CARDIAC ELECTROPHYSIOLOGY AN UPDATE FOR PHYSICIANS

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SYNOPSIS

Cardiac electrophysiology is a recently available technique for the investigation of patients with major or recurrent troublesome cardiac arrthymias. The indications and limitations of cardiac electrophysiological techniques are discussed in this article. Sinus node dysfunction may be intermittent giving diagnostic problems. Assessment of sinus node function in the laboratory can then reveal the diagnosis. In patients with heart block localisation of the site of conduction disturbance allows prognostic evaluation. In supraventricular and ventricular tachycardias not only can definitive diagnosis of the arrhythmia be established but the mechanisms elucidated. This allows a more rational approach to their subsequent therapy. The ability to initiate and terminate tachycardias with programmed stimulation enables selection of appropriate antiarrhythmic therapy for the patient. With these cardiac electrophysiological techniques it is hoped that the diagnosis and management of patients with major cardiac arrhythmias can be improved.

INTRODUCTION

Cardiac arrhythmias has been and will be diagnosed from surface electrocardiograms. Deductive analysis of the electrocardiogram has led to advances in our knowledge of cardiac arrhythmias (1-3). Many of these interpretations are empirical. Recent electrophysiological techniques have enabled some of these presumptions to be validated or otherwise. As the electrocardiogram represents a net summation of the electrical forces of the heart, recorded from the surface of the body, limitations occur in the ability to analyse some cardiac arrhythmias. For example, in supraventricular tachycardias, it is difficult to deduce the mechanism of the tachycardia from the electrocardiogram. Similarly, wide complex tachycardias can result in diagnostic and therapeutic problems which may be difficult to resolve with the surface electrocardiogram. As a result of these limitations a large amount of work and knowledge has recently been accumulated in cardiac electrophysiology. This has confirmed some of the deductive reasoning of surface electrocardiograms. More important still, a better understanding of the various mechanisms of cardiac arrhythmias has been obtained. Besides being of academic value, knowledge of the mechanism of cardiac arrhythmia enables us to tailor our therapy to the patient more rationally. New cardiac electrophysiological techniques have also evolved for the treatment of cardiac arrhythmias; for example, development and assessment of newer drugs, specialised arrhythmia pacemakers and cardiac electrosurgery. The aim of this article is to review our present concepts of clinical cardiac electrophysiology and to outline the various indications for which this modern investigative tool is invaluable. This review is appropriate at this stage because a cardiac electrophysiology laboratory has just been established in Singapore General Hospital.

TECHNIQUE

The cardiac electrophysiological investigation is basically straightforward (4): intracardiac electrical potentials are measured from various areas of the heart. This is achieved by placing multiple catheters in various chambers of the heart at the same time (Figure 1). The intracardiac electrical potentials are processed through a series of bioelectric amplifiers, from which it is fed into various output modalities. These usually include a osciloscope, a multichannel recorder as well as a tape recorder. A programmable stimulator provides electrical stimuli in different permutations to the various heart chambers (Figure 2).

