radiofrequency applications to left-sided accessory pathways but only in 5% of radiofrequency applications to right-sided accessory pathways. This finding points to the increased cooling effect of the circulating blood pool on the right side of the heart as well as to a possible sliding effect of the catheter electrode along the tricuspid annulus. Electrode sliding has been shown in an experimental study to reduce the incidence of impedance rises and allow for the application of higher electrical power6.

The temperature-controlled mode of radiofrequency current delivery has been introduced into clinical practice with the objectives of improved control over current-induced lesion size and, in particular, the prevention of sudden impedance rises and their accompanying sequelae such as carbonization of tissue and myocardial tearing. At the present time, it must be stressed that measurement of catheter tip temperature does not correlate with efficacy. In experienced centres, ablation of right- and left-sided accessory pathways using this modality or, for that matter, the power-controlled modality is equally effective. Compared with the latter, temperature-controlled radiofrequency current delivery may reduce the incidence of sudden impedance rises, but it does not completely prevent them because current catheter technology does not provide for a precise and reliable measurement of electrode–tissue interface temperature. Preliminary animal studies have shown that electrode–tissue interface temperature can be kept below boiling point in most instances with the recently developed ‘chilled-tip’ catheters; consequently, higher electrical power can be applied to achieve larger lesions1,2.

Using the ‘conventional’ thermistor-tipped catheters, it appears advisable for ablation of left-sided accessory pathways to set the catheter tip temperature at 70 °C or lower. In conjunction with the firm electrode-wall contact at the ventricular aspect of the mitral valve annulus this usually suffices to produce lesions large enough to ablate the majority of the subendocardially located accessory pathways. On the other hand, the combined effects of convective cooling and electrode sliding should allow for higher pre-set temperatures to ablate right-sided accessory pathways. This, of course, is a hypothesis in need of confirmation by further investigation.

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References

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Cardio-oesophageal reflex and ‘linked angina’ — is the way to a man’s (or woman’s) heart through the stomach?

See page 407 for the article to which this Editorial refers

For many years, clinicians have been aware that chest pain of oesophageal origin may be clinically indistinguishable from true cardiac angina. Up to one quarter of patients admitted with acute chest pain prove to have an alimentary cause1. In such patients, the culprit oesophageal lesions involve either gastrooesophageal reflux of acid, or motility disorders

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e.g. diffuse oesophageal spasm, 'nutcracker' oesophagus or both together. Recently, the diagnostic overlap between cardiac and oesophageal chest pain has been increased by the demonstration that exertion can produce gastro-oesophageal reflux of acid not present at rest. Oesophageal pain may thus be exercise-induced, a relationship which hitherto has been a cornerstone in the diagnosis of true cardiac angina[2].

In 1962 Smith and Papp[3] first suggested that genuine cardiac angina, as distinct from clinically identical but non-cardiac pain arising elsewhere, could be induced in patients with established coronary artery disease 'if one of the organs which has nervous connexion with the heart and which lies in its proximity has become diseased'. To support this concept, which they termed 'linked angina', they referred to earlier work showing that raising bile duct pressure produced typical pain in anginal patients; that angina could be more easily induced in patients with distended (full) stomachs; that gall bladder 'reflexes' could induce angina, transient ECG changes and arrhythmias; and that operative repair of hiatus hernia could bring swift relief of co-existent angina. Subsequently, oesophageal acid perfusion has been shown to induce the usual pain in 76% of patients with angina and angiographic CAD and was accompanied by ECG changes in two-thirds of those not on beta-blockers[4]. Alban Davies et al.[5] have also shown in a similar cohort that oesophageal acid instillation reduces the exertional angina threshold.

