

Ventricular arrhythmias

Dr Edward Keelan

Ventricular arrhythmias range from benign ventricular ectopy to fatal ventricular fibrillation (VF). Because at one extreme they are potentially life-threatening, the presence of any ventricular arrhythmia often engenders anxiety and concern which may lead to unnecessary and inappropriate treatment. One must therefore attempt to distinguish those arrhythmias which are benign, requiring reassurance and follow-up, from those which are potentially more sinister, when aggressive intervention may be indicated.

Probably the two most important factors are the underlying cardiac status and the mode of presentation. If the cardiac function is normal with no underlying structural heart disease or genetic disorder such as the long Q-T syndrome, then almost all arrhythmias will be tolerated without acute haemodynamic collapse. This means that even those patients with sustained ventricular tachycardia (VT) will be able to seek medical attention and appropriate treatment can then be administered (see below).

On the other hand, if patients have a history of cardiac disease or if there is evidence of this on examination (one must remember that ventricular arrhythmias may be the presenting feature of coronary disease or cardiomyopathy) then the risk posed is greater. One should also pay special attention to any patients who present with syncope or near syncope (excepting the momentary dizziness often associated with isolated ventricular ectopics or 'missed beats') as these may be markers of a more malignant arrhythmia substrate.

Appropriate work-up starts with a complete cardiac history and examination. One should ask if there is a family history of sudden death as this may point to a possible genetic disorder. The ECG is helpful. Apart from the usual features, one may be able to determine the morphology of any ectopics, whether they are unifocal or multifocal and of right ventricular or left ventricular origin (usually LBBB and RBBB morphology, respectively). The morphology of any documented VT and additional features such as the Q-T interval (corrected for the heart rate) must also be scrutinised. The echo provides further objective assessment of ventricular function and any underlying valvular disease which may be followed over time.

Holter monitoring is helpful in determining the frequency and complexity of ventricular ectopy/arrhythmia. More subtle features, such as possible trends associated with changes in autonomic tone and/or diurnal variation in frequency, may become apparent. Event monitors, either those using standard external ECG leads or the newer implantable devices, may document the arrhythmia at the

time of symptoms in those with infrequent episodes. Stress testing can also be helpful, as benign ventricular ectopy usually disappears during exercise and re-emerges during the recovery phase, while exercise-induced/aggravated arrhythmia may be a more sinister finding.

More invasive investigations such as cardiac catheterisation and electrophysiology (EP) study are reserved for those with known or suspected structural heart disease and/or sustained or malignant arrhythmias. In patients with documented cardiac disease, one should always try to optimise the treatment of the underlying condition before undertaking EP studies. In addition to the manipulation of medical therapy, this may require angioplasty or even bypass surgery in those with significant coronary disease causing ischaemic injury.

Risks with therapy

If one decides to initiate therapy with antiarrhythmic drugs, one must always remember that all of these agents have potential proarrhythmic effects which may themselves prove fatal. There are now a number of multicentre studies which had to be terminated prematurely because of excess mortality in the active drug treatment arm(s). Most prominent among these is the oft-quoted CAST (Cardiac Arrhythmia Suppression Trial) study. In this, Class I antiarrhythmic agents given to suppress ventricular ectopics in

Figure 1a. Early implantable defibrillators were large, required open-heart surgery to place pericardial patches and the device was placed in a pocket in the anterior abdominal wall.

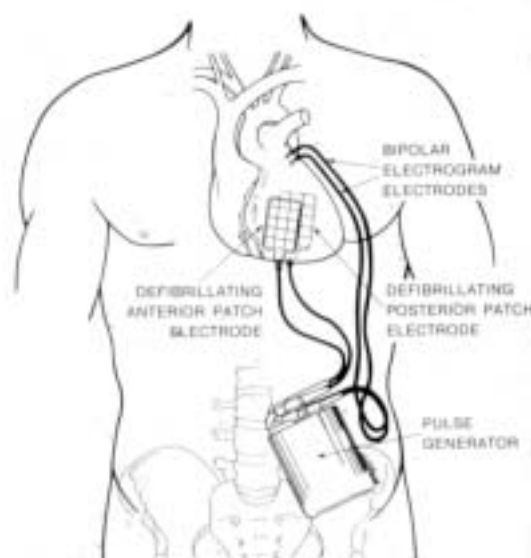
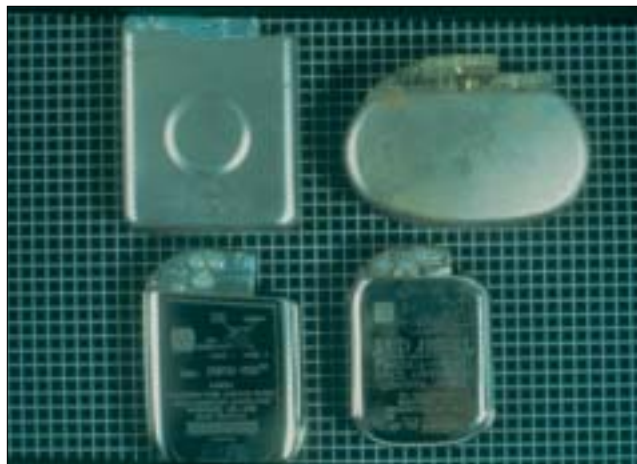