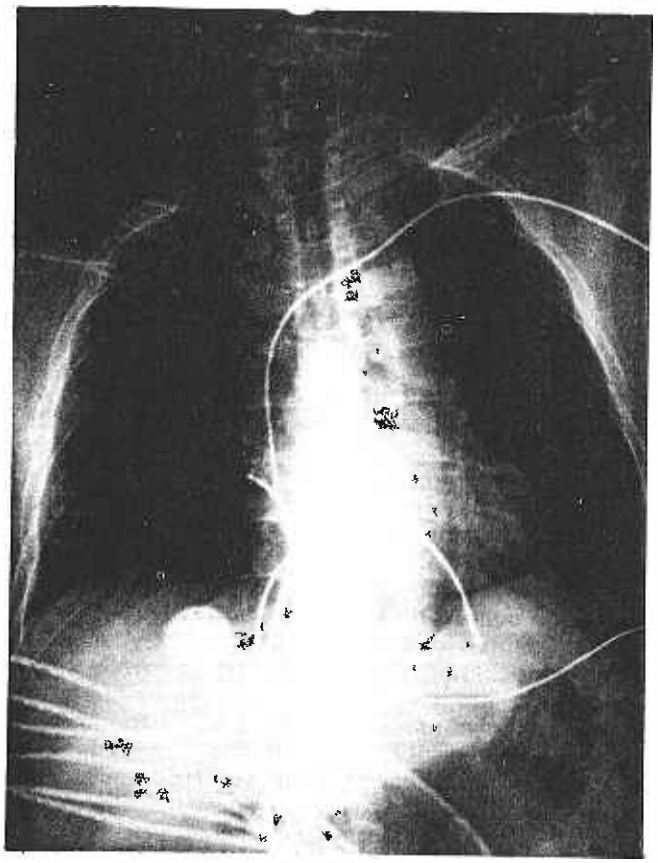

FIGURE 1
The chest X-ray shows the positions of the multipolar electrode catheters in the heart. The catheters are initially positioned in the Right Atrium, Right Ventricle, Coronary Sinus and just across the Tricuspid valve. They are used for stimulation and recordings at multiple cardiac sites.

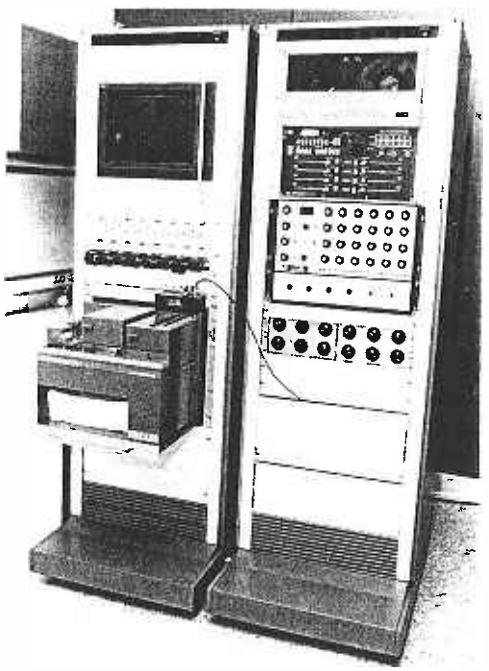

FIGURE 2
The cardiac electrophysiology equipment in the Singapore General Hospital. The left rack contains the 8 channel oscilloscope, bioelectric amplifiers and the ink injector recorder. The right rack houses the tape recorder, intracardiac stimulator and junction boxes.

INDICATIONS

Sinus node disorders

In some patients with sick sinus syndrome, the resting 12-lead electrocardiogram is adequate for diagnosis. The presence of electrocardiographic abnormalities in association with symptoms is diagnostic. In other patients symptoms and electrocardiographic abnormalities are, however, transient. Ambulatory electrocardiographic monitoring is then extremely useful in these patients. It may be necessary to monitor the patients for several days. When prolonged pauses are present (that is more than 3 seconds) in association with symptoms, permanent pacemaker implantation is indicated (5). Despite prolonged ambulatory monitoring, electrocardiographic abnormalities or symptoms may not be detected. In these patients, electrophysiological assessment of the sinus node may be necessary for the diagnosis. The automaticity of the Sinus node and Sinoatrial conduction times can be assessed by electrophysiological techniques. To assess sinus node automaticity, sinus node recovery time (SNRT) or corrected sinus node recovery time is measured. The atrium is stimulated at varying rates from 100 to 200 per minute for 30 seconds to one minute. On cessation of overdrive suppression of the sinoatrial node the time taken by the node to recover is measured, giving the sinus node recovery time (4) (Figure 3). The corrected sinus node recovery time is obtained by substracting the bassic sinus cycle length from the SNRT. Patients with sick sinus syndrome have prolonged SNRT and if symptomatic from the bradycardia will require pacemaker implantation. In some patients sinus node dysfunction may be occult but unmasked by drugs such as, verapamil, betablockers or digitalis. Assessment of the sinus node recovery time before and after exhibitation of these drugs can reveal sinus node dysfunction in these patients. Such drugs should not be used in these patients though if absolutely necessary, a pacemaker should be implanted before their use.

