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Atrial Tachycardias Arising from the Atrial Appendages and Aortic Sinus of Valsalva

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Abstract: Focal atrial tachycardias arising from the atrial appendages and the aortic sinuses of Valsalva are less frequently encountered in clinical practice. This review article describes the clinical presentation, surface P wave morphology, electrophysiologic characteristics and treatment of these arrhythmias. Catheter ablation of these focal tachycardias has a high success rate. It is however important to be aware of specific anatomic considerations in these locations for optimal treatment outcomes with low complication rates.

Keywords: Ablation, aortic sinus of Valsalva, atrial appendage, atrial tachycardia, non-coronary cusp.

1. INTRODUCTION

Focal atrial tachycardias (ATs) are a distinct subset of supraventricular tachycardias (SVT) characterised by centrifugal spread of activation wavefronts from an atrial point-source. The sites of origin of AT are not randomly distributed throughout the atrial myocardium but instead cluster around well-described regions of anatomic and electrophysiologic heterogeneity. These include the crista terminalis [1], tricuspid annulus [2], coronary sinus ostium [3], pulmonary veins [4], mitral annulus [5], parahisian region [6] and inter atrial septum [7-9] .

The atrial appendages and the aortic sinus of Valsalva are less commonly encountered sites of origin of focal AT [10-19]. This review will examine these subgroups of AT in detail.

2. FOCAL ATRIAL TACHYCARDIAS ARISING FROM THE ATRIAL APPENDAGES

2.1. Clinical Features

As with other ATs, patients with AT of appendage origin may present with palpitations, dyspnoea, presyncope or, rarely, syncope. A male gender predominance has been described more characteristically for right atrial appendage (RAA) than left atrial appendage (LAA) tachycardias [11-15, 20].

A particular feature of appendage ATs is their frequent propensity to present with incessant tachycardia. Consequently, an association between these tachycardias and tachycardia-mediated cardiomyopathy (TCM) is well described. This diagnosis is often missed in patients presenting with heart failure where a tachycardia is assumed to represent a physiological reaction to the low-output state. Whilst sinus tachycardia could be considered a possible compensatory response to left ventricular systolic dysfunction, the P wave morphology will readily exclude a sinus origin in most cases. However, tachycardia foci arising from right-sided pulmonary veins and crista terminalis may generate a similar P wave morphology to sinus rhythm and result in an incorrect diagnosis. Medi *et al*. describe a large series of 345 patients who underwent radiofrequency ablation for focal atrial tachycardia, 14 of whom had left ventricular (LV) systolic impairment in the setting of prior structural heart disease [20]. Incessant tachycardia was seen in 25% (82 of 331) of the remaining cohort and TCM was seen in over a third of these (30 of 82). Foci arising from the atrial appendages and pulmonary veins were the most frequent site of incessant tachycardia. Importantly of the 19 patients who had an appendage foci, 16 patients presented with incessant tachycardia and 8 (50%) of these patients had TCM. A similar observation was made with atrial tachycardias from the pulmonary veins. This was significantly higher than the incidence seen in association with other anatomic locations. The ventricular response rate was slower and the tachycardia cycle length was longer in those with TCM in comparison to those without. Normalisation of left ventricular function occurred in 29 of the 30 patients (1 patient with an appendage focus was only partially controlled on medications) at 2.8 +/- 2 months after successful ablation and no sudden deaths were reported at a mean follow up of 2 years. In another study of tachycardias arising from the RAA by Freixa *et al.* 27% of patients had left ventricular systolic dysfunction (defined as left ventricular ejection fraction <50%) [14].

2.2. Anatomic Considerations and Mechanisms

The appendages are anatomically and embryonically distinct from the atria. Unlike the smooth walled atria (sinus venosus remnant) they contain numerous trabeculae, termed pectinate muscles. The base of the appendages is the usual focus of atrial tachycardia origin [13], however tachycardias

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arising from the mid and distal portion of the left atrial appendage [12, 15] and the tip of the right atrial appendage [11, 14] have also been reported. There has been a report of atrial tachycardia arising from a congenital left atrial appendage aneurysm [21].

It has been proposed that automaticity is the most common underlying mechanism in this group of tachycardias [22]. Iwa *et al.* proposed that the LAA ATs may originate from the surviving tissue of the left sinus node as the LAA AT foci appeared to be a mirror image of the right sinus node on the study of surgically resected specimens [23]. However, triggered activity and microreentry may also be operative in many cases.

