Ventricular tachycardia: a life-threatening dysrhythmia

Melanie Humphreys, MA, BSc (Hon), ENB 124, RGN, ONC, Senior Lecturer, Post-registration Studies, School of Health, New Cross Centre, University of Wolverhampton, Wolverhampton, UK

Senior Lecturer, Post-registration Studies, School of Health, New Cross Centre, University of Wolverhampton, Wolverhampton WV10 0QP, UK. e-mail: in5277@wlv.ac.uk

INTRODUCTION

Ventricular tachycardia (VT) is the most important differential diagnosis of a regular broad complex tachycardia, because it carries the least favourable prognosis of the potential causes. The results of misdiagnosing VT as supraventricular tachycardia (SVT) can be fatal, both at the acute presentation, and in the long term. Ischaemic heart disease is the commonest cause of VT, particularly in the middle-aged patient and the elderly. Indeed, following an acute myocardial infarction (AMI), most sudden deaths that occur do so as a result of VT or ventricular fibrillation (VF). As a result of the increased awareness of the dangers of misdiagnosis, diagnostic strategies have been developed to promote VT as the default diagnosis of a regular broad complex tachycardia (Forsey and Griffith, 1996; Connaughton, 2001; Edhouse and Morris, 2002). The critical care nurse can apply a number of well-supported morphological criteria facilitating a confident diagnosis of VT in most cases from a standard 12-lead ECG recording.

PATHOPHYSIOLOGY

Although there is no universally accepted definition of what is clinically important VT, most authorities agree that VT can be defined as three or more ventricular extrasystoles in succession, at a rate of more than 120 beats/minute. Ventricular tachycardia may develop without any warning signs but is most often preceded by frequent or dangerous forms of premature ventricular contractions (PVCs), i.e. occurring in pairs, runs or R-on-T type. The duration of the QRS complex must exceed 0.12 seconds, i.e. three small squares on a standard electrocardiogram (ECG). Generally speaking, the wider the complex, the more convincing is the diagnosis. Indeed, some authorities would assert a QRS duration of >0.16 seconds is more conclusive (Garcia and Holtz, 2001; Edhouse and Morris, 2002). However, durations can be relatively short – as in fascicular tachycardia, where the QRS duration ranges from 0.11–0.14 seconds (Wagner, 1994). The tachycardia may be self-terminating but it is described as 'sustained' if it lasts longer than 30 seconds (Thompson, 1997; Edhouse and Morris, 2002).

Ventricular ectopic beats may occur in normal individuals as well as in patients with organic heart disease. Rapid runs of consecutive ectopic beats lasting less than 10 seconds are defined as 'non-sustained' VT, and has little prognostic significance and rarely require specific symptomatic therapy. Indeed, these brief episodes may not produce any signs or symptoms (Dracup, 1995).

Ventricular tachycardia is described as 'monomorphic' when the QRS complexes have the same general appearance and 'polymorphic' if there is wide beat-to-beat variation in QRS morphology. Monomorphic VT is the commonest form of sustained VT, and this review will focus upon this phenomena. Monomorphic VT usually occurs after myocardial infarction and is a sign of extensive myocardial damage; it is associated with a high in-hospital mortality, more often resulting from impaired ventricular function with ischaemia, than recurrence of the arrhythmia (Edhouse and Morris, 2002).

The mechanisms responsible for VT include re-entry within the ventricles or increased myocardial automaticity. Re-entry requires two conditions. Firstly, a potential circuit made up of two pathways of conduction with differing electrical properties. Secondly, a transient or permanent block in one direction of the pathway, in order that an impulse can be conducted along one pathway and return in the opposite via the other pathway, thus a re-entry circuit. The re-entry circuits that support VT can be 'micro' and 'macro' in scale and often occur in the zone of ischaemia or fibrosis surrounding damaged myocardium. Ventricular tachycardia in a patient with ischaemic heart disease is probably caused by a re-entry phenomenon involving infarct scar tissue, and thus the arrhythmia tends to be recurrent (Edhouse and Morris, 2002). Enhanced automaticity can either be spontaneous or triggered after depolarisation, which leads to early activation of the myocardium. Damage and disease of the myocardial cells can result in enhanced automaticity.

