

**VENTRICULAR TACHYCARDIA**



*“Self-portrait Shading the Eyes” c. 1747-9, oil on canvas, Sir Joshua Reynolds, National Portrait Gallery London.*

*Sir Joshua Reynolds once remarked of Titian, “by a few strokes he knew how to mark the general image and character of whatever object he attempted; and produced, by this, alone, a truer representation than his master Giovanni Bellino, or any of his predecessors, who finished every hair”. This disinclination to “finish every hair” would become one of his own trademarks, though not one of which everyone approved: after Reynolds’s death, George III, who was short sighted and therefore tended to view pictures at very close quarters, spoke of Reynolds’s portraits as “course and unfinished”. The remark was made to Sir William Beechey, to whom the King was sitting at the time, and, Beechey, more of a courtier than Reynolds had ever been, attempted a diplomatic response. “Your majesty,” he said, “who is so perfect a judge of music, knows that the effect of the finest overture may be harsh and unpleasant when we are too close to the orchestra; so the pictures of Reynolds may appear course if we look at them too near, but at the proper distance they are all harmony”. The King, however, was not to be fobbed off, and by way of return of service produced a magnificent non sequitur: “Very good; but why did he paint red trees?”*

*In cases of wide complex tachycardias it is important to not to get too involved in “splitting hairs” over an exact ECG diagnosis. Rather is it best to heed Sir William’s advice to stand back and observe the “big picture”, as Sir Joshua does in his self-portrait. The “big picture” is not the exact ECG diagnosis, but how “unwell” the patient is and whether or not they require immediate cardioversion.*

## **VENTRICULAR TACHYCARDIA**

### Introduction

- Ventricular tachycardia which is **sustained** is a cardiac emergency that requires immediate treatment.
- The mode of treatment will depend on how compromised the patient is and the underlying cause.
- Initial diagnosis will often be problematic but in these cases VT should always be assumed in the first instance.

### Pathophysiology

#### VT Classification

From an electrophysiological viewpoint VT can be classified as:

1. **Monomorphic:**

This is VT where the morphology of all complexes is identical.

- This type of VT tends to be the result of a **fixed anatomical** abnormality within the myocardium.

2. **Polymorphic:**

This is VT where the morphology of the complexes is variable.

- This type of VT tends to be the result of a **migratory physiological** ventricular abnormality, hence the changing morphology.
- A subtype of polymorphic VT is that which is induced by a prolonged QT interval and results in a characteristic “torsades de points”. The distinction is important as treatment is different to polymorphic VT, which is not induced by prolonged QT interval, (see Long QT syndrome and Torsade guidelines)

### Causes

The causes of VT can be broadly considered in two groups:

1. *Primary cardiac disease*

- IHD (including old scarring)
- Cardiomyopathies, (of any type)
- Other structural heart disease eg ventricular aneurysms.
- Myocarditis.
- Congenital electrophysiological abnormalities.

Note that these abnormalities although they may show characteristic features on ECG, cannot be definitively diagnosed on ECG alone, they will require specialist electrophysiologist investigation.

Examples include, (in order of importance):

- ♥ Long QT syndromes, (which produce a characteristic form of polymorphic VT, torsades de pointes, but may also produce a monomorphic VT)
- ♥ Brugada's syndrome.
- ♥ ARVC/D, (arrhythmogenic right ventricular cardiomyopathy / dysplasia). There are also other variants of this syndrome.
- ♥ Idiopathic VT, seen in young patients, classically exercise induced VT and has a relatively benign prognosis. This is a rare condition.

Note that conditions of “aberrant pathways” such as WPW syndrome may lead to VF, but they do **not result in VT**, as the abnormal pathway generating the abnormal rhythm (apart from cases of VF) is from the supraventricular tissue, (an SVT by definition). This is not to say, however that a patient who has WPW cannot develop VT due to another reason!

## 2. *Secondary causes*

- Hypoxia, hypercarbia
- Electrolyte, (hypokalemia, hyperkalemia) and acid/base abnormalities
- Drugs and poisons, (classically, TCAs, digoxin, chloral hydrate, anti-arrhythmic agents and sympathomimetic agents in general)
- Environmental causes, hypothermia, hyperthermia, electrocution, (though more commonly this will result in VF or asystole)
- Mechanical, myocardial contusions.
- Severe systemic sepsis.

## Investigations

**Note that investigation should never delay definitive initial treatment in the compromised patient**

### Blood tests

- FBE
- U&Es (urgent potassium)
- Glucose
- Magnesium and calcium.
- Cardiac enzymes
- Others as clinically indicated, (eg digoxin levels, TFTs).