2.3. P Wave Morphology

In the absence of structural heart disease or extensive prior ablation, and presupposing a normal geometric relationship between intrathoracic cardiac location and the chest wall, the 12 lead surface electrocardiograph generally provides a reliable guide to the myocardial breakout site of focal arrhythmias. In the case of focal AT, the P wave morphology (PWM) reflects the site of origin of the tachycardia. Whilst precise localisation of tachycardia foci ultimately depends on intracardiac mapping techniques, PWM provides a valuable tool in directing mapping to a particular area of interest. However, during tachycardia, the P wave is commonly superimposed on the adjacent T wave and any assessment of P wave morphology made during T-P fusion may overlook the initial electrical forces. It is therefore important to analyse only those P waves with a preceding isoelectric baseline [24]. This can be performed during periods of atrioventricular block induced pharmacologically or with rapid ventricular pacing.

Kistler *et al.* presented a prospectively evaluated algorithm to determine the site of origin of tachycardia based on their analysis of 186 patients who underwent ablation of focal AT [25]. They demonstrated that the tachycardias originating in the RAA had a very similar morphology to those arising from the superior portion of the tricuspid annulus. The PWM characteristics included a negative polarity in lead V1, negative or isoelectric in aVL and positive in the inferior leads (Fig. **1**). Other studies showed the P wave became progressively more positive in the precordial leads and was negative in aVR [11, 14]. This precordial transition serves as a useful distinguishing feature from the tricuspid annular tachycardias where the P wave remains persistently negative [14].

The PWM of tachycardias arising from the LAA was similar to that arising from a left sided pulmonary vein. The P wave polarity is positive in lead V1 and bifid in lead II and or lead V1 and positive in the inferior leads. In addition, the presence of a deeply negative P wave in lead I suggested a LAA origin [25]. Yamada *et al.* demonstrated that the criterion of negative P wave polarity in leads I and aVL in predicting a LAA focus was associated with a sensitivity of 92.3% and a specificity of 97.3% [13]. In addition, the amplitude of the P wave in the inferior leads was ≥0.1mV for those with foci in the medial LAA and <0.1mV for those originating in the lateral LAA. Negative P wave polarity in leads aVL and I in ATs with an LAA focus was also seen in another series from Wang *et al.* [12]. In this series the precordial leads V2-V6 showed either an isoelectric or a low amplitude upright component.

2.4. Electrophysiologic Characteristics and Endocardial Mapping

Appendage ATs often present spontaneously in the electrophysiology laboratory given their frequently incessant nature. If not, induction is attempted with programmed stimulation, or atrial burst pacing, sometimes with the use of isoproterenol. The mean tachycardia cycle length reported in the different series is variable ranging from 364 to 516ms for ATs from the RAA [11, 14] and 381 to 433ms for ATs from the LAA [12, 13, 15].

Precise localization of the site of origin is best accomplished with activation mapping during the tachycardia (or

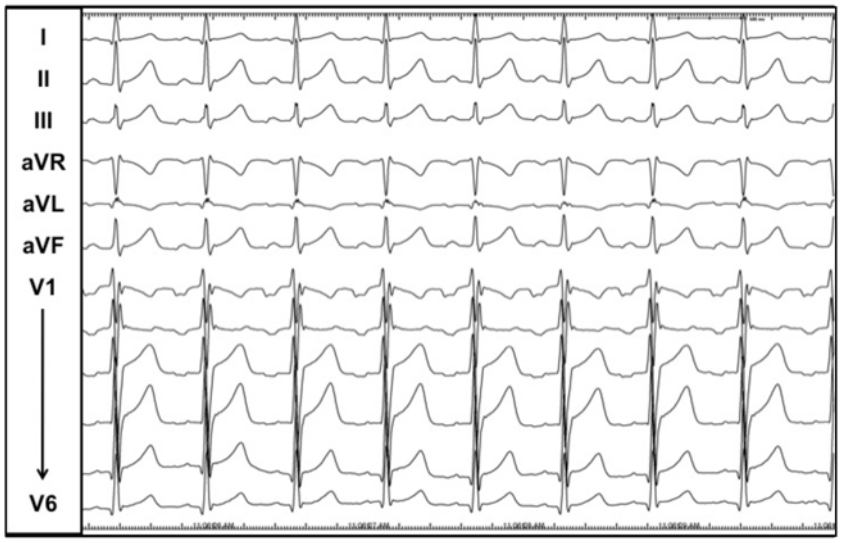

Fig. (1). Typical surface P wave morphology of a focal right atrial appendage (RAA) tachycardia with a deeply negative, notched P wave in lead V1.

during atrial ectopy) with point-by-point mapping to locate the site of earliest endocardial activation relative to the surface P wave onset [11]. Activation mapping can be performed with conventional catheters under fluoroscopic guidance. Three-dimensional electroanatomic mapping systems facilitate this process and significantly reduce radiation exposure to both patient and operator. This also allows for the delivery of lesions at sites of mechanical catheter-induced terminations or additional lesions at sites of successful ablation. Intracardiac echocardiography (ICE) can also be used to assist with mapping, confirming catheter contact and lesion formation, as well as to monitor for complications.

