In this installment of teaching rounds, Dr Shenthar illustrates what makes a clinical teaching round interaction so memorable for the learner. First, as with bedside demonstration of a physical sign, we have a unique ECG that compels the electrophysiology student to explain a clearly documented event that we may not have otherwise imagined possible—a wide complex, bidirectional, otherwise stable, ventricular tachycardia (VT). Then, the author briefly historically reviews important definitions with regard to VT morphology and subsequently provides an insightful analysis of possible mechanisms and the relationship of this arrhythmia to a distinct underlying pathological substrate.

Intervals the Same: Morphology Varies
Ventricular bigeminy typically has fixed coupling intervals, and each bigeminal beat looks identical to each other. Similarly, sustained VT is usually monomorphic with fixed intervals and morphology. When we see bigeminy with fixed intervals but marked variation in morphology or a VT of constant cycle length but with a change in morphology, we generally anticipate a single source of arrhythmia that has variable routes through which the rest of myocardial activation occurs, creating each exit's own characteristic QRS complex.

Origin in the conduction system particularly more proximally, such as with the bundle branches or even the His bundle, and with functional block through one of the exiting bundles or fascicles would be expected to have a constant cycle length or coupling interval, but a morphology that would depend on which bundle branch or fascicle conducts the impulse to the ventricle.

Origin of arrhythmia in any cul-de-sac of the heart, such as the supra semilunar valve myocardium, the distal portions of a papillary muscle, or a pathologically created cul-de-sac—viable myocardium amid or distal to a scar—may also generate a regular ventricular impulse but with discrete and distinct exits to the rest of the heart and varied QRS morphology.

Morphology the Same: Interval Varies
Bigeminal or other regularly occurring ventricular ectopy may occasionally show variation in the coupling interval. Autonomic tone (the baseline sinus conducted rate) and the mechanism for the PVCs may impact why such interval variation occurs. For example, with a nontriggered automatic focus, variation in the amount of entrance block or propagation of a conducted impulse to the arrhythmogenic site could cause considerable interval variation. In the extreme case, mutual complete entrance block, but without exit block from the focus, produces 2 distinct competing rhythms, each with their own cycle length, and resulting variable fusion of ventricular activation gives rise to a parasystolic rhythm.

Cul-de-sac origin reentrant or automatic arrhythmia, as in the sites mentioned above, may also be affected by varying entrance block to their site of origin with or without varying exit block, producing significant irregularities in interval but with essentially the same morphology for the PVC.

During a sustained VT, abrupt changes in the interval suggest exit block from the site of origin and indirectly imply arrhythmogenic substrate within or near scar or significantly diseased tissue.

Linked Events: Triggered Automaticity
Shenthar references and summarizes an entirely different mechanism for regularly varying morphology during an arrhythmia. Here, it is not the exit or subsequent propagation of an event that produces the changes in interval or morphology but rather the influence of one focus on another. In this ping-pong type of abruptly changing directionality, one site's threshold for triggered activity produces in turn an increased rate that then allows another site to reach its threshold for a delayed afterdepolarization and may thus initiate and maintain a pattern of marked change in access or morphology during a sustained arrhythmia.

Linked Reentry?
The patient described in their case illustration has myocardial scar and discrete pathology well demonstrated by the cardiac imaging modalities shown. Unlike the patient with normal heart outflow tract VT or digoxin toxicity, we do not expect triggered automaticity in scar-related VT but rather reentry. Can one reentrant site or circuit influence another in an analogous mechanistic recreation of bidirectional VT?

Dual loop reentry and figure-of-8 reentry do involve interaction between reentrant circuits. However, with dual loop
reentry, the predominant circuit (shorter cycle length) entrains the secondary circuit, producing a fixed cycle length (cycle length of the dominant circuit) and fused but constant activation sequence. Similarly, in figure-of-8 reentry, with a discrete exit site, neither the cycle length nor the morphology would be expected to vary. It is only during ablation or when observing atypical responses to entrainment or activation mapping sequences do we suspect and diagnose these less common interactive reentrant arrhythmias.

With large circuit macroreentry, there may be >1 exit site from the protected reentrant circuit to the remaining myocardium. Here 1 exit may result in the first beat of tachycardia, but the same exit site may still be refractory when the impulse attempts to reenter the region, and a more downstream exit, which had previously been refractory, may allow exit with a different QRS for the second beat. If the phenomenon continues, potentially significant changes in the QRS morphology may result, but some cycle length variation, because the exits do not need to be equidistant from each other, would be expected.

Macroreentry, but with >1 limb present in the circuit, each with its own exit and total circuit length, could produce changing intervals, as well as morphology. However, is it possible with reentry that the cycle length remains constant but morphology changes? Microreentry, where a particular region of the myocardium exhibits extremely slow conduction, thus, enabling reentry despite a short circuit length, may result in varied morphology and interval much in a similar manner as what Shenthar describes and mentioned above for point-source automatic tachycardia.1

The Great Mimic

As students, we learned of the great mimics in general medicine. Historically, syphilis and then systemic lupus erythematosus with their protean manifestations, insidious onset, and lack of defining characteristic inciting circumstances made these conditions a part of many differential diagnostic algorithms. With regard to VT for the invasive electrophysiologist, sarcoidosis is another such mimic.1 In some cases, where a common manifestation, such as PVCs, is seen, we do not consider this rare diagnosis (sarcoid), but in others, as in Shenthar’s case, with an unusual manifestation—bidirectional tachycardia—we would tend to think of even more unusual disorders (catecholaminergic polymorphic VT, aconitine poisoning, etc) rather than this less esoteric entity!

Shenthar not only guides us through the vocabulary of VT in the mechanisms for bidirectional tachycardia but also teaches us to not forget this largely treatable condition—sarcoidosis—when confronted by common or rare ventricular arrhythmia.1

Disclosures

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References


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