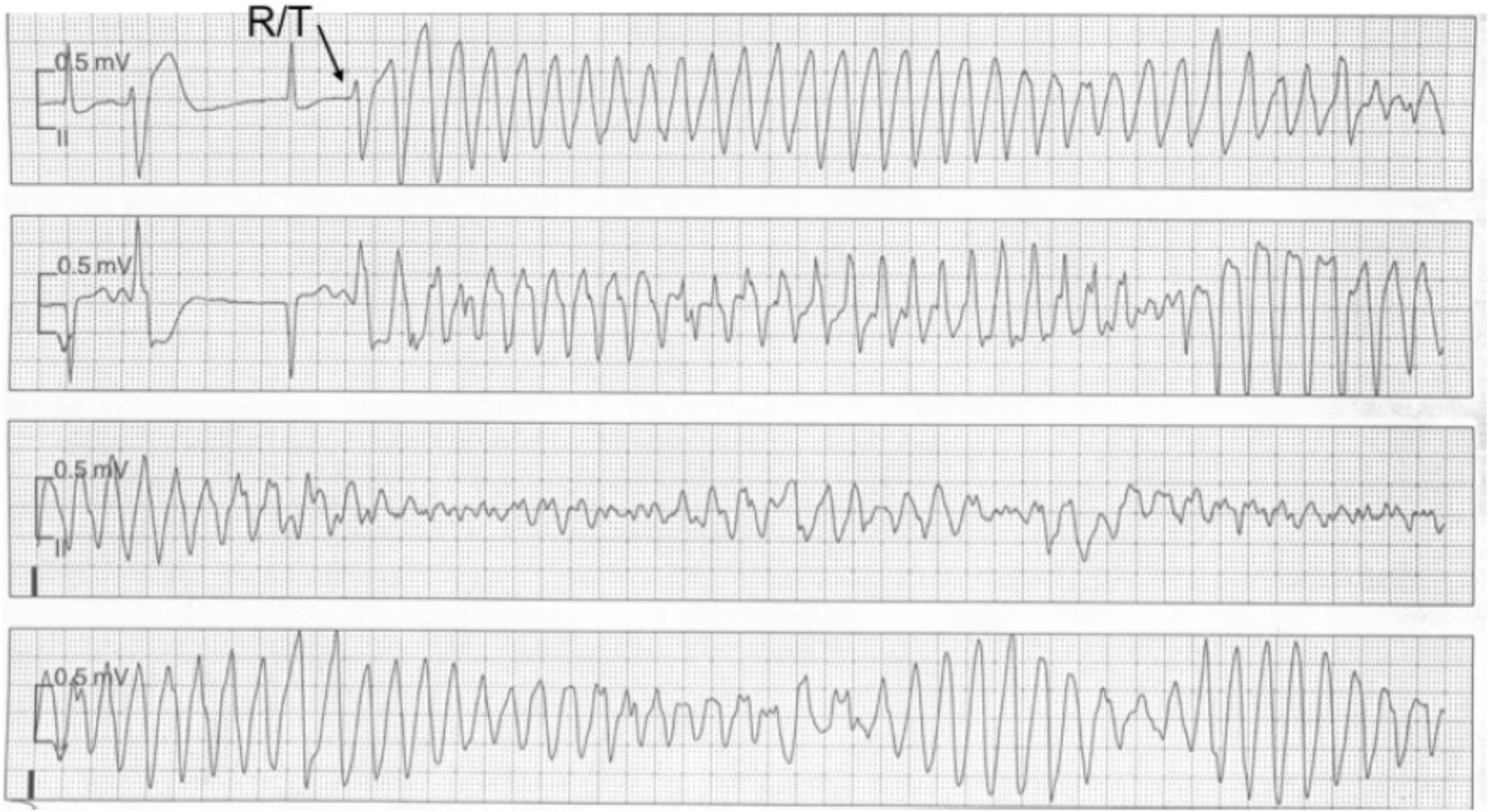
- **Torsades de Pointes (TdP)**



- Polymorphic VT characterized by:
  - Irregular RR intervals
  - Ventricular rates of 150–300 BPM (usually 200–280 BPM)
  - Sinusoidal cycles of changing QRS amplitude and polarity resulting in characteristic appearance of a twisting of the QRS complex around an isoelectric baseline
  - Prolonged QT interval (often > 600 msec; QTc > 470 msec in males and 480 msec in females)

    Often starts with short runs and progresses to sustained VT.

    QRS morphology varies from beat to beat.

    Cycles usually consist of 5–20 complexes but can become sustained.

    The wide QRS and rapid ventricular rate often make it difficult to distinguish the QRS and T waves.

    May be confused with AFIB with aberrant conduction, though, unlike TdP, AFIB may be intermixed with narrower (typical) QRS complexes and the RR intervals are more irregular.

    Rapid arm movements (such as brushing hair or teeth) can cause artifact that mimics TdP, particularly if an electrode adhesive pad is loose or has detached from skin.

    May appear monophasic in a single lead (and therefore missed), but characteristic appearance is evident in other leads.

    May be missed during very short cycles (typical twisting morphology may be absent).

