An unusual cause for a road traffic accident

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This 42y.o. man was transferred to us after a prolonged stay on ITU following a severe RTA in which he sustained polytrauma with bifrontal contusions, left hemisphere hypertension and ischaemia with fractured left orbital sinus and fractures around the left temporal bone (image 1), fractured right leg, right arm and multiple ribs. When paramedics arrived on the scene he was found to be in cardiac arrest and was successfully resuscitated.

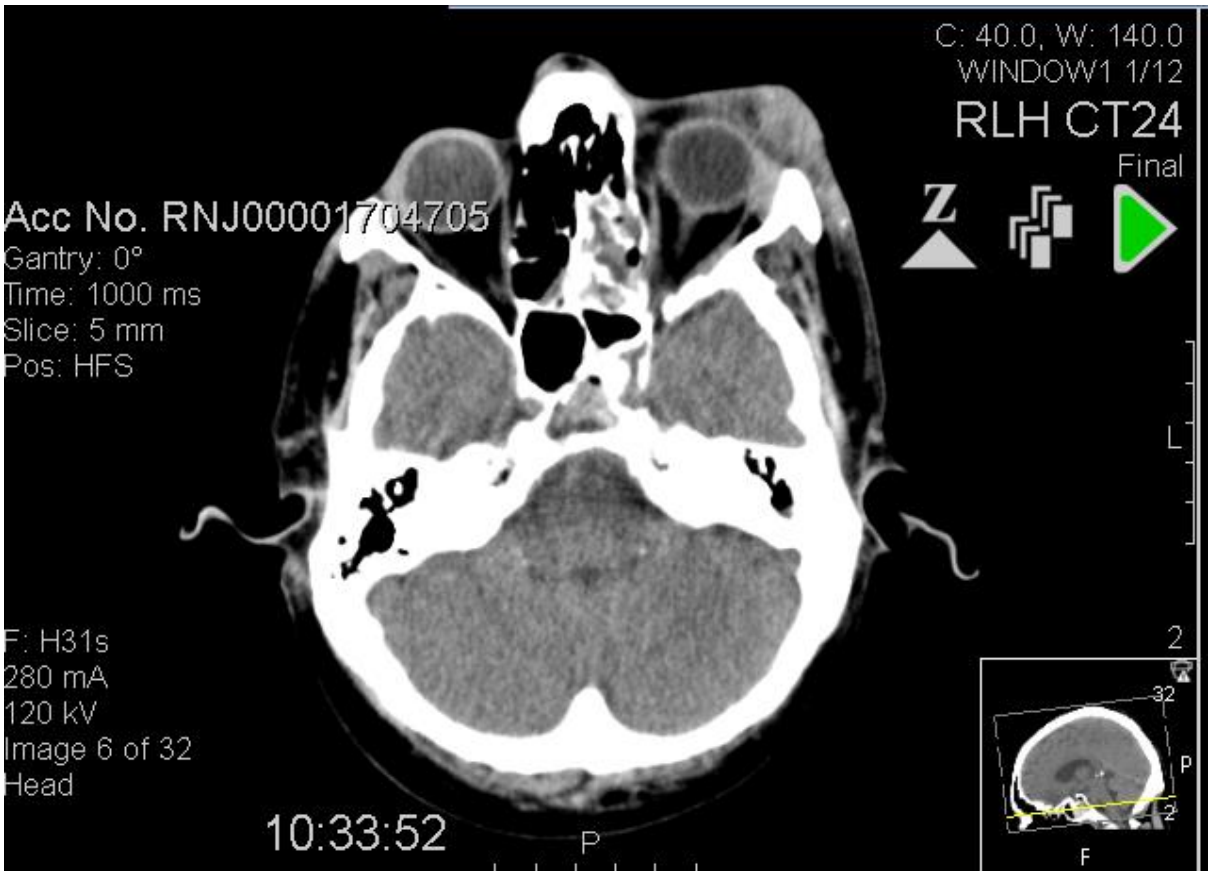


Image 1. CT head showing fractures

He recalled nothing of the accident due to short term memory loss. The witnessed accident occurred on a straight stretch of road during the day.

