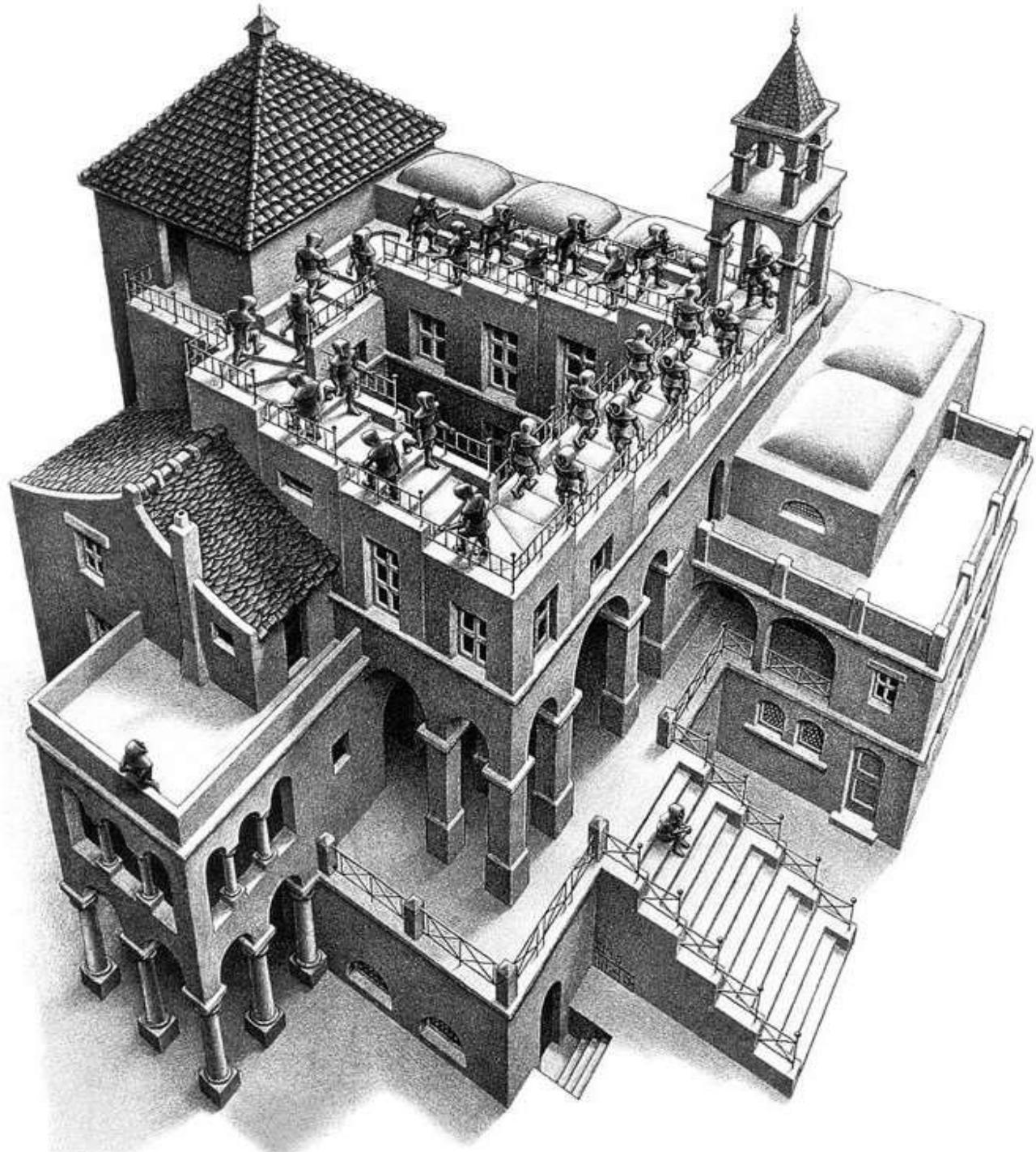


**SUPRAVENTRICULAR TACHYCARDIA**



*"Ascending and Descending"*, M.C Escher, Lithograph 1960.

*I'm busy designing a new print showing a staircase that goes on endlessly ascending - or descending, if you see it that way. This would normally have to be a spiraling thing in which the top would disappear into the clouds and the bottom into Hell. Not in my version... Nevertheless, it's possible to draw it with correct perspective: each step higher (or lower) than the last one. A large number of human figures walk on it in two directions. One procession climbs wearily up ad infinitum; the other descends endlessly...*

*That staircase is a rather sad, pessimistic subject, as well as being very profound and absurd... Yes, yes, we climb up and up, we imaging we are ascending; every step is about ten inches high, terribly tiring - and where does it all get us? Nowhere; we don't get a step farther or higher. And descending, running down with abandon, is not possible either.*

*People don't like to talk about falling; they'd much rather talk about ascending. How absurd it all is sometimes it makes me feel quite sick.*

*M.C Escher 1960.*

*Like the figures in Escher's haunting lithograph trapped in a never-ending "other dimensional" cycle, most symptomatic SVTs are the result of impulses similarly trapped in a never ending "electrical" cycle. Rather than of the "Ascending, Descending" variety, these impulses are caught within an "anterograde - retrograde" cycle.*

## **SUPRAVENTRICULAR TACHYCARDIA**

### **Introduction**

SVT is a relatively common presentation to the ED.