Of a standard intracardiac catheter set, the earliest endocardial activation in ATs from the RAA is seen in the duodecapolar mapping catheter placed around the tricuspid valve annulus. It corresponds to 10 to 11 o'clock on the tricuspid annulus followed by the proximal and distal activation of the duodecapolar catheter, proximal His catheter, and then the proximal coronary sinus (CS) catheter [11]. The endocardial activation at the site of successful ablation preceded the P wave onset by 38 ± 14ms in the study by Roberts-Thomson *et al.* and 28.4 ± 13ms in the study by Freixa *et al.* [11, 14].

In ATs arising from the LAA the earliest activation occurs at the distal coronary sinus (CS) catheter [12]. A Lasso catheter in the left superior pulmonary vein demonstrates a far field atrial potential that precedes the pulmonary vein potential as well the CS and surface P wave suggesting an origin in the LAA [15]. The mean activation time relative to the P wave onset at the site of successful ablation ranged from 42.3 to 51.6ms in different studies [12, 13, 15]. Non-sustained tachycardias or those difficult to induce may be mapped by use of the non-contact multielectrode array or by paced activation sequence mapping. The lack of a robust endpoint is a potential limitation in all non-mappable tachycardia situations, regardless of the specific technique employed.

2.5. Ablation Strategies

Despite the use of electroanatomic mapping systems, catheter manipulation and mapping in the appendages can be challenging due to their trabeculated nature. Angiography or computed tomography (CT) is useful in excluding abnormal anatomy or aneurysms of the appendage, and the latter can particularly be integrated into the 3D mapping system. Radiofrequency current is the commonest form of energy delivered for lesion formation. Acceleration is often seen at the site of successful ablation prior to termination of the tachycardia.

The RAA is dominated by pectinate muscles, which can often limit adequate power delivery for radiofrequency ablation (RFA) in the RAA. Catheter manoeuvrability may be challenging for the same reason. Roberts-Thomson *et al.* describe 10 patients who underwent RFA for RAA AT and half of these required an irrigated tip catheter for adequate power delivery [11]. The power was set at 50W (with a 60°C temperature cut-off) for a standard 4mm tip catheter, and 30W for a 3.5mm open-irrigated catheter. Similarly Friexa *et al.* report the use of an irrigated catheter in 5 out of 15 patients [14]. Both series had a 100% acute success rate with extremely low recurrence rates and no major complications.

The LAA is usually approached through a transeptal puncture. In their series of 7 pts who underwent RFA in the LAA; Wang *et al.* used non-irrigated catheters with lower temperature and power in the LAA apex (55°C, 20-30W) compared to the base and the mid segment (60°C, 30-50W). An irrigated tip catheter was used in one patient who had a recurrence [12]. In another series by Yang *et al.,* of 14 patients with AT from the distal LAA, RFA with an irrigated tip catheter was used (45°C, 20-30W) and was successful in 13 patients. One patient however required surgical excision of the LAA [15]. Yamada *et al.* also report a 100% acute success rate in their series of 13 patients [13]. Contrast angiography performed in a couple of the above series post ablation demonstrated preserved LAA mechanical function and excluded perforation. There have been reports of 3D mapping system guided cryoablation in the LAA [26]. The use of ICE for placement of the catheter in a safe position in the LAA to minimize the risk of perforation has also been described [27].

Potential complications include cardiac perforation, phrenic nerve injury, reduced left atrial appendage transport function and thromboembolic events, particularly with RFA in the LAA. Pacing at high output to exclude phrenic nerve capture pre-ablation is essential to minimize the risk of phrenic nerve injury during all appendage AT ablations.

Overall the above series would suggest an excellent result with catheter ablation of focal atrial appendage tachycardias with acute procedural success rates of 98 to 100% and extremely low recurrence rates at medium to long term follow up which ranged from 2-8 years. No significant complications were reported. Normalisation of left ventricular function was seen post procedure in those with TCM.

Strategies described for patients who fail percutaneous endocardial ablation include; epicardial ablation [28, 29] (Fig. **2**), cryoballoon isolation of the RAA [30], epicardial ablation followed by minimally invasive appendectomy [31] and epicardial exclusion of the appendage with a minimally invasive occlusion device [32] as isolated reports in literature. Open surgical appendage ligation remains a last resort option for the cases refractory to all other techniques [15].

2.6. Conclusion

Focal atrial tachycardias arising from the appendages are uncommon and constitute <5 % of all ATs. They should be suspected in a patient with the appropriate P wave morphology particularly in the setting of incessant tachycardia. Frequently, TCM is present in these patients and can be reversed with successful treatment. They respond well to radiofrequency ablation with low recurrence rates however cardiac perforation, phrenic nerve injury, thromboembolic events and decreased left atrial transport function are potential complications.