### CXR

- This should never delay treatment, even in the non-compromised patient.

### 12 lead ECG

This should be done before treatment in all cases, providing the patient's clinical condition allows this.

ECG Definition:

- Wide complex QRS (> 3 small squares)
- Rate > 100 (usually 140-200)
- 3 or more successive ventricular complexes.
- Regular (in vast majority of cases).

VT may be difficult to distinguish from SVT with aberrancy. Various morphological criteria, such as those of Wellens and Brugada may be used to help distinguish the two, however these criteria will often not be helpful in providing a definitive diagnosis. (See ECG criteria for VT guidelines)

**If there is any known structural cardiac disease, VT will be the likely diagnosis.**

## Management

**Management of VT can be problematic, and will depend on how compromised the patient is and the cause of the VT.**

The following basic principles apply:

- **When confronted with a wide regular and fast tachyarrhythmia, VT should be assumed in the first instance and it should be treated as such. There should not be excessive rumination on the analysis of the ECG.**
- If the rhythm is regular and the patient stable adenosine is safe to use, (if it is thought that the rhythm is probably SVT with aberrancy). Verapamil however is not safe as severe hypotension may result if the rhythm is VT and not SVT with aberrancy.
- All cases should be managed in a resuscitation area.

### Management Options:

1. Any **arrested** patient **without a pulse**, whatever the cause or type of VT, should be **defibrillated**, (ie the synchronizer is **off**) according to standard guidelines.
2. Any unstable patient should be **cardioverted**, (ie, the synchronizer is **on**).

Unstable may be defined as

- An altered conscious state.
  - Acute pulmonary edema
  - Ongoing ischemic chest pain
  - Hypotensive, (generally defined as a systolic pressure of less than 90 mmHg)
3. Patients who appear relatively stable may be cardioverted or considered for drug treatment.
    - **The best treatment for all VT (with respect to attaining sinus rhythm) of whatever cause or type in these cases however is still cardioversion, though the attendant risks of sedation that is required for the procedure need to be kept in mind.**

### Drug option considerations:

**In general, one drug only should be tried and if this fails, then cardioversion should be used rather than a second drug.**

**In cases of VT that are not clearly ischemic in origin, the best treatment will be cardioversion as some cases of non ischemic VT may respond adversely to certain drugs, depending on the underlying pathology.**

### *VT due to ischemic heart disease*

- **Amiodarone** is a reasonable first line treatment, its rate of reversion is probably not much superior to other agents, however it has less toxicity than other agents.

**Give amiodarone 5 mg/kg IV over 10- 20 minutes.**

- **Sotalol** may be considered if amiodarone is unsuitable. It has been shown to be superior to lignocaine (for structural heart disease)<sup>3</sup>

**Give sotalol 1 to 2 mg/kg IV, over 10 to 30 minutes**

*VT due to Brugada's syndrome, (see separate guidelines)*

- Any class I agent, (especially flecainide and including lignocaine) ie sodium channel blocking agents, are problematic and should be **avoided**.

*VT due to prolonged QT syndrome, (which may be the classic "torsade" but may also on occasions be monomorphic)*

- Any class Ia, Ic or class III (amiodarone /sotalol) agent should be avoided.

*Idiopathic VT of the young*

- This form of VT can respond to adenosine and verapamil. Verapamil, however should only be used on patients who have had this condition proven electrophysiologically and only then on the advice of the treating cardiologist

4. Treat the underlying cause.

- VT will be very resistant to any drug treatment if it is due to a secondary cause such as digoxin toxicity or hypothermia.
- Again cardioversion is a more effective option, but the underlying cause must also be treated, (eg: FAB fragments for digoxin toxicity, sodium bicarbonate for TCA overdose, warming for hypothermia, oxygenation for hypoxia etc)

Disposition

- All cases of VT should be admitted to CCU.
- Prophylactic amiodarone infusions in ischemic cases is not necessary unless there is ongoing / recurrent episodes of VT.

References:

1. Lancet vol 341, June 12 1993: 1512.
2. O'Donnell D, Cox D. Clinical and electrophysiological differences between patients with arrhythmogenic right ventricular dysplasia and right ventricular outflow tract tachycardia. Eur Heart J. 2003 May; 24 (9): 801-10.
3. HO DS et al, Double-blind trial of lignocaine versus sotalol for acute termination of spontaneous sustained ventricular tachycardia. Lancet 1994 Jul 2; 344(8914):18-23.

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