Mechanisms and management of stroke in the elderly

Ashfaq Shuaib, MD, FRCPC; Vladimir C. Hachinski, MD, FRCPC, DSc (Med)

Objective: To highlight the mechanisms, common causes and management of stroke in the elderly.

Data sources: MEDLINE was searched for articles published from 1967 to 1990. The following key words were used: "stroke," "cerebrovascular disease," "elderly," "aging," "hypertension," "drug interactions," "etiology," "evaluation," "management" and "recovery of function." Original articles with large series of patients were reviewed in detail.

Study selection: Of about 750 original articles reviewed 116 were finally selected for detailed analysis. Those that dealt with cause, pathophysiologic features and management of stroke with emphasis on the elderly were chosen.

Data synthesis: With increasing age the incidence of stroke increases, cardiovascular reserve decreases, catecholamine responsiveness diminishes and cardiac arrhythmias become more common. Blood pressure, especially systolic, rises, and the benefits of its treatment become both more difficult to assess and less certain. In the elderly population embolic stroke, particularly that due to nonvalvular atrial fibrillation, is seen with increasing frequency. Because of postural hypotension, cardiac arrhythmias and overmedication, watershed infarction occurs more frequently with increasing age. Amyloid angiopathy now represents the most common cause of spontaneous intracerebral hemorrhage.

Conclusions: Because of the altered drug metabolism and pharmacodynamics in the elderly the therapeutic armamentarium is growing, and so are the risks of such treatments. Stroke in the elderly poses unique problems that deserve distinctive solutions. Further research is needed to study the effect of cerebral ischemia to understand better how the older brain handles the stress of the ischemic insult.

Objectif : Souligner les mécanismes, les causes habituelles et le traitement de l'accident cérébrovasculaire chez les personnes âgées.

Sources de données : MEDLINE a fait l'objet d'une recherche sur des articles publiés de 1967 à 1990 au moyen des mots clés suivants : «accident cérébrovasculaire», «affection vasculaire cérébrale», «personnes âgées», «vieillissement», «hypertension», «interactions médicamenteuses», «étiologie», «évaluation», «traitement» et «rétablissement de fonction». Les articles originaux sur d'importantes séries de malades ont été examinés en détail.

Sélection d'études : Sur environ 750 articles originaux examinés, en définitive, on en a choisi 116 pour une analyse détaillée. Ceux qui ont été choisis traitaient de la cause, des aspects physiopathologiques et du traitement de l'accident cérébrovasculaire, en particulier chez les personnes âgées.

Synthèse de données : En fonction du vieillissement, on observe une augmentation de l'incidence de l'accident cérébrovasculaire, une diminution de la réserve cardiovasculaire, une baisse de la réactivité aux catécholamines et une plus grande fréquence des arythmies cardiaques. La tension artérielle augmente, en particulier la tension systoli-

Dr. Shuaib is the director of the Clinical Investigative Stroke Unit, Royal University Hospital, Saskatoon, Sask., and Dr. Hachinski is a career investigator of the Heart and Stroke Foundation of Ontario, Toronto, Ont.

Reprint requests to: Dr. Ashfaq Shuaib, Division of Neurology, Royal University Hospital, Saskatoon, SK S7N 0X0

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que, et les avantages du traitement deviennent plus difficiles à évaluer et moins certains. Chez les personnes âgées, on observe une fréquence accrue de l'embolie, en particulier celle qui est causée par une fibrillation auriculaire non ventriculaire. En raison de l'hypotension posturale, des arythmies cardiaques et de la surconsommation de médicaments, l'infarctus critique survient plus fréquemment en fonction du vieillissement. L'angiopathie amyloïde représente maintenant la cause la plus fréquente d'hémorragie intracérébrale spontanée.

Conclusions : En raison de l'altération du métabolisme et de la pharmacodynamique des médicaments chez les personnes âgées, l'arsenal thérapeutique est en train d'augmenter, et il en va de même des risques de tels traitements. L'accident cérébrovasculaire chez la personne âgée pose des problèmes particuliers qui méritent des solutions distinctes. Il faut poursuivre la recherche sur l'étude de l'effet de l'ischémie cérébrale pour mieux comprendre comment le cerveau des personnes âgées réagit au stress de l'accès ischémique.

Stroke and coronary artery disease account for over half the deaths in North America.[1] Age is the most important risk factor. The incidence of stroke is 30 per 100 000 population before 44 years of age and more than 1230 per 100 000 after 75 years.[2] With increasing age the vascular response to stress and injury changes, multiple organ dysfunction becomes a problem, and consumption of multiple medications is common. Combined, these problems magnify the insult from stroke, increase the number of complications and may delay recovery. Therefore, management of stroke in the elderly poses special problems that need careful attention.