3. FOCAL ATRIAL TACHYCARDIAS ARISING IN THE REGION OF THE AORTIC SINUS OF VALSALVA

Septal ATs with earliest atrial activations in the right and left perinodal regions are not an uncommon subgroup of AT. Whilst these may be mapped and ablated from either the

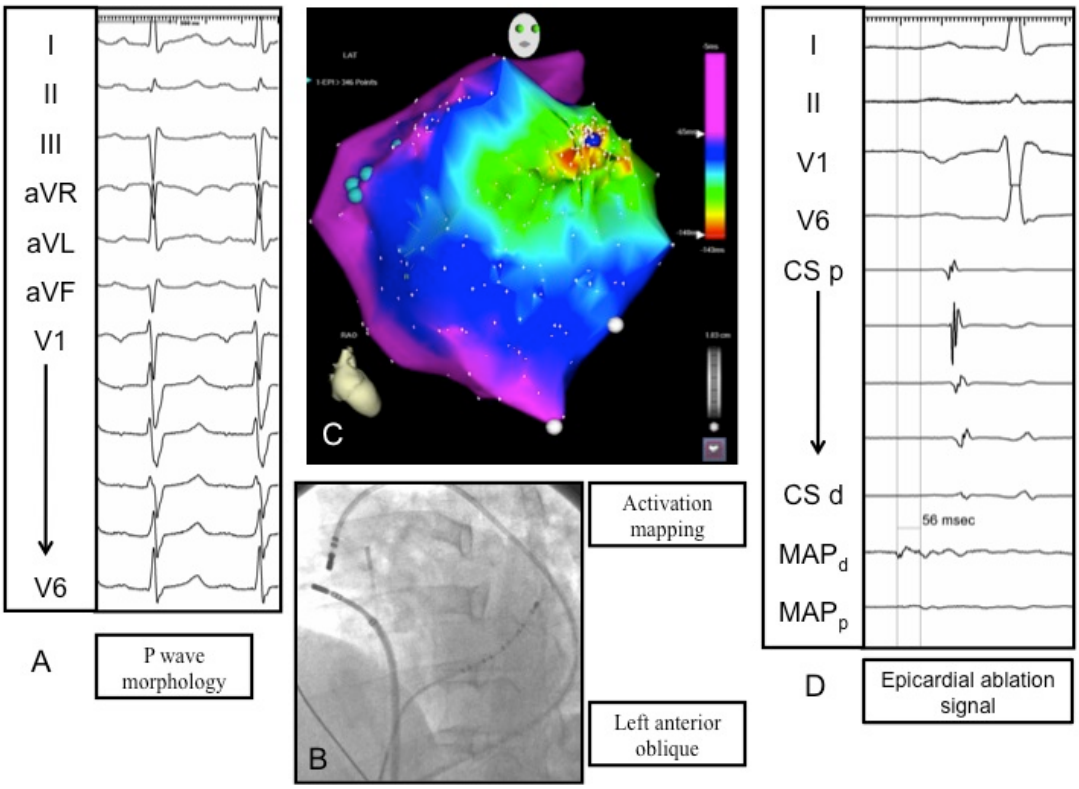

Fig. (2). A 26 year old man presented with near incessant focal tachycardia from the RAA and a tachycardiomyopathy (LV ejection fraction of 40%). Panel A shows the surface P wave morphology in tachycardia. Endocardial ablations performed at sites up to 40ms pre P wave with an irrigated tip radiofrequency ablation catheter and cryocatheter were unsuccessful. Percutaenous pericardial access was obtained and an epicardial site 56ms pre P wave was found opposite to the site of the earliest endocardial activation (Panel B). Radiofrequency ablation here resulted in termination of the tachycardia (Panel C and D). Left ventricular systolic function normalized within one week.

right or left atrial side of the septum, an increasing number have been targeted for ablation from the aortic sinus of Valsalva (ASOV) [16-19]. Most commonly, the non-coronary cusp (NCC) of the ASOV has been identified as the successful site of ablation to eliminate the arrhythmia and rarely, the right coronary cusp (RCC) and even the left coronary cusp (LCC) [16-19, 33].

Patients with atrial tachycardia arising in the region of the ASOV include palpitations, dizziness and, rarely, syncope. The duration of symptoms prior to presentation can range from 3 to 5 years [17, 18, 33]. Structural heart disease is rarely seen in this patient group. Reported series in literature would suggest a trend toward a higher incidence in females with an average age of presentation between 53-62 years of age [6, 18, 33]. In contrast to patients presenting with AT from the atrial appendages, tachycardia-mediated cardiomyopathy is rare in patients with tachycardia from the ASOV with only one reported case in the literature thus far [33].

