describe being struck down by an illness, especially one which rendered the victim unconscious or paralytic (Millikan *et al.* 1987). The use of the word stroke to indicate apoplexy (*plexus* meaning stroke and *apo* meaning from) began in the 17th century. The recognition, however, of apoplexy as an important medical condition is evident from the writings of Hippocrates (460–370 BC). He noted that unaccustomed attacks of numbness and anesthesia were a sign of impending apoplexy and that when patients, who were not febrile, complained of headache, noises in the head, vertigo, slowness of speech, or numbness of the hands, there was a high likelihood that they would become epileptic or suffer from apoplexy (Millikan *et al.* 1987). It was not until the development of the microscope by Leeuwenhoek (1632–1723), however, that an understanding of the nature of stroke occurred. Bichat (1771–1802) was the first to describe cerebral softening and Rostan (1823) identified that brain softening found in victims of apoplexy was not due to inflammation. Hughlings-Jackson (1875) wrote that cerebral softening was always focal, "it is localized by vessels, mostly arteries." Information about the physiology of the cerebral circulation, however, began with the observations of Monro (1783). He reported that the volume of blood in the brain was constant and that blood was continually flowing in through arteries and out through veins. Hughlings-Jackson also noted that the middle cerebral artery, or some of its branches, were almost always the vessels occluded. Furthermore, Hughlings-Jackson distinguished between cerebral softening due to arterial disease and cerebral softening due to embolism.

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The earliest reports of emotional disorders related to stroke were descriptions of patients by neurologists and psychiatrists. Disturbances of language function have frequently been associated with emotional disorders because aphasia is a common and easily recognized behavioral manifestation of dominant hemisphere brain damage. Furthermore, speech is intimately associated with emotions and thoughts. Pierre Paul Broca, a surgeon and anthropologist (1861) described a single case of aphasia in a patient who was able to utter only one syllable "tan." At autopsy the patient was found to have a large lesion of the left hemisphere including the posterior aspect of the inferior frontal gyrus which has been named Broca's area (Fig. 2.1). Carl Wernicke (1874) proposed that aphasia was the result of impairment in elementary psychic processes which were localized in different brain areas. Wernicke described the first patients with impairment in verbal comprehension with lesions involving the temporal occipital–parietal junction of the left hemisphere which has become known as Wernicke's area.

Broca (1878) also first described an area of cortex which he called the limbus cortex that surrounded the midbrain. This included phylogenetically older areas of cortex such as the inferior temporal lobe, hippocampal gyri, cingulate cortex, and

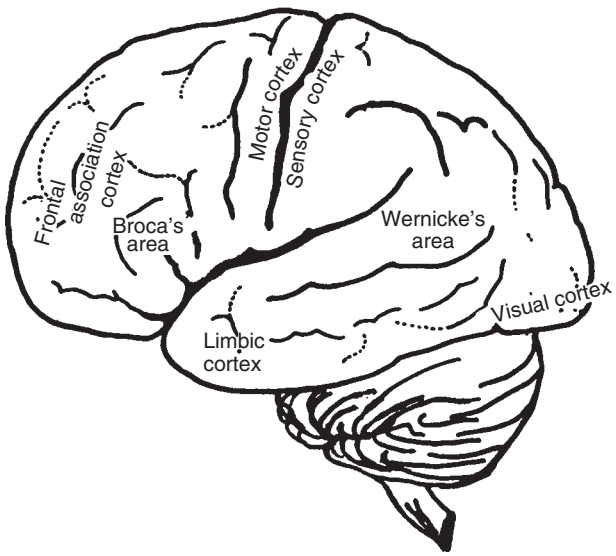

Figure 2.1 Schematic lateral view of the brain indicating functional brain regions. Broca's and Wernicke's areas play an important role in the production and comprehension of language. The anterior basal portion of the temporal cortex is part of the limbic circuit and the frontal association cortex plays an important role in coordinating motor and cognitive functions of the frontal lobe. The motor, sensory, and visual corticis have topographic and columnar organization for focal motor, sensory, and visual functions.

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inferior frontal cortex. He noted that all mammals had this large cerebral convolution which formed a ring or limbus around the lower brain stem structures. Limbus cortex has given rise to the term "limbic system." In 1937, Papez (1937) suggested, based purely on anatomical observations (i.e., the older phylogenetic age of the limbic cortex and its circular anatomical organization), that this limbus cortex or limbic system formed the anatomical basis for emotion (this proposal will be discussed more fully in Chapter 3).

Some of the earliest writings about the cerebral basis of emotion, however, were published by Hughlings-Jackson (1915). Hughlings-Jackson, widely regarded as the father of English neurology, was appointed to the National Hospital in 1862. Jackson was the first to identify that brain lesions were associated with a duality of symptoms. That is, brain lesions produced both a loss of specific function as well as a positive symptom such as a movement disorder or a mental process not previously seen. He explained this emergence of "positive symptoms" as a result of removal of higher controls normally inhibiting these functions. This culminated in his view that there was a hierarchy in the organization of the nervous system with the prefrontal cortex mediating the highest level of mental function.

Similar to his concept of duality of symptoms associated with brain injury, Hughlings-Jackson conceptualized language as an expression of brain function existing in two forms. Language was either intellectual (conveying content) or emotional (expressing feelings). He proposed that these components might be separated by disease. This was the first suggestion that emotional expression was distinct from spoken language and that stroke might produce disorders of emotion without producing disorders of language function.

Adolph Meyer (1904), on the other hand, conceptualized emotion, as he did psychopathology, as a response to multifaceted influences including both physiological and mental processes that he referred to as psychobiology (Lewis 1957):

> In the concept of emotion one is able to see most clearly the basic psychobiological premise of the unity of "mental" and "bodily" functions. Emotion as, in fact, the middle ground serves as a striking refutation of an artificial parallelism. An emotion is a reaction which by its very nature covers the entire range of human reactivity, including in the response the non-conscious vasomotor and other organic changes as well as the conscious feelings of rage, joy, etc. In fact, it is in the emotion, permeating and coloring as it does the entire psychobiological activity that one has most definitively and convincingly the feeling of self.

Based in part on his background in neuropathology, Adolph Meyer became very interested in focal brain injury associated with trauma. He stated, "considering the meager knowledge of unmistakably reliable facts of etiology in mental disease, traumatism would seem to furnish unusually clean-cut conditions of interference with the mechanisms of motor sensory plasticity. The first question is: has the localization

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