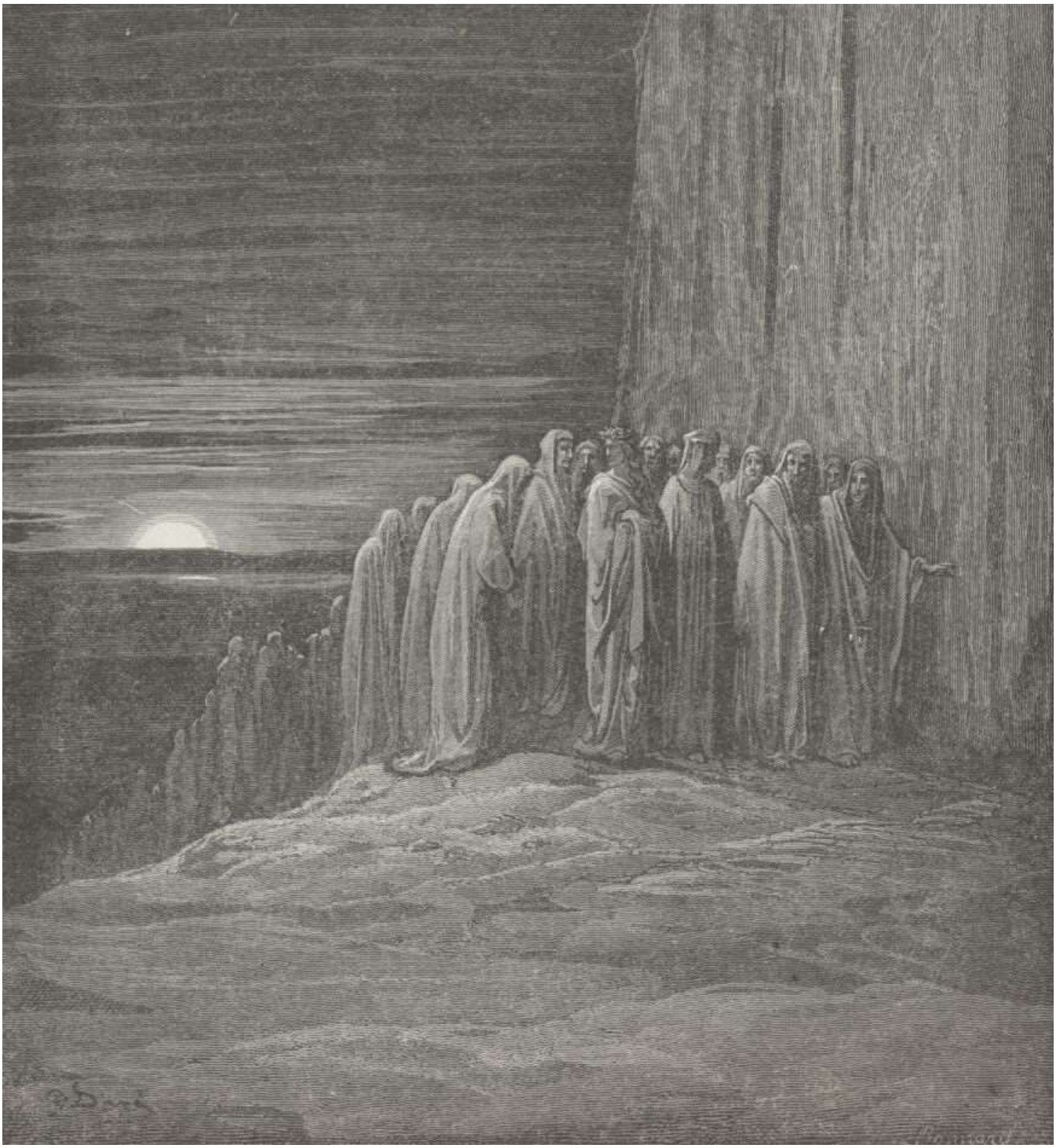
THE NORTHERN HOSPITAL

PULSELESS ELECTRICAL ACTIVITY



"…Look how the sun's rays on the ground are cut off to his left…" Gustave Dore, woodcut print 1865.

"…And as the air, when it is full of rain,
is adorned with rainbow hues not of its making
but reflecting the brightness of another,

so here the neighbouring air is shaped
into that form of the soul, which stays with it,
imprints upon it by its powers.

and like the flame that imitates its fire,
wherever that may shift and flicker,
its new form imitates the spirit.

a shade we call it, since the insubstantial soul
is visible this way, which from the same air forms
organs for each sense, even that of sight…"

Dante Alighieri, Purgatorio, XXV, 91-102 (1306-1317)

"…Look how the sun's rays on the ground
are cut off to his left
and how he moves and seems like one alive.

