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The Internet Book of Critical Care

Torsades de Pointes

November 22, 2016 by **Josh Farkas**

(https://i2.wp.com/emcrit.org/wp-content/uploads/2016/11/torsadesbanner.jpg)
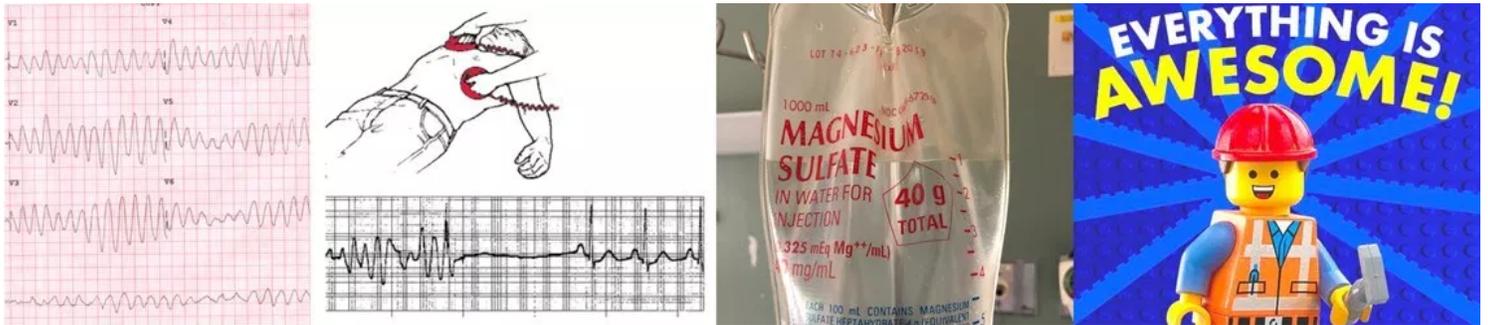
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Diagnosis of torsades

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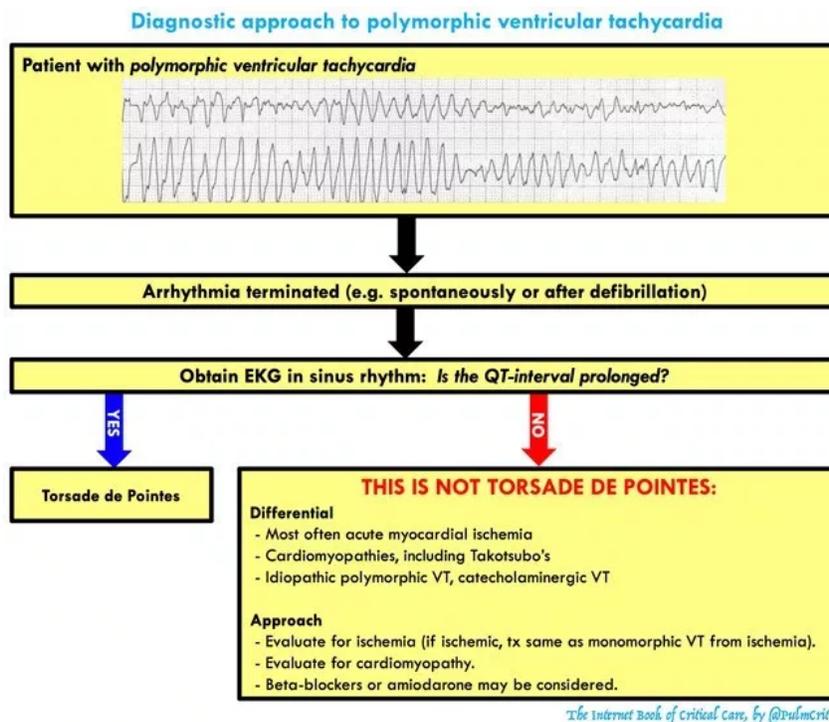
getting some definitions straight

