

A New Paradigm of Severe Pressure Injury Formation

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Disclosures

- CMO of Intellicure, Inc
- Executive Director of the US Wound Registry

(A pandemic is a bad time for your computer camera to break so pretend you can see me.)



Injury vs. Ulcer terminology in this talk



I really am trying to call these injuries but it's hard when there is a booboo.

“Apply dressing to injury bed . . .”

NPIAP Pressure Injury Definition

Pressure Injury:

A pressure injury is **localized damage** to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can **present** as intact skin or an open ulcer and may be painful. The injury **occurs as a result of intense and/or prolonged pressure** or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, co-morbidities and condition of the soft tissue.

**The tissue damage is caused by (intense and prolonged)
LOCAL pressure.**

Here's a localized area of pressure



Localized tissue damage . . .from
intense or prolonged pressure

The same patient had other
pressure injuries after surgery.

The Angiosomal Theory of Pressure Injury



On the Origin of Intraoperative Pressure Injury: An Angiosomal Theory of Pressure Injury Formation

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We review a case of a 22-year-old healthy man who underwent a 5-h maxillofacial surgery while continuously supine with foam pads placed prophylactically over elevated heels. Immediately after surgery, Stage 1 pressure injuries appeared on the left lateral heel and right lateral ankle, despite the absence of local pressure to these areas. Both lesions eventually resolved. Eight months later, a Doppler evaluation was performed of the patient's lower extremities, the peroneal artery and its tributaries were marked, and the intraoperative positioning was simulated to determine if a wedge at the back of the calf could have obstructed blood flow in these vessels. In this position, the feet naturally abducted so that the lateral calcaneal and posterior malleolar arteries became positioned immediately underneath the wedge. We propose a vascular mechanism of pressure injury development, postulating that some heel pressure injuries are not the result of localized pressure but rather angiosomal ischemia, based on the observation that the anatomical pattern of these lesions frequently follow the distribution of a named vessel. We hypothesize that in this case, intraoperative positioning along with permissive hypotension may have occluded arterial or venous flow to the relevant angiosomes, causing an ischemia reperfusion injury to the downstream tissues.

Keywords: angiosomes, deep tissue injury, pressure injury, ischemia reperfusion, pressure ulcer



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22-year-old healthy man who underwent a 5-h maxillofacial surgery while continuously supine with foam pads placed prophylactically over elevated heels.

Immediately after surgery, Stage 1 pressure injuries appeared on the left lateral heel and right lateral ankle, **despite the absence of local pressure to these areas since the patient was supine throughout surgery.**

Both lesions eventually resolved.

Left Lat Heel and Right Lat Malleolar PI – after 5 hours in the OR . . . while SUPINE