Hearing these words, I turned to look at them
and saw that they were staring in amazement
at me, at me and at the interrupted light…

…When they perceived my body stopped
the rays of the sun from shining through,
their voices faded to a hoarse and drawn out, "Oh"…"

Dante Alighieri, Purgatorio, V, 4-9 (1306-1317)

"…The souls who at my taking breath
could see that I was still alive,
turned pale with wonder,

and as people crowd to hear the news
around the messenger who bears the olive-branch,
and no one minds the crush,

So all these fortunate souls
kept their eyes fastened on my face,
as though forgetful of the road to beauty.

I saw one of them come forward
with such affection to embrace me
That I was moved to do the same.

Oh empty shades, except in seeming!
Three times I clasped my hands behind him
Only to find them clasped to my own chest…"

Dante Alighieri, Purgatorio, II, 67-81 (1306-1317)

Dante is the only mortal in history to have been miraculously granted a glimpse of all three realms of the afterlife or, "the other world" as it was widely known in his day, and allowed to return to tell the tale.

First he is shown the unimaginable horrors of the circles of Hell, then the torments of the Terraces of Purgatory. Eventually to his great relief, he will be shown the Nine heavenly Spheres of Paradise. In this way he is given a second chance at life, providing he changes his ways.

As Dante is guided by Virgil through the Seventh Terrace of Purgatory, wherein wander the souls of the lustful, he explains the nature of a "shade" or spirit to his readers. A shade is like a rainbow. Just as a rainbow imprints itself upon thin air, so too does the soul. In this manner the soul transforms the surrounding air into a memory or reflection of its former body. This reflection is as inseparable from the shade, as a flame from its fire.

On the whole Dante passes through the Seven Terraces of Purgatory unnoticed by the host of tormented souls, but occasionally an observant shade notices that the stranger is not "one of them". On the Second Terrace of the Envious he is betrayed by his breathing, which spirits have no need for. A great crowd of curious shades quickly gather around him. In an attempt to reach out to one of them his hands pass straight through the image, just as if he were attempting to grab hold of a rainbow, "Three times I clasped my hands behind him, only to find them clasped to my own chest." On the Fifth Terrace of the Avaricious he is noticed again, this time by the fact that he is casting a shadow, which no spirit can do, "...Look how the sun's rays on the ground are cut off to his left."

When confronted with a patient who has appeared to have left this world for the afterlife, we must quickly establish if this is to be forever, or whether they are merely engaged in a momentary glimpse of the other world from which they may return. In the situation of PEA the ECG may assist us in this assessment. It will give us a useful indicator of the true state of the patient. An agonal rhythm, suggests that our patient is not for this world again. Their non perfusing type rhythm is like a rainbow or a shade. There is no true substance to it. But if there is a rhythm of the perfusing kind, the situation is very much more optimistic! We also correlate the ECG with careful observation of the patient's clinical status. We must be keenly observant, like the envious shade of the Second Terrace or the Avaricious one of the Fifth, to the tell tales signs that life still exists with a chance of return. We look for signs of breath, as one who "seems alive". We must act quickly in this second group in order to give our patient a second chance. For those who are given this second chance it is to be hoped that they mend their ways by paying close attention to the potentially reversible risk factors that may have led them to this momentary vision of the other world.

PULSELESS ELECTRICAL ACTIVITY

Introduction

Pulseless electrical activity (PEA), is broadly defined as the presence of ventricular electrical activity as recorded on an ECG monitor, yet without any **clinical** evidence of a cardiac output, (i.e the patient is **pulseless** and **unconscious**).

VF and Pulseless VT are *by convention* not included in the definition.

PEA is one of the 4 cardiac arrest rhythms.

The 4 cardiac arrest rhythms are:

1. VF

2. Pulseless VT

3. Asystole

4. Pulseless Electrical Activity

In the past PEA was defined as having a "very poor prognosis", however a *blanket* statement cannot in truth be made for this **heterogeneous** condition, as it will depend on the **exact cause**, which in turn will determine the ease (or otherwise) of reversibility, and hence the prognosis.

For example an ECG rhythm which shows a narrow complex electrically organized rhythm, due to hypovolemia is a very different entity from an "agonal" (< 10 complexes per minute) wide complex rhythm due to irreversible hypoxic injury - and yet both, by traditional definitions may be termed "PEA".

Some causes are **readily reversible**, while other causes are **irretrievable**, thus the lumping together of all "PEA" with **asystole** in current ACLS protocols is not appropriate, given the heterogeneity of conditions that lead to PEA.