Without a clear mechanism(s), however, linked angina has remained a somewhat hypothetical concept. Mellow et al.[4] found significant rises in rate-pressure products during acid-induced chest pain suggesting a 'demand-related' effect, probably sympathetically mediated since it could be attenuated by beta-blockers. By contrast, animal evidence[3] has favoured altered coronary blood supply since oesophageal (and biliary) distension was associated with a 50% reduction in coronary blood flow. The authors conclude that oesophageal acid stimulation in humans can induce typical angina by reducing coronary blood flow via a neural cardio-oesophageal reflex. Furthermore, since the diameter of the left anterior descending artery remained unchanged they infer that the reduction in coronary blood flow results from a neurally mediated increase in microvascular resistance due to vasoconstriction either directly or after the secondary release (locally or systemically) of vasoactive substances.

This evidence of a neural reflex underlying the cardio-oesophageal link in humans should come as no surprise. Not only are the two organs anatomically juxtaposed but they also share a common innervation. In addition to the animal work cited above[3], oesophageal stimulation in man can cause clinically important, vagally mediated, reflex bradycardia and atrio-ventricular block, particularly in the presence of structural oesophageal disease. Furthermore, the appreciation of angina requires the involvement of intact afferent sympathetic nerves.

A number of issues, however, remain unresolved and await further study. Firstly, the absence of tests of oesophageal structure or function leaves open the question as to whether this reflex occurs in the normal oesophagus or only in abnormal ones with motor dysfunction or sensitization/exposure of pain receptors by disease such as oesophagitis. Secondly, the lack of simultaneous ambulatory ECG and pH recording leaves unestablished the temporal link necessary for the attractive hypothesis that gastro-oesophageal reflex of acid during daily activities initiates a neural reflex reducing coronary blood flow and hence induces a true cardiac (linked) angina. It remains possible (though unlikely) that the neural reflex does exist but the chest pain was actually of oesophageal origin. Lastly, further work is required before extrapolation of these findings from syndrome X to patients with coronary artery disease can be justified.

What are the implications of this for the practising clinician? It reminds us that cardiac and oesophageal pain can be indistinguishable by patient...
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and doctor alike. It confirms the growing belief that the term syndrome X is no more than a flag of convenience encompassing a miscellany of heterogeneous conditions of differing pathogenesis. The current list includes impaired coronary flow reserve (microvascular angina), early cardiomyopathy, visceral sensory dysfunction, reduced efficiency of the potassium pump and insulin resistance. Chauhan et al. have now added a further subgroup in which the chest pain results from reduced coronary blood flow reflexly induced by oesophageal acid stimulation such as would occur with gastro-oesophageal reflux.

Importantly, this work has helped establish the existence of linked angina by providing a probable underlying mechanism i.e. the ability of oesophageal acid stimulation not only to cause anginal-type chest pain arising in its own pain receptors but also to induce true cardiac angina — at least in patients with syndrome X and possibly in those with coronary artery disease. Since cardiac and oesophageal disease commonly co-exist, clinicians should be aware that identical episodes of pain occurring at different times in the same patient may have entirely different origins. Consideration should, therefore, be given to simultaneous ambulatory ECG and pH monitoring or to well constructed therapeutic trials e.g. with omeprazole to distinguish cardiac from oesophageal episodes. In addition, treatment of both conditions may be necessary in order to render the patient symptom-free.

Increasingly, however, it does seem as though one route to a man’s (or woman’s) heart may be through the stomach!

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References

Spectral analysis of RR variability during transient myocardial ischaemia: have we moved from the computer console to the bedside?

See page 388 for the article to which this Editorial refers

Spectral analysis of RR variability during transient myocardial ischaemia: have we moved from the computer console to the bedside?

While improvements in medical technology have rendered the clinical appreciation of the mechanical and metabolic effects of acute myocardial ischaemia a standard diagnostic procedure, the assessment of the attendant autonomic effects is still largely a matter of experimental investigation, which has not been resolved with simple determinations of plasma catecholamines.

Since the early observation that acute regional myocardial ischaemia in anaesthetized cats was capable of initiating a cardio-cardiac sympathetic excitatory reflex[1], the search for practical ways to address in the clinic the link between sympathetic