During his time in ITU an echocardiogram was performed which showed a severely dilated LV with global severely impaired systolic function, EF of 15%. He and his wife denied any heart failure symptoms prior to his accident. He had no history of MI and his one risk factor was smoking.

He was discussed with the EP team due to long paroxysms of tachycardia that were unable to be controlled with adenosine, amiodarone or beta blockers. He was transferred to our institution and taken to the lab.

Figure 1 - baseline 12 lead ECG with tachycardia termination.

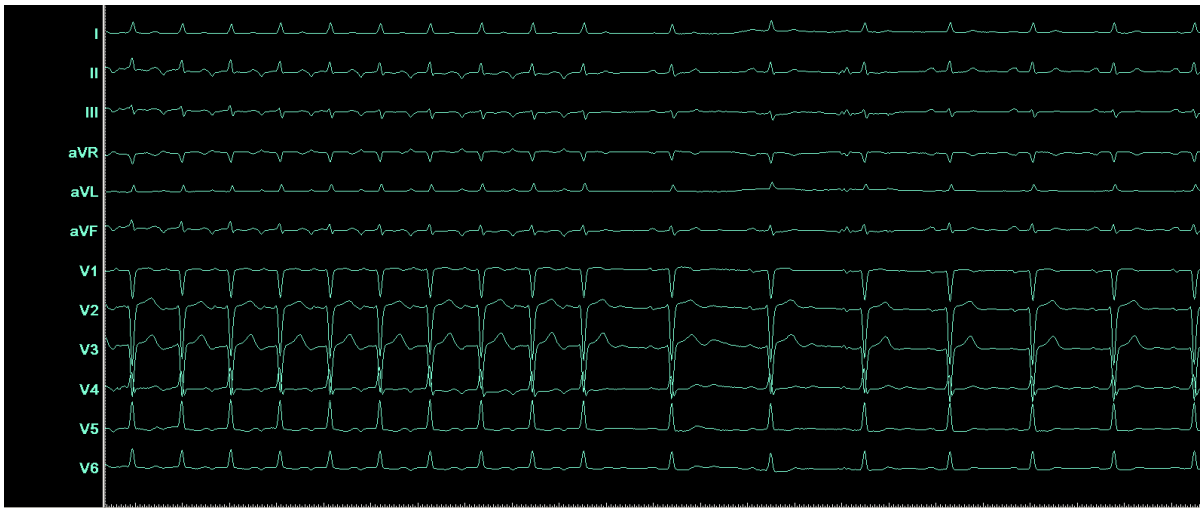


Figure 1- The ECG shows a long RP tachycardia with a negative P wave in the inferior leads and termination in the ventricle. Sinus rhythm on the latter half of the tracing shows a normal axis and flattened T waves in the inferior and lateral leads.

A hexapolar cather was advanced to the His position followed by a decapolar catheter only partway into to the CS so that CS 3-4/5-6 were at the ostium both via the RFV. Baseline showed normal AH & HV intervals. Spontaneous tachycardia resumed almost immediately.

