intrarenal function for renin becomes a crucial part of the argument. Renin could have a direct effect on the blood supply of the glomerulus if it caused the formation of angiotensin locally. Recent work by K.K.F. Ng and Vane\(^1\) casts doubt on this possibility. Renin acts on a plasma substrate to release a decapeptide, angiotensin I, which has much less pharmacological activity than the octapeptide, angiotensin II, which is formed from it as the blood passes through the lungs. These authors found that angiotensin I had relatively little effect on the kidney when it was infused into its arterial circulation, and they inferred that the kidney was incapable of converting it to the more potent angiotensin II. Unless it is supposed that the intrarenal effect of renin is mediated through some other agency than angiotensin II, this observation seems to weaken the hypothesis of Brown and his colleagues, as they themselves admit.

No firm conclusion is possible on the present evidence. Sufficient renin is released during haemorrhagic shock in experimental animals and is found in the plasma of patients with acute oliguric renal failure for the angiotensin it releases to exert some vasoconstrictor action. However, the evidence is not yet strong enough to prove that it is the sole or even necessarily the most important cause of the reduction in renal blood flow. There may be an important role for a neurogenically mediated vasoconstriction as well as for renin, and possibly it is in the interaction of these factors that the true explanation lies.

**Lacunes**

Some medical terms, like clothes, fall in and out of fashion for variable periods of time. Most clinicians who attend necropsies, and all pathologists, are familiar with lacunes, but many of the clinicians at least may not realize what they are, for it is a term that nowadays is rarely used. Yet at one time it was the cause of extensive publications and considerable controversy.

Coined first by M. Durand-Fardel,\(^1\) the name is given to small, irregularly jagged cavities in the brain ranging in size from 0.5 to 15 mm. in diameter. C. M. Fisher\(^2\) described them as "small, deep cerebral infarcts" and considered that they outnumber all other varieties of cerebrovascular lesion combined. They are principally found in the lenticular nucleus, pons, thalamus, caudate nucleus, internal capsule, and corona radiata, in that order of frequency, and are conspicuously absent from the cerebral and cerebellar cortex. Since the early part of this century the use of the term, and interest in these lesions, both seemed to have been lost until a series of publications by Fisher in the 1940s. Being scattered through several journals, they are not so widely known as their value warrants. T. R. Browne and D. Poskanzer\(^3\) have recently drawn attention to these publications and summarized Fisher’s views.

He recognized, as did the older authors, that lacunes are associated with atherosclerosis and hypertension, but pointed out that they are not features of internal carotid occlusion or stenosis, cerebral embolism, or diabetes.\(^4\) In 1,042 unselected necropsies they were found in 114 brains, but a total of 376 lacunes were identified in these brains. Browne and Poskanzer, discussing the treatment of cerebrovascular disease, consider that the recognition clinically of syndromes produced by lacunes would be of considerable value, for modern methods of investigation and treatment make it additionally important for clinicians to try to define at the bedside which of the different types of cerebrovascular lesion has occurred in order to decide whether or not to carry out arteriographic studies, whether or not to consider the use of anticoagulants, and whether or not to recommend hypoten- sives. And it is in making such a diagnosis that clinicians find themselves in great difficulty in many patients. Fisher has attempted to associate certain specific syndromes with lacunes in special sites, such as a pure motor hemiplegia without sensory, visual-field, or speech defect, due to localized infarction in the internal capsule or the basis pontis, and pure sensory loss in the face, arm, and leg due to such a lesion in the thalamus. Rather more tentatively he has suggested, by anatomical reasoning, that a pyramidal-tract type of weakness of one side affecting the leg more than the arm, with a cerebellar ataxia on the same side, may be due to a lacunar lesion in the capsular corona radiata, and that clumsiness of one hand, with dysarthria (not dysphasia), may follow a lesion deep in the pons. If these syndromes could be relied on to be due to lesions in the areas suggested, arteriography would show nothing of value other than the presence of cerebral atheroma, and anticoagulants would be positively dangerous, for many of these patients are hypertensive and many of the lacunes contain a blood-vessel, diseased though apparently patent, but unsupported by surrounding cerebral tissue.

Unfortunately lacunes are nearly always multiple, so that it may be difficult to ascribe a particular physical finding to a particular lacune. It was this multiplicity which P. Marie called the "état lacunaire", and he and J. Ferrand\(^4\) associated this pathological picture with the clinical state which nowadays is usually termed pseudobulbar palsy. These are patients with a history of transient strokes, occurring on different sides at different times, and leaving the patient with dysarthria, clumsiness of all fine movements, the distressing and embarrassing symptom of forced laughter or crying, and the characteristic gait to which Dejerine gave the name "marche-à-petits-pas". This state may progress to dementia, but without bilateral hemiplegia, gross spasticity, or contractures. However, as modern radiographic studies of the cerebral circulation have advanced and more detailed pathological studies of the blood vessels have been carried out, the clinician’s difficulty at the bedside in differentiating between infarction and haemorrhage, or between disease of proximal blood vessels and distal, has been emphasized. Indeed the whole emphasis now is on a dynamic concept of the cerebral circulation and its deficien- cies. This idea allows for periods of arterial insufficiency to occur sufficient to produce symptoms but without permanent occlusion or infarction. Hence the traditional practice of trying to diagnose in life a static lesion as seen post mortem has waned. While the value of re-establishing interest in lacunes is therefore undoubted, and may help to explain some curious cerebrovascular phenomena, it would be unwise to assume that the syndromes defined earlier are always due to lacunes at specific sites. Fisher, with his wide experience of cerebrovascular disease, would be one of the last to wish it to be thought that he was saying this.