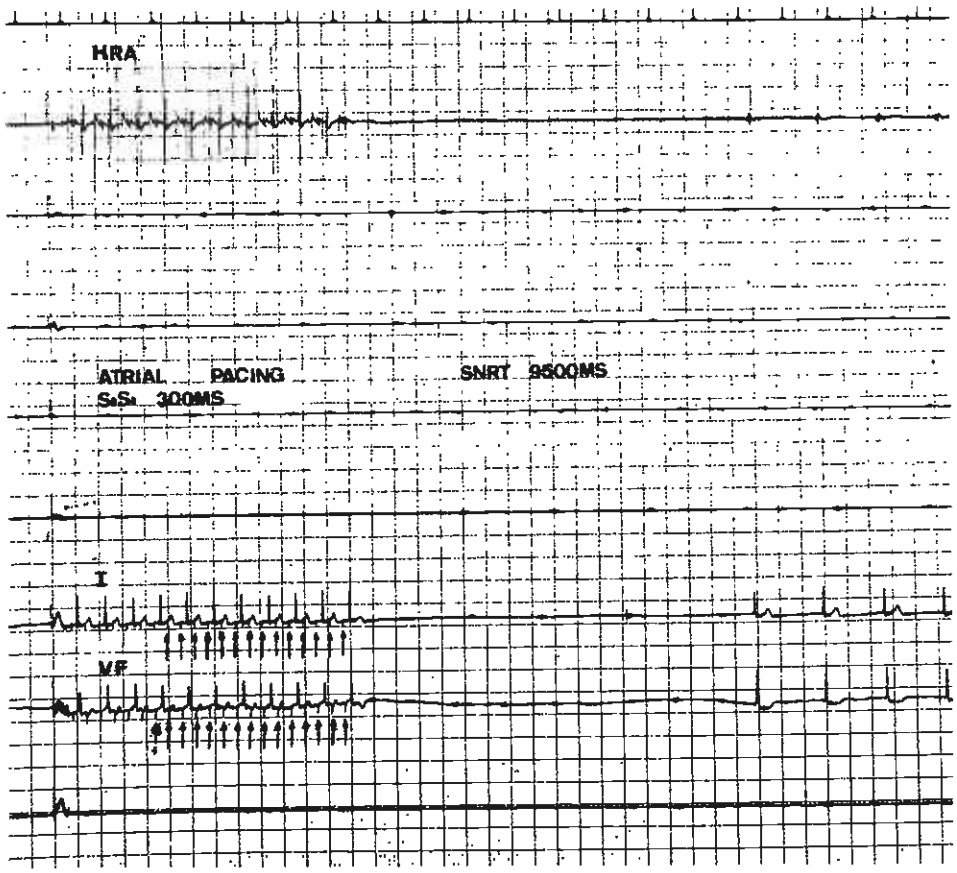

FIGURE 3

Assessment of Sinus node automaticity is done by the Sinus Node Recovery time (SNRT). The atrium is paced (arrows) at a stimulus interval of 300 ms (200 beats/minute) for 1 minute. On cessation of overdrive suppression of the Sinus node the node took 9500 ms to recover resulting in a extremely prolonged SNRT.

Atrio-Ventricular Blocks

The site of atrio-ventricular block can be reasonably deduced from the surface electrocardiogram. Wenckebach block (Mobitz type I) is usually due to atrio-ventricular nodal disease while Morbitz Type 2 block is due to His Purkinje conduction system disease. These electrocardiographic phenomenae are related to the different electrophysiological properties of the atrio-ventricular node and His Purkinje tissue. Recently, it has been recognised that Wenckeback block in the electrocardiogram may be caused by disease in the His Purkinje conducting system (6). Furthermore significant atrio-ventricular conduction delays can occur in some patients without electrocardiographic abnormalities. The use of electrophysiological recordings enables precise differentiation between the various sites of block: the site of atrio-ventricular block can be localised to the atrio-ventricular node, the bundle of His or distal to the His bundle. Patients symptomatic from infranodal block should have pacemarker implantation. Besides localising the site of block, the automaticity of the escape junctional or ventricular pacemaker can be assessed. For example, in some patients with Wenckeback block the escape junctional pacemaker may be unstable causing syncopal spells. Pacemaker implantation will then relieve symptoms.