Further some **cardiac output may exist** *despite* being unable to palpate a pulse **clinically**, e.g. in hypovolemia or hypothermia. This situation is sometimes referred to as "**Pseudo-Pulseless Electrical Activity"** and does not necessarily carry a "dismal" prognosis at all.

Additionally, reversible causes require **specific interventions** that are not part of ACLS protocols.

The traditional mnemonic of the **4 Hs and the 4 Ts** does provide a useful **list** for potentially reversible causes, however the complete list is variably defined and difficult/ impossible to be recalled in the heat of an arrest situation.

This thinking is therefore too complex, and does not provide guidance as to the **most likely cause in any given scenario** nor does it **guide effective initial interventions**.

A more **practically useful** approach to PEA (**in the non-trauma setting**) is an assessment via the triad of:

1. **Clinical setting**

2. **ECG rhythm (narrow versus wide)**

3. **Point of care ultrasound**

The best chance of survival rests in finding a **reversible** cause.

If this can be **quickly** found and **promptly corrected**, then prognosis may not be nearly so "dismal" as is generally quoted for PEA as an undifferentiated group.

History

Pulseless electrical activity (PEA), is the current term for what was previously known as **electro-mechanical dissociation (EMD)**

Pathophysiology

Types of Electrical Activity in PEA

Clinically, it is useful to distinguish between 2 types of ECG rhythm in PEA:

1. A "**perfusing**" or organized type:

    - This is usually a **narrow QRS complex**, (unless there is a pre-existing BBB).

      These are **electrically organized** rhythms, which would normally be expected to provide some cardiac output, such as **sinus rhythm, sinus bradycardia, sinus tachycardia, rapid or slow AF.**

      It is much more likely that PEA is due to a reversible cause (see below) if the rhythm is of this type.

      It is also more likely that the PEA may in fact be a "Pseudo-Pulseless Electrical Activity".

2. A "**non-perfusing**" or non-organized type:

    - This is a **wide QRS complex**

      These are **chaotic** rhythms, which would normally not be expected to provide (a significant) cardiac output.

      These rhythms include:

        - **Agonal** (wide, irregular, very slow (<10), sometimes referred to as "pre-asystole" or "dying heart") The term "brady-asystole" is also used for this type of rhythm.

- ♥ **Idioventricular rhythm** (wide QRS, no atrial activity, rate about 10-40.) (In distinction to *accelerated* IVR (rate 40-100) which normally does provide a reasonable cardiac output)

Causes of PEA

The traditional mnemonic of the **4 Hs and the 4 Ts** does provide a useful **list** for potentially reversible causes, however the complete list is **variably defined** and difficult/ impossible to be recalled in the heat of an arrest situation.

This thinking is therefore too complex, and does not provide guidance as to the **most likely cause in *any given scenario*** nor does it **guide effective initial interventions**.

Note that **trauma**, is a special specific setting that has unique management strategies **separate** from current ACLS guidelines and so are is not included here.

A more practically useful approach in considering the differentials (in a non-trauma setting) is to consider the **ECG rhythm**, as follows:

Narrow complex (= hypovolemia / hypothermia & mechanical causes):

1. Pseudo-PEA

2. Potentially reversible causes:

   H: **Hypovolemia**

   H: Hypothermia (more likely this will be pseudo-PEA).

   T: Tension pneumothorax

   T: Massive pulmonary embolism.

   T: Cardiac tamponade / myocardial rupture.

   Also: Hyperinflation on mechanical ventilation

   Note that if there is a **pre-existing BBB**, then QRS complexes for the above conditions will be wide.

Wide complex (= metabolic / toxic causes):

1. Irreversible causes

   - Agonal rhythm (< 10) - "dying heart".

2. Potentially reversible causes:

   T: Toxins - **Sodium channel blockade**

   H: **Hyperkalemia**

   T: Myocardial infarction - end stage pump failure

Note that some traditionally listed causes are excluded.

The following **rarely - if ever -** present primarily with PEA:

- Hypoxemia
- Hypokalemia
- Hydrogen ions i.e acidosis - Metabolic acidosis *without hyperkalemia* is usually the **consequence of,** rather than the cause of PEA.
- Hypoglycemia

Hypothermia and **trauma** are usually obvious with the clinical scenario.

Of "toxins", the initial presentation of β-blocker, calcium channel blocker and digitalis toxicity is almost always hypotension, sinus bradycardia, sinus arrest or atrioventricular block. Some of these do progress to PEA but by that time the diagnosis is usually well established.

All untreated narrow complex PEA will *eventually* become wide complex PEA / asystole **pre-terminally.**

Clinical assessment

Making the Diagnosis:

By definition there is electrical activity on the ECG, but patient is pulseless and unconscious.