- **Polymorphic ventricular tachycardia**
  - Defined as ventricular tachycardia with varying QRS amplitude.
  - This is commonly referred to as torsades de pointes, but it's actually *not* the same thing. Polymorphic ventricular tachycardia may be caused by several etiologies (e.g. congenital QT prolongation, acquired QT prolongation, ischemia, Takotsubo's cardiomyopathy).
- **Torsades de Pointes**
  - Torsades is defined as the combination of polymorphic ventricular tachycardia *plus* a prolonged QT-interval.
  - Torsades can be caused by either *congenital* long-QT syndrome or *acquired* long-QT syndrome (due to electrolyte abnormalities and/or medications). The vast majority of torsades results from acquired long-QT syndrome, which is the focus of this chapter.
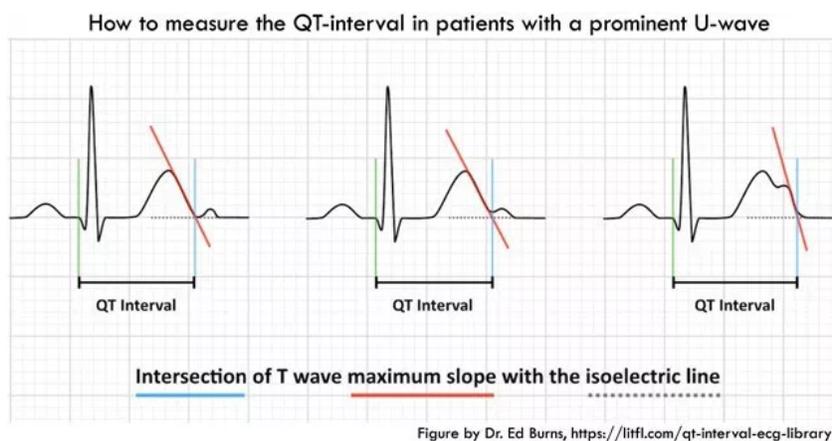
(https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/qtcmeas.jpg) **how to measure QT & QTc interval**

- The EKG computer will generally get this right, but it cannot be relied upon entirely.
- QT may vary between leads. The lead with the longest easily measurable QT interval should be used.
- If there is a U-wave, the QT interval should be determined as the intersection of the T-wave maximum slope with the isoelectric line (figure above).
- The QTc may be determined using a calculator (e.g. *MDCalc* (https://www.mdcalc.com/corrected-qt-interval-qtc)).
  - QTc > 475 ms is statistically elevated (above the 99th percentile).[1]

- Torsades is unusual unless QTc is >500 ms.[2]
- Bundle branch blocks will prolong the QT *without* increasing the risk of torsades.[3] Interpretation of QT intervals here is murky; consider comparing to prior EKGs.

clinical presentations

- Depending on the length of the episode and whether torsades degenerates into ventricular fibrillation, clinical presentations include:
  - Syncope or presyncope
  - "Seizure" episode(s)
  - Cardiac arrest
- Telemetry monitoring during an episode makes the diagnosis more straightforward.

EKG differential diagnosis: things that masquerade as torsades

- Severe hyperkalemia with "sine wave" pattern
- Coarse ventricular fibrillation
- Atrial fibrillation with Wolf Parkinson White (varying morphology may create polymorphic appearance)
- Polymorphic ventricular tachycardia with normal QT interval (may be caused by acute myocardial ischemia, Takotsubo cardiomyopathy, or such genetic conditions as catecholaminergic VT).

Tx #1: breaking active polymorphic VT

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unstable: immediate defibrillation

- Patients with unstable polymorphic VT need immediate defibrillation.
- The defibrillator will often be unable to lock onto any QRS complexes, so you generally need to perform an *unsynchronized defibrillation*.
- Technically the appropriate amount of energy is 200J biphasic. However, it might also be reasonable to use more energy particularly in a larger patient (with the rationale that unsuccessful defibrillation could be very dangerous, potentially pushing the patient into ventricular fibrillation).

"stable polymorphic VT"

- Polymorphic VT is never truly stable. This is a short-lived *transitional state* which usually flips back into sinus rhythm, or less commonly degenerates into ventricular fibrillation. Even if the patient looks OK, they're not really stable: attach pads and don't leave the room.
- Given the risk of degeneration into VF, urgent defibrillation is reasonable if the patient shows any hint of instability.
- Intravenous magnesium is a reasonable treatment, but a practitioner should be in the patient's room with a defibrillator ready to shock the patient.
  - Give 2 grams (8 mM) IV magnesium sulfate over 10-15 minutes, repeat if no response. If the patient remains stable and the rhythm persists, repeat an EKG and consider the possibility of an alternative diagnosis.
  - Note that most treatments associated with torsades are used for the *prevention* of VT, not *breaking* an episode of VT. Thus treatments such as isoproterenol or pacing have no role here.