Bundle Branch Block

The prognosis of patients with bundle branch block depends on the underlying heart disease, the severity of the conduction disturbance and the presence of symptoms (7). Young patients with isolated right bundle branch block have a benign prognosis and do not require further evaluation. Acute right or left bundle branch block during acute myocardial infarction on the other hand, carries a totally different prognosis. Mortality in these patients have been shown to be increased 3 to 4 fold and is related to the severity of myocardial damage, likelihood of progression to high grade atrio-ventricular block and presence of ventricular tachyarrhythmias (8). Stoke Adam attacks in patients with bundle branch block are due to either transient high grade atrio-ventricular block or ventricular tachyarrhythmias. Ambulatory monitoring can differentiate between them. However, though haemodynamically catastrophic these episodes may be infrequent and difficult to document during ambulatory monitoring.

Measurement of intracardiac conduction interval at rest and during cardiac stimulation enables a more accurate assessment of the conduction reserve of the His Purkinje system. The "HV" interval as measured from the intracardiac electrogram represents the conduction time of a cardiac impulse in the His Purkinje system. Abnormalities in the His Purkinje conducting tissues would result in abnormal HV intervals. Several prospective studies have shown that patients with HV intervals more than 70 ms are more likely to develop high grade atrio-ventricular block (9) (Figure 4). A subset of patients with a markedly prolonged HV interval of more than 100 ms have a even higher risk of progression. During electrophysiological studies, the possibility of ventricular tachyarrhythmias causing Stoke Adams attacks can also be evaluated with ventricular stimulation techniques.

Supraventricular Tachycardias

Narrow QRS complex supraventricular tachycardias

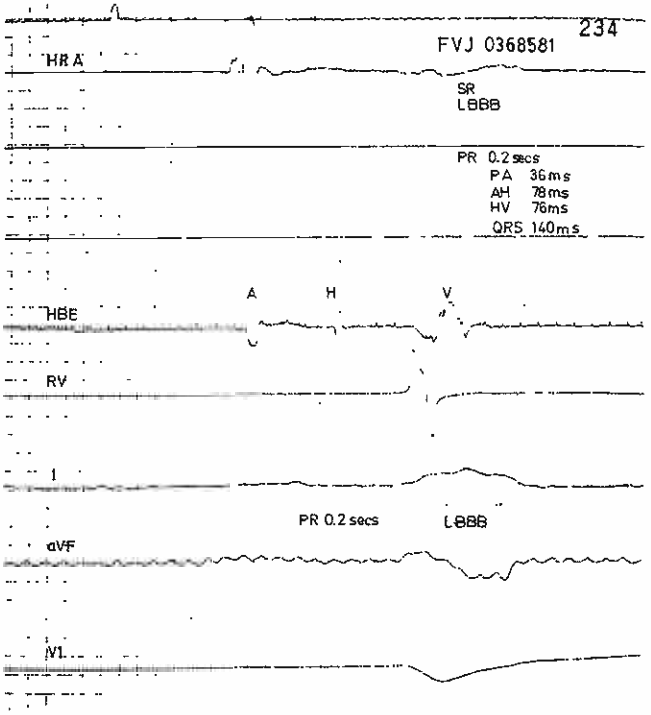

FIGURE 4

The patient has complete left Bundle Branch Block with a normal PR interval. Intracardiac recordings however, reveal a prolonged HV interval, showing the presence of trifasiccular disease. HRA = High Right Atrium electrogram; RV = Right ventricular electrogram; HBE = Septal electrograms; A = Atrial Septal electrogram; H = His Bundle Electrogram. V = Ventricular Septal electrogram.

are quite easily diagnosed by a 12-lead electrocardiogram. However, the 12-lead electrocardiogram, although sometimes offering "clues" (10) cannot reliably differentiate between the various types of supraventricular tachycardias. Electrophysiological studies have shown that supraventricular tachycardias are predominantly due to "re-entry or circus movement" mechanisms, with a minority due to automatic foci. The "re-entry circuit" is confined solely to the atrio-ventricular node or involving both the atrio-ventricular node and a acessory atrio-ventricular pathway (AAVC). About a third of patients with supraventricular tachycardia involving a AAVC do not have any evidence of Wolffe Parkinson White Syndrome on the electrocardiogram. This is because there is no antegrade conduction through the AAVC. These patients are said to have "concealed AAVC".