3.1. Septal Atrial Tachycardias

Septal ATs are difficult to categorise precisely due to the close proximity of a variety of anatomic structures in the perinodal, interatrial septal region [34]. They account for ~12% of all atrial tachycardias [9, 25]. The diagnosis of these tachycardias has remained challenging because there are no specific electrocardiographic or electrophysiologic characteristics that define their true anatomic origin [34]. There is a degree of overlap in relation to septal P wave morphologies and whilst V1 has been shown to be the most useful lead in differentiating left from right-sided foci, this does not reliably distinguish foci in close proximity to the septum (left vs right septal or perinodal) [10, 25]. Electrophysiology study will confirm earliest atrial activation in the His catheter which is suggestive of a parahisian or anteroseptal foci but the target for ablation may remain unclear until endocardial mapping of the right and left side of the interatrial septum and the ASOV is performed [17, 25, 35]. The risk of inadvertent AV block while ablating near the compact AV node and His-bundle is concerning, particularly in the left perinodal region.

3.2. Anatomy

Spatially, the aortic root occupies a central location within the heart and the NCC of the ASOV is anterior and superior to the paraseptal region of the left and right atria close to the superior atrioventricular junctions. The non-coronary ASOV has exclusively fibrous walls whereas the left and right ASOV incorporate a part of ventricular muscle at their base. In a structurally normal heart, the non-coronary sinus is adjacent to atrial myocardium on the epicardial aspect. The rightward or anterior margin of the NCC is related to the paraseptal region of the right atrial free wall whereas the leftward margin is related to the left atrial wall [18, 19].

Liu *et al.* investigated the spatial relationship between the NCC and the contiguous atria using CT and histologic specimens. CT was performed in 25 patients with structurally normal hearts and slices perpendicular to the aortic root were reconstructed from the bottom of the NCC to the midpoint of the NCC with a thickness and distance of 1mm each. In each slice the minimum distance between the endocardium of the NCC and the endocardium of the RA and LA was measured using digital calipers. In all patients, the NCC was adjacent to the paraseptal regions of the atria and furthermore, in 88% (22/25) of patients, the NCC was closest to the RA than the LA. The minimum distance between the NCC and atria was always documented in the lower part of the NCC and measured 1.6 ± 0.6mm (RA) versus 2.1 ± 0.9mm (LA) [16].

In the anatomic specimens, the NCC could be seen adjacent to the atrial myocardium. Interestingly, on histological examination, the wall of the NCC did not contain any atrial myocardium. Instead the deepest sinus wall adjoined the atrial walls in some hearts or was separated by a thin layer of fibrofatty tissue in others. The upper parts of the NCC were further away from the atria concurring with observations made on CT imaging [16].

Thus it is most likely that the perinodal ATs successfully ablated from the non-coronary ASOV are most likely originating epicardially from paraseptal atrial myocardial tissue lying adjacent to the NCC. Additionally, this myocardial tissue lies in closer proximity to the lower part of the NCC than to the atrial endocardium causing access from the atria to be difficult endocardially and access via the NCC viable [16].

Importantly, the central fibrous body of the heart, containing the bundle of His, is located in the interleaflet triangle between the NCC and the RCC. Therefore these two aortic sinuses are in close proximity to the bundle of His when it penetrates the central fibrous body and passes to the ventricular septum [18, 19].

Embryologic studies in mice have traced the specialised conduction system around the aortic root and AV canal in early stages and found marked regression in later stages. It has been postulated that the persistence of conduction system tissue in this region may be responsible for this form of atrial tachycardia [6, 36, 37].

3.3. P Wave Morphology

Atrial tachycardias arising from the interatrial septum are associated with variable P wave morphology. This is due to the fact that they originate from a variety of anatomic structures in close proximity to the septum. However, the most common distinguishing feature suggestive of a septal origin is a narrow, biphasic lead V1 [34] (Fig. **3**).

There have been some small series that have described the P wave morphology of perinodal atrial tachycardias ablated from the NCC of the aortic valve. The P wave duration is narrow compared to the sinus rhythm P wave in most cases and an average shortening of 30ms can be seen during tachycardia [6]. Lead V1 was found to be negative/positive