In this review we describe the pathophysiologic features of the cardiovascular changes associated with aging, highlight the effect of the metabolic change and increased propensity for drug interactions on management, identify certain types of stroke that occur with increased frequency in the elderly and outline an approach to evaluate and manage acute stroke in this population. A detailed description of the pathophysiologic changes is necessary because cardioembolism and watershed infarction are significantly more common in the elderly than in younger groups and are a result of cardiovascular dysfunction. As well, cardiac disease is the most common cause of death in patients with cerebrovascular disease.[3] Similarly, altered metabolism and drug interactions may be directly responsible for major problems with patient evaluation, management and recovery of function after acute stroke.

We searched MEDLINE for relevant articles published from 1967 to 1990 using the key words "stroke," "cerebrovascular disease," "elderly," "aging," "hypertension," "drug interactions," "etiology," "evaluation," "management" and "recovery of function." We read the abstracts of over 750 articles and selected about 250 papers for detailed review. These dealt with aging, drug interactions in the elderly, and stroke evaluation and management with some degree of emphasis on elderly patients. Finally, we reviewed several major monographs that dealt with cerebrovascular disease. For the final selection of the 116 articles cited in this review we restricted ourselves to papers that either were directly relevant to elderly stroke patients or had important bearing on stroke in general.

Pathophysiologic changes

Cardiovascular changes

Cardiac muscle: In disease-free older people aging has no or little effect on cardiac function.[4] In the West, however, over 50% of people over 50 years of age who have died have shown evidence of ischemic vascular disease.[5] The presence of atherosclerosis in one region of the vasculature is associated with an increased prevalence of similar changes in other organs. Also, most patients with stroke have some degree of coronary atherosclerosis.[5]

In the absence of coronary artery disease the cardiac muscle in the aging heart maintains its ability to develop tension, but cardiac responses to β-stimulation decrease strikingly with age. This decreased responsiveness is manifested by a reduced inotropic response, arterial vasodilating response and heart rate.[4,6]

In experimental models with animals the rate of rise and the maximum contraction achieved by cardiac muscles under isometric conditions remains unchanged with aging.[4] However, contraction and relaxation of isolated cardiac muscle are consistently prolonged in senescent animals.[7] Noninvasive studies involving humans have shown a similar prolongation of the duration of contraction and a delayed relaxation phase.[8,9] Although these changes are not of sufficient magnitude to affect the resting cardiac muscles, it is believed that with added ischemic heart disease the delayed relaxation may compromise the subendocardial blood flow. Similar changes could also occur with increased sympathomimetic stimulation during an acute cerebral infarction.[10] The prolonged relaxation may also be responsible for symptoms of arrhythmia (i.e., syncope) at a

lower heart rate. The arrhythmia or syncope can result in cerebral hypoperfusion and may be responsible for the watershed infarction seen with increasing frequency in the elderly.

Arrhythmia: There is a progressive loss of conduction tissue cells and pacemaker function with aging.[11] This predisposes to sinus node dysfunction and abnormal conduction in the atrioventricular node and bundle-branch fibres. In addition, the frequency of supraventricular arrhythmia has been found to be increased among elderly people.[12] These changes may be present without associated ischemic heart disease.

The detection of cardiac arrhythmia has improved with the increased availability of 24-hour Holter monitoring.[13] A few studies have addressed the effect of age on the frequency of cardiac arrhythmia, but little evidence supports the view of this association in the absence of cardiac disease.[13] However, the frequency of cardiac arrhythmia is increased among elderly patients with symptomatic ischemic heart disease (which often coexists with cerebrovascular disease).[14] Cardiac arrhythmia, especially atrial fibrillation, is a common cause of cardioembolic stroke.[15-19] Also, elderly stroke patients with atrial fibrillation have been found to have a poor prognosis.[20]

Valvular heart dysfunction: The occurrence of primary degenerative changes in the aortic valve increases with age, and in the elderly such changes lead to calcific aortic stenosis. This sometimes leads to significant compromise within a short time.[21] The presence of left ventricular hypertrophy differentiates calcific aortic stenosis from benign aortic sclerosis, both of which cause a systolic ejection murmur. If aortic stenosis is suspected a Doppler flow study should be performed to determine the extent of stenosis and the flow gradient. Patients with aortic stenosis are at risk of bacterial endocarditis.