Ischaemic conditions have produced a substrate for the initiation and maintenance of arrhythmias. In an acute event, absence of collateral flow in the area distal to the occlusion, and platelet microemboli downstream may contribute to the development of sudden ischaemia (Davies, 1990). These microemboli are seen in the arterioles and small arteries (downstream from an artery with a fissured plaque) in up to half of all patients who die suddenly. Platelet microemboli may be associated with the scattered foci of myocardial necrosis and may conceivably trigger fatal disturbances of rhythm and conduction. Ischaemia may alter regional conduction velocity and initiate some forms of automaticity. Additionally, when the ischaemic region is reperfused, an inward flux of calcium occurs, and this influx

correlates with bursts of ventricular ectopy. Thus, ischaemia may contribute to sudden death in several ways (Woods *et al*., 1995).

Ventricular tachycardia should be suspected in any patient with a regular broad-complex tachycardia who has significant damage (acute or long-term) to the ventricular myocardium. A broad-complex tachycardia is likely to be VT in at least 95% of the time in these patients; therefore the default diagnosis should be VT (Griffith, 1994; Connaughton, 2001). In VT, with rare exceptions, there is complete atrioventricular dissociation, so that every so often the right atrium will contract against a closed tricuspid valve. This causes intermittent cannon 'a' waves in the jugular venous pressure (JVP) (positive predictive value 82%) and a first heart sound varying in intensity from beat to beat (positive predictive value 100%). Thus, these features can be reliably used to determine the presence of VT (Garratt, 1994). Various conditions associated with the development of VT are listed in Table 1.

Clinically, the seriousness of VT varies depending primarily on the duration of the tachycardia, the rate, and the underlying condition of the heart (Lilly, 1993). When VT provokes symptoms, hypotension and loss of consciousness due to decreased cardiac output are the major manifestations (Lilly, 1993). Short runs of VT, lasting for only a few seconds, are seldom dangerous in their own right. However, they represent a warning of sustained VT and the possible development of VF. In contrast, when VT persists and becomes an established rhythm, serious haemodynamic consequences can be expected. The rapid ventricular rate, reduction in ventricular filling time, and loss of atrial kick significantly reduce cardiac output, which leads to left ventricular failure, cardiogenic shock, and myocardial and cerebral ischaemia (Humphreys, 2001). Hypotension and loss of consciousness are the major manifestations; the haemodynamic deficit may be so great that sudden death can occur (Lilly, 1993). Ventricular tachycardia tends to be unstable and at any time during its course, VT may suddenly change into VF. For this reason sustained VT must be considered to be in the same life-threatening category as VF (Huff, 1997; Wagner, 1994; Resuscitation Council, 2001).

CLINICAL FEATURES

Most patients with VT are immediately aware of the sudden onset of rapid cardiac activity and describe dyspnoea, palpitations, and light-headedness. Coronary blood flow occurs predominantly during diastole. High heart rates reduce filling time, resulting in poor coronary blood flow and myocardial ischaemia, which presents as angina. When angina is present, the patient senses that a catastrophe has occurred, and marked apprehension is evident. In contrast, some patients have remained haemodynamically stable, when first seen, and it is commonly this group that receive an erroneous diagnosis of supraventricular tachycardia (SVT). If an SVT is conducted to the ventricles in the presence of a bundle branch block (BBB), the complexes will appear broad, as they do in sinus rhythm with BBB. Unfortunately,this relatively rare group of tachycardias is frequently over-diagnosed. This has grave practical consequences if verapamil is given to abort a presumed SVT, as immediate haemodynamic deterioration often occurs (Forsey and Griffith, 1996), in the form of catastrophic hypotension, and as asystole if the rhythm is VT (Connaughton, 2001). Inaccurate diagnosis will in any case compromise the possibility of successful treatment, particularly in an emergency (Resuscitation Council, 2001). One of the key factors determining a patient's tolerance to a tachyarrhythmia is the severity of the underlying clinical problem and associated conditions (Wagner, 1994). When the heart has good cardiac reserve (otherwise healthy myocardium) haemodynamic stability will be maintained for longer.

The clinical features and the ECG criteria, described below, will generally allow a definitive diagnosis in at least 95% of cases of VT. However, if in doubt, treat as VT (Thompson,1997; Connaughton, 2001).