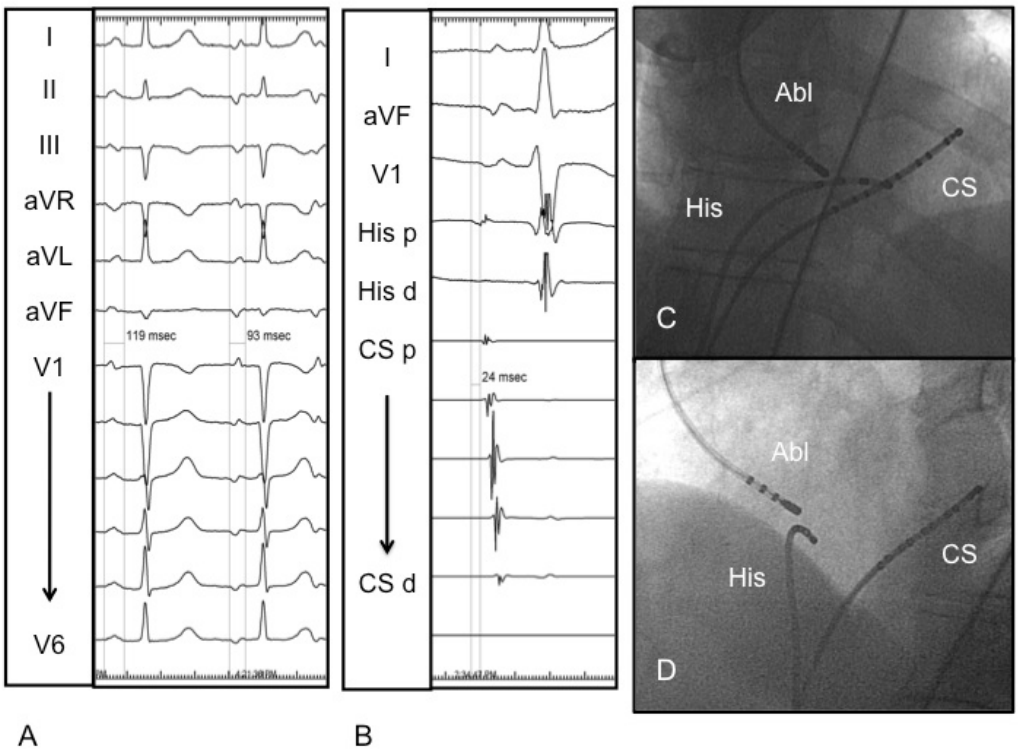

Fig. (3). A 76 year old woman presented with frequent palpitations and focal atrial tachycardia arising from the NCC. Panel A shows the sinus rhythm P wave morphology adjacent to the tachycardia P wave with a typical biphasic negative/positive V1. Significant narrowing of the P wave duration can be seen during tachycardia. The His A EGM is 24ms pre P wave that is consistent with a perinodal origin (Panel B). Fluoroscopic right anterior oblique (RAO) and left anterior oblique (LAO) views shows the relative proximity of the ablation catheter within the NCC to the His catheter (Panel C and D).

in all but 1 patient in a combined total of 25 patients. Secondly, positive P waves in lead aVL and I were seen in 22 out of 25 patients [18, 33]. The remaining leads carried marked variability in all the series described. Overall, it has been recognised that there is not a consistent global P wave morphology associated with these perinodal ATs and the surface morphology can be altered by the variation in anatomy between individuals.

3.4. Electrophysiologic Characteristics and Endocardial Mapping

Sustained perinodal AT originating near the NCC may occur spontaneously or be induced by pacing manoeuvres. Programmed stimulation and burst pacing of the atrium are reliably used to induce tachycardia and pharmacological agents are not usually required. The mean tachycardia cycle lengths varied between 341ms and 391ms in different series. The most defining feature of these atrial tachycardias is the earliest atrial activation which is always seen in the His catheter [16-18, 33, 38].

Most series describe utilization of 3D mapping systems to create endocardial activation maps and localise the origin of tachycardias arising in close proximity to the AV node and the His bundle. Often an RA and LA activation map is performed prior to proceeding to the NCC (Fig. **4**) Liu *et al.* described earliest RA activation in the parahisian region 23ms pre P wave and earliest LA activation in the anteroseptal region 20ms pre P wave [16]. A separate series by Ouyang *et al.* also showed that the earliest LA activation was consistently later than the RA by 2-5ms [18]. This is most likely explained by the anatomical proximity of the NCC to the His bundle in the RA compared to the anteroseptal region of the LA. Activation times up to 40ms pre P wave could be observed in the NCC in the above series by Liu *et al.* [16]. Other studies have reported activation times in the NCC preceding the P wave by 21-35ms, [17-19, 33].

Wang *et al.* reported a series of 22 consecutive patients with ASOV atrial tachycardias where the NCC was the site of origin in 16 patients and the LCC in 6 patients. They were able to demonstrate a statistically significant difference between the NCC and the LCC activation times in reference to P wave onset. The earliest activation time pre P wave was 21.3ms ± 8.8ms in the NCC group and 47.5ms ± 14.9ms in the LCC group ($p<0.01$) [33].

No studies have been able to postulate a specific electrophysiologic marker in addition to P wave morphology to immediately suggest proceeding to the NCC to perform RFA. In a small series by Rillig *et al.* of 6 patients with successful catheter ablation of atrial tachycardia in the NCC, the His A EGM during tachycardia preceded P wave by at least 24ms in all patients. Additionally, results strongly suggest that if activation is early in the RA parahisian region, this highly favours mapping in the NCC before the LA [19]. Consideration of the NCC as a potential target site should also be considered in patients with previous failed ablation attempts in the parahisian region.