The incidence of mitral valve disease is now less common among elderly people and usually results from ischemic heart disease or myxomatous degeneration. Patients with mitral valve disease are often asymptomatic unless the condition develops suddenly after myocardial infarction. The impact of these valvular changes on cardioembolic stroke is not fully understood but may contribute to its rising incidence with age.

Hypertension

Like cardiac tissue peripheral vasculature becomes increasingly stiff with age and less responsive to β-stimulation.[22] The resultant increase in blood pressure, especially systolic, has been found to peak among patients 75 to 84 years of age.[22-24] In the Framingham study 57% of the men and 65% of the women with hypertension who were 66 to 89 years of age had isolated systolic hypertension (systolic pressure greater than 160 mm Hg and diastolic pressure less than 90 to 95 mm Hg).[22,24] Isolated systolic hypertension has recently become recognized as an important risk factor for both cardiovascular and cerebrovascular disease.[24-26]

Control of hypertension has been an important factor in the decreased incidence of various vascular diseases in the last four to five decades.[27-29] The treatment of hypertension in elderly patients reduces the incidence of death from cardiac and cerebrovascular diseases.[29] However, there is no consensus on which agents are most appropriate for use in the elderly or on the level of reduction of the blood pressure required.

Overzealous or inappropriate treatment of hypertension often leads to serious complications,[30-34] and the protective effects of hypertension therapy may not be as obvious in the elderly. Elderly people may be more prone than younger patients to the side effects, including hypotension. In a study by Carter[31] 97 patients with hypertension and ischemic stroke were randomly assigned to receive antihypertensive therapy or placebo and were followed up for 5 years; among those over 65 years of age there seemed to be no benefit in the treatment group. Meissner, Whisnant and Garraway[35] conducted a community-based study involving 1680 patients with stroke (mean age 72 years), of whom 368 suffered a second stroke during follow-up. The rate of recurrence was unaffected by the presence of hypertension before the stroke or the subsequent management of the hypertension. Finally, in another study 200 stroke survivors over the age of 60 years were randomly assigned to receive therapy with either reserpine and triamterene–hydrochlorothiazide or else placebo and were followed up for 3 years.[36] The reduction in the incidence of stroke did not differ significantly between the two groups.

Stroke as a complication of aggressive treatment of hypertension has been reported and appears to be an important problem among elderly patients admitted with acute stroke. Jansen and associates,[34] through a questionnaire sent to medical directors of nursing units, identified 30 patients who had a completed stroke within days after their hypertension medications had been changed. In a subsequently published prospective study involving 100 patients with stroke or transient ischemic attack they paid particular attention to recent changes (within the previous 3 weeks) in the hypertension medications.[30] They found that such changes had been made in seven cases and felt that they directly contributed to the stroke in at least four of them. In two other studies, one an uncontrolled trial by Jackson and collaborators[32] involving seven patients

and the other a study by Graham[33] involving two patients, stroke developed within 1 week after the start of antihypertensive therapy.

Rapid reduction of blood pressure in the elderly therefore appears to be dangerous, perhaps in part because of the vascular changes associated with aging. In people under 66 years of age a reduction of 25% in the mean arterial pressure is required to reach the lower limit of cerebral blood flow autoregulation, and a further reduction to 55% would result in symptoms of cerebral hypoperfusion.[37] In elderly patients symptomatic hypoperfusion can develop with a reduction of only 25%.[38,39] This suggests that cerebral ischemic damage is more likely to develop in elderly people with the same degree of reduction of blood pressure.

Although evidence is scant the blood pressure should probably be reduced gradually several days after the stroke. In many cases as the stress and sympathetic activity settle down the blood pressure may decrease without any intervention. Acute cerebral ischemic events are frequently associated with blood pressure elevations that tend to settle with time. In a study involving 334 consecutive stroke patients 84% had hypertension (supine blood pressure greater than 150/90 mm Hg) in the initial 24 hours after admission.[40] Only half had a history of hypertension. Within 1 week after admission the blood pressure returned to normal without intervention in two-thirds of the patients.

Pseudohypertension results from poor contraction of stiff atherosclerotic blood vessels and is frequently seen in the elderly. It should be considered when the blood pressure differs significantly between the two arms, there is no evidence of end-organ damage despite a long history of "poorly controlled" hypertension, multiple drugs are ineffective in controlling the blood pressure, and orthostatic problems complicate treatment.[41] It is important to recognize pseudohypertension, because inappropriate treatment may result in dangerous reduction of the cerebral perfusion pressure.