Defining the mechanism of the patient's supraventricular tachycardia enables a more rational plan of management. If the supraventricular tachycardia is due entirely to atrio-ventricular node re-entry, drugs acting solely on the atrio-ventricular node will be effective. If the re-entry circuit involves a accessory atrio-ventricular pathway, drugs with selective action on this pathway may be more useful. During the study the effects of drugs on the initiation and perpetuation of the tachycardia can also be investigated. Drugs, shown to be effective during electrophysiological studies, can then be selected for the patient. Drug selection based on the results of electrophysiological studies has enabled better control of the tachycardias (11). However, not all patients with supraventricular tachycardia will require detailed electrophysiological

assessment. If the tachycardia is occasional and not associated with syncope, it will be quite reasonable to use drugs such as digoxin or Propranolol empirically to control these attacks. Caution should be employed when Digoxin or Verapamil are used in patients with the Wolffe Parkinson White Syndrome. These drugs can increase conduction along accessory atrio-ventricular pathways. In some patients supraventricular tachycardia can degenerate into atrial fibrillation. In the presence of an accessory pathway (12) with a short refractory period, atrial fibrillation results in rapid conduction down this pathway causing ventricular rates of 300/- per minute or more (Figure 5). Ventricular fibrillation may precipitate sudden death in these patients with Wolffe Parkinson White Syndrome.

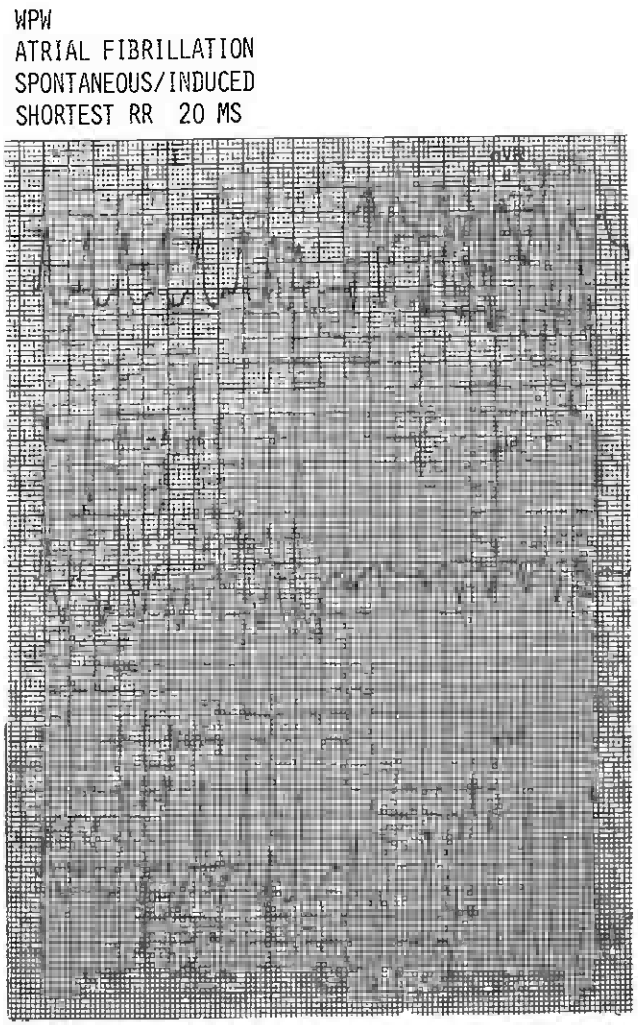

FIGURE 5

A patient with Wolffe Parkinson White syndrome having atrial fibrillation with rapid conduction in the accessory pathway giving a ventricular response of 300 beats per minute. Such patients have a high risk of sudden death from ventricular fibrillation. Note the wide QRS complexes due to conduction down the accessory pathway.