Tx #2: Basic tx to prevent recurrent torsades

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If the EKG shows a prolonged QT-interval, the patient is diagnosed with torsades. If you simply break torsades but do nothing else, it is likely to recur. The following therapies will prevent recurrence of ventricular tachycardia in this situation:

magnesium = 1st line therapy

- Patients with torsades should receive magnesium, even if they have a normal magnesium level.
- **Four grams magnesium sulfate IV (16 mM)** is a reasonable place to *start*. Unfortunately, if you stop after four grams then the magnesium level will fall over several hours and torsades may recur.[4]
- Additional magnesium should be provided based on the patient's renal function:

- (a) If GFR > 30 ml/min, a **magnesium infusion** is useful (see protocol below).
  - (b) If GFR < 30 ml/min, cycle magnesium levels and bolus intermittently to target a magnesium level of 3.5-5 mg/dL (1.5-2 mM).
- Magnesium has a very wide safety margin. A protocoled magnesium infusion may seem aggressive, but overall this is far safer than the risk of recurrent cardiac arrest.
- Below is a magnesium protocol (one version in American units, one in SI units).[5,6] This will work best if pasted directly into the patient chart so that everyone is literally on the same page (physicians, nurses, and pharmacists).
- The rationale for a magnesium infusion is explored further here (https://emcrit.org/pulmcrit/tdp-magnesium/) .

Cardiac Magnesium Infusion Protocol

- **[1] Loading dose & starting infusion**
  - Load with 4 grams magnesium sulfate over 1 hour
  - Then start infusion at 1 gram/hour
- **[2] Monitor electrolytes & magnesium q6hr x 24 hours**
  - Magnesium
    - Target level = 3.6-4.9 mg/dL
    - If Mg is 5-7 mg/dL → reduce infusion by 50%
    - If Mg >7 mg/dL → stop infusion (do not re-start)
  - Potassium: replete for target K > 4.5 mM
- **[3] Clinical monitoring**
  - For weakness or somnolence, check Mg level
  - For bradycardia or dyspnea, stop infusion and check a Mg level
- **[4] Stop magnesium infusion after 24 hours**
References: J Intensive Care Med 2008; 23:61 and Critical Care Med 1995; 23: 1816.

(https://i0.wp.com/emcrit.org/wp-content/uploads/2016/11/amfixed3.jpg)

Cardiac Magnesium Infusion Protocol (SI Units)

- **[1] Loading dose & starting infusion**
  - Load with 16 mMol magnesium sulfate over 1 hour
  - Then start infusion at 4 mMol/hour
- **[2] Monitor electrolytes & magnesium q6hr x 24 hours**
  - Magnesium
    - Target level = 1.5-2 mM
    - If Mg is 2.1-2.9 mM → reduce infusion by 50%
    - If Mg >3 mM → stop infusion (do not re-start)
  - Potassium: replete for target K >4.5 mM
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potassium

- Aggressively treat any hypokalemia, targeting a high-normal potassium level (>4.5 mEq/L).[7]

cessation of all QT-prolonging medications

- The medication list should be carefully reviewed for any medications which may prolong QT interval. Patients are often on several QT-prolonging medications, all of which should be stopped if possible.
- Evidence regarding which drugs cause torsades is extremely murky. In some cases it seems that drugs have been incorrectly maligned (e.g. azithromycin (https://emcrit.org/pulmcrit/myth-busting-azithromycin-does-not-cause-torsade-de-pointes-or-increase-mortality/) , olanzapine (https://emcrit.org/pulmcrit/intravenous-olanzapine-haloperidol/)). One complicating factor is that there isn't a simple relationship between QT prolongation and torsades (some drugs such as amiodarone increase the QT *without* causing much torsades).
- A comprehensive list of medications implicated in causing torsades is here (https://crediblemeds.org/pdftemp/pdf/CombinedList.pdf) . The most common offenders are listed below.

more common medications linked to torsades[2,3,7]