The ECG will typically show rapid, regular and narrow QRS complexes.

Occasionally the complexes will be wide due to pre-existing bundle branch blocks or due to accessory pathways. In these cases it will be very difficult if not impossible to distinguish the rhythm from VT. If there is doubt the rhythm should always be considered to be VT in the first instance and treated accordingly.

### **Pathophysiology**

#### **Mechanism:**

Tachyarrhythmias may be due to one of 2 mechanisms:<sup>1</sup>

1. Accelerated automaticity of a pacemaker (atrial / junctional tachycardias)
2. Triggered activation due to a re-entry circuit, which may be either:

- Micro re-entry (occurring within the AV node)
- Macro re-entry (occurring via an accessory pathway)

Note that the acceleration of automaticity is limited by the maximal rate of impulse formation in pacemaker cells and therefore rarely results in a clinically important tachyarrhythmia. Most symptomatic tachyarrhythmias therefore are due to the re-entrant circuit type capable of much faster rates.

Causes:

1. Organic heart disease including:
  - IHD and ACS
  - Cardiomyopathies
  - Valvular heart disease (in particular mitral valve lesions).
2. Aberrant conduction pathways:
  - WPW in particular.
3. In association with intrinsic electrophysiological defects, usually as a result of inherited genetic factors.
4. Myocarditis
5. Drugs
  - In particular sympathomimetic agents, including caffeine, digoxin, alcohol and salbutamol.
6. Thyrotoxicosis
7. Electrolyte abnormalities, (ie hypokalaemia)
8. Anxiety.
9. Smokers.

Complications:

This rhythm is usually fairly well-tolerated, however the following complications may be seen, especially in those with significant co-morbidities or pre-existing ischaemic heart disease.

- Hypotension
- Ischaemic chest pain
- Dyspnoea

### **Clinical Features**

The following symptoms and signs may be present:

1. Palpitations.
  - A useful question is whether or not the palpitations were regular or irregular, (may point to SVT or AF)
2. Light headedness or (uncommonly) syncope.
3. Shortness of breath
4. Chest pain, especially in those with pre-existing coronary artery disease.
5. Prominent jugular venous pulsations may be seen, (these are A waves due to atrial contraction against a closed tricuspid valve). These may be experienced as a “pulsating” sensation by the patient.
6. Onset and offset
  - The onset and offset of symptoms is usually very abrupt. This is in contrast to sinus tachycardia where the onset and offset are gradual.

### **Investigations**

#### **Blood tests**

- FBE
- U&Es and glucose
- TFTs
- Others should be done as clinically indicated eg, digoxin level, cardiac enzymes.

#### **CXR**

- Especially for first presentations.
- To rule other pathology, such as infection.

## 12 lead ECG

ECG features show:

1. Rate is typically between 140 - 250.
2. Regular.
3. QRS is normal duration (unless aberrant conduction or pre-existing bundle branch block)
4. P waves can be:
  - Buried within the QRS complexes and not seen (usually with A-V nodal micro re-entry circuits)
  - Inverted and often after the R wave (may be seen in aberrant “macro re-entrant” circuit pathways)
  - Preceding upright P waves (automatic focus, SA re-entry)
  - The P waves, if seen have a fixed relationship to the QRS complexes.

## Management

1. Monitored cube and IV access.
2. Try vagal manoeuvres such as:
  - Carotid Sinus Massage, providing the patient does not have significant carotid artery disease
  - Valsalva
  - Ice packs to the face may be useful in children unable to Valsalva.

**Treatment then depends on how unwell the patient is. Most will respond well to drug therapy, a few may require cardioversion.**

Options will include:

### **Cardioversion:**

- DC cardioversion if the patient is very unstable (BP < 90 especially with an altered conscious state)
- Failed medical treatment.

## **Drug Options:**

Before considering drug options it is important to check that the patient is not in fact in AF. This is because if they are in AF and this is due to an underlying pre-excitation syndrome, then A-V nodal blocking drug options will be contra-indicated (see also [AF guidelines](#)).

### Adenosine:

**This is the best first line option for SVT**

**If adenosine is not successful reconsider the diagnosis of SVT**

#### Advantages include:

- Half-life is very short (seconds), therefore any adverse reactions will be short lived
- Is safe in VT.
- It is relatively safe in the hypotensive patient
- Will not revert a flutter, but will make the diagnosis more apparent by increasing the degree

#### Disadvantages include:

- Relatively contra-indicated in severe asthma/ COAD
- Patients will often experience unpleasant “flushing” or “impending doom” sensation on reversion to sinus rhythm, however this usually responds well to firm explanation and reassurance.

