HYPERTENSION is an important risk factor for many forms of cerebrovascular disease, including cerebral infarction, intracerebral hemorrhage and subarachnoid hemorrhage. Although the statistical importance of hypertension as a risk factor for stroke is well known, the only clinical neurological disorder that is caused solely by elevating the blood pressure is hypertensive encephalopathy. Two other neurological conditions are also linked with hypertension: Binswanger's disease (BD) and lacunar syndromes. Two hundred and twenty lesions were seen in all stroke patients admitted to King Khalid University Hospital during the period of June 1982 to May 1987. Hypertension was invariably present in all patients with Binswanger's disease and in 75% of patients with lacunar syndromes, as compared to 26% to 37% of patients with arterial infarcts, 54% of patients with cerebral hemorrhage and 30% of patients with subarachnoid hemorrhage (Table 1). In this series, we will review the pathogenesis, clinical characteristics, and the management of these syndromes: Binswanger's disease, lacunar syndromes, and hypertensive encephalopathy.

**Subcortical Arteriosclerotic Encephalopathy - Binswanger's Disease**

Binswange~ in 1894 described eight patients with progressive intellectual deterioration, seizures, and stroke-like events. The pathological changes of the brain were those of extensive white matter atrophy, predominantly in a periventricular and occipitotemporal distribution. He suggested that these white matter changes resulted from deficient blood supply caused by arteriosclerosis. In 1902, Alzheimer3 emphasized that the cortex was relatively unaffected while the white matter had focal degeneration and glial proliferation. Olszewski,4 in 1962, presented two new cases and emphasized the presence of associated lacunes and cortical infarcts. He suggested the title "Subcortical Arteriosclerotic Encephalopathy." Since then, several welldocumented cases were published. More recently, emphasis has been on pre-mortem recognition of the disease.5

**Epidemiology**

The average age of onset of symptoms is 57 years (range, 40 to 78); men and women are affected equally.4-10 Hypertension is invariable, and other risk factors reported include diabetes mellitus and ischemic heart disease.5 The incidence reported from Japan, 3.8% in the elderly is highest in those with cerebrovascular disease (6.7%).11

**Pathogenesis and Pathological Findings**

The pathogenesis is that of ischemic changes in the white matter due to prolonged hypertension, the thickness of media of arterioles in BD being greater than in comparable cases with intracerebral hemorrhage or hypertensive encephalopathy.12 The vascular changes are similar to lipohyalinosis of penetrating arterioles in lacunar infarction. DeReuck indicated that the periventricular area is an "end zone" for penetrating vessels. Chronically decreased perfusion of these zones without actual occlusion could cause a
deficit, confusion, change in character, paranoia, apathy, and loss of interest. Change of mood and behavior are prominent. Aphasia, 16 hemianopia, anosognosia and apraxia are uncommon. There is usually evidence of multiple long tract manifestations as mild asymmetrical pyramidal signs (reflex asymmetry, hemiparesis), extrapyramidal rigidity, cerebellar signs with limbs or gait ataxia. Dysarthria with or without pseudobulbar features are not rare. Partial or generalized seizures occur in 15% to 20%.5

**Diagnosis**

The definitive diagnosis depends upon the pathological findings, although the combination of the clinical features and neuroradiological imaging abnormalities (computerized tomography [CT] and magnetic resonance imaging [MRI]) can make the diagnosis with reasonable certainty. Cerebrospinal fluid (CSF) examination is not helpful. The electroencephalogram (EEG) may show diffuse slow background activity or focal asymmetrical slowing. 7,8,18

The neuroradiological findings reflect the described pathological changes and show cortical atrophy, dilated ventricles, lacunes, and cortical infarcts. In addition, the characteristic lesions are the hypodensities (or high intensity lesion at the second thoracic vertebra (T2) in MRI) in the periventricular distribution spreading to centrum semiovale, with the regions just anterior to the frontal horns of the lateral ventricles which are frequently abnormal.7,21 These white matter changes are related to the loss of myelinated small cystic infarcts of variable size. The extent of these lesions determines the severity of dementia.7,21,23 Because these changes are not specific and occur in normal or pathologic aging,23-25 the combination of the radiological and the clinical features are essential parameters to make the diagnosis.

**Relation to Multi-Infarct Dementia**

Multi-infarct dementia and Binswanger's disease form a pathophysiological spectrum. At one end, there is the circumscribed intellectual impairment from destruction of particular brain areas, for example, memory loss after inferior temporal lobe infarct26 while at the other end there is a more insidious progressive dementia from diffuse small-vessel pathology (Binswanger's disease).9,19,26-28 It may be difficult to distinguish between multi-infarct dementia and BD on clinical grounds alone, but it is likely that an individual with hypertension, intellectual loss progressing to frank dementia, significant gait impairment, signs of corticospinal tract disorder and diffuse periventricular white matter low absorption on CT or MRI makes the diagnosis of BD more likely.

**Management**

There is no treatment for the disorder other than control of hypertension. Whether this will halt the progression of the disease is uncertain, although some recent reports suggest that aggressive control of blood pressure close to normal levels was associated with clinical improvement and relative stability.5 Other drugs tried are vasodilating agents to improve cerebral blood flow, antiplatelet agents, and anticoagulants but their values are questionable.5,19

**Illustrative Case**

A 63-year-old Saudi male developed progressive deterioration of intellectual function manifested by memory deterioration, lack of concentration, slight confusion and difficulty in judgement for 8 months. Neurological examination showed, in addition to deterioration of higher cerebral function, slurred speech, bilateral pyramidal signs, and trunkal ataxia. Cranial nerves were all intact and fundi were unremarkable. His blood pressure was 220/130 mm Hg. The CT brain scan is shown in Figure 1. Attempts were made to control his blood pressure but his compliance was poor. Five months later he was admitted unconscious; his blood pressure was 230/150 mm Hg and his CT scan at this stage is shown in Figure 2. He recovered consciousness and his blood pressure was well controlled at 130/80 mm Hg. Neurological assessment showed further deterioration of his intellect.

Nine months later, he was readmitted with further deterioration of his higher cerebral functions, his speech was slurred, he had marked bilateral pyramidal signs with clonus and pseudobulbar palsy, and he was incontinent and bedridden. His blood pressure was not controlled at this stage, and after controlling his blood pressure, he was referred to the rehabilitation unit.
Figure 1. CT brain showing confluent periventricular white matter low attenuation mainly around the anterior and the occipital horns of the lateral ventricles (A) and also involving the centrum semiovale (B).

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References

12. Okeda R. Morphometrische Vergleichsuntersuchungen