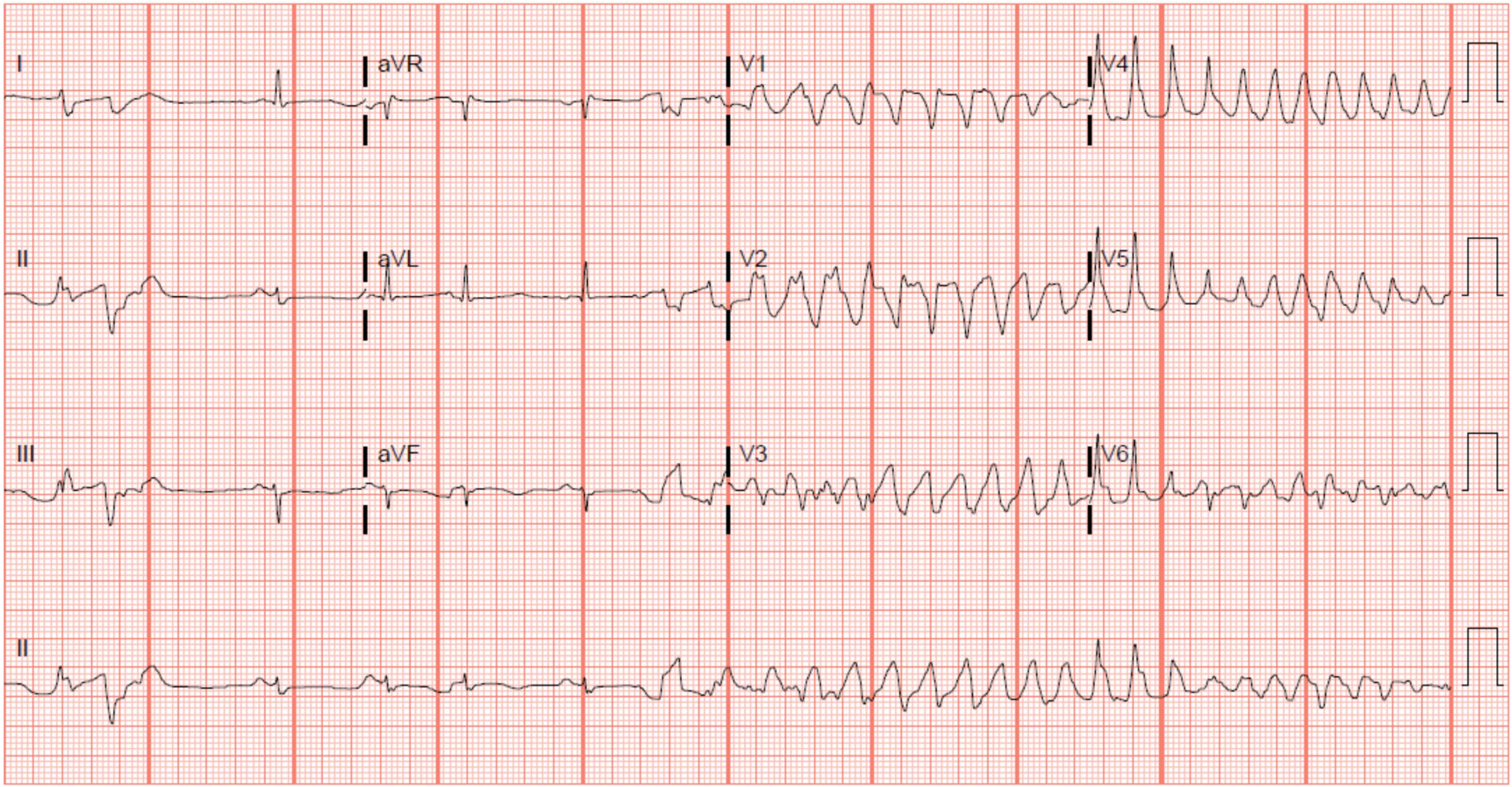
- Polymorphic VT (rapid VT with changing morphology) can be due to TdP or can be secondary to myocardial ischemia. Polymorphic VT due to TdP occurs typically in the setting of a prolonged QT interval, is preceded by a “short-long-short” RR sequence and is triggered by a “late-coupled” VPC that occurs during repolarization of the preceding complex (R-on-T phenomenon). VPCs that occur on the T wave (R-on-T) occur on the “vulnerable” portion of ventricular repolarization and are particularly dangerous due to their propensity to trigger malignant ventricular tachyarrhythmias.

  The first beat of the “short-long-short” sequence is often a VPC, resulting in a short RR interval. This is followed by a compensatory pause and then a supraventricular beat with a long RR interval and a longer QTc interval. The third beat is usually a VPC that has a relatively short cycle length (short RR interval) and falls on the preceding T wave (R-on-T), which initiates TdP.

  TdP is triggered by a “late-coupled” VPC (i.e., occurs at end-diastole) and is associated with a prolonged QTc interval.

  Polymorphic VT secondary to ischemia (in contrast to polymorphic VT due to TdP) is triggered by a “close-coupled” R-on-T VPC (i.e., occurs earlier in diastole) and is usually associated with a normal QTc interval.

- AV-dissociation is present.



Overview: TdP is an uncommon and distinctive form of polymorphic VT associated with a long QT interval that usually spontaneously reverts to normal sinus rhythm (or occasionally to marked bradyarrhythmias) within a few seconds. It usually presents as recurrent episodes of palpitations, dizziness, and syncope. However, TdP can also be life-threatening, resulting in sustained VT or degenerating into VF and presenting as sudden cardiac death. In the setting of congenital long QT syndrome, TdP can occur in patients of any age, including newborns. TdP can also occur at any age in acquired long QT due to medications, electrolyte abnormalities, and other causes. TdP is 2-3 times more common in women than in men and often occurs in the setting of organic heart disease, heart failure, LVH, marked bradyarrhythmias, intracranial (subarachnoid/intracerebral) hemorrhage, mitral valve prolapse, hypothermia, alcoholism, hypoxia/acidosis, malnourishment, renal/hepatic failure, electrolyte abnormalities (hypomagnesemia, hypocalcemia, hypokalemia), drugs (digitalis toxicity, antiarrhythmics, phenothiazines, tricyclic antidepressants, cocaine, alcohol, nicotine), and other causes of prolonged QT interval. It can also occur in structurally normal hearts.