- Antiarrhythmics

- Class IA: Quinidine, disopyramide, procainamide
- Class IC: Flecainide
- Class III: Dofetilide, ibutilide, sotalol, dronadarone
- Psychotropic
  - Haloperidol, droperidol, chlorpromazine, pimozide
  - Citalopram, escitalopram
  - Tricyclic antidepressants
- Antibiotics
  - Clarithromycin, erythromycin
  - Fluoroquinolones
  - Fluconazole, itraconazole, voreconazole, posaconazole
  - Pentamidine
- Other
  - Methadone
  - Cocaine, loperamide (when abused in massive doses)
  - Ondansetron
  - Propofol
  - Arsenic trioxide, sunitinib, vandetanib

Tx #3: Refractory torsades storm

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Occasional patients will have recurrent episodes of torsades ("Torsades storm"). Each individual episode may be treated with magnesium or defibrillation, if needed (Treatment step #1 above). However, additional therapies are required to stop recurrence and end the storm.

re-load magnesium PRN

- Recurrent torsades may reflects inadequate magnesium dosing (e.g. patient is bolused with 2-4 grams, without an infusion). The first step when managing recurrent torsades is therefore to ensure that the patient has truly received an adequate dose of magnesium.
- If the patient was bolused with magnesium a few hours ago without an infusion, re-load with 2-4 grams IV immediately (8-16 mM).
- If the patient is a candidate for magnesium infusion (GFR >30 ml/hr), this should be ordered.
- If the patient has renal failure and has already received 4-6 grams of magnesium (16-24 mM), then check magnesium levels and ensure that a high level is achieved.   Note that a therapeutic level for torsades is roughly 3.5-5 mg/dL (1.5-2 mM) – not a "normal" level.
- More on magnesium above (Treatment step #2).

optimize the potassium

- The target potassium level here is probably >4.5-4.7 mEq/L.
- Giving potassium alone is unlikely to work, but it might help a bit.

speed up the heart

- Speeding up the heart rate will decrease the QT interval and reduce the risk of acquired torsades (but it may be contraindicated in some forms of congenital torsades).
- The usefulness of chronotropy depends on the patient's **baseline heart rate**.
  - Chronotropy is most beneficial for patients starting out with bradycardia.
  - If the patient is already significantly tachycardic, chronotropy is unlikely to provide benefit. The usual target heart rate is 100-110 b/m, but occasionally heart rates up to 140 b/m may be needed.[2] There's no high-quality data on this.
- **Medical chronotropy** is generally the easiest & fastest way to stabilize the patient. The ideal chronotrope depends on the patient's hemodynamics and baseline blood pressure:
  - Baseline severe hypotension: epinephrine infusion.
  - Baseline normotension or mild hypotension: dobutamine or isoproterenol infusion.
- **Electrical chronotropy** may be used if medical chronotropy fails:
  - Transcutaneous pacing may work, but this is painful for conscious patients.

- Transvenous pacing is more comfortable, but this is more invasive and takes a bit longer to achieve.
- Patients with a pacemaker may have the device rate increased.

lidocaine

- Lidocaine is the preferred anti-arrhythmic drug for torsades, although there isn't a ton of evidence supporting its use.
  - **Do not use amiodarone, procainamide, beta-blockers, or most other antiarrhythmics.** Most of these will stretch out the QT interval even further! Beta-blockers will slow down the heart rate, increasing the risk of torsades.
- Start with a loading dose of 1-1.5 mg/kg followed by a 1 mg/min infusion. For recurrent arrhythmias, re-load with another 1 mg/kg bolus and increase the maintenance infusion.

consider an alternative diagnosis

- Torsades is generally fairly easy to control with a combination of high-dose magnesium, heart rate augmentation, and occasionally some lidocaine. Failure to respond to these interventions suggests an alternative diagnosis (e.g. polymorphic VT due to ischemia or catecholaminergic ventricular tachycardia).

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algorithm

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podcast

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questions & discussion

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To keep this page small and fast, questions & discussion about this post can be found on another page *here* [(https://emcrit.org/pulmcrit/tdp/)](https://emcrit.org/pulmcrit/tdp/).

