most general practitioners do not employ the full quota of practice staff for which they can claim reimbursement.

Rigorous evaluation of preventive activities is needed so that they may be further modified and improved in the light of experience. In particular, a comparison of programmes with and without measurements of the plasma cholesterol concentrations would allow assessment of the relative costs, acceptability, effectiveness, and logistical problems. Questions still remain about the optimum diet for reducing cardiovascular risk (and cancer), but a reduction in fat intake and perhaps an increase in vegetable intake (including sources of dietary fibre) seem likely to reduce both.¹²⁻¹⁵⁻¹⁷ Other factors, particularly haemostatic variables, are likely to be independent determinants of cardiovascular risk which may also be susceptible to dietary influences.¹⁸⁻²⁰

The uncertainties which persist should not, however, be a justification for inaction. If the poor record of the United Kingdom in ischaemic heart disease is to be reversed general practitioners and other primary care professionals will have to rise to the challenge both in their day to day work and by participating in research to resolve the remaining elements of uncertainty.

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Irritability

In ordinary speech irritability implies poor control of temper resulting in an external display such as quarrelling. As such we are all irritable at some time or other and are so, we hope, without any disorder of our mind. Irritability is also, however, a common complaint in general practitioners' surgeries and the outpatient clinics of psychiatrists, neurologists, and neurosurgeons. If the patient does not admit to it, which is unusual, the relatives certainly will. In these patients irritability has become abnormal either because it is a new characteristic in a person not normally irritable or it is present to an abnormal degree.

So what do we mean by "irritability," and if we can define it is it important? Should the definition include any mood change which results in external physical aggression, and, by contrast, can a person be irritable even without external signs?

An initial attempt at a definition used the words "irritable aggression."¹ Unfortunately, this definition contained within it the attempt to harm, which most of us would not accept as a feature of irritability. Recognising this, a recent paper has suggested that, although "irritability" and "irritable aggression" are probably at opposite ends of a spectrum, the important feature of irritability is that it does not include any attempt to harm.² The authors proposed a definition of irritability as "a fuming state characterised by reduced control over temper which usually results in irascible, verbal, or behavioural outbursts, although the mood may present without observed manifestations. . . ." Included in this was the fact that irritability may occur for either brief or prolonged periods, is always unpleasant for the individual (lacking the cleansing effect of justified outbursts of anger), and may be present without any overt verbal or physical

manifestations. Irritability may be associated with other symptoms such as depression or anxiety, but it is an independent mood and is not merely symptomatic of these states.

Brain damage is an important factor in generating irritability. The limbic system has been suggested as the site of the damage, but no specific area has been implicated with certainty. Chemical changes are also important. Alcohol is well known for its effect on mood: both irritability and aggressive behaviour may be symptoms of alcohol intoxication or, more likely, withdrawal. Indeed, people recovering from head injuries should be warned to use alcohol sparingly for this reason—as well as for fear of precipitating seizures. In any individual case, however, it may be difficult to establish a causal relation between alcohol and irritability; personality factors, existing psychiatric disorders, and social problems may all complicate the picture. Hormones too play a part: irritability may be a prominent feature within the postnatal³ and paramenstrual periods.^{2,4}

In the clinical setting a head injury, even a minor one, is one of the most common causes of irritability.⁵⁻⁸ It forms part of the post-traumatic syndrome and is usually the result of brain damage.⁸ Even a minor head injury may cause some slight brain damage, which may impair the processing of information.^{9,10} Possibly this subtle deficit, with its resultant frustration, produces the irritability. The relevance of a compensation claim is often exaggerated.⁷

Managing irritable patients with head injuries may be awkward but is not difficult. Both the patient and the relatives should be told about the possible organic basis, especially after a minor head injury. Alcohol intake should be limited. Other than this, time and sympathy is all that can

be offered—but fortunately they are all that is needed. Especially after a minor head injury the symptom is self limiting, though it may take up to two years to disappear and sometimes never does. When dealing with adolescents the normal child-parent conflicts should be identified and not attributed to the head injury, even though parents might often wish this were the case.