#### Dosing:

- Initial dose is a **6 mg IV**, given *rapidly* into a large bore cannula (cubital fossa is best if possible), followed by a *rapid* saline bolus push. Its action is dependent on a rapid bolus effect on the heart.

If 6 mg IV is not successful, then a **second dose of 12 mg IV** rapid bolus injection should be tried.

Occasionally a **third dose of 18 mg** may be necessary.<sup>3</sup>

**On occasions a second line agent may be required. This may be because adenosine was unsuccessful or only temporarily so.**

**Note that one reason for failure of adenosine treatment is that the rhythm is not SVT, it may be an extreme sinus tachycardia or it may in fact be a VT.**

One study has also shown that the ingestion of caffeine (an adenosine receptor blocker) within four hours significantly reduces the effectiveness of a 6 mg IV dose of adenosine. Higher doses may be required in these cases.<sup>4</sup>

**Second line options for SVT include:**

[Verapamil](#)

This was historically the first line option, till replaced by adenosine.

[Indications](#)

- Verapamil may be used if there is an absolute contra-indication to adenosine providing the blood pressure is adequate (> 90 mmHg).
- It may also be considered in cases where adenosine brings only temporary reversion, as verapamil has a much longer half-life.

[Disadvantages:](#)

- Verapamil is absolutely contra-indicated in VT. In if doubt about the diagnosis then do not use it and treat as for VT.
- Verapamil is also contra-indicated in ages < 2 years.
- It is best avoided in patients on **beta-blockers**.
- It should be used with caution in patients with significant pre-existing cardiac disease due to its myocardial depressant effects.

[Dosing:](#)

- **Give 1 mg IV per minute up to a maximum of 15 mg.**

[Beta-blockers](#)

- These are an alternative to verapamil.
- The most widely used agent is metoprolol.

[Dosing:](#)

- Give metoprolol 5 mg IV *slow* bolus over 1-2 minutes.
- Three doses (total 15 mg maximum) may be given IV over a 15-minute period.

### Aramine

In significant hypotension small boluses of aramine may be tried as an alternative to cardioversion.

- Give **0.5 -1.0 mg IV** boluses titrated to response, (2 to 3 doses)
- This agent is thought to work via a “medical valsalva” mechanism, ie the sudden increase in blood pressure invokes a reflex baroreceptor vagal response, which terminates the arrhythmia.

### Post reversion

Following successful reversion:

1. Do a *post reversion* ECG in all cases, look for evidence of ischemia or aberrant pathways.
2. Observe the patient for 1-2 hours post reversion in monitored cube.

### Disposition

Patient may be discharged home providing:

- They have remained stable with normal bloods and post reversion ECG.
- No significant co-morbidity.
- Adequate social supports/follow up arrangements.
- A precipitating ischaemic event does not need to be ruled out.

Follow-up:

- An echocardiogram should be organized as an outpatient for first presentations, especially in those without an obvious cause.
- **Referral to an electrophysiologist should always be considered, especially in younger patients, as electrical ablation therapies will often be able to affect a cure.**

### Prophylactic Medication:

The best longer-term treatment is radiofrequency ablation therapy performed by an experienced electrophysiologist.

Some patients may not be suitable for this treatment or decline it. They may be considered for medical prophylactic treatment; however there is no current ideal drug in this setting and all options have significant side effects, especially in the long term.

Patients who suffer infrequent and minimally symptomatic episodes of relatively short duration will not require any prophylactic medication.

The options for prophylaxis to be considered will include:

- Atenolol
- Metoprolol
- Sotolol
- Verapamil sustained release.

**For full prescribing details, see the latest edition of the CVS Therapeutic Guidelines.**

If the above agents are not successful the following 2 agents may be considered *on the advice of a cardiologist*:

- Flecainide.
- Amiodarone

There are two main approaches:

1. The *stat* or *one off* dose, *as needed*:
  - These are for those patients with sporadic episodes, which are prolonged (> 1 hour) and/ or have significant symptoms.
  - Calcium channel blockers or beta-blockers have been advocated (providing pre-excitation has been ruled out) or alternatively flecainide.
2. Longer term maintenance therapy:
  - This may be an option for those unsuitable/ unwilling to have ablation therapy and who suffer from frequent debilitating episodes.
  - Again calcium channel blockers or beta-blockers have been advocated (providing pre-excitation has been ruled out) or alternatively flecainide.

References:

1. Marriot's Practical Electrocardiography: Mechanisms of Arrhythmias with Increased Rates, p.203. 9<sup>th</sup> Ed 1994.
2. Delacretaz E, Supraventricular Tachycardia, NEJM, 354:10, 9 March 2006
3. Cardiovascular Therapeutic Guidelines, 3<sup>rd</sup> ed 2003.
4. Cabalag M.S et al. Recent Caffeine Ingestion Reduces Adenosine Efficacy in the Treatment of Paroxysmal Supraventricular Tachycardia. Academic Emergency Medicine, 2009; 16:1 - 6

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