Carotid sinus hypersensitivity

Massage or compression of the carotid sinus in healthy people is frequently associated with slowing of the heart rate and a mild decrease in blood pressure. In some people, especially the elderly, there is marked slowing of the heart rate and severe hypotension; this condition, known as carotid sinus hypersensitivity, can lead to syncope. Diffuse atherosclerosis or other cardiac disease is also common in such patients.[42] The combination of acute slowing of the heart rate and hypotension after stroke can exacerbate the neurologic deficit. Severe cases that do not respond to preventive measures may require medical therapy (with sympathomimetic agents) or pacemaker implantation. Before such aggressive measures are taken a cardiologist should evaluate the condition, because not all types of carotid sinus syndromes respond to pacemakers.[42,43]

Baroreceptor function

One of the main afferent pathways for control of circulatory dynamics originates in mechanoreceptors in the aortic arch and the carotid sinus. As the arterial pressure increases there is increased activation of the nerve fibres arising from these receptors; this results in a decrease in the muscle tone of the blood vessels and in the heart rate. The reverse occurs if the blood pressure suddenly decreases. With aging there is a progressive loss in function of the mechanoreceptors, especially in the aortic arch.[44] A similar decrease in sensitivity also occurs in patients with hypertension.[45]

The mechanism for the lowered sensitivity is not well understood. Some evidence suggests that the decrease may be due to reduced distensibility of the aortic vessel wall at the site of the baroreceptor. There has been autopsy evidence of such decreased distensibility of the carotid sinus.[46] Physicians should realize the clinical importance of the decreased baroreceptor sensitivity when they consider lowering the blood pressure in elderly patients with acute stroke.

Cerebral autoregulation

There is a gradual decrease in the cerebral blood flow with increasing age. Davis and colleagues,[47] in a study of cerebral blood flow and carbon dioxide reactivity involving 55 volunteers aged 18 to 88 years, found a definite decrease in the blood flow in the cerebral grey matter with increasing age. Shaw and coworkers[48] suggested that this age-related decrease occurs more rapidly in people with risk factors for stroke than in others. They found the blood flow to be significantly decreased in people with cerebrovascular risk factors.

Two mechanisms have been proposed to explain this age-related decrease in blood flow: (a) a progressive loss of neurons and (b) a loss of hemodynamic reserve. Further decrease in cerebral blood flow in patients with cerebrovascular disease may mean an added loss of neurons. In addition, positron-emission tomography has shown that a small lesion in the cortex (related to cerebral ischemia) can severely decrease the blood flow.[49]

Failure of cerebral autoregulation can result in symptomatic cerebrovascular ischemic disease. In a study involving 11 elderly patients with postural hypotension Wollner and associates[50] found 7 with

relatively minor hypotension who had symptoms of cerebral ischemia. All seven had altered cerebral autoregulation. In the other four patients the cerebral autoregulation was intact. Patients with acute stroke have been found to have altered cerebral blood flow:[51] the cerebral blood vessels are maximally dilated, and any decrease in blood pressure may then result in serious compromise.[52] This should be kept in mind when hypertensive patients with acute stroke are given medication to lower their blood pressure.

Altered metabolism and drug interactions

Drug pharmacokinetics

Most of the information on the efficacy and adverse effects of medications comes from experiments involving young volunteers.[53] The assumption that the drug will have similar effects in the elderly can be potentially dangerous. Because gastric acidity, gastric emptying, the intestinal absorptive surface, liver size and blood flow are affected by aging, absorption and elimination of commonly used medications can be particularly unpredictable in the elderly.[53] In addition, drug distribution can be similarly affected, for a variety of reasons, including decreased total body size and lean body mass, increased body fat stores and declining renal function.[53]

With the decrease in the plasma albumin level drugs such as warfarin that are strongly bound to plasma proteins can become increasingly "free and unbound" and cause "overanticoagulation" and potentially serious bleeding. The anticoagulant effect of warfarin has been found to be higher in the elderly than in other age groups.[54] Elderly patients with cardioembolic stroke often receive warfarin and therefore need careful monitoring.

Other commonly prescribed drugs whose metabolism may be significantly altered with aging include certain antihypertensives (e.g., guanethidine), cardiac glycosides, benzodiazepines, hypnotics (barbiturates), major tranquillizers and tricyclic antidepressants.[53,55] Many such medications, including diazepam,[56] haloperidol[57,58] and phenytoin,[59] have been shown to slow recovery of function after experimental brain injury. Early clinical data suggest that similar effects may be seen after acute stroke.[60]

Altered pharmacodynamics

Pharmacodynamics involves the measurement of the duration and intensity of a drug's effects at a particular concentration.[38] Several age-related changes have been noted in homeostatic mechanisms, including derangement in postural control,[61] thermoregulation,[62] visceral muscle function and orthostatic circulatory responses.[63,64] These changes are believed to lead to a gradual reduction in homeostatic reserve — in other words a diminished ability to adapt to changing environmental stresses.[38] In terms of pharmacodynamics this means that there is a general decrease in handling the effects of drugs at their target organs.