3.5. Ablation Strategies

The NCC of the ASOV is targeted via a retrograde aortic approach. Earlier reports of successful catheter ablation of focal ATs from the NCC were based on conventional electrophysiologic mapping with confirmation of catheter position and coronary artery location by aortic and coronary angiography [39]. The NCC is located at the most inferior and posterior aspect of the aorta in the right anterior oblique

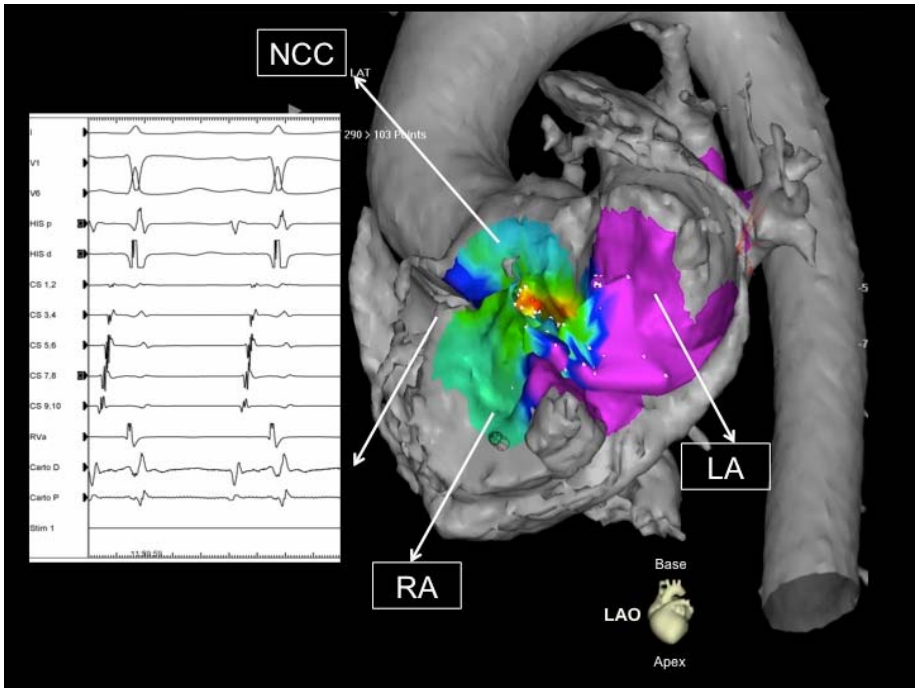

Fig. (4). Activation mapping of a non-coronary cusp (NCC) focal atrial tachycardia using electroanatomic mapping with image integration. Right paraseptal sites were slightly earlier than any left atrial breakouts but the activation at the nadir of the NCC was discretely earliest. Focal ablation here resulted in early tachycardia termination.

view, which is close to the interatrial septum and His bundle region. The LCC is easily recognised as the most leftward aspect of the aortic root in the left anterior oblique view. The A:V (atrial to ventricular) ratio from the local electrogram will be >1 in the NCC due to the relative thickness of atrial myocardium behind the aortic wall. However, when foci are located near the junction of the NCC and RCC or from the LCC, an A:V ratio of <1 will be observed due to the relatively thicker ventricular myocardium beneath the RCC and LCC [33] (Fig. **5**).

The use of 3D electroanatomic mapping has been described in some cases and allows spatial reconstruction of chamber geometry including the right atrium, His bundle and ASOV. It provides a unique tool for tagging the earliest activation site and creating a high resolution map in the area of interest [24]. In addition, image integration with CT or MRI allows precise analysis of the spatial relationship between the ablation target and surrounding structures [39] (Fig. **4**).

Intracardiac echocardiography (ICE) provides unparalleled real-time imaging with the ability to accurately visualise catheter position within the ASOV. Short-axis imaging of the aortic root from the base of the infundibulum allows identification of each aortic sinus and identification of the catheter within a particular sinus. Long-axis imaging enables assessment of the catheter tip in relation to the coronary artery ostia and/or the His bundle region [39]. Of additional benefit is the reduction in fluoroscopy time and radiation exposure to both patient and operator [24, 39].

A variety of energy modalities have been used to create ablation lesions in the ASOV including radiofrequency with irrigated and non-irrigated tip catheters as well as cryoenergy, and all have reported successful outcomes. Blood flow in the aortic cusps alternates from very high flow to minimal flow during the cardiac cycle. For this reason, lesion characteristics resulting from RF application in this region cannot be directly extrapolated from previous endocardial ablation data. Of particular concern is the formation of coagulum on the catheter tip causing thromboembolism, hence the requirement for systemic anticoagulation before mapping and ablation in the ASOV commences. Coagulum formation is directly linked to catheter tip temperature and therefore irrigated tip catheters are thought to be advantageous in some series because they allow constant catheter tip cooling [17]. However, irrigated tip catheters also create larger lesions that may increase the incidence of AV block. Cryoablation is thought to have the least thrombogenic potential however energy delivery in the area of high flow may be challenging [17]. Overall, RF is most widely used because of its general availability and efficacy for most ablation procedures [19].

