Non-torsade VT/VF storm

December 19, 2016 by Josh Farkas

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definition

what is VT/VF storm (a.k.a. “electrical storm”)?

- Definition
  - Generally defined as three or more episodes of VF or sustained VT within 24 hours.
  - Patients can be shocked out of VT/VF reasonably easily, but then they keep flipping back into VT/VF (this differentiates VT storm from refractory VT wherein the patient is continually in VT/VF and never goes back to sinus rhythm).
  - If a patient is shocked out of VT/VF and then has a recurrence very soon thereafter, this is a harbinger of VT/VF storm (and might be considered storm by some).
**Presentation typically dramatic:**
- (a) If patient has an ICD (implanted cardiac defibrillator), this may present with recurrent ICD firing.
- (b) If patient doesn't have an ICD, this may cause recurrent symptoms. Depending on the heart rate and cardiac function, symptoms may range from palpitations to recurrent cardiac arrest.
- The problem with VT storm isn't generally breaking any individual episode of VT. Rather, the problem is often that the VT keeps on coming back – so it's difficult to keep patients out of VT for a prolonged period of time.

**Torsade vs. non-torsade storm**

- Torsade de pointes (TdP) is defined by the combination of polymorphic VT plus a prolonged QT interval. This should be managed according to the chapter on Torsades [here](https://emcrit.org/ibcc/tdp/).
- Patients with polymorphic VT with a normal QT interval (following cardioversion) don't have Torsade de Pointes. Such patients often have ischemia-mediated VT and may be managed by the treatment pathway described in this chapter.

**Pathophysiology**

**VT storm is a vicious cycle**

- VT/VF increases intracellular calcium levels, which may be pro-arrhythmic.
- Shocks and episodes of cardiac arrest (e.g. treated with epinephrine) may cause myocardial injury.
- Myocardial injury & pain stimulate outpouring of endogenous catecholamines, promoting recurrent arrhythmia.
- Key point: The natural history of VT storm is generally to deteriorate (even despite the use of standard ACLS algorithms to interrupt each individual episode of VT). Aggressive therapy is needed to stop this process.

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VFib arrest. Just after CPR is held, valves still move a bit, but there is no change in chamber size. EF is zero. ⚠️⚠️⚠️

#PoCUS
common causes

underlying substrate

- Most patients have severe structural heart disease (usually ischemic cardiomyopathy).
- If the patient has a structurally normal heart, this suggests a rare form of arrhythmia requiring specific management (e.g. Brugada syndrome, catecholaminergic polymorphic VT).

trigger

- Sometimes present (but only in ~10% of cases)¹
- Triggers may include:
  - Acute MI
  - Heart failure exacerbation, volume overload
  - Electrolyte abnormality (especially hypokalemia, hypomagnesemia)
  - Medication toxicity, substance abuse (e.g. sympathomimetics)
  - Medication non-adherence
  - Pro-arrhythmic drug toxicity
  - Thyrotoxicosis (thyroid storm with VT/VF storm)
  - Sepsis²

treatment

Non-torsade VT/VF storm Pathway

- Intubation
  - Generous analgesia (e.g. fentanyl)
  - Deep sedation (e.g. high dose Propofol)
- Access & monitoring
  - Arterial line & central line
  - Consider crash double lumen access
- Vasopressor infusion
  - Target MAP >90 mmHg
  - Use agent without much beta-activity (e.g. phenylephrine or norepinephrine)
- Amiodarone bolus & infusion
  - Start ~300 mg load & 1 mg/min infusion
  - (May re-load to total of 900 mg over time if refractory)
- Electrolytes
  - Magnesium 2-4 grams IV if deficiency or level unknown
  - Replete potassium if hypokalemic
- Esmolol bolus & infusion
  - (skip if severe hypotension)
  - Load 0.5 mg/kg then infuse 0.05 mg/kg/min
  - (May re-load and up-titrate by 0.05 mg/kg/min)
  - Max infusion rate 0.3 mg/kg/min
  - (Alternative: Propofol 0.5-1 mg/kg IV over 18 min then 3-5 mg IV q5 - QR 20 mg IV 90 s split)
- Lidocaine
  - (skip if hemodynamic instability)
  - Bolus 1-1.5 mg/kg, infuse 1-4 mg/min
  - May re-bolus and up-titrate infusion for refractory VT

Approach to non-torsade VT storm. Each individual episode of VT is treated per ACLS algorithms (which aren’t shown here). Early intubation, sedation, and definitive access is fundamental. Meanwhile, anti-arrhythmics should be escalated as needed to control repeat episodes of VT. If the patient remains hypertensive, it may be sensible to up-titrate beta-blockers (even in the absence of any VT recurrence). Patients will often require catheterization or ablation once they are stable enough for transport.

