Types of pancreatitis

SIR,—A recent paper[1] on the classification of pancreatitis has stimulated the renewal of interest dating back at least 25 years.[2 3]

The desire to understand disease entities dates back beyond Hippocrates and the Coan school of the 4th Century BC. That school was quite properly concerned with the symptoms of disease, their interpretation, its therapy and prognosis. Precise diagnosis was considered to be of less significance (as indeed it was) than prognosis and treatment. Classification of disorders, though of academic (scientific) interest, was considered to be less important; it, indeed, was the major interest of the rival and ultimately less successful Cnidian School.

For all that, once we know a good deal about a disease, attempts at classification will always be made and are entirely rational. With the remarkable expansion of medical and scientific knowledge of the past century, many new disease entities have been recognised. Understandably, there have been attempts at classification so that we might know, by a better understanding of a disorder (in the framework of related disorders), what might be useful therapeutically and what might be the prognosis. We have earlier favoured the term 'progressive' rather than 'relapsing' pancreatitis.[4 5] I, for one, would not do so now, as I feel that the term 'relapse' should be used as a strictly clinical term. It is a clinical expression which may have many features on investigation, biochemical, imaging or pathological. No feature in itself indicates a 'relapse' which is essentially a clinical description. In an acute attack, when a patient describes it as being similar to an earlier attack (and where alcoholic excess has not preceded the attack) one should search for gall stones, hypercalcaemia, hyperlipidaemia, some drug usage or other cause. A diagnosis of relapsing acute pancreatitis would often be reasonable. Appropriate treatment may be available. If older patients, sometimes recognisably alcoholic, admit to this episode being one of many, the presence of the relapsing chronic disorder is not inappropriate. Is it helpful to simplify the diagnosis of pancreatitis to no more than 'acute' and 'chronic' by eliminating these useful subdivisions of 'relapsing acute' and 'relapsing chronic'?

It behoves a doctor, who has made a diagnosis of 'acute' pancreatitis, to determine why the attack has occurred; otherwise the patient may suffer from another attack – a relapse. If the patient has gall stones, this should be treated so that another attack will not occur. If the doctor neglects to carry out a reasonably thorough study of the attack and if a relapse should occur, I believe that he may have been negligent. If the patient has not been recognised as an alcoholic and what that means, an opportunity to advocate abstinence will have been missed. The progress of even alcoholic pancreatitis into the full chronic disease can be delayed, as in alcoholic liver disease, by abstinence. The advice to abstain from alcohol is more likely to be listened to when the patient has recently experienced the typically severe pain of a relapse rather than warn about the rather different symptomatology of the chronic disorder which he has not yet experienced.

The term 'relapse' is important. It must not be discarded because there may be a difficulty of interpretation by some doctors, or because parallels between the clinical investigation and pathological pictures are not easily made in the present state of knowledge. The greatest challenge in the field of pancreatitis is in the clear recognition of non-alcoholic chronic pancreatitis in older people. We have a ready made model of the chronic disorder in the well accepted form of relapsing chronic pancreatitis due to alcohol progressing into the chronic disorder. That model should not be discarded but studied; for chronic pancreatitis is not uncommon. Thus we will achieve sound progress. We will not do so if a major criterion for diagnosis of chronic pancreatitis be the presence of pancreatic insufficiency. In this day and age, we ought to be able to recognise chronic pancreatitis earlier, perhaps at a stage of the relapsing chronic disease. We need to expand, not contract our clinical classification of pancreatitis and to relate it more precisely to pathophysiology, to pathology and to prognosis.

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References

1 Sarner M, Cotton PB. Classification of pancreatitis. *Gut* 1984; **25:** 756–9.
2 FitzGerald O, FitzGerald P. Pancreatitis. *Brit Med J* 1959; **1:** 1188
3 FitzGerald O, FitzGerald P, Fennelly J, McMullin P, Boland SJ. A clinical study of chronic pancreatitis. *Gut* 1963; **4:** 193–216.
4 Howat HT. Chronic pancreatitis. *Lancet* 1963; **2:** 1234–5.
5 FitzGerald O, FitzGerald P, McMullin JP. Chronic pancreatitis. *Lancet* 1963; **2:** 1333.