In summary, the indications for electrophysiological assessment of patients with supraventricular tachycardia will include:

1. Patients whose episodes of tachycardias are associated with severe haemodynamic upsets, such as hypotension, cardiac failure or syncopal attacks. In these patients, it will be necessary to identify accurately the optimal drug or drug regime to prevent further recurrences.
2. Patients with recurrent incapacitating episodes of tachycardias despite being on empirical therapy. Detailed electrophysiological testing with acute drug testing during the studies will enable a more rational and effective regime of management to be devised.
3. Patients with Wolffe Parkinson White Syndrome having tachycardias and syncope. In these patients, it will be necessary to investigate the functional properties of the accessory pathway accurately (Figure 6). Those with pathways capable of rapid conduction are at a higher risk of sudden death. Detailed electrophysiological studies enables selection of appropriate drug regimes that can effectively reduce conduction in the pathway. In patients considered for cardiac electro-surgery a pre-operative electrophysiological investigation is mandatory.

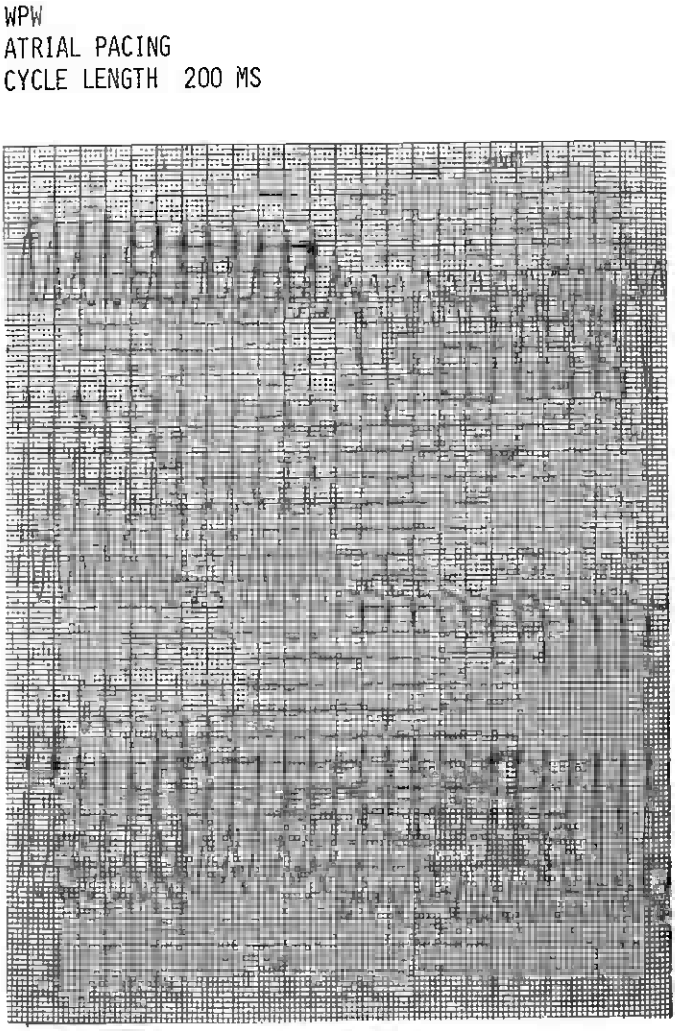

FIGURE 6

Rapid Atrial pacing in a patient with Wolffe Parkinson White Syndrome shows that the accessory pathway can maintain 1:1 conduction to a rate of 300 per minute. This identifies the patient with a increased risk for sudden death.

4. It is controversial whether asymptomatic patients with Wolffe Parkinson White Syndrome should be studied to identify those prone to sudden death. Most electrophysiologists feel that a study is not indicated unless the patient is engaged in heavy sports or hold positions like a air pilot or bus driver. Non-invasive methods such as exercise testing, use of Ajmaline or procainamide have been used to identify patients with rapidly conducting accessory pathways. At present the most accurate method of assessing bypass tract condition is still by electrophysiological testing, although stimulation of the left atrium through oesophageal pacing requires further research.

Ventricular Tachycardia

Wide complex tachycardias can pose diagnostic and therapeutic problems. With a 12-lead electrocardiogram supraventricular tachycardia with aberrancy can be differentiated from ventricular tachycardia. In some patients differentiation may be difficult and intracardiac electrophysiological recordings required to establish the diagnosis.