Figure 1b. With advancing technology, device size has become progressively smaller.



patients with coronary disease and frequent ectopy (known to be at risk for sudden arrhythmic death) actually caused an increase in mortality.

This finding was a particularly chastening one which served to warn electrophysiologists and all prescribing physicians that these agents have to be used very carefully and in a controlled way. It also highlighted the need for in-patient monitoring when initiating therapy, which should be considered for all those with structural heart disease. Finally, one should not forget the proven safety and efficacy of beta-blockers in patients with heart failure or post MI.

For patients at risk of life-threatening ventricular arrhythmias, or those surviving a cardiac arrest, the best way to prevent sudden death is the implantable defibrillator. These devices were first implanted in 1980. Early models were very large, required open-heart surgery to place pericardial patches and an abdominal pocket for the generator, had limited battery charge and could not distinguish between supraventricular or VT (Figure 1a). However, they saved lives. Today, technology has advanced to the extent that these devices are much smaller (like a large pacemaker), their implantation is technically much easier and consequently safer, they have multiprogrammable features with sophisticated detection algorithms and their battery longevity is increased (Figures 1b and 1c).

Unfortunately, they are still very expensive (£10,000-15,000 for the standard models and over £20,000 for the latest technology). However, when faced with patients at high risk, one must consider this option. The best that drug therapy can achieve in some of these patients is an annual sudden death rate of about 10% compared with a rate of 1% with a defibrillator (Figure 1d). These devices are therefore the best way to prevent sudden arrhythmic death.

Different ventricular arrhythmias

There follows a brief discussion of the more common forms of ventricular arrhythmia and their treatment.

Ventricular ectopic beats (Figure 2)

As discussed in Part 1 of this series, these are a

Figure 1c. Implantation is now in the pectoral region, similar to pacemakers.



common cause of symptoms in otherwise healthy adults. Patients will describe or recall 'skipped or missed beats' which are often most troublesome at rest or in bed at night. On questioning, patients will often admit to a fear of possible heart attack or cardiac arrest which magnifies their concern and worry.

Once structural heart disease has been excluded, the correct course of action is explanation and reassurance. Rhythm monitors can be helpful by confirming the cause of symptoms and excluding any more complex arrhythmia. Stress testing may also provide further reassurance as discussed above. Drug therapy should be avoided as it will only serve to reinforce any lingering anxiety about possible heart disease. Also, one might find oneself changing from simple beta-blockers to more potent (and risky) antiarrhythmic agents when the symptoms recur, as they usually do.

In contrast, patients with significant structural heart disease in whom frequent ventricular ectopic beats may be a marker of increased risk of sudden death are frequently

Figure 1d. Once these devices detect VT/VF, they charge internally and deliver a defibrillation shock which restores sinus rhythm, all within a number of seconds.

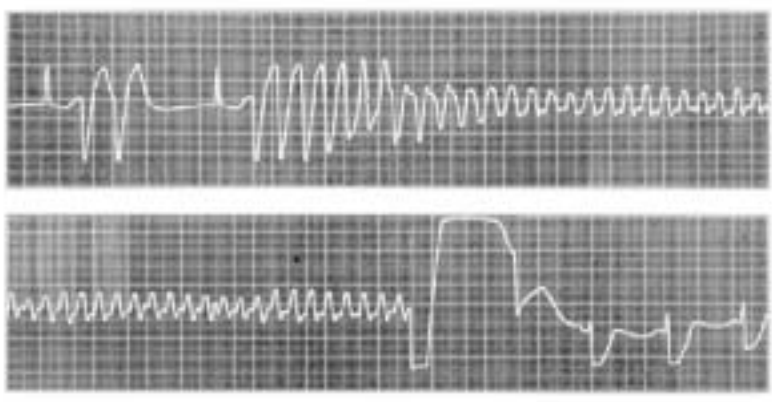


Figure 2. Isolated ventricular ectopic beats.



asymptomatic. While further invasive investigation may not be indicated, our index of suspicion is raised and we should certainly ensure that their medical therapy is optimised.