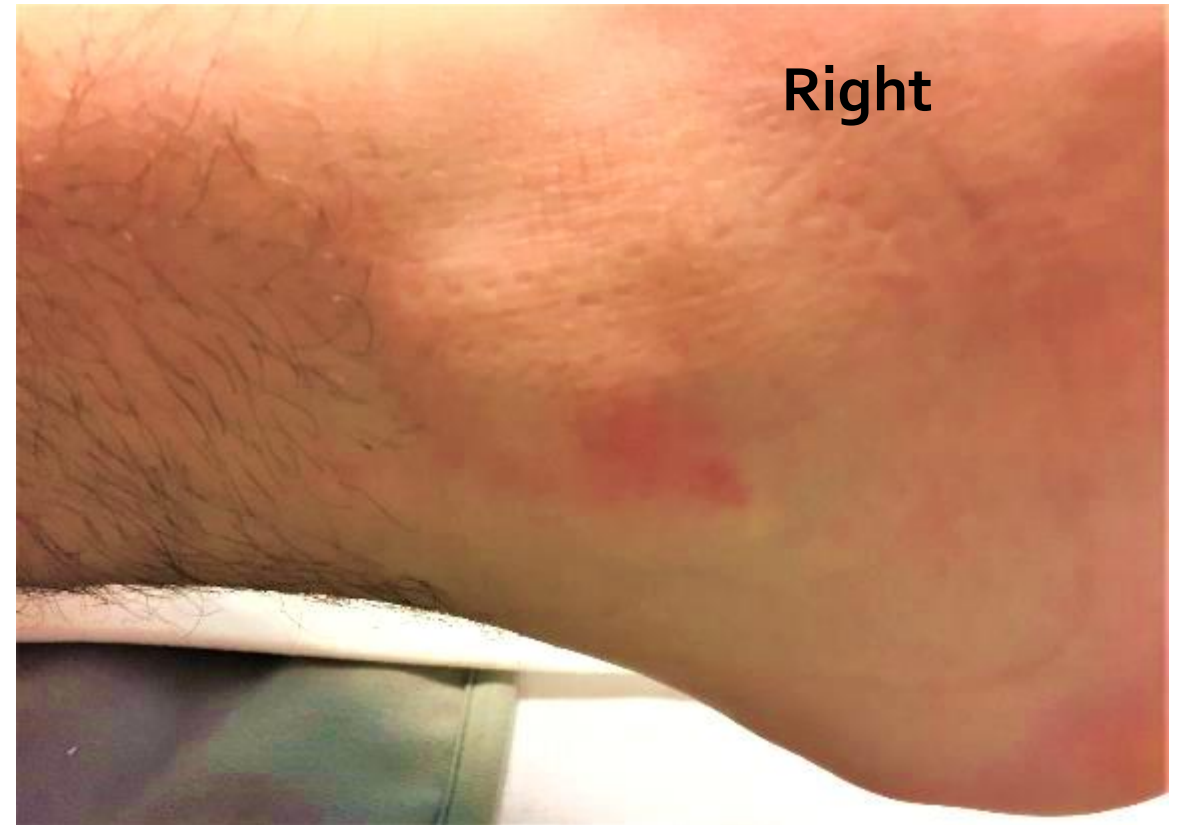


Figure 1. An excruciatingly painful, but blanching, pressure injury on the *left* lateral heel pictured here immediately after a maxillofacial operation. Note that a foam pad placed prophylactically before surgery was removed so that the heel can be examined.

How did he get lateral ulcers while supine?



Left **lateral calcaneal artery depicted in red ink** with it's vascular territory; **sural nerve in blue**, fibula in black



Look what happens when the feet abduct over a heel wedge.

How did he get lateral ulcers while supine?



The **right lateral malleolar artery** in red;
the location of the **ankle pressure injury** in
brown, and **fibula** in black.



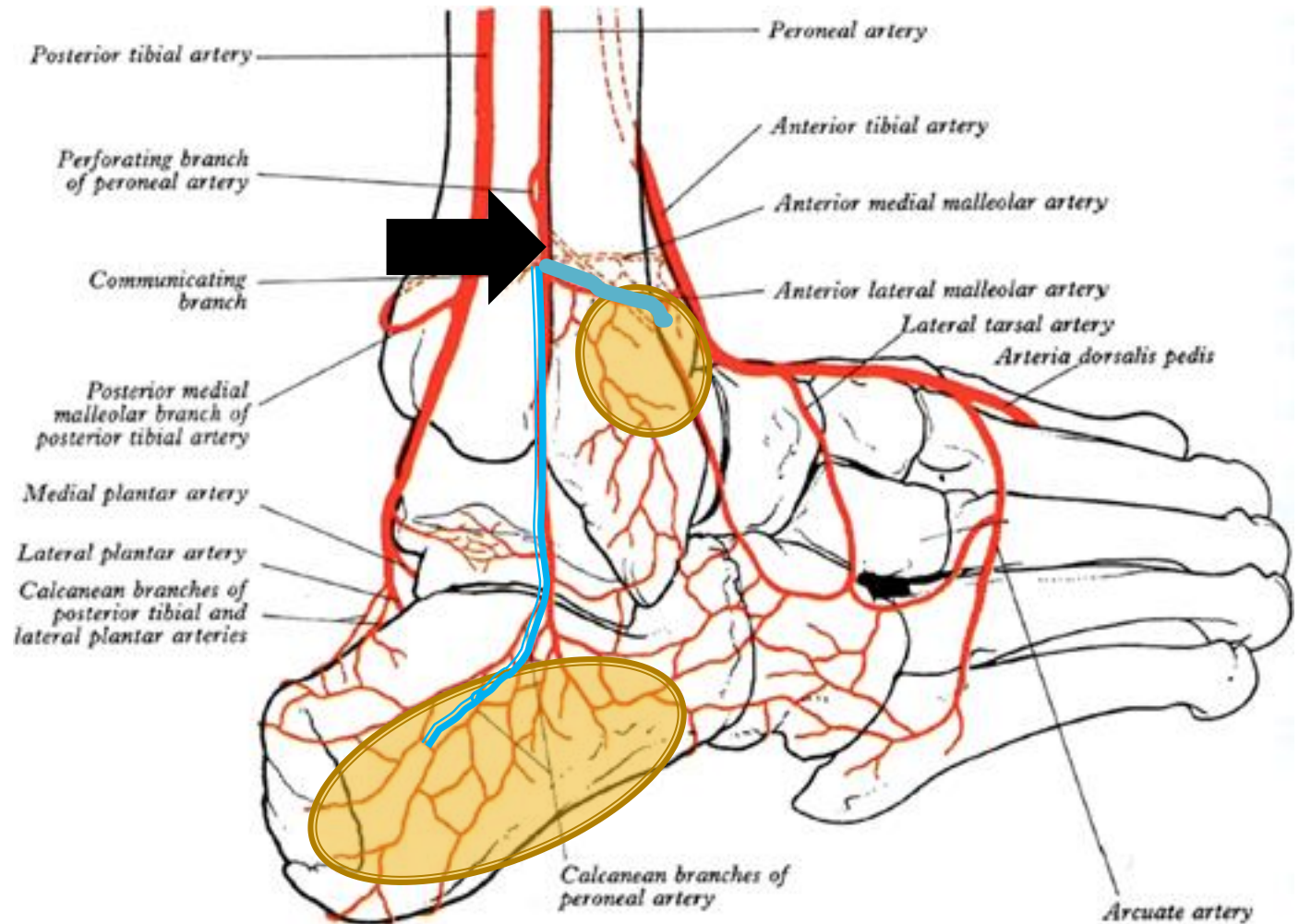
Look what happens when the feet
abduct over a heel wedge.