There is an increase in the corrective responses to standing up as a person grows older. The mechanisms are not well understood but may be related to striatal dysfunction with a reduction in D_2 receptors in the basal ganglion. This results in an increased risk of postural instability and falls with the use of sedatives. Epidemiologic data have shown an association between the use of sedatives and an increased incidence of falls in the elderly.[38]

Orthostatic circulatory responses are important in maintaining normal blood pressure when a person rises to an upright position. With increasing age such reflexes slow, the result being a postural fall in blood pressure of over 20 mm Hg in 17% of elderly people and over 40 mm Hg in 5%.[63,64] These effects become particularly important in those taking drugs to control hypertension.[45] Other medications such as phenothiazines, tricyclic antidepressants, antihistamines and barbiturates that are commonly used in patients with acute stroke can similarly affect orthostatic postural responses.[38]

Elderly people have an increased sensitivity to benzodiazepines. Several studies have shown changes in cognitive function with commonly used compounds such as lorazepam,[65] diazepam,[66] nitrazepam[67] and temazepam[38] in elderly patients, even though there appeared to be no differences between the young and elderly patients in the serum levels of the drugs. The mechanism for this age-related difference is not well understood; however, a decrease in central γ-aminobutyric acid receptors, a redistribution in the regional concentration of benzodiazepines in the brain and an alteration in postreceptor function have all been considered.[38]

Angiotensin-converting-enzyme inhibitors are now becoming the drug of choice in the management of hypertension and are frequently prescribed for the elderly.[38] The fall in blood pressure associated with enalapril has been found to be greater in elderly patients than in younger volunteers.[68] The lower rate of elimination of enalaprilat (an active metabolite of enalapril) in the elderly accounts for the greater activity of a given dose.[68]

Adverse drug reactions

About 10% of all admissions to geriatric units are because of drug reactions.[69] However, this may be the tip of the iceberg, as physicians tend to

underreport such complications.[70] The incidence of drug reactions increases with age, and nearly identical trends have been reported in the United States,[71] Germany,[72] Israel,[72] Northern Ireland[73] and Scandinavia.[74] Physicians must beware of such problems developing in acutely ill elderly patients with cerebrovascular disease.

Risk factors of adverse reactions include female sex, polypharmacy, previous reactions and progressive decrease in cognitive function with increasing dosage.[75]

Stroke types

Stroke with atrial fibrillation

The frequency of nonvalvular atrial fibrillation increases with age. In Denmark its prevalence was found to be 4% among people over the age of 60 years.[76] In the Framingham study its prevalence increased from 0.2 per 1000 among people 30 to 39 years to 19 per 1000 among those 74 to 84.[14] The incidence of stroke among those with nonvalvular atrial fibrillation was 6.7% among those 50 to 57 years and 36.2% among those 80 to 89.[14] In most cases the mechanism is cardioembolism, and the risk appears to be highest among patients with a large left atrium and paroxysmal atrial fibrillation.[16] Although the diagnosis is easy in most cases, patients with paroxysmal atrial fibrillation may require Holter monitoring for 24 hours or longer.

Until recently most elderly patients with nonvalvular atrial fibrillation and no neurologic complications were simply observed and not given anticoagulants. In two recent reports[15,77] the risk of embolism seemed to outweigh the risk of complications from anticoagulant therapy. Petersen and collaborators[15] reported that therapy with acetylsalicylic acid (ASA), 75 mg/d, apparently failed to reduce the risk, whereas therapy with warfarin resulted in a significant reduction. In the second study[77] the incidence of cardioembolic stroke was significantly lower in the group given 300 mg of ASA daily than in the group given a placebo. The same group is currently comparing the effectiveness and safety of ASA and warfarin. Until the results of the clinical trial are available we suggest that most patients with isolated atrial fibrillation be given warfarin and their prothrombin time monitored closely. ASA should be used in patients with relative contraindications to warfarin. Careful follow-up is necessary to avoid complications.

