HISTORY OF ELECTROCARDIOLOGY

The Initial (Earliest) Report of Polymorphous Ventricular Tachycardia

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In these short historical notes, we describe the early history of polymorphic ventricular tachycardia. Polymorphous ventricular tachycardia was probably first noted in 1918 by Wilson and Robinson. In a publication describing complete heart block and ventriculophasic arrhythmia, they noted a tachyarrhythmia characterized by multiple extrasystoles of different types at a rapid rate. Also, we briefly discuss the earliest recognized torsades de pointes by Dessertenes in 1966 and the first description of catecholaminergic polymorphic ventricular tachycardia, by Reid in 1977.

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polymorphic ventricular tachycardia; early history; torsades de pointes; catecholaminergic polymorphic ventricular tachycardia

Polymorphous ventricular tachycardia (PVT) is characterized by continuous change in the QRS morphology at a rate between 150 and 300 bpm. PVT is mostly seen in patients with congenital or acquired long QT syndrome and less frequently in ischemic heart disease, other structural cardiac abnormalities or in patients without organic heart disease.¹ Dessertenne in 1966 reported a specific form of PVT called "torsades de pointes" in a patient with atrioventricular block.² This short historical note discusses what we believe was the first reported PVT.

In 1918, Wilson and Robinson³ published an article in *Archives of Internal Medicine* titled "Heart-Block" with a subtitle "Two cases of complete Heart Block showing unusual features." The main purpose of this article was the description of ventriculophasic sinus arrhythmia in complete AV block. According to Wilson and Robinson³ this arrhythmia was rare with only one previously reported case in 1913 by Hecht.⁴ The first patient was a 39-year-old housewife known to have slow pulse since the age of 19. The second patient was a cigar-maker who was hospitalized because of multiple

syncope episodes, vertigo, exertional shortness of breath and exercise induced PVT. His resting and postexercise ECGs are shown in Figures 1 and 2.

In Figure 1, lead 3 shows complete AV block with an atrial and ventricular rate of 67 and 33 bpm, respectively. The QRS complexes are slightly wider and there is left axis deviation. The P–P interval with and without QRS complex varied between 870 ms and 970 ms, respectively, consistent with ventriculophasic sinus arrhythmia. In Figure 2, there are two rhythm strips of lead II recorded shortly after exercise showing two paroxysms of PVT. The upper strip starts with an escape beat followed by a ventricular premature beat, a pause, another escape beat and a paroxysm of six beats PVT. Thus, the tachyarrhythmia is initiated by long–short sequence. The lower strip shows an eight-beats PVT initiated by a late ventricular premature beat and more prominent changes in the QRS complex morphology. Both paroxysms of PVT were terminated spontaneously. The authors called this tachyarrhythmia "multiple extrasystoles after exercise" with the following comment, "the extrasystoles were not always of the same type and

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Figure 1. Lead III shows complete AV block and slightly wider QRS complexes.

they occurred at a rapid rate.³ Soon after Wilson and Robinson's article,³ additional cases of ventricular tachycardia induced syncope were reported in patients taking quinidine, having AV block or both.^{5,6}

As mentioned earlier, the main purpose of Wilson and Robinson's article was not PVT, but ventriculophasic sinus arrhythmia and its mechanism. Normalization of the PP intervals by atropine and exercise supported the role of the parasympathetic tone in the pathogenesis ventriculophasic sinus arrhythmia. The authors also used atropine to localize the site of AV block. In the first patient, the ventricular rate increased from 43 to 60 bpm while in the second one there was no change in the ventricular rate after atropine. Wilson and Robinson³ correctly assumed that improvement of AV conduction after parasympathetic blockade was consistent with AV nodal block, while no effect suggested conduction defect below the AV node.

TORSADES DE POINTES

It was not until 1966 that Dessertenne of France described the undulating nature of a type of PVT and coined the term "torsades de pointes."² This too

Figure 2. Two runs of PVT in lead II. The upper one is initiated by the long-short sequences and the lower strip which is the longer episode, is initiated by a late ventricular premature beat.

was in a patient with AV block. The patient's syncope episodes were due to self-terminating irregular ventricular arrhythmias resulting from intermittent 3rd degree AV block and bradycardia and not directly because of the AV block as was originally thought (Fig. 3).

Subsequent articles quickly reported that, prior the onset of torsades, QT lengthening was almost universally seen on ECG.⁷ Soon after this, the characteristic ECG pattern of long-short cycle was recognized and it was postulated that early after depolarizations play a role in arrhythmia genesis.^{8,9}

CATECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA

It is now clear that not all polymorphic ventricular tachycardias require a long-short cycle or even a long QT interval for initiation. Catecholaminergic induced polymorphic VT is a rare arrhythmia seen primarily in children and adolescents characterized by a normal QT interval and the lack of a preceding pause. It is induced by stress or exercise and classic ECG findings include ventricular ectopy of RBBB morphology, bidirectional VT and polymorphic VT.¹⁰ It is recognized as having a familial

Figure 3. The first irregular ventricular rhythm termed "torsades de pointes" by F. Dessertenne.

inheritance, in both autosomal dominant and recessive fashion. Mutations of either the ryanodine receptor (RyR2) or the calsequestrin gene (CASQ2) can result in abnormal calcium sequestration and concentrations within the sarcoplasmic reticulum and an increased vulnerability to ventricular arrhythmias.¹⁰

The first description of ventricular tachycardia induced by catecholamines came in 1977 by Reid et al.¹¹ The patient, a 6-year-old girl, had symptoms precipitated by stress or effort. Reid was able to induce bidirectional ventricular tachycardia through rapid atrial pacing or isoproterenol infusion. He was careful to mention that the patient had no structural heart disease and had never received digitalis. The induced arrhythmia was proven to be ventricular in origin through the use of His electrocardiography.

CONCLUSIONS

The management and prevention of ventricular tachycardias have become a focus for much of cardiology research today. As we look to the future for advances in medications and device therapy, it is always interesting to reflect back on the origins of an arrhythmia or disease process.

This short historical note not only describes the first PVT but also shows the insightful thinking of Wilson and his coworkers almost 90 years ago. As we discussed in our two previous articles,^{12,13} Wilson was one of the first who reported supraventricular tachycardia in preexcitation¹⁴ and together with his coworkers first correctly classified right and left bundle branch block.¹⁵

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