VT in patients with heart disease (Figure 3)

Patients with heart disease, especially those with impaired ventricular function, are at risk of VT. This may be nonsustained (defined as up to 30 seconds duration) and asymptomatic or sustained and associated with presyncope or collapse. Both forms are markers for increased risk of sudden death and consideration should be given to formal EP study during which programmed stimulation is used to test the inducibility of sustained tachycardia. There are now several controlled studies demonstrating the survival benefit afforded by implantable defibrillators in patients with inducible sustained VT. Antiarrhythmic therapy, predomi-

Figure 3. Sustained, haemodynamically unstable VT in a patient with coronary heart disease. The patient subsequently had a defibrillator implanted and received life-saving shocks for recurrent VT.



nantly with amiodarone, remains an alternative for those who are not suitable for or who decline defibrillator implantation.

'Normal heart' VT (Figure 4)

VT with focal origin in either the right or left ventricle may occur as a primary electrical problem in patients with otherwise normal cardiac function. The outlook for these patients is quite different from those with structural heart disease. Most particularly, the risk of sudden death is very low. Beta-blockers or even verapamil (for those with left ventricular VT) may provide good control and radiofre-

Figure 4. Tachycardia of focal origin from the right ventricular outflow tract in an otherwise healthy young adult. The patient subsequently underwent curative radiofrequency ablation.

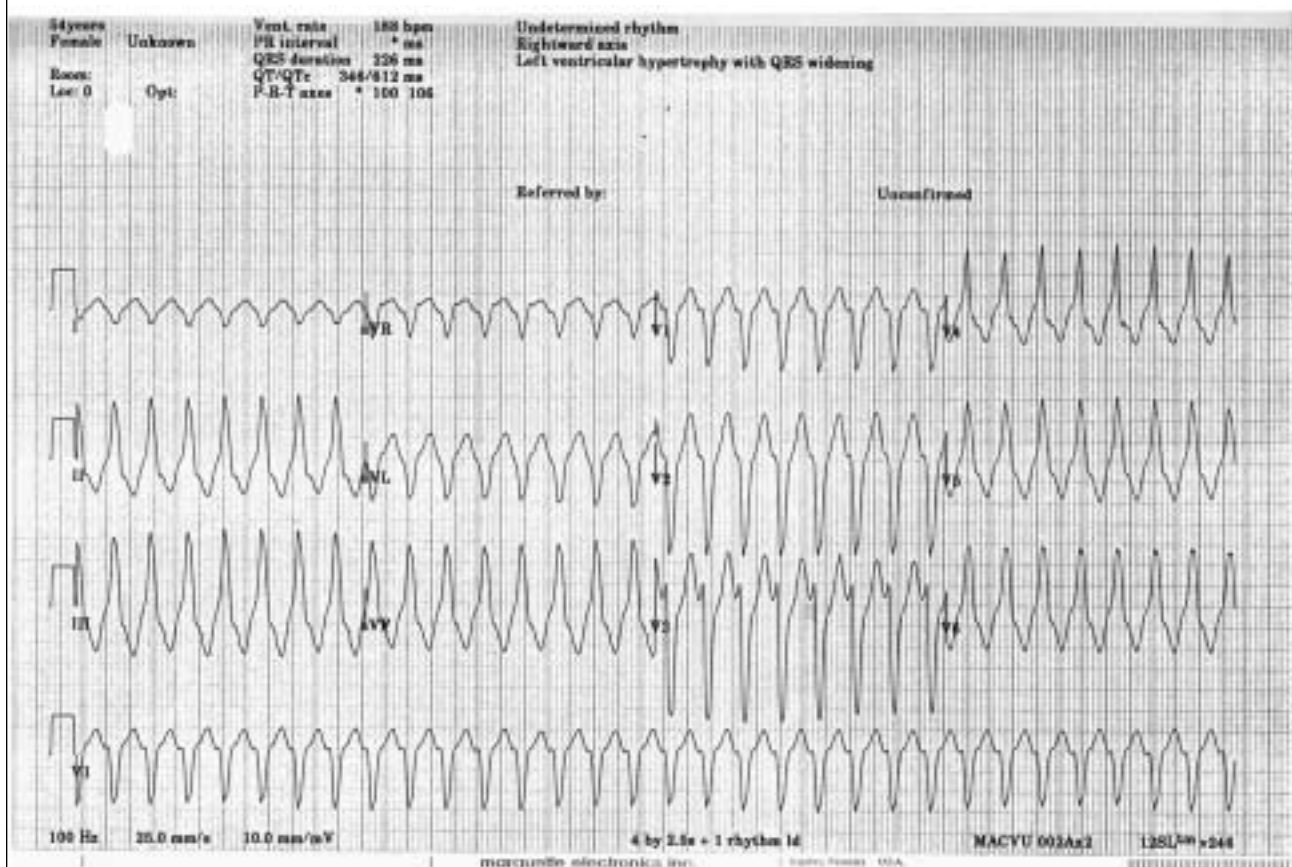
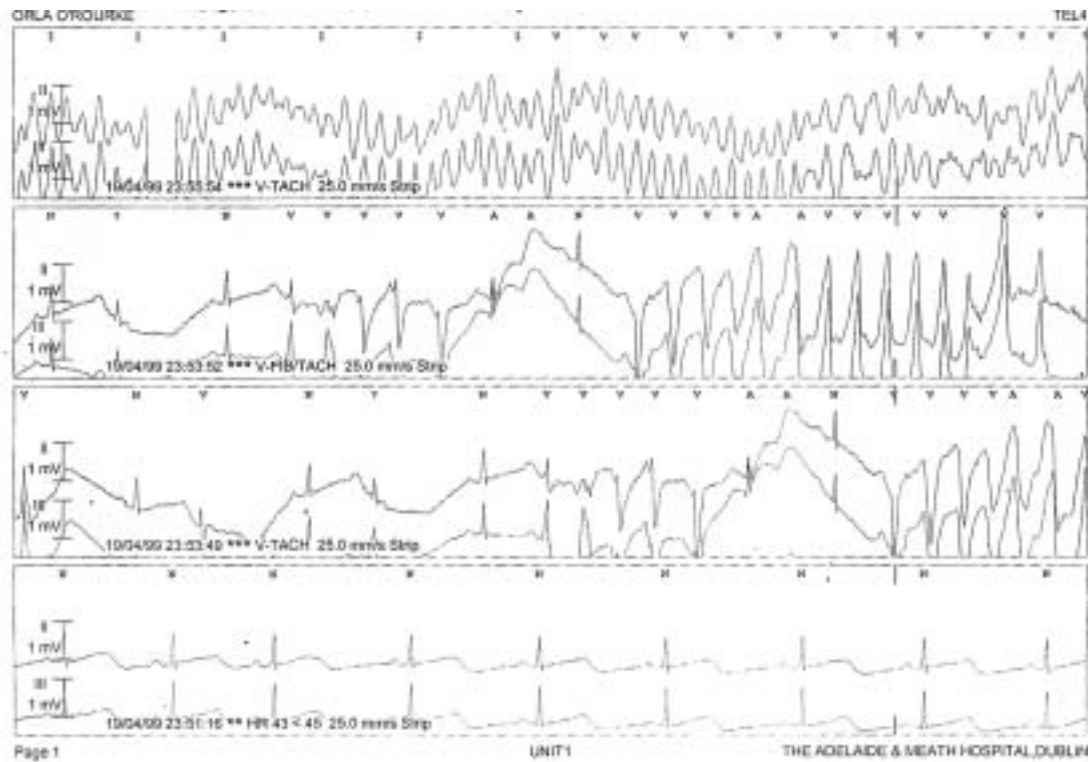