Table 1. Conditions associated with the development of ventricular tachycardia (VT)*

- Underlying heart disease
  - Myocardial infarction
  - Cardiomyopathy
  - Mitral valve prolapse
  - Congestive heart failure
- Certain medications, as these may prolong the QT interval, causing the ventricles to be particularly vulnerable to VT
  - Digoxin toxicity
  - Class I antiarrythmics (e.g. flecainide, quinidine and disopyramide)
  - Tricyclic antidepressants
- Electrolyte disturbances
  - Especially hypokalaemia and hypomagnesaemia
- Reperfusion following thrombolytic therapy or angioplasty

*Woods *et al*., 1995; Huff, 1997; Edhouse and Morris, 2002

ECG IDENTIFICATION

No diagnostic criteria can differentiate all wide complex tachycardias (Brugada *et al*., 1991). However, factors favouring monomorphic VT are:

- AV dissociation, fusion beats, capture beats: V_1 (MCL_1) is the superior lead for analysis of atrial activity. However, AV dissociation is seen in less than 50% of patients, while fusion and capture beats are seen in 20–30% of cases of VT.
- Very wide complexes >0.14 seconds (3.5 small squares on standard ECG paper).
- The same morphology in tachycardia as in ventricular premature beats.
- Previous MI.
- Absence of any rS, RS or Rs complexes in the chest leads. If an RS morphology is present, the interval from the onset of the QRS to the nadir of the S wave is >0.10 seconds (Brugada's sign).
- Concordance: all the QRS complexes in the chest leads are either predominantly positive or predominantly negative. Positive concordance probably indicates that the origin of the tachycardia lies on the posterior ventricular wall; the wave of depolarisation moves towards all the chest leads and produces positive complexes. Similarly, negative concordance is thought to correlate with a tachycardia originating in the anterior ventricular wall and is considered virtually diagnostic of VT.

ECG characteristics of VT

The ECG characteristics of VT are as follows:

- Rate: ventricular rate is normally 120–300 beats/minute.
- Rhythm: usually regular but may be characterised by slight irregularities of both rate and morphology. They maybe further disturbed by the presence of capture or fusion beats.
- P waves: often dissociated from QRS complexes. If sinus

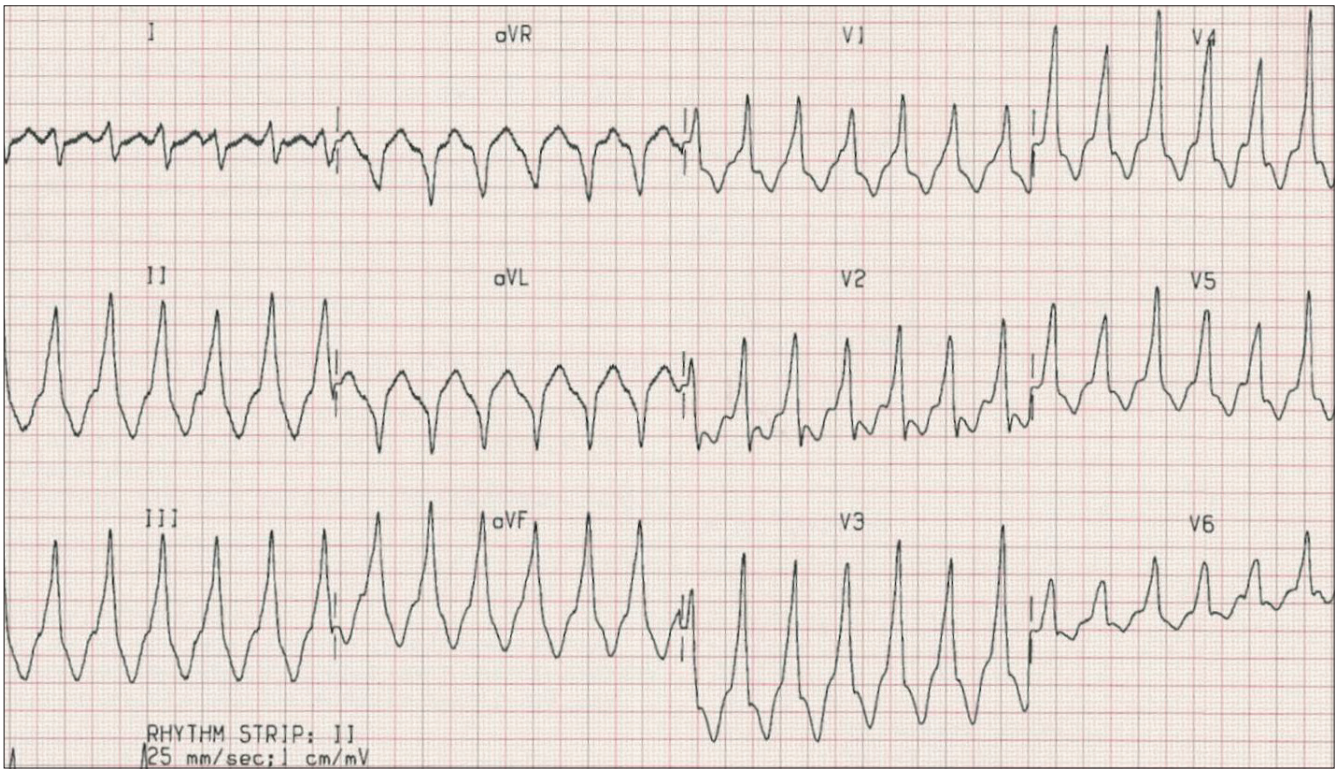
rhythm is the underlying basic rhythm, regular P waves may be seen but are not related to QRS complexes. P waves are often buried within the QRS complexes. Occasionally, VT conducts retrograde to the atria, and P waves can be seen after each QRS.

