drug dependence on which young people are being continually hooked. At the same time more encouragement could well be given to research into the possibility of harmless smoking. Can it exist? A cigarette that gave the same or similar satisfaction as the tobacco-filled article but lacked its noxious effects would still be sociable and taxable. But it may prove to be a pipe dream.

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Atypical Angina

In 1959 M. Prinzmetal[1] called attention to an unusual form of angina pectoris in which the pain occurred at rest but was not related to physical exercise or emotional stress. Though not nearly as common as angina of effort, it is not a rare syndrome, and it has the same age and sex distribution. The pain generally lasts longer than in effort angina, and it is usually more severe, while the attacks are frequently recurrent in a cyclical and regular pattern. Prinzmetal referred to this syndrome as a variant form of angina pectoris. He noted that if myocardial infarction occurred in a patient with variant angina the pain usually disappeared—unlike that of effort angina. The electrocardiographic changes also differ; instead of the generalized ST depression of classical angina in variant angina ST elevation is accompanied by reciprocal depression in the standard leads. The occurrence of ST depression without reciprocal elevation in classical angina is explained by the diffuse distribution of areas of myocardial ischaemia. In variant angina the extent of the ischaemia is restricted to a discrete area supplied by a large, narrowed coronary artery,[2] and Prinzmetal postulated that the syndrome is due to an increase in vascular tone. Quite a modest increase in tone in such a vessel could lead to complete though temporary obstruction. Angiographic studies in six patients with variant angina have confirmed this explanation by showing a single stenotic lesion of one major vessel without significant disease elsewhere.[3]

The assumption that symptoms of variant angina are due to coronary spasm seems to contradict generally accepted concepts. Angina of effort develops only when there is severe myocardial hypoxia as a result of inadequate perfusion. In these circumstances the affected coronary vessels are already under the maximum physiological stimulus to dilate. This is probably true for classical angina, in which there is generalized disease of coronary vessels limiting their response to variations in tone and in which the ischaemic areas are scattered throughout the myocardium. Nevertheless, angina of effort as a result of myocardial hypoxia has been recorded in the absence of obstructive disease of visible coronary arteries.[4-6] More recently a remarkable case of variant angina has been reported in which myocardial hypoxia was confirmed despite the absence of any coronary artery disease demonstrable by angiography.[7] In this patient the pain occurred at rest or during sleep, but not with exercise. Marked ST elevation was associated with the pain, and episodes of ventricular tachycardia and even ventricular fibrillation were recorded. After an episode of syncope due to complete heart block associated with pain a pace-maker was inserted. No abnormalities of haemoglobin-oxygen dissociation were found, and the basi for this patient's variant angina remained unexplained.

It is possible that in these patients vascular disease is present but not seen, for the state of the microcirculation cannot be assessed by angiography. This is, however, unlikely in view of reports of such patients who have died after infarcts with no apparent disease of the large or small coronary arteries at necropsy.[8] It is possible that myocardial ischaemia might arise from impaired oxygen diffusion or inappropriate oxygen utilization by the myocardial cells. Abnormalities in dissociation of oxygen from haemoglobin have been described in young women with angina and normal coronary angiograms.[8] Furthermore, the energy needs of the myocardium may be abnormal. These are largely determined by the tension of the muscle wall of the ventricle, which is governed by the pressure within the cavity of the ventricle and the mean radius of the heart chamber.[9] It has recently been suggested that the beneficial effect of nitroglycerine in angina is related to the reduction in ventricular volume and hence myocardial oxygen consumption associated with its use.[10]

The pathogenesis of angina is more complex than a mere obstruction to perfusion of the myocardium by atheromatous narrowing of the coronary arteries. The lack of correlation between the degree of atheroma and the presence of clinical disease suggested this many years ago. Vascular spasm, changes in the microcirculation, and physicochemical disturbance within the myocardium are certainly relevant, but their frequency and importance await further elucidation.

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Relapsing Polyneuropathy and Corticosteroids

Though many cases of acute idiopathic polyneuritis, or the Guillain-Barré syndrome, have been reported, clearcut criteria for its diagnosis have proved difficult to define. In some patients the interval between the onset of symptoms and maximum neurological loss may be only a day, while in others apparently identical in every other way the interval may be several months.

G. Guillain, J. A. Barré, and A. Strohl[1] divided their cases into those with a relatively acute onset and the atypical forms. L. D. Osler and A. D. Sidell[2] suggested that the diagnosis should not be made when the interval exceeded two weeks. However, H. Ravn[3] found that the interval had no effect on the later course of patients with this syndrome. The longest interval in his series of 127 patients was 45 days. Similarly, D. E. Pleasure and colleagues,[4] accepting cases with an interval of up to two months, found no difference in prognosis between those with a shorter and those with a longer interval. When the symptoms progress for more than