¹The Internet Book of Critical Care, by @EMcrit
²https://emcrit.org/ibcc/storm/
Treatment of VT storm requires an organized approach, with coordination between multiple services (e.g. critical care, interventional cardiology, electrophysiology, unit pharmacist). Call for help early and try to think a couple steps ahead (e.g. start ordering esmolol or propranolol before you actually need it).

### Intubation & Sedation

**Intubation**

- This is generally required for a true VT. Intubation offers numerous benefits:
  - Patients may lose airway control during episodes of VT/VF
  - Sedation itself is therapeutic
  - Intubation may facilitate safe performance of procedures (e.g. VT ablation).

**Analgesia**

- Analgesia is important for any intubated patient, but it’s especially important in VT storm because untreated pain can drive sympathetic tone and promote recurrent arrhythmia.
- Initially err on the side of over-aggressive analgesia (until the VT storm is controlled).

**Sedation**

- Deep sedation can help break VT storm.
- **Propofol** seems to work particularly well here.³
  - Propofol may cause hypotension due to vasodilation; this may be counteracted with the use of vasoconstrictors (e.g. phenylephrine).
  - Dexmedetominedine may also reduce sympathetic tone, so it’s not a terrible choice here. However, dexmedetominedine has some drawbacks which probably make it 2nd line here (at least initially until the storm has abated):
    - Dexmedetominedine is sluggish to titrate.
    - Dexmedetominedine can't achieve the same depth of deep sedation that propofol can.
    - To date, no evidence on dexmedetominedine in VT storm.
- Benzodiazepine may be used if unable to tolerate propofol/dexmedetominedine due to severe hypotension.

### A-line & Pressors

**Arterial Line**

- These patients are often very hemodynamically labile (e.g. with recurrent episodes of cardiac arrest).
- By definition, in every case of VT storm we will be aggressively titrating numerous drugs which affect hemodynamics (e.g. propofol, amiodarone, beta-blockers).
- An arterial line is usually indispensable and should be placed as soon as possible.
  - These patients may be good candidates for [crash femoral access](https://emcrit.org/pulmcrit/hemodynamic-access-for-the-crashing-patient-the-dirty-double/).

**Pressors**

- Phenylephrine may be the best choice, because it won't stimulate cardiac beta-receptors.
- Pressor support may facilitate the use of propofol and beta-blockers to treat VT storm.
- The coronary circulation is perfused in diastole, so don't allow the diastolic Bp to decrease too much.

### Antiarrhythmics
1st line antiarrhythmic: amiodarone

- Usual regimen: 300 mg bolus, then 1 mg/min x 6 hours, then 0.5 mg/min.
- Additional boluses can be given for recurrence, up to a total of ~1,200 mg in boluses.
  - Avoid >2.2 grams total dose within 24 hours.

magnesium & electrolyte management

- 2-4 grams of IV magnesium sulfate may be considered, particularly if patient is deficient.
  - There is no strong evidence to support the use of magnesium. Given that intracellular calcium overload may be implicated in VT/VF storm, magnesium might be expected to alleviate this problem given some antagonism of calcium's effects.
- Correct other electrolyte abnormalities (e.g. hypokalemia)

2nd line antiarrhythmic: beta-blockers (might actually be the most effective therapy)

- IV Esmolol infusion is fairly well supported by evidence.\(^4\)\(^5\)
  - Loading dose is 0.3-0.5 mg/kg IV (~30 mg).
  - Start infusion at 0.050 mg/kg/min (~3 mg/min).
  - May re-load & up-titrare infusion in increments of 0.05 mg/kg/min every 10 minutes.
  - Max dose is 0.3 mg/kg/min.
  - The advantage of esmolol is titratability. From a mechanistic standpoint, it may be less effective than propranolol because it lacks efficacy at beta-2 receptors.
- IV Propranolol
  - Superior to metoprolol (and probably esmolol) because antagonizes both beta-1 and beta-2 receptors. However, may be harder to get access to and more difficult to titrate.
  - IV regimen: Bolus 0.15 mg/kg IV over 10 minutes, then 3-5 mg IV Q6hr.\(^5\)\(^6\)
  - Oral regimen: Propranolol 40 mg PO q6hrs has been proven superior to metoprolol (50 mg q6hr).\(^7\)
  - IV metoprolol may be used if nothing else is available (5 mg IV every 5 minutes for total of 15 mg).