- PR interval: not measurable because of dissociation of P waves from QRS complexes.
- QRS complex: wide and bizarre; greater than 0.16 seconds in duration. However, as previously mentioned, the duration can be relatively short as in fascicular tachycardia. The complexes are predominantly positive (RBBB morphology) or negative (LBBB morphology) in V_1. Furthermore, when the QRS complex is mainly positive in V_1 a monophasic or diphasic morphology is indicative of VT, in the context of broad complex tachycardia. If the complex has two positive peaks in this lead (RSr'), it is widely accepted that these are termed rabbit ears. If the left 'ear' is taller then ectopy is suggested. If the right 'ear' is taller, then this is not helpful in indicating aberrancy from ectopy as it is common in both (Marriott and Conover, 1989).
- When QRS complexes have the same morphology, the term monomorphic VT is used. When the QRS complexes are different morphologies, the term polymorphic VT is used.
- T waves: large and travel opposite in direction to the main QRS deflection.
- Axis: with the onset of VT, the mean frontal plane axis changes from that seen in sinus rhythm and is often bizarre. A change in axis of greater than 40° to the left or right is suggestive of VT.
- Conduction: the conduction impulse originates in one ventricle and spreads through muscle cell-to-cell conduction through both ventricles.

(Brugada *et al*., 1991; Wagner, 1994; Woods *et al*., 1995; Forsey and Griffith, 1996; Thompson, 1997; Jenkins and Gerred, 1999; Edhouse and Morris, 2002).

BOX 1

Undertake a systematic analysis of the following 12-lead ECG



COMMENTARY

The 12-lead ECG shows the following features:

- Heart rate:160bpm
- Rhythm: regular pattern
- Axis: +90 (borderline right axis deviation, but essentially normal)
- QRS complex: slightly varied in morphology; probable AV dissociation; positive concordance in chest leads; RBBB morphology and monophasic R wave is present in lead V_1
- P waves: not clearly evident on the 12-lead ECG, however, irregularities in QRS morphology would suggest independent atrial activity
- PR interval: not measurable
- QRS duration: 0.16 seconds; prolonged and suggestive of ventricular origin
- T wave: opposite direction to QRS complex; normal secondary change in a ventricular rhythm.

The diagnosis is ventricular tachycardia; it is a very fast ventricular rate and is dissociated from an underlying atrial rate. There are some irregularities of the QRS morphologies at regular intervals. These irregularities are the underlying sinus beats.

There may be retrograde conduction through the atria, but often the sinus node continues to fire regularly and depolarises the atria normally. Occasionally, a sinus beat will fall on a spot that allows some innervation of the ventricle to occur through the normal ventricular conduction system. This forms a fusion beat, which has a morphology somewhere between the abnormal ventricular beat and the normal QRS complex. This type of complex is literally caused by two pacemakers, the SA node and the ventricular pacer. Because two areas of the ventricle are being stimulated simultaneously, the result is a hybrid – or fusion – complex with some features of both.

A capture beat is completely innervated by the sinus beat and is indistinguishable from the patient's normal complex. It occurs in the middle of the chaos that is VT, and is caused by chance timing of a sinus beat at just the right millisecond to 'capture' or transmit through the AV node and depolarise the ventricles through the normal conduction system of the heart. Fusion and capture beats are hallmarks of VT; if they are evident, they make the diagnosis definitive (Garcia and Holtz, 2001). However, fusion and/or capture beats are seldom seen and then only at the less rapid rates (under 160 beats/minute).