Stroke with other types of heart disease

Cardioembolic stroke accounts for 15% to 25% of ischemic stroke. The incidence of cardioembolic stroke increases with age. Data from the Stroke Unit, Toronto, showed that the incidence of ischemic heart disease and congestive heart failure was significantly increased among patients with ischemic stroke over 65 years of age (V.C.H.: unpublished data). In addition, stroke patients were found to have a significant increase in potential cardiac sources of stroke with increasing age.[78-80] Early recognition of a cardiac source is important, because anticoagulant therapy may prevent recurrences.[81,82]

Hypotension and watershed infarction

Watershed infarction occurs between two main arterial territories. Although bilateral infarction frequently develops after severe hypotension, unilateral watershed infarction can occur in patients with severe ipsilateral carotid disease and hypoperfusion. Such infarction is common in patients with cardiac dysfunction or postural hypotension. Bogousslavsky and Regli[83] found that 75% of 51 patients with unilateral watershed infarction had severe cardiac disease, hypotension or an increased hematocrit in addition to significant ipsilateral carotid stenosis. Correct diagnosis and management of the underlying cardiac disease or hypotension can sometimes prevent recurrences.

Amyloid and stroke or cerebral hemorrhage

Cerebral amyloid angiopathy is unrelated to systemic amyloidosis, and its incidence rises with age. It may be seen in over 90% of patients with advanced Alzheimer's disease.[84] Patients with cerebral amyloid angiopathy may remain asymptomatic or present with cerebral hemorrhage. The condition may account for 4% to 10% of cases of cerebral hemorrhage and is the most common cause of spontaneous hemorrhage in people over the age of 65 years.[85] The hemorrhage tends to recur and be lobar in distribution, at the junction of the grey and white matter, and is commonly located in the parietal or occipital lobes. Some patients with cerebral amyloidosis may also present with a small infarction. Treatment is currently symptomatic, and recurrences cannot be prevented.[84]

Cancer-related stroke

According to autopsy findings in one study, cerebrovascular disease was found in up to 30% of patients with systemic cancer;[86] after cerebral metastases it accounts for most neurologic findings. In another study the commonest mechanism was nonbacterial thrombotic endocarditis (in 18.5% of 500 patients at autopsy), the next commonest being intravascular coagulation (in 9.6%).[87] In cancer pa-

tients with cerebrovascular manifestations the clinical presentation is more often one of diffuse encephalopathy and can therefore be easily missed.[87] Also, such patients have an increased risk for subdural hematomas.[88] Since the incidence of cancer increases with age, cancer-related stroke should be suspected in cancer patients when progressive encephalopathy develops, especially with associated focal signs.

Management of elderly stroke patients

Evaluation[89]

Since cerebrovascular disease is a systemic problem a complete history and physical examination, especially of the neurologic and cardiac systems, is essential in all age groups. It is important to obtain details of currently used medications from elderly patients; information from other family members or even referring physicians is often needed, because the patients may be unable to give details. The blood pressure should be checked with the patient lying and sitting to detect any postural hypotension. Because of altered cerebral autoregulation in the elderly, such fluctuations in blood pressure could predispose to cerebral damage. Elderly people often have compromised cognitive function, and an acute stroke may lead to further deterioration. Patients who become severely confused in the "new environment" of the hospital may require urgent medical attention. The cardiac rhythm must be carefully assessed, and because of an increased incidence of multiple-organ dysfunction with increasing age careful attention should be paid to such organs as the lungs, the intestines, the kidneys and the endocrine organs. Since neoplasia is associated with a hypercoagulable state and secondary ischemic or hemorrhagic stroke, the examination should include the search for any evidence of systemic cancer.

An increase in the serum cholesterol and lipoprotein levels has been linked with an increased risk of cardiovascular disease.[90-92] Fasting blood levels of cholesterol, low-density-lipoprotein cholesterol and high-density-lipoprotein cholesterol are therefore routinely measured in patients with ischemic heart disease. Since no relation between abnormal serum lipoprotein levels and stroke has been found[93-95] we believe that routine checking of those levels is of limited value in elderly stroke patients.

Investigation[89]

In large stroke units the diagnosis "acute stroke" may be incorrect in up to 13% of cases.[96] Conditions frequently confused with acute ischemic infarction include seizure with Todd's paralysis, brain tumour and subdural hematoma. During the initial 48 hours after presentation tests are done to establish the exact diagnosis and to define a possible cause. In all cases routine investigation (regardless of age) should include a complete blood count, measurement of serum electrolyte levels, kidney and liver function tests, measurement of blood glucose levels (random and fasting), urinalysis, electrocardiography with a rhythm strip and chest radiography.