3rd line antiarrhythmic: lidocaine (unclear value)

- Lidocaine is traditionally recommended as third-line antiarrhythmic after amiodarone & beta-blockers.
- Efficacy may be limited to patients with acute ischemia.\(^1\)
- However, caution is advised, as class-I anti-arrhythmics may worsen cardiac function due to negative inotropic effects.\(^2\) Given lack of proven benefit in the context of VT/VF storm, it's unclear whether this risk is worth it. Furthermore, combining systemic lidocaine with stellate nerve block(s) might increase the risk of systemic lidocaine toxicity.
  - Growing evidence is suggesting that it might be best to move directly to stellate ganglion block at this point, rather than trialing IV lidocaine (see below).
- Dose: Bolus with 1-1.5 mg/kg and then infuse at a rate of 0.02 mg/kg/min.\(^1\)
  - May re-bolus with 0.5-0.75 mg/kg IV up to a total dose of 3 mg/kg.
  - May up-titrare infusion up to ~4 mg/min.

stellate ganglion block

- Nerve block in the neck cuts off sympathetic outflow to the myocardium.
- Supported by case-study level data, which show a dramatic reduction in arrhythmia burden.\(^8\)
- Seems fairly safe (e.g. used as an outpatient procedure for control of neuropathic pain).

[back to contents]
Sensible intervention for patients failing to respond to intubation & anti-arrhythmics.
The block should cause an ipsilateral Horner’s syndrome (pupillary constriction), which is evidence of a successful block.

**left stellate ganglion block versus bilateral blocks?**

- The left stellate ganglion is more important in autonomic regulation of the heart, so most literature has described unilateral left-sided blockade.
  - In a recent systematic review of the literature, 34/38 reported cases received left-sided block, with the remaining four receiving bilateral blocks.\(^8\)
- However, bilateral blockade may be most effective. Some authors are currently advocating for bilateral blockade.\(^9\)

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**basic anatomic principles**

- (1) Operate at the level of the C6 transverse process (Chassaignac’s tubercle; figure above).
  - This bony structure might be appreciated with deep palpation adjacent to the trachea at the level of the cricoid membrane.
  - The C6 transverse process should be visible during the procedure as a bright, bony signal.
  - The C6 transverse process provides a back-stop which blocks the needle from hitting the vertebral artery (although ideally you should never hit the bone).
- (2) The injection target is just anterior to the longus coli muscle, which runs underneath carotid artery.

**overview of two different techniques**

Two general techniques may be used. There’s no firm evidence regarding which is better, so both will be described here.

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“landmark technique” approach

- Traditionally, stellate ganglion blocks were performed using a landmark technique as follows:
  - Palpate the neck adjacent to the trachea to locate the lateral process of C6 (Chassaignac's tubercle)
  - Insert a needle straight into the neck until it hits Chassaignac's tubercle.
  - Inject some local anesthetic, then withdraw 1-2 mm and inject some more anesthetic.
- This is a fast and simple technique (see video below). The problem is that if you’re off a little (either too cephalad or caudad) the needle could slip past Chassaignac's tubercle and hit the vertebral artery.
- This approach can be replicated using ultrasonography, which allows needle visualization and prevents going too deep. Ideally anesthetic should be placed just anterior to the longus coli muscle.
The Ultrasound Podcast provides a nice video illustrating the ultrasound views that would be used for this technique (start at 5:10 if you're in a hurry).

