Financial audit of antitachycardia pacing for the control of recurrent supraventricular tachycardia.

Sir—Griffith et al reported on the financial issues affecting the choice between medical treatment and antitachycardia pacing for atrioventricular nodal reentrant tachycardia. They attempted to include data on radiofrequency catheter ablation in their analysis but their analysis is flawed on two accounts: they overstate the cost of catheter ablation and overstate the risks and hazards.

Indeed, in another article on the same topic, Connolly et al conclude that the widespread use of curative techniques for supraventricular tachycardia (catheter ablation) has reduced the risks of the procedure. Griffith et al’s results favoured pacing but they report a high rate of complications, with 10 of 25 patients being re-admitted to hospital for wound-related problems. The reasons for these wounds were varied and some were as young as 22 years—so cosmetic considerations might have to be set against a purely financial gain. It would have been useful to have had an indication of patients’ perception of the acceptability of this form of treatment compared with alternatives.

When pacing and ablation were compared by Griffith et al they chose a worst case analysis. Medical data used from Jazayeri et al represent a learning curve and experimental experience rather than an established treatment, and included initial attempts at ablation of the fast pathway in 40% of the patients. This is now regarded as a last resort approach, because ablation of the slow pathway is much safer and usually curative. Special mention is made of the three patients who required repeated procedures, pacing for iatrogenic atrioventricular block (after a fast pathway approach) but were cured of tachycardia. Nonetheless, 94% of patients were cured of tachycardia by ablation. Many of these patients are permanent pacemakers with an antitachycardia pacemaker. The more extensive and recent report of Jackman et al provides evidence that catheter modification is now the best treatment for atrioventricular nodal reentrant tachycardia, with 80 patients (100%) undergoing successful treatment with a 2-5% complication rate (one patient had a pulmonary embolism and one an atrioventricular block) and no recurrence without medication over 15-5 (11 mean (SD)) months of follow-up. At the University of California, San Francisco, the last 62 patients to be treated for atrioventricular nodal reentry by catheter ablation of the slow pathway had a 100% success rate (free from symptoms and medical therapy over a mean of more than nine months) and the procedure required a single overnight hospital stay in almost all cases, complicated only by inadvertent atrioventricular block requiring permanent DDD pacing in one elderly patient (1-4%).

All other contemporary electrophysiological studies aimed at identifying various causes of paroxysmal supraventricular tachycardia, with the help of available non-invasive data, before catheter ablation during the same procedure. The mean total procedure time for the diagnostic and therapeutic study was 191 (49) min, with 33 (36) min for fluoroscopy. Furthermore, several centres have reported performing single diagnostic and therapeutic electrophysiological studies as day cases. As this becomes the norm, there will be simply no financial contest from treatments other than catheter ablation. A curative therapy is more acceptable for itself in terms of patient acceptability.

While there has been concern about the long-term effects of radiofrequency ablation in growing hearts, with experimental data in infant sheep suggesting that scarting grows with the heart, there is no evidence of long-term progression in the adult heart.

Radiofrequency catheter ablation of an atrioventricular nodal reentrant pathway creates a small well circumscribed lesion at a site distinct from the compact node or His bundle. Long-term follow up is expected to show continuing excellent results, and there have been no reports of early or late ventricular arrhythmias with radiofrequency energy, despite the concerns of Griffith et al. Acknowledging the revolutionary impact of curative radiofrequency catheter ablation on quality of life of patients with paroxysmal supraventricular tachycardia will surely help to provide the necessary training and resources to cure more British patients by this technique.

ADAM P FITZPATRICK
LAURENCE M EPSTEIN
HARRY JAZAYERI
LEANDER D LESH
Department of Medicine and CPRl, Moffitt Hospital, Room 0132 Box 0214, University of California, San Francisco, California 94143, USA


This letter was sent to the authors, who reply as follows:

Sir,—We are disappointed that Fitzpatrick et al failed to recognise that our paper was a cost benefit analysis of high technology, and therefore apparently expensive techniques, in relation to conventional management with drug therapy and antitachycardia pacing experience as an example and did not attempt to make a direct comparison between antitachycardia pacing and other invasive treatments of node reentrant tachycardia.

Fitzpatrick et al point out, as did we in our paper, that selective ablation of one atrioventricular nodal pathway may become the preferred treatment for atrioventricular node reentrant tachycardia. We also stated clearly that radiofrequency ablation, particularly when combined with a single diagnostic electrophysiological study may be a cheaper treatment than antitachycardia pac-
Arrhythmogenic right ventricular dysplasia

Sir,—The three consecutive articles on arrhythmogenic right ventricular dysplasia (ARVD) published in the February issue of the British Heart Journal demonstrate the increasing interest in this clinical entity.1,2

Gerlis et al conclude that there is a clear distinction between ARVD and Uhl’s anomaly. This accords with our limited experience of these two conditions.4 The confusion probably arose because Uhl’s original description was based on only one case.4 Because Uhl’s anomaly was regarded as a simple case of a condition affecting the right ventricular musculature any disorder of the right ventricular myocardium was viewed as a form of Uhl’s anomaly.4 Later it was realised that infiltration of the myocardium of the right ventricular free wall by fatty tissue with few remaining myocardial fibres did not fit Uhl’s original description.2