In selected cases of pulmonary disease, blood gas values should be determined. The incidence of cardiac disease may be significantly higher among elderly patients than among those less than 65 years of age;[78-80] arrhythmias and congestive heart failure are the cardiac diseases that cause the most deaths among elderly patients.[97] Echocardiography and Holter monitoring should be performed in most cases, especially those with a history suggesting cardiac disease. The use of transesophageal echocardiography may increase the detection of cardiac abnormalities.[78-80,98]

Neurologic investigation should include computed tomography (CT) of the head within the first 24 hours after presentation (to rule out intracerebral hemorrhage or subdural hematoma). If available, magnetic resonance imaging (MRI) may be preferable because it is more sensitive than CT scanning in detecting an early ischemic infarction.[99-101] MRI may sometimes reveal findings suggestive of embolic stroke and therefore help in management.[101,102] Noninvasive carotid Doppler studies should be done in most cases. In the presence of moderate to severe carotid stenosis or a suspected unusual cause (e.g., vasculitis) cerebral arterial angiography may be performed in otherwise healthy patients less than 80 years of age.

Recent data from the North America Symptomatic Carotid Endarterectomy Trial suggest that carotid endarterectomy may be significantly better than ASA therapy alone in preventing strokes in selected patients with carotid stenosis of 70% to 99%.[103] The role of carotid endarterectomy in cases of partial stenosis of 30% to 69% has yet to be established. In all patients requiring the procedure arterial carotid angiography is essential beforehand for a careful overview of the entire cerebral circulation. Therefore, cerebral angiography should be considered only if surgical therapy is being seriously contemplated. Recently, MRI angiography has shown promise as a clinical application.[104]

The incidence of aspiration pneumonia is high among stroke patients. Recent evidence suggests that aspiration immediately after a stroke may be "silent" and may account for many cases of pneumonia. Unfortunately the type of stroke and clinical evaluation are not helpful in predicting patients at risk for such a complication. Since a modified barium swallow can detect swallowing dysfunction in

most affected people[105] it is routinely used in cases of large cortical infarction at the Clinical Investigative Stroke Unit, Saskatoon. The test is easy to perform and can be done within 2 to 3 days after the onset of stroke. We give our patients fluids intravenously until the test results are available. In a recent study from Duke University Medical Center, Durham, NC, the occurrence of aspiration pneumonia was found to be rare when such a protocol was followed for all patients with acute stroke.[106]

Careful attention must be paid to electrolyte balance and hydration. Cognitive impairment contributes to the problem, and the combination of dehydration and decreased mobility puts the patient at increased risk of deep vein thrombosis.

Medical management[107,108]

Patients should be admitted to a specialized stroke unit or an acute medical care facility, where specially trained nurses can do regular evaluations and are able to detect complications.[89] To decrease the risk of joint contractures and venous thrombosis assessment for occupational and physical therapy should be done early so that the patient can be mobilized as soon as possible. There is no justification for prolonged bed rest after a stroke.

The hazards of lowering the blood pressure have already been discussed. The blood pressure should not be lowered unless there is clear evidence of end-organ compromise (e.g., hypertensive encephalopathy or congestive heart failure). In two-thirds of stroke patients an elevated blood pressure will return to normal within a week without any intervention.[40] The risk of the infarction becoming worse or a new infarction developing is highest among elderly patients with compromised cerebral autoregulation[47] and decreased sensitivity of the baroreceptors.[45]

Some commonly used drugs may retard the recovery of function in cerebral injury.[56,57,59,109] Preliminary evidence that such slowing occurs after stroke has recently been published.[60] Such drugs include benzodiazepines, phenothiazines, phenytoin and clonidine. Although the clinical data are only preliminary, it may be wise to use alternative medications until more information becomes available. We use codeine for sedation or analgesia and phenobarbital or carbamazepine to control seizures.

Fecal impaction is frequent. The lack of oral intake and decreased mobility contributes to the problem. We routinely prescribe a stool softener. The routine use of bladder catheters should be discouraged, because it increases the risk of bladder infection. In most male patients condom catheters will solve the problem. Women with urinary incontinence may require diapers.

Because of an increased risk for venous thrombosis we recommend prophylaxis with heparin given subcutaneously in all cases. Anticoagulant therapy is recommended only in cases of definitive cardiac embolism. In patients with ischemic stroke ASA, 1300 mg/d, will decrease the risk of recurrence by 25% to 35%.[110] Lower doses may also be effective; however, this has yet to be shown in a clinical study. In the only study comparing high-dose ASA therapy and 325 mg of ASA daily the latter significantly decreased the number of non-vascular-related deaths.[111] Unfortunately, because of the small sample the question of whether a lower dose (300 mg) is as effective as a higher dose (1000 to 1300 mg) in the prevention of stroke remains unanswered. However, 30 mg of ASA has been shown to be as effective as 300 mg in stroke prevention among patients with transient ischemic attacks or minor stroke.[77]