Figure 2 - Tachycardia

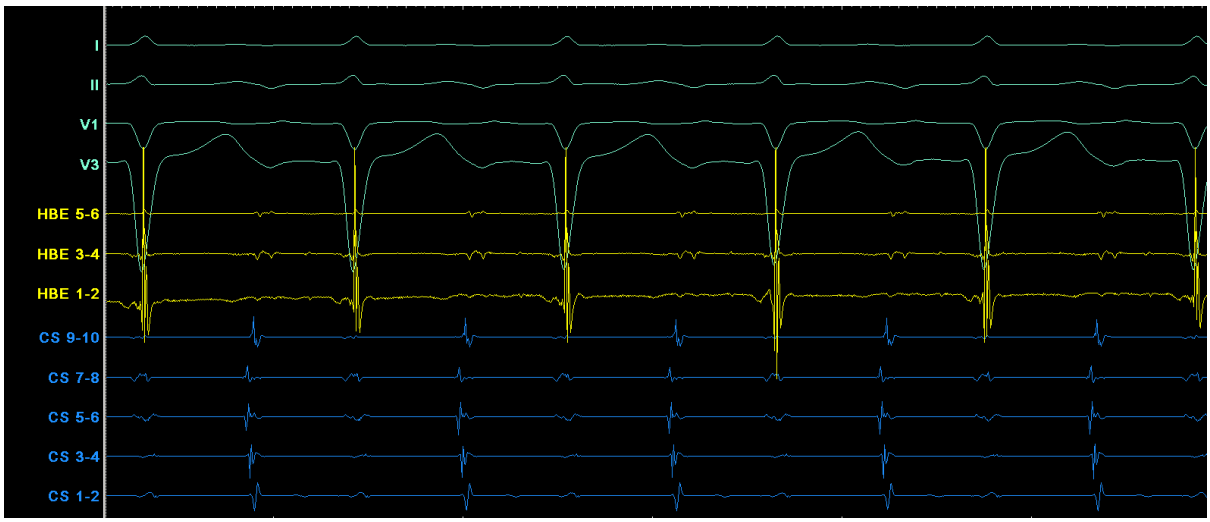


Figure 2. Tachycardia – The ECG leads show a narrow complex slow tachycardia with CL 550ms. Atrial activation on the His catheter is preceded by poles 5-6 on the decapolar catheter.

Ventricular pacing at 600ms was unable to be performed as tachycardia initiated during the drive each time. Ventricular pacing at 400ms is shown in figure 3.

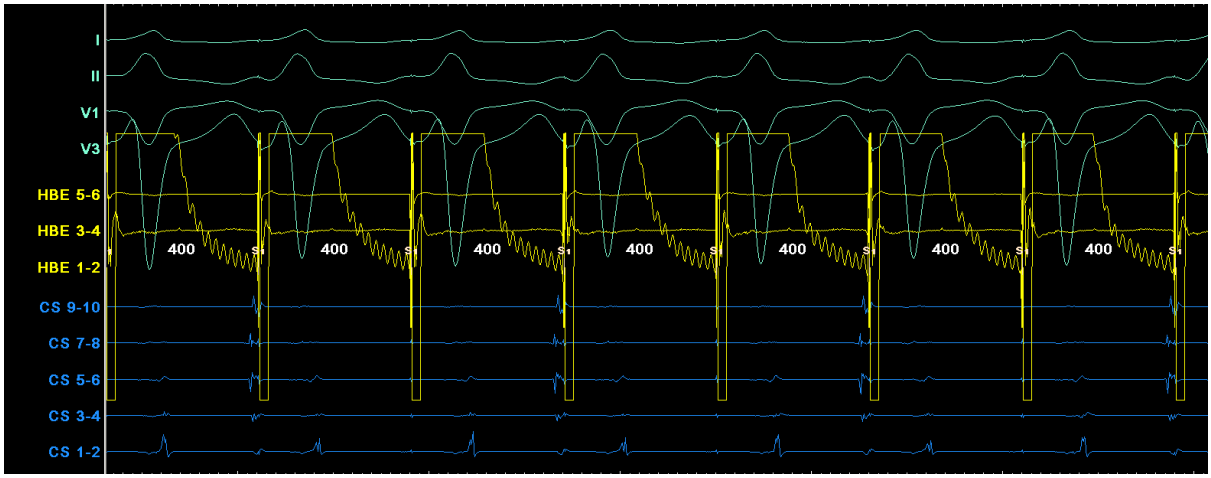


Figure 3 shows ventricular pacing at 400ms with 2:1 conduction. Interestingly earliest atrial activation still occurs at decapolar 5-6.

At this point an ablation catheter (F curve thermocool 35Q53R, Biosense Webster) was inserted and utilised as a roving catheter to look for earlier atrial activation, shown in figure 4a & 4b.