Most of ventricular tachycardias are re-entry in mechanism and can be reproducibly initiated (Figure 7) and characterised in the laboratory. The mode of initiation and site of origin of the tachycardia can be studied. Sustained ventricular tachycardia, especially if associated with haemodynamic upsets, convey an adverse prognosis. These tachycardias have previously been managed with drugs empirically. Prognosis of these patients is however, poor if the ventricular tachycardias are not controlled. In the laboratory the effect of various drugs on the initiation and perpetuation of the ventricular tachycardia can be studied. Various studies have shown that drug therapy, based on electrophysiological drug testing gives better control of the tachyarrhythmias compared to empiric trials.(13)

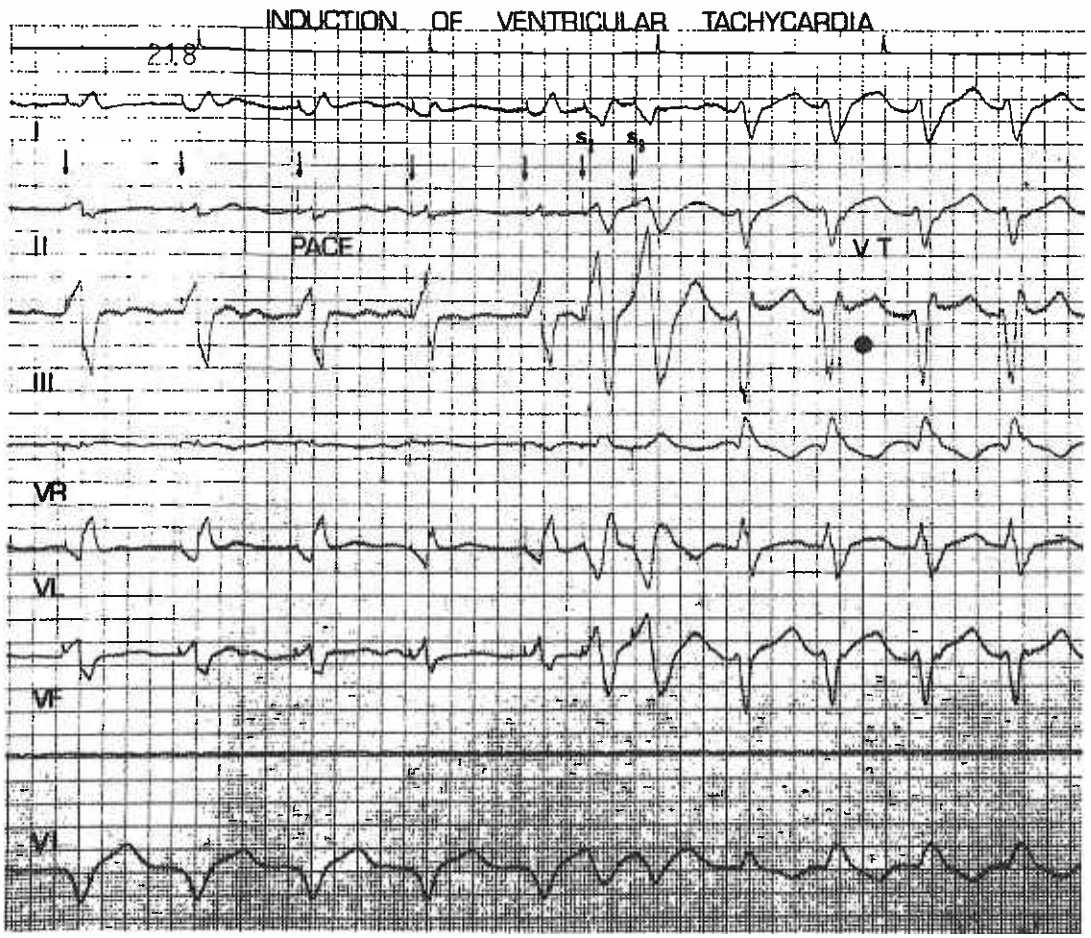

FIGURE 7

The electrogram shows the initiation of Ventricular Tachycardia with two ventricular stimuli (S2S3). The ventricle is paced at 100 beats per minute before two extrastimuli are delivered. The efficacy of different antiarrythmics in preventing further initiation of ventricular tachycardia can be assessed. This enables more certain prevention of future arrhythmias.