Drug therapy to prevent further damage from stroke or to enhance recovery of function is at present experimental.[112-114]

Summary and future directions

The proportion of elderly people in the population has been progressively increasing. Because age appears to be the most important risk factor for stroke, the evaluation and management of elderly stroke patients needs particular attention. We have given a detailed overview of the age-related changes in the heart, vasculature and cerebral autoregulation that are of pathophysiologic significance to the development of stroke in the elderly. Altered metabolism and drug interaction make evaluation and management difficult and may retard recovery of function after acute stroke. Cardioembolism, watershed infarction and stroke secondary to cancer occur more frequently with increasing age. Amyloid angiopathy and related hemorrhage are the most common causes of cerebral hemorrhage. Careful control of hypertension and prevention of complications after acute stroke will help in decreasing the morbidity and mortality rates.

There is little prospective information on stroke prevention in the elderly. Whether the decrease in the incidence of stroke during the last five decades has helped the elderly as much as it has younger people is uncertain. Management of hypertension and hypercholesterolemia in elderly patients has not been clearly outlined and needs further attention. Recovery of function after acute stroke may be slower in the elderly than in younger groups. The limited animal experimental data on recovery after stroke in different age groups[115,116] suggest that recovery of function may be slower in older animals. This may be related to their low risk for neuroplastic effects.[117] Similar evidence remains to be collected in prospective clinical studies. During the "decade of

the brain,"[118] from 1990 to 2000, we hope some of these important questions will be answered.

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Conferences

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Oct. 19, 1991: Breast Imaging Update (cosponsored by the University of Toronto)
Toronto Hospital, General Division
Mrs. Donna Deelarbi, Toronto Hospital, General Division, Department of Radiology, 200 Elizabeth St., Toronto, ON M5G 2C4; (416) 340-3372, fax (416) 340-3390

Oct. 19-26, 1991: International Cardiology '91
London, England
Meeting coordinator, *British Journal of Hospital Medicine*, Croxted Mews, 288 Croxted Rd., London SE24 9DA, England

Oct. 20-23, 1991: Joint Annual Research Conference on Obesity and Food Intake (sponsored by the North American Association for the Study of Obesity and the Society for the Study of Ingestive Behavior)
Hyatt Regency Hotel, Sacramento, Calif.
Judith Stern, Department of Nutrition, Rm. 3150 B, Meyer Hall, University of California-Davis Medical Center, Davis, CA 95616; (916) 752-6575, fax (916) 752-1297

Oct. 22-24, 1991: Biotechnica '91 — 7th International Congress and Exhibition for Biotechnology
Hannover Fairgrounds, Hannover, Germany
Hannover Fairs USA Inc., 103 Carnegie Center, Princeton, NJ 08540; (609) 987-1202

Oct. 23-26, 1991: American Medical Writers Association 51st Annual Conference — World Class: Worldwide Biomedical Communications
Royal York Hotel, Toronto
American Medical Writers Association, 9650 Rockville Pike, Bethesda, MD 20814; (301) 493-0003

Oct. 23-27, 1991: Canadian Association on Gerontology 20th Annual Scientific and Educational Meeting
Toronto
Jean-Claude Carisse, Canadian Association on Gerontology, 110-1565 Carling Ave., Ottawa, ON K1Z 8R1; (613) 728-9347, fax (613) 725-0514

Oct. 24-25, 1991: Gairdner Foundation Lectures
University of Toronto
Sally-Anne Hrica, Gairdner Foundation, 220-255 Yorkland Blvd., Willowdale, ON M2J 1S3; (416) 493-3101, fax (416) 493-8158

Les 24 et 25 oct. 1991 : 9e Assemblée scientifique annuelle du Collège des médecins de famille du Canada (section Québec) — Culture et santé : défis et perspectives
Montréal
Micheline Guilbault, secrétaire administrative, CP 146, Succ. Champlain, LaSalle, QC H8P 3J1; (514) 762-9889, fax (514) 762-9870

Oct. 24-27, 1991: Canadian Association of Gerontology 20th Annual Scientific and Educational Meeting
Regal Constellation Hotel, Toronto
Canadian Association of Gerontology, 110-1565 Carling Ave., Ottawa, ON K1Z 3R1; (613) 728-9347

Oct. 25-27, 1991: The Power of Laughter and Play (cosponsored by the Institute for the Advancement of Human Behavior and *Punch Digest for Canadian Doctors*)
Hilton International Toronto
Some workshops offer CE/CME Category I credits.
Institute for the Advancement of Human Behavior, PO Box 7226, Stanford, CA 94309; (415) 851-8411, 1-800-258-8411, fax (415) 851-0406

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