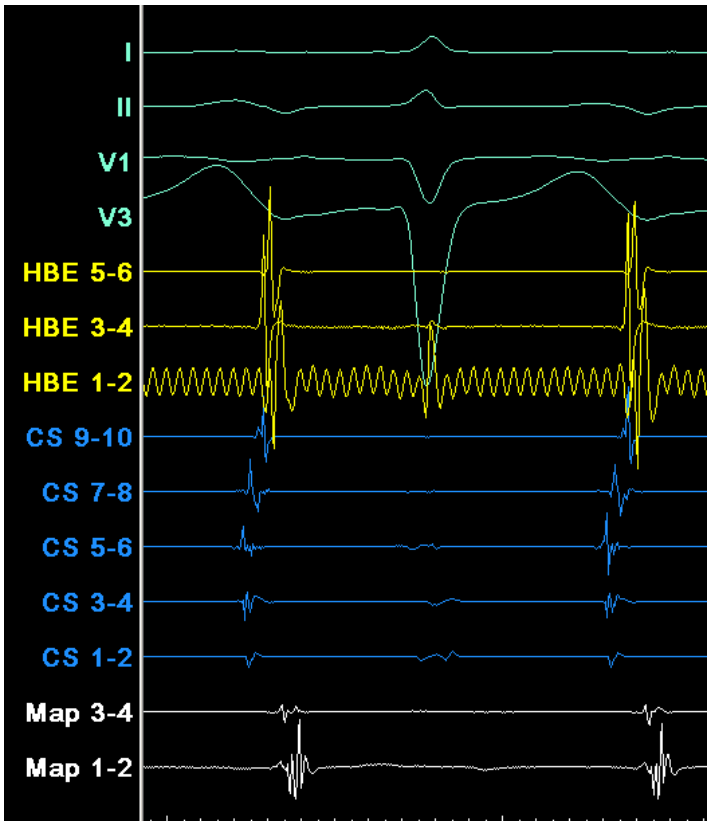


Figure 4a Map catheter at lateral RA

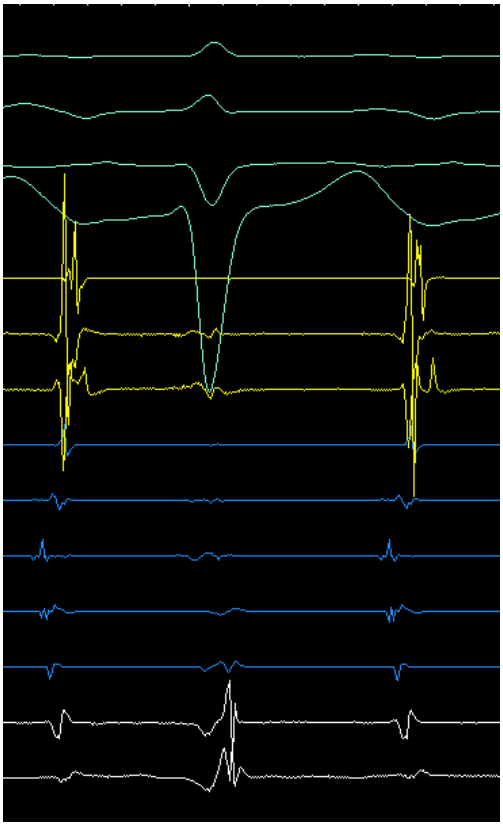


Figure 4b Map catheter in distal CS

After mapping the RA and tricuspid annulus earliest atrial activation remained at decapolar 5-6, the ostium of the CS.

Further diagnostic testing proved very difficult. Each time atrial pacing was attempted tachycardia initiated therefore AV nodal properties were unable to be assessed. All attempts at performing a retrograde curve resulted in tachycardia during the drive also. All attempts at ventricular entrainment resulted in termination.

One potentially useful 'diagnostic manoeuvre' occurred when RBBB was accidently induced during catheter manipulation during tachycardia – figure 5a and b.