There has been some confusion about the so-called “partial” and “complete” forms of the disease. The term “partial” was originally used by Sugita et al who described a heart obtained at necropsy that had “aspiration of the epicardium on the endocardium”, which appeared as a small window (12 mm wide and 15 mm long) on the free wall of the right ventricle. This was an anomaly because the patient had no cardiac symptoms.6 Another striking example of Uhl’s anomaly was seen at necropsy in a patient with a normal cardiac examination before death. In this patient, only the anterior free wall of the right ventricle was paper-thin and translucent.7 Therefore, the term “partial” should be restricted to patients in whom an area of the heart is completely devoid of muscle and should not be applied when there is only a “partial replacement” of the right ventricular musculature by fatty tissue in the free wall of the right ventricle. When some cardiomyocytes remain between the epicardium and endocardium in the most affected area, Uhl’s anomaly should not be diagnosed. Dr Lino Rossi, a pathologist of great experience, told me that in 40 years he had never seen a typical case of Uhl’s anomaly.

We saw our first adult case of Uhl’s anomaly in 1974.4 The patient was referred to us because of our special interest in the surgical treatment of ventricular tachycardia. The right ventricle was monstrously dilated and the wall of the right ventricle was so thin that at surgery blood could be seen flowing inside the right ventricular cavity. This case was the first in which the reentrant pathway of ventricular tachycardia was mapped on the anterior aspect of the infundibulum where some fibres remained, making a two dimensional structure.6 We recorded late potentials from this area 260 ms after the onset of the QRS complexes.11 This demonstrated the presence of atrial fibrillation and uncontrollable heart failure after operation. The biopsy specimen taken at operation showed “abrupt interruption of the myocardium” in the right ventricular muscle and apposition of epicardium against endocardium in most of the free wall of the right ventricle.

Our second case was a patient who died of pulmonary embolism. Ventricular tachycardia could be done. The cardiac pathology in this case has been reported elsewhere.10,11 Histological examination showed absence of myocardial muscle and again the abrupt interruption of the myocardium. These two cases were reviewed by our cardiac pathologist Dr Fabrice Fontainier, who confirmed that these patients had Uhl’s anomaly. Therefore, we think that the cases reported by Vedel et al7 in which the pathology was not fully described were probably two adult cases of Uhl’s anomaly.

Miani et al12 described some of the most difficult aspects of the differential diagnosis.2 They presented two families that illustrated the role of left ventricular involvement in ARVD. Fatty change can be a minimal component of the right ventricular wall (but not the left), and the common feature of the pathological material available from the two families was fibrosis of both ventricles.12 In ARVD a modest amount of fibrous tissue is generally found around the surviving myocardial fibres that are embedded in the fatty tissue. This was not the pattern of fibrosis shown in the cases of both families. The description of pathological examination in these cases (especially the first family) is more compatible with the fibrous form of idiopathic dilated cardiomyopathy. In addition, the progression of the disease is quite different in these two families. In the first family, the involvement of the left ventricle was the most salient feature in all the cases, and ventricular tachycardia, probably originating from a focus that had been seen only in the third case. The presence of lymphocytic and plasmocytic infiltration suggests that cardiomyopathy was the result of earlier myocarditis. The progression of the disease in both families only resembles that of ARVD.12,13,14 However, we cannot exclude the possibility that the pathological findings could be the result of a healed myocarditis in which the signs of a previous infection had disappeared.

An abnormal host immune response could explain the familial cases and the histological “fibrous pattern” that was more frequently seen in the Veneto region than in our own series.15 The cases reported by Gerlis et al are examples of the congenital form of dysplasia and those reported by Miani et al are probably examples of the acquired form of the disease. It is likely that both ARVD and Uhl’s anomaly are the result of abnormal development16 rather than a pathological entity caused by myocarditis alone.12 ARVD could be a congenital anomaly with superimposed myocarditis. In either event the term dysplasia, which means abnormal development as well as a pathological process resulting from an inflammatory process, deserves to be re-instated.19

McLay et al reported their interesting experience with treating ventricular tachycardia in patients with ARVD who did not respond to antiarrhythmic drugs.2 Even when patients with ARVD have considerable distortion of the right ventricle, there should be progression of right ventricular enlargement and ventricular tachycardia with multiple configurations, we still consider ablation with radiofrequency or DC energy, provided that strict protocols are followed. This approach is used alone or in combination with drug treatment at our hospital and others, and was effective in the short and long term.20,21 We believe that surgery should be performed only when there is an additional indication—such as, correction of abnormal venous return, removal of pacing leads in case of sepsis, etc.

I hoped that the long-term study of outcome in the patients who have had the operation described by McLay et al (disconnection of the right ventricle) will include not only patients with ARVD who have this procedure but also matched controls.

Finally, I agree that ARVD is not a rare clinical entity, as indicated by the two cases of McLay et al ARVD could cause sudden death in a young adult who has had ARVD but has not experienced only minor cardiac symptoms before the terminal event. Because ARVD may be uncommon it is important to have an individual to apply for positions in which sudden illness or even of consciousness caused by ventricular tachycardia or ventricular fibrillation would pose a considerable risk to others. They could be screened by echocardiography for several conditions, including hypertrophic cardiomyopathy and ARVD. Independently, patients could be screened for ARVD by ordinary and signal averaged electrocardiography because the QRS vector ratio would depend upon the prevalence of ARVD in the population that is studied, and this is as yet unknown.

Guy Fontaine
Hôpital Timothée Roux,
94200 Ivry, France