Brain tumours, especially those of the frontal lobe, may lead to personality changes which include irritability. This rarely happens in isolation, however, but is usually associated with other frontal lobe behavioural changes. Temporal lobe abnormalities may also produce various types of aggressive behaviour, but even here irritability is unusual.¹¹

We need to remember, then, that irritability is not necessarily abnormal. When it is it is often clearly associated with neuronal damage, usually from a head injury—but it is rarely the sole feature of an intracranial organic lesion. Reassurance, time, and sympathy are the main props of treatment. Even so, many patients will continue with

symptoms for some considerable time, possibly indefinitely. Fortunately, irritability is not a debilitating disease.

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Biochemical tests for acute pancreatitis

Faced with a battery of biochemical tests and new imaging techniques what laboratory aid should the clinician seek when considering the diagnosis of acute pancreatitis? The value of serum amylase estimations was reported first in 1929, and it remains the most requested test in this disorder.¹ Measurements of the enzyme by a variety of simple kits are available as a 24 hour service in most laboratories. The activity rises within two to 12 hours of the onset of symptoms and generally returns to normal within three to five days. And yet as a diagnostic test the serum amylase activity has many drawbacks. It is not specific; there are many other causes for hyperamylasaemia.² Raised activities have no prognostic significance. Activities may be normal in 10% of fatal cases³ and when there is associated hypertriglyceridaemia.⁴

More worrying, perhaps, is the failure to agree on what activity is diagnostic of acute pancreatitis. A survey of six commonly consulted textbooks of general medicine and of gastroenterology reflects this imprecision, with statements ranging from a twofold, or threefold, or fourfold rise being significant to a comment that the greater the hyperamylasaemia the more certain is the cause to be acute pancreatitis.

The total serum amylase activity represents a mixture of salivary and pancreatic amylases, the latter accounting for 35-40% of total activity. Commercial kits are available to measure serum pancreatic isoamylase activities. Although there have been claims that the isoenzyme estimation is clinically helpful,^{5,6} recent reports suggest that it has little diagnostic advantage as a routine assay for acute pancreatitis.⁷

Urinary amylase activities, the amylase:creatinine clearance ratio, and isoamylase activities have no advantage over serum measurements and pose similar problems in interpretation.^{2,8}

Serum lipase and trypsin(-ogen) activities may also be raised in acute pancreatitis. Lipase estimations have disadvantages compared with amylase and may be raised in non-pancreatic disease. The only source of trypsin in the blood is

from the pancreas (a radioimmunoassay for trypsinogen is available⁹) and raised activities strongly suggest acute inflammation.¹⁰

Many other biochemical alterations occur in acute pancreatitis but they are non-specific. Methaemalbuminaemia may accompany other causes of intra-abdominal bleeding than acute necrotising pancreatitis.¹¹ A rise in the blood or serum concentrations of glucose, urea, and lactic dehydrogenase, and a lowering of calcium and albumin concentrations and of the arterial oxygen saturation are individually non-specific but collectively provide a guide to outcome.¹²

Which, then, of the four—amylase, isoamylase, lipase, or trypsin—should be used in routine clinical practice? Using ultrasonography, computed tomography,¹³⁻¹⁵ and laparotomy to establish a diagnosis of acute pancreatitis, Steinberg and his colleagues have assessed the sensitivity and specificity of the four biochemical tests.⁷ All gave good sensitivity but specificity was less satisfactory. More importantly, when they analysed their data using a cut off level that best separated acute pancreatitis from controls with abdominal pain they found that most non-specific rises were of between one- and twofold. Activities in each of the four tests greater than twice normal were almost 100% specific for acute pancreatitis.

Ultrasonography is widely available. Why should we not use that as the definitive emergency test for acute pancreatitis? Unfortunately, sonograms are normal in one in three patients with mild acute inflammation. Its greatest value is in the diagnosis of pseudocysts,¹³⁻¹⁶ and in the detection of gall stones in association with acute pancreatitis.¹⁷ Endoscopic retrograde cholangiopancreatography is contraindicated in acute pancreatitis, and computed tomography is not generally available as an emergency investigation.

Because the serum amylase activity is easy to measure and readily available as a 24 hour service it remains the initial assay of choice. Should it be necessary to show that the hyperamylasaemia is pancreatic in origin either the isoamylase or trypsinogen activity may be measured; but