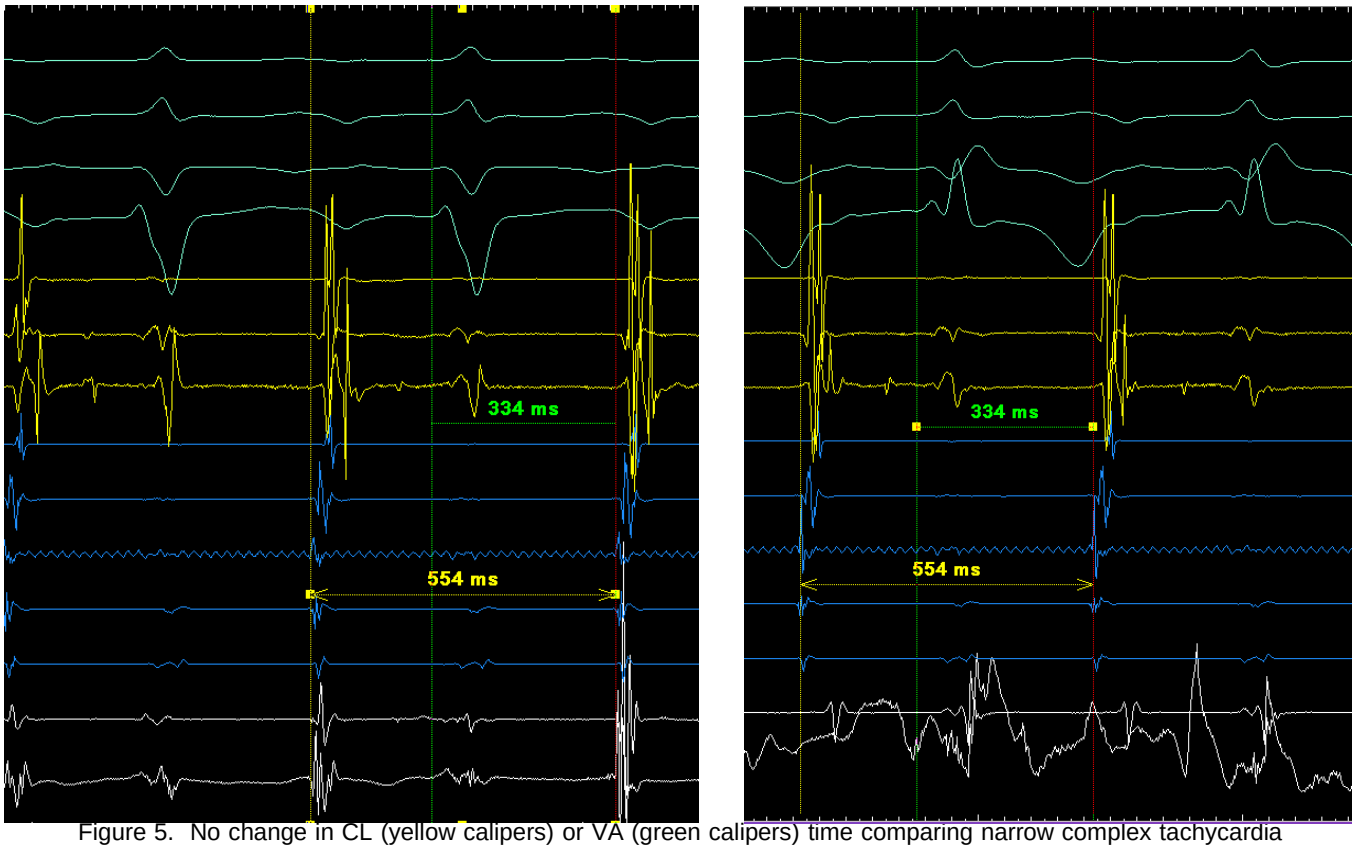


Figure 5. No change in CL (yellow calipers) or VA (green calipers) time comparing narrow complex tachycardia (a) with RBBB during tachycardia (b).

Without changes in CL or VA time between narrow complex tachycardia and that with RBBB we can exclude a right sided accessory pathway.