Newer methods in the treatment of ventricular tachycardia have evolved in the electrophysiology laboratory. These include pacing techniques, either by ectopic stimuli (Figure 8) or overdrive stimulation or the combination of both to terminate ventricular tachycardia. Needless to say, before a pacemarker is implanted for control of tachycardia it must be established that it will control or terminate the tachycardia in the laboratory. In some patients, various drug regimes may be unsuccessful in controlling life threatening ventricular tachyarrhythmias and these patients will require cardiac electrosurgery.

CONCLUSION

The cardiac electrophysiological laboratory is one of the newer techniques used in the investigation of major cardiac arrhythmias. These studies can be done with minimal morbidity and mortality, similar to that of cardiac catheterisation for an atrial septal defect. However, as the test is invasive, the benefits and indications of such an investigation must be clear. One must also be aware of the limitations of this technique. The results of the electrophysiological investigation must never be interpreted in isolation but form part of the total input on a patient's problem. Only then, will the cardiac electrophysiological investigations contribute meaningfully to the management of patients having major cardiac arrhythmias.

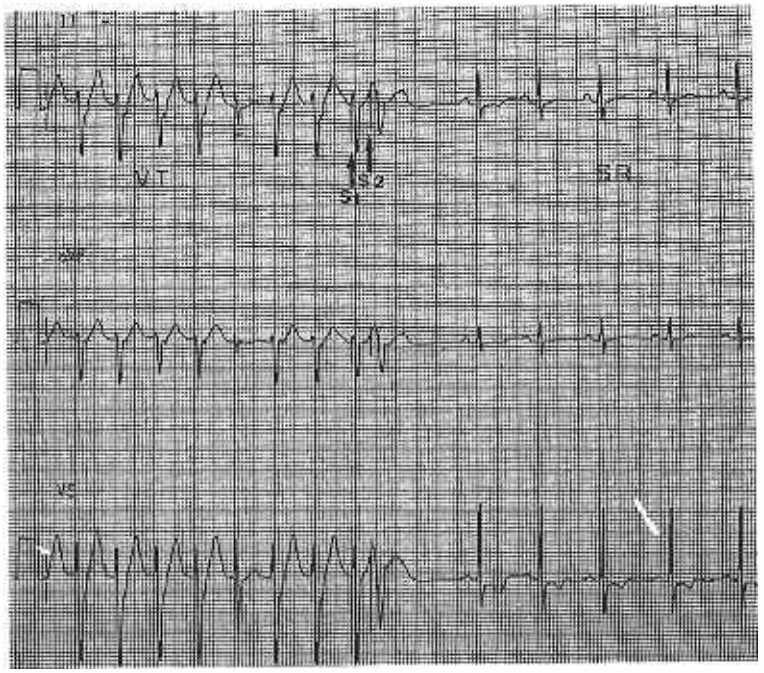

FIGURE 8

This electrocardiogram illustrates the termination of ventricular tachycardia with two ventricular extrastimuli (S1S2). In patients with ventricular tachycardias resistant to medical therapy but responsive to ventricular extrastimuli special "tachycardia pacemakers" will provide a alternative mode of therapy.

A similar problem exists in patients who have been resuscitated from a cardiac arrest. Ventricular tachyarrhythmias can be induced in up to 76% of these patients (14). As these patients were fortunate enough to survive a cardiac arrest, it will be necessary to establish a drug regime that will prevent a recurrence of the tachycardia. This can be achieved by drug studies during the electrophysiological investigation.

Syncope

In many patients who have syncope, it is difficult to establish a cause. Besides a thorough clinical examination patients should also undergo non-invasive neurological investigations. If these investigations are negative, a cardiac electrophysiological study can be done to try to establish a cause for the syncope. Electrophysiological abnormalities may be found in up to 68% of these patients (15). Appropriate therapy based on these abnormalities often result in a resolution of symptoms.

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