[(https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif)](https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/pitfalls2.gif)

- Under-dosing magnesium (e.g. giving two-four grams magnesium and walking away). This will often cause recurrence of ventricular tachycardia in a few hours after the serum magnesium levels fall.
- Leaving patients on QT-prolonging meds (make sure to scour the medication list for any problematic drugs).
- Don't give amiodarone or other QT-prolonging antiarrhythmics (e.g. procainamide).   These may actually aggravate the situation.
- Recognize that polymorphic VT with a normal QT interval *isn't* torsades, this requires an entirely different treatment strategy.

Going further:

- [New approach to Torsades (PulmCrit)](https://emcrit.org/pulmcrit/tdp-magnesium/) & [Prior post on Mg infusions](https://emcrit.org/pulmcrit/magnesium-infusions-for-atrial-fibrillation-torsade/)
- [QT-interval](https://litfl.com/qt-interval-ecg-library/) and [Torsades & polymorphic VT](https://litfl.com/polymorphic-vt-and-torsades-de-pointes-tdp/) (Ed Burns, LITFL)
- [Podcast on torsades](http://blog.ercast.org/torsades-de-pointes/) by Rob Orman and Joe Bellezzo (ERCast). This is a pretty epic episode and well worth listening to. I wholeheartedly agree with Joe Bellezzo regarding the need to do *something* pro-active to prevent recurrent torsades, but would suggest that a magnesium infusion can be used instead of temporary transvenous pacing.
- [Polymorphic VT](http://hqmeded-ecg.blogspot.com/2013/10/polymorphic-ventricular-tachycardia.html) (Steve Smith's ECG blog)
- [Torsades de Pointes](https://first10em.com/torsades-de-pointes/) (Justin Morganstern, First 10 in EM)
- [Evaluation of QTc drug interactions](https://www.pharmacyjoe.com/how-to-evaluate-qtc-drug-interactions/) (Pharmacy Joe)
- [EKG pointers: Is it Torsades de Pointes?](http://www.emdocs.net/ecg-pointers-is-it-torsade-de-pointes/) (emDocs)

References:

1. OK, technically >470 ms in men and >480 ms in women. But if you just remember 475ms that is close enough. .
2. Thomas S, Behr E. Pharmacological treatment of acquired QT prolongation and torsades de pointes. *Br J Clin Pharmacol.* 2016;81(3):420-427. [[PubMed](https://www.ncbi.nlm.nih.gov/pubmed/26183037)]
3. Drew B, Ackerman M, Funk M, et al. Prevention of torsade de pointes in hospital settings: a scientific statement from the American Heart Association and the American College of Cardiology Foundation. *Circulation.* 2010;121(8):1047-1060. [[PubMed](https://www.ncbi.nlm.nih.gov/pubmed/20142454)]
4. Biesenbach P, Mårtensson J, Lucchetta L, et al. Pharmacokinetics of Magnesium Bolus Therapy in Cardiothoracic Surgery. *J Cardiothorac Vasc Anesth.* 2018;32(3):1289-1294. [[PubMed](https://www.ncbi.nlm.nih.gov/pubmed/29169799)]
5. Moran J, Gallagher J, Peake S, Cunningham D, Salagaras M, Leppard P. Parenteral magnesium sulfate versus amiodarone in the therapy of

atrial tachyarrhythmias: a prospective, randomized study. *Crit Care Med*. 1995;23(11):1816-1824. [PubMed (https://www.ncbi.nlm.nih.gov/pubmed/7587256)]

6. Sleeswijk M, Tulleken J, Van N, Meertens J, Ligtenberg J, Zijlstra J. Efficacy of magnesium-amiodarone step-up scheme in critically ill patients with new-onset atrial fibrillation: a prospective observational study. *J Intensive Care Med*. 2008;23(1):61-66. [PubMed (https://www.ncbi.nlm.nih.gov/pubmed/18320707)]

7. Al-Khatib S, Stevenson W, Ackerman M, et al. 2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: Executive Summary. *Circulation*. 2018;138(13):e210-e271. [PubMed (https://www.ncbi.nlm.nih.gov/pubmed/29084733)]

The Internet Book of Critical Care is an online textbook written by Josh Farkas (@PulmCrit), an associate professor of Pulmonary and Critical Care Medicine at the University of Vermont.