Energy delivery is applied during tachycardia with close attention to AV conduction. Early termination of tachycardia is commonly seen sometimes preceded by tachycardia acceleration (Fig. **5**). No near field His recording on the ablation catheter was noted in any reported study. Additionally, the absence of any reported PR prolongation or junctional rhythm during energy delivery may suggest that the compact AV node or fast pathway is a significant distance away from the nadir of the NCC in most cases [18]. A single successful lesion causing tachycardia termination and subsequent non-inducibility has been described in a large percentage of patients.

The acute success rates of ablation within the NCC are between 70%-100% with good long-term outcomes [17-19, 33]. In the series by Das *et al.*, where ablation within the NCC failed to eliminate the arrhythmia in 3/10 patients, there was a significant difference in activation timing in relation to P wave onset. In the 7/10 successful cases activation was 34.7 ± 14.4ms pre P wave compared to 6.7 ± 0.6ms in the 3 unsuccessful cases. Subsequent ablation on the left atrial septum successfully eliminated these arrhythmias [17]. These left septal tachycardias have recently been described

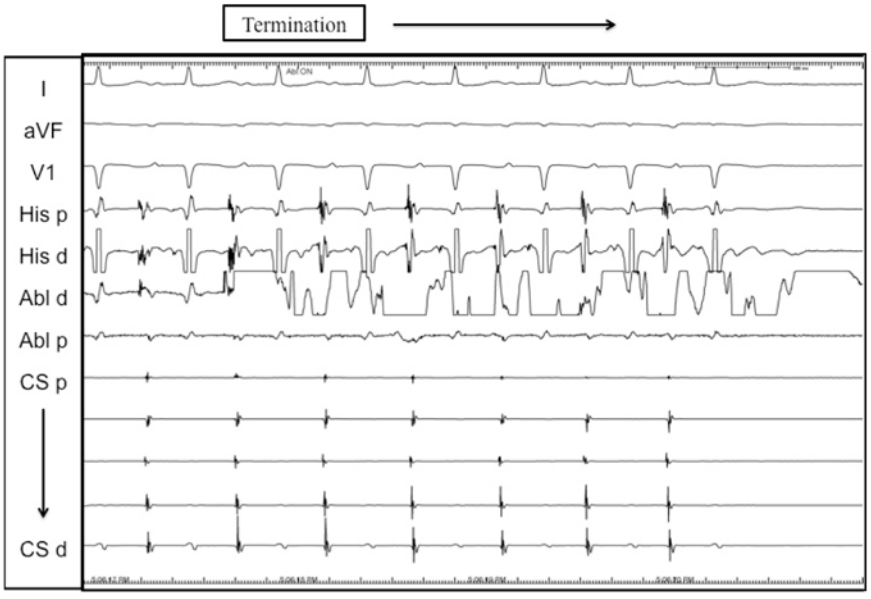

Fig. (5). Typical electrogram characteristics at earliest non-coronary cusp sites (NCC) are shown with a large A:V ratio in the mapping signal. Termination of tachycardia is seen within 2.3 seconds of radiofrequency application in the NCC.

by Wong *et al*. as having later His catheter activation times than the average NCC reports and likely represent a distinct subgroup of perinodal ATs [9].

3.6. Complications

There is a range of potential complications related to ablation within the aortic sinuses of Valsalva including aortic valve damage leading to regurgitation, thromboembolism, aortic mural damage or dissection, coronary artery stenosis or dissection and AV block because of the proximity to the normal AV conduction system [17-19].

Rillig *et al*. did the first study to look at the safety and efficacy of aortic ablation with regard to the potential risks of aortic wall and valve damage and cerebral embolism. In the reported cases of tissue damage in the literature, high target temperatures >70°C were noted [40]. A target temperature of 55°C with a target energy of 30W was used and assessment via TEE and MRI found no atrial or ventricular tissue damage at this temperature [19]. Due to the close anatomical location of the coronary artery ostia and risk of coronary artery occlusion [41] which have been previously described, coronary angiography or ICE is employed prior to ablation to confirm the location of both coronary arteries and identify the contour of the ASOV [19]. Overall, radiofrequency ablation of these atrial tachyarrhythmias can be safely and effectively performed within the NCC with minimal risk of complications [16-19, 33].

3.7. Conclusion

The non-coronary cusp of the ASOV is a potential site from which perinodal atrial tachycardias can be successfully mapped and ablated. Atrial septal myocardium lies immediately subjacent to the NCC and is the likely arrhythmogenic target. Ablation here is safe and effective and has been shown to produce good short and long-term clinical outcomes. Mapping in the NCC of the ASOV should be considered early in perinodal AT cases with suggestive P wave morphologies or where previous right and left septal ablations have been unsuccessful.

DISCLOSURES

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CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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