Clinical detection of pulses may be difficult in some cases such as patients with severe hypothermia or severe hypotension.

Hence in practice it is sometimes difficult to make any definitive comment on whether there is mechanical cardiac contraction based purely on palpation for the pulse.

In some cases of apparent PEA, some **cardiac output may exist** *despite* being unable to palpate a pulse **clinically**, e.g. in hypovolemia or hypothermia. This situation is sometimes referred to as "**Pseudo-Pulseless Electrical Activity"** and does not necessarily carry a "dismal" prognosis at all.

The following can help in making the diagnosis:

1. If the patient is conscious or responsive then there must be some cardiac output.
2. In intubated patients the $ETCO_2$ monitor can be checked and if there is a reading above zero there must be at least some cardiac output.
3. Ultrasound at the bedside can detect the presence of cardiac contractions - or establish that there aren't any.
4. If an arterial line is in place, look for an arterial waveform and a recorded blood pressure.

5. Doppler ultrasound devices may detect the presence of a pulse.

There may therefore be some cardiac output despite being unable to palpate a pulse clinically. This situation is sometimes referred to as "Pseudo-Pulseless Electrical Activity". This is more likely the more normal the rhythm is on ECG monitor.

The commonest causes of pseudo-pulseless electrical activity are **severe hypotension** and **hypothermia.**

Assessing the cause:

A useful approach to PEA (**in the non-trauma setting**) is an assessment via the triad of:

1. **Clinical setting**

2. **ECG rhythm (narrow versus wide)**

   See above.

3. **Point of care ultrasound**

   See below

Consider the following **clinical settings**:

1. The hypothermic patient.

2. Trauma

3. Volume loss (from any cause)

4. Known DVT/PE - consider PE

5. Malignant disease - consider cardiac tamponade / PE

6. Renal failure /dialysis patient - **hyperkalemia, till proven otherwise**.

7. Overdose - consider sodium channel blockade, beta blockade, calcium channel blockade, digitalis toxicity.

Investigations

Urgent Potassium level on VBG:

This is the only relevant blood test that may be obtained in a timely to make some difference. Check for **hyperkalemia**.

Ultrasound:

Doppler ultrasound may assist in the detection of a pulse, (and so pseudo-PEA).

Bedside ultrasound / echocardiography - in the hands of the most experienced operator - is the best immediate imaging modality.

In general terms:

A **hyperdynamic LV** will tend to correlate with **narrow complex PEA,** and:

1. If the RV is "full" - consider:
    - Massive PE
2. If the RV is "collapsing" consider:
    - Cardiac tamponade - confirm by the presence of pericardial fluid.
    - Tension pneumothorax
    - Hyperinflation on mechanical ventilation

A **hypokinetic or akinetic LV** will tend to correlate with **wide complex PEA**, consider

1. Agonal rhythm
2. Cardiogenic shock
3. Hyperkalemia
4. Sodium channel blockade

Management

1 Initiate immediate BLS and ALS.

- Consider the clinical setting / what is known about the patient

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2. Check **cardiac rhythm** on ECG - **narrow** versus **wide:**

Narrow QRS - give fluids, via a pump set.

Ultrasound:

- **Full RV & hyperdynamic LV**

Consider **massive PE**:

50 mg tPA administered as a rapid IV push over 1 minute while CPR is ongoing.

The *timely* administration of 50 mg of tPA over 1 minute in patients with PEA due to confirmed PE is safe and effective leading to restoration of spontaneous circulation in the majority of such patients.

Subsequently 5000 units of heparin is given as an IV bolus, and the patient is started on an initial maintenance infusion of heparin at 10 U/kg per hour.

Despite chest compressions and other invasive manoeuvres during CPR, bleeding complications during the PEAPETT trial were surprisingly minimal.

- **RV is "collapsing" & hyperdynamic LV** consider:
  - ♥ Cardiac tamponade - needle aspiration
  - ♥ Tension pneumothorax - Needle decompression / finger thoracostomy.
  - ♥ Hyperinflation on mechanical ventilation - disconnect from the ventilator and adjust ventilator settings.

Wide QRS

Ultrasound:

- **Hypokinetic / akinetic LV** consider:
  - ♥ **Calcium chloride** for **hyperkalemia**
  - ♥ **Sodium bicarbonate** for sodium channel poisoning

Agonal or slow idioventricular type rhythms in the presence of severe hypoxic / metabolic injury, usually due to massive myocardial infarction, have a prognosis of virtually zero.

- This situation should be treated as for **asystole.**
- Calcium has been advocated, but there is no good evidence for its effectiveness, **unless the cause is hyperkalemia**.
- Cardiac pacing is ineffective

References

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