Figure 5. Nonsustained polymorphic VT (torsade de pointes variety) in a 19 year old student with long Q-T syndrome who had stopped beta-blocker therapy a few weeks previously. Tachycardia settled when adequate beta-blockade was re-established.



quency ablation offers the prospect of complete cure.

VF/cardiac arrest

With the exception of patients who have a cardiac arrest in the setting of acute myocardial infarction (MI), or where there is a clearly identifiable and reversible cause (e.g. significant electrolyte abnormality), all survivors should be considered for defibrillator implantation. Those with poor functional status due to advanced heart disease or other cause may be deemed unsuitable, but it should now be considered the treatment of choice for the remainder.

Genetic disorders predisposing to ventricular arrhythmias

The best characterised of these is the long Q-T syndrome, the most common form of which is also known as the Romano-Ward syndrome (after Prof OC Ward from our own Crumlin Hospital). Patients are at risk of polymorphic VT, which may cause syncope and even sudden death. The arrhythmias occur most commonly at times of increased adrenergic tone such as fright or sudden arousal

and beta-blockers remain the treatment of first choice (Figure 5). For those with adverse prognostic features, defibrillator implantation is now advised. Other disorders with a genetic basis include arrhythmogenic right ventricular dysplasia and the recently described Brugada syndrome. These rare conditions must always be considered during work-up of unexplained VT or cardiac arrest.

Conclusion

The spectrum of ventricular arrhythmia ranges from benign ectopics to life-threatening tachycardia or fibrillation. The first step in evaluation remains a careful history and exam. This will usually help to direct work-up towards either a simpler, non-invasive approach or a more detailed and intensive one. Some patients require simple explanation and reassurance, while others will need drug therapy or defibrillator implant. The good news is that, even for those at highest risk, much can now be done to avert the spectre of premature sudden death.

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