CONCLUSION

Established criteria for the positive diagnosis of VT can be complex. However, with careful consideration, ventricular tachycardia can be readily recognised from a 12-lead ECG recording, and should be the default diagnosis of regular broad-complex tachycardia (Connaughton, 2001); the presence of concordance would also suggest that the tachycardia has a ventricular origin (Edhouse and Morris, 2002). Ventricular tachycardia tends to be unstable and at any time during its course, VT may suddenly change into VF. For this reason, sustained VT must be considered to be in the same life-threatening category as VF. Thus, early recognition and appropriate intervention are vital for patient welfare.

ACKNOWLEDGEMENTS

My thanks are given to Andrew Smallwood, Charge Nurse, CCU, New Cross Hospital, Wolverhampton, UK, for his valued comments and reflections in the development of this paper.

REFERENCES

Brugada P, Brugada J, Mont L, Smeetes J, Andries EW. (1991). A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*; **83:** 1649–59.

Connaughton M. (2001). *Evidence-Based Coronary Care*. London: Churchill Livingstone.

Davies MJ. (1990). A macro and micro view of coronary vascular insult in ischaemic heart disease. *Circulation*; **82**(Suppl 3)**:** II38–II46.

Dracup K. (1995). *Meltzer's Intensive Coronary Care: A Manual for Nurses. (5th edn.)* California: Prentice-Hall International.

Edhouse J, Morris F. (2002). ABC of clinical electrocardiology: Broad complex tachycardia: Part 1. *British Medical Journal*; **324:** 719–22.

Forsey P, Griffith MJ. (1996). The electrocardiographic diagnosis of ventricular tachycardia. *British Journal of Cardiology*; **March:** 43–8.

Garcia TB, Holtz NE. (2001). *12-lead ECG: the art of interpretation*. London: Jones and Bartlett.

Griffith MJ. (1994). Ventricular tachycardia as default diagnosis in broad complex tachycardia. *Lancet*; **343:** 386–8.

Garratt CJ. (1994). Value of physical signs in the diagnosis of ventricular tachycardia. *Circulation*; **90:** 3103–7.

Huff J. (1997). *ECG Workout: Exercises in Arrhythmia Interpretation. (3rd edn.)* New York: Lippincott.

Humphreys M. (2001). Patients with inferior MI: right ventricular involvement. *Connect Critical Nursing in Europe*; **1:** 46–47.

Jenkins RD, Gerred SJ. (1999). *ECGs by Example*. London: Churchill Livingstone.

Lilly LS. (1993). *Pathophysiology of Heart Disease: A Collaborative Project of Medical Students and Faculty*. London: Lea and Febiger.

Marriott HJ, Conover MB. (1989). *Advanced Concepts in Arrhythmias*. London: Mosby.

Resuscitation Council (UK). (2000). *Advanced Life Support Course Provider Manual. (4th edn.)* London: Resuscitation Council (UK) and European Resuscitation Council.

Thompson PL. (1997). *Coronary Care Manual*. London: Churchill Livingstone.

Wagner GS. (1994). *Marriott's Practical Electrocardiography. (9th edn.)* London: Williams and Wilkins.

Woods SL, Froelicher ESS, Halpenny CJ, Underhill-Motzer S. (1995). *Cardiac Nursing. (3rd edn.)* Pennsylvania: Lippincott.

GLOBAL CONNECTIONS – LETTERS

Dear Editors,

I am writing to express my sincere thanks to all the support I have received from your readers. In the winter 2001 issue of *Connect* you published an article about my experience as an intensive care nurse in Kenya (O'Keefe, 2001). I have had a wonderful response. Many nurses from units in Britain have sent encouraging and supportive e-mails and letters and I have also received generous donations of books and journals as well as thoughtful parcels including sterile gloves, micropore tape and nasal airways. I was also given a very generous donation of GBP200 worth of new books from the BACCN (British Association of Critical Care Nurses). What once a bare store room is now a well stocked and up to date resource centre.

A few senior nurses and resuscitation officers have written to ask if I wanted to set up links with their hospitals and Consolata. This is a wonderful idea, and could really help us here. However, at the moment we are without a matron and as I am about to leave, I think it would be better to wait or such links might be neglected with the appointment of a new matron. I am staying in Kenya as I have a new job managing an abandoned babies home in Nairobi and I plan to maintain strong links with the hospital. if anyone is still interested, they are welcome to contact me and I will let them know any new developments.

Thanks again to everyone for their generous and thoughtful support; it has certainly helped me a lot through times of complete despair and I am confident that I am leaving Consolata with a team of nurses who are competent in intensive care nursing, which is a very satisfying feeling. I just hope they keep it up.

Megan O'Keefe
Voluntary Services Overseas
E-mail: ftcmegan@africaonline.co.ke