So in summary to this point we have:

- A 'slow' long RP tachycardia easily induced from the A and V
- Termination of tachycardia with a V
- 2:1 retrograde conduction with V pacing at 400ms
- Can't fully assess anterograde AV nodal properties
- Can't fully assess retrograde AV nodal properties
- Can't perform V entrainment to differentiate atrial tachycardia

This leads us to the following differential diagnosis:

- Atrial tachycardia originating from CS ostium
- Atypical AVNRT (fast/slow)
- Slowly conducting posterior left sided pathway

The next step was delivery of a HSVPB shown below in figure 6

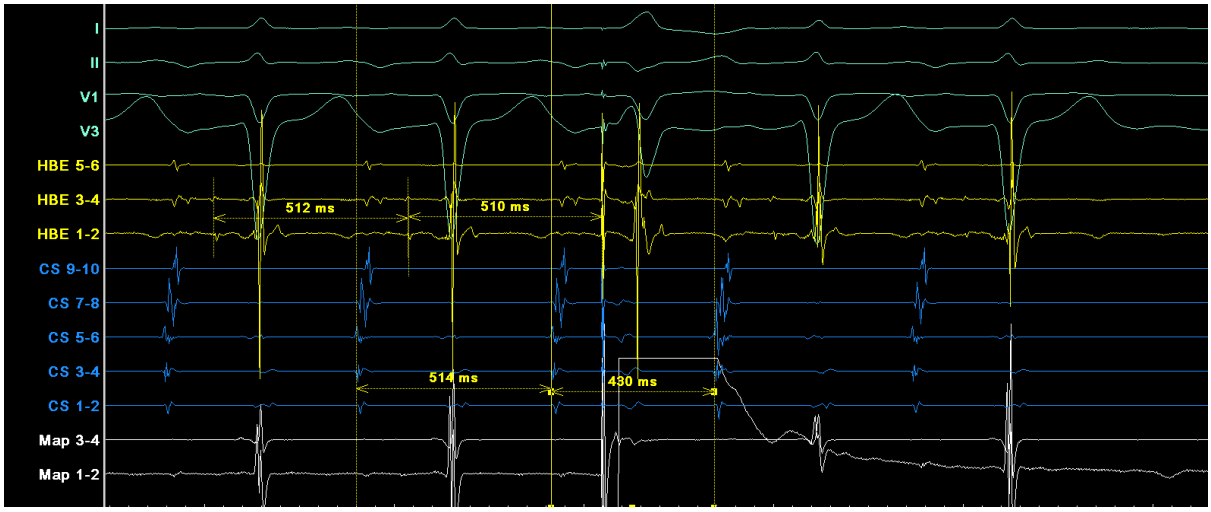
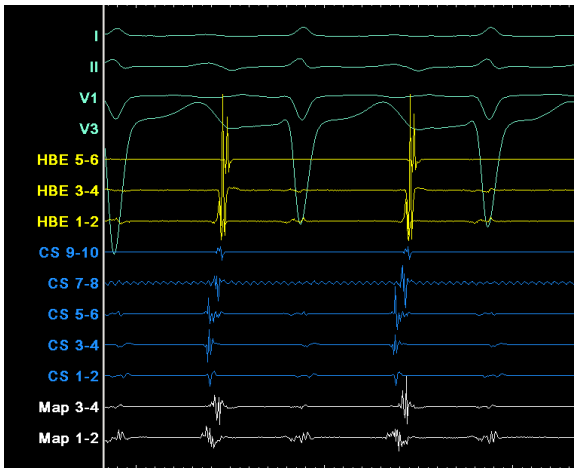


Figure 6 shows atrial advancement with HSVPB and thus proving the presence of an AV bypass tract.

HSVPB demonstrated atrial advancement and advancement of the next ventricular depolarisation. This gives us the diagnosis of a slowly conducting accessory bypass tract.

With the presence of a slowly conducting accessory bypass tract the ablation catheter was placed back into the coronary sinus to look for the earliest atrial activation. shown in figure 7.



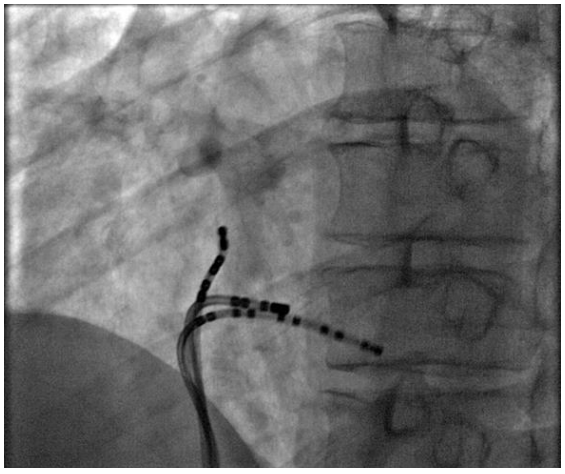


Figure 7 Pre ablation signal on Map 1-2. Note the earlier onset compared to AEGMs on the CS.