**lateral approach**

- In this approach, the patient's head is turned to the contralateral side. The longus coli is approached lateral to the carotid artery (figure below). As with any technique, the goal is to deposit anesthetic just *anterior* to the longus coli muscle.
- This technique has the advantage of providing a greater margin of safety between the needle and vital structures (e.g. the carotid artery and thyroid).
A more detailed illustration of the sono-anatomy at the level of C6 is shown below.\textsuperscript{10}

- The key sono-landmarks are the carotid artery and the Chassaignac's tubercle (marked "at" in the figure below).
- The longus coli (with the ganglion anterior to it) are sandwiched between the carotid artery and the anterior tubercle.
- Using some gentle posterior-medial pressure with the ultrasound probe may displace the carotid artery medially, opening up this space between the carotid and Chassaignac's tubercle.\textsuperscript{11}

The following video illustrates this general approach, albeit at the C7 level (not my preferred level because, as you can see in the video, incorrect angulation of the needle could risk laceration of the vertebral artery).
Another short video to reinforce the anatomic structures:

Ultrasound guided Stellate Ganglion for CRPS
Implanted Cardioverter-Defibrillator (ICD) optimization

- Any patient with an ICD requires device interrogation.
- Ensure that the device is truly detecting VT (rather than over-responding to artifact).
- Anti-tachycardia overdrive pacing may be optimized to break episodes of VT without requiring shocks.

Revascularization & therapy for myocardial infarction

- MI is an important cause of VT storm.
- Recurrent VT/VF should always prompt consideration for emergent cardiac catheterization.
- Additional therapies for acute coronary syndrome should also be considered (e.g. aspirin, P2Y12 inhibitor).

Catheter ablation of VT/VF

- Case series suggest that ablation has a surprisingly high initial success rates in monomorphic VT storm.
- Recent meta-analysis of 417 patients with VT storm demonstrated clinical arrhythmia suppression in 92% of patients, with an impressive safety profile (complication rate of 1% and peri-procedural mortality of <1%).

ECMO

- May serve as bridge to revascularization and/or ablation.
- May facilitate use of very high-dose beta-blockers and sedation, to settle arrhythmias.
- Unavailable in most centers (and patients are usually too unstable to transfer).

post-cardiac arrest care

- Patients who have had a VT/VF arrest causing anoxic brain damage require temperature management to prevent neurologic injury. Post-cardiac arrest management is discussed [here](https://emcrit.org/ibcc/post-arrest/).
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The Internet Book of Critical Care, by @PULMCrit

[https://emcrit.org/ibcc/storm/]

podcast

[https://i1.wp.com/emcrit.org/wp-content/uploads/2016/11/apps.40518.14127333176902609.7be7b901-15fe-4c27-863c-7c0dbf2c65c5.5c278f58-912b-4af9-88f8-a65ff2da477.png]


The Podcast Episode

Want to Download the Episode?
Right Click Here and Choose Save-As [http://traffic.libsyn.com/ibccpodcast/IBCC_EP_33_-_VF_Storm_Final.mp3]

questions & discussion

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


- Unawareness of the entity of VT storm and its specific treatment pathway.
Glimming together Torsades de Pointes (TdP) with non-torsade electrical storm, as is done in some articles. This is a pitfall, because Torsade de Pointes requires a specific magnesium-based therapeutic plan (https://emcrit.org/ibcc/tdp/#Tx_#2:_Basic_tx_to_prevent_recurrent_torsades).

A wait-and-see approach, which will often fail these patients (VT storm is a vicious cycle which often deteriorates without aggressive management). Once a patient has had two-three episodes of VT within a day, strongly consider progressing down the VT storm pathway (with intubation and sedation).

Under-utilization of sedation, analgesia, and beta-blockers.

Failure to consider Stellate Ganglion Blockade in refractory VT storm.

**Going further:**

- [Propranolol vs. metoprolol for electrical storm](https://emcrit.org/emcrit/propranolol-versus-metoprolol-for-electrical-storm/) (Kristina Kipp, EMCrit)
- [Electrical storm or refractory VF/VT](http://lifeinthefastlane.com/ccc/electrical-storm/) (Blog, LITFL)
- [Management of electrical storm](https://first10em.com/electrical-storm/) (Justin Morgenstern, First10EM)
- [Electrical storm](https://wikem.org/wiki/Electrical_storm) (WikEM)
- Stellate ganglion block
  - [Ultrasound guided stellate ganglion block for refractory VF](https://resusreview.com/2013/ultrasound-guided-stellate-ganglion-block-for-refractory-ventricular-fibrillation/) (Charles Bruen, Resus Review)
  - [Stellate Ganglion Block](https://www.ultrasoundpodcast.com/2018/05/stellate-ganglion-block-for-ventricular-storm-save-a-life-with-this-nerve-block/) (Ultrasound Podcast)

**References**