Image 2 – LAO projection of the ablation catheter in the CS alongside the decapolar catheter.

Irrigated ablation at 30W was performed at this site with termination of tachycardia after 3 seconds.

Non-selective angiography of the CS was taken after successful ablation. Image 3a shows an RAO projection of the ablation catheter at the successful ablation location. Image 3b shows the anatomy of the proximal CS also in RAO – note the successful ablation point is at the anterior junction of the CS main body and the middle cardiac vein (arrow).

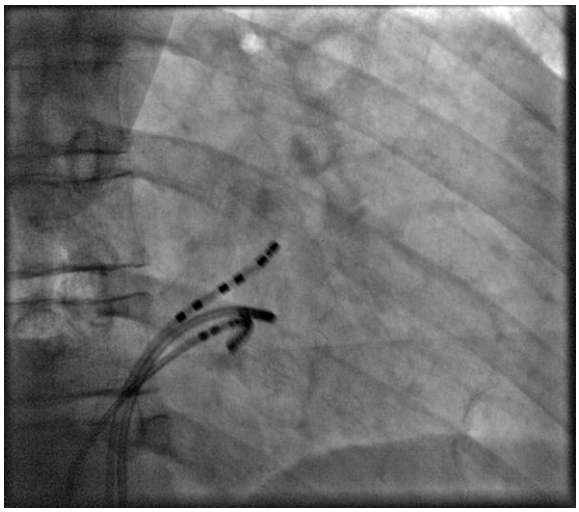


Image 3a – RAO projection of successful ablation site.

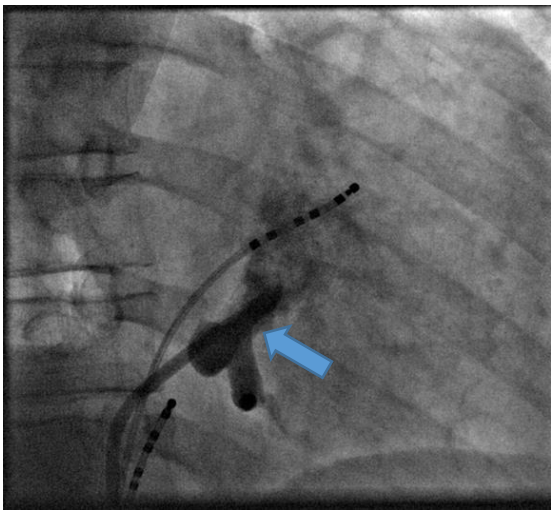


Image 3b – RAO projection with contrast

DISCUSSION

This demonstrates a case of permanent junctional re-entry tachycardia (PJRT). Characteristics of PJRT are as follows:

- most commonly found in the posteroseptal location

  In a study of 33 people Gaita etal found the site of the earliest retrograde atrial activation was posteroseptal in 25 patients (76%), midseptal in 4 (12%), right posterior in 1 (3%), right lateral in 1 (3%), left posterior in 1 (3%) and left lateral in 1 (3%)

- usually concealed and exhibit slow and decremental retrograde conduction
- HSVPD preexcites A, delays A, or terminates with no A
- If HSVPD delays A – diagnostic of decremental pathway

The patient had significant improvement in LV function at 3 months and normal LV function at 12 months. Unfortunately the head injuries he sustained have left him with short term memory loss.

Prolonged periods of tachycardia are a well-established cause for cardiomyopathy. There have been published reports of PJRT leading to cardiomyopathy from foetal stages to adults. This is the first case the author can find which shows tachycardia induced cardiomyopathy which led to cardiac arrest while driving causing a road traffic accident.

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