The “Angiosome” Mechanism

A wedge at the posterior Achilles would put pressure on the calcaneal and/or the lateral malleolar branches of the peroneal artery.

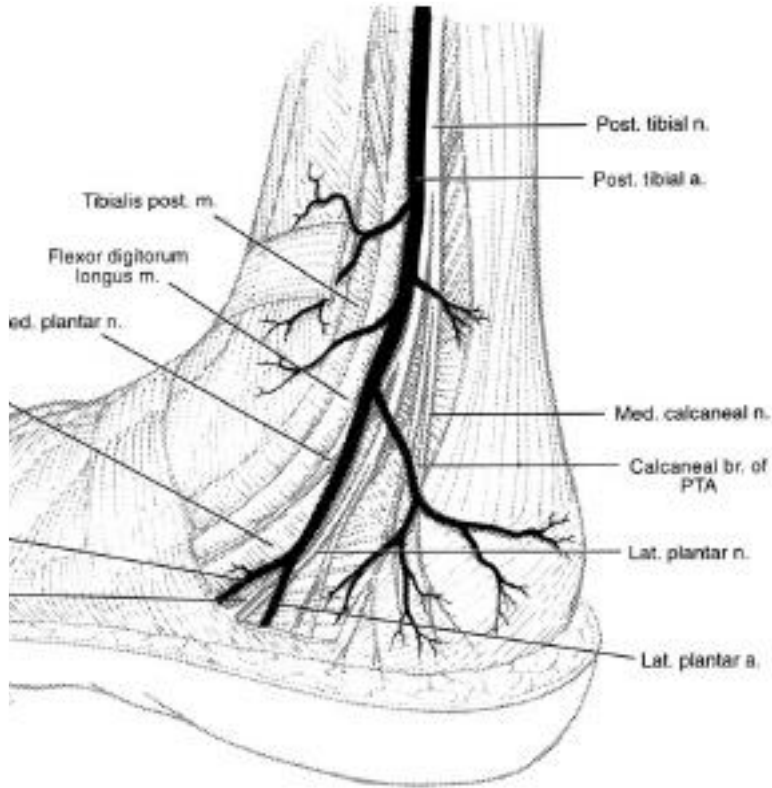
It caused an **ischemia reperfusion injury** downstream.

These PI's were not due to local pressure, but occlusion of the vascular territory of a named vessel.



Source: R.S. Dieter, R.A. Dieter Jr., R.A. Dieter III:
Peripheral Arterial Disease, www.cardiology.mhmedical.com
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I started to notice that heels ulcers had vascular supply issues (Calcaneal branch of the Post tibial)



This looks like a heel
“pressure ulcer” doesn’t it?



*(Cadaver Picture Provided
by Dr. C. Attinger M.D.)*

Most heel “pressure ulcers” are manifestations of ischemia of the calcaneal branch of the posterior tibial artery.

Pressure Injury of the Buttock *Cheeks* (?)

- 48 yo morbidly obese (BMI 42) WM
- PMHx: CAD, Left ventricular dysfunction, AMI, HTN, sleep apnea, anemia, ischemic cardiomyopathy
- CABG 5 .5 hrs:
 - MAP ~ 50 mmHg
 - Acute blood loss anemia
 - Hypotensive in ICU (78/44) for >12 hours on vasopressors
- Post op day #2, large purple discoloration noted over buttocks



What are these “holes”?

- 18 days post CABG surgically debrided to reduce the risk of colonization because his sternum was open.
 - Intraoperatively, plastics noted hematomas on either side of the sacrum.
 - A 2.4 cm tunnel ran along the sacroiliac ligament on either side which persisted for 12 weeks
- He remained insensate over the buttock cheeks bilaterally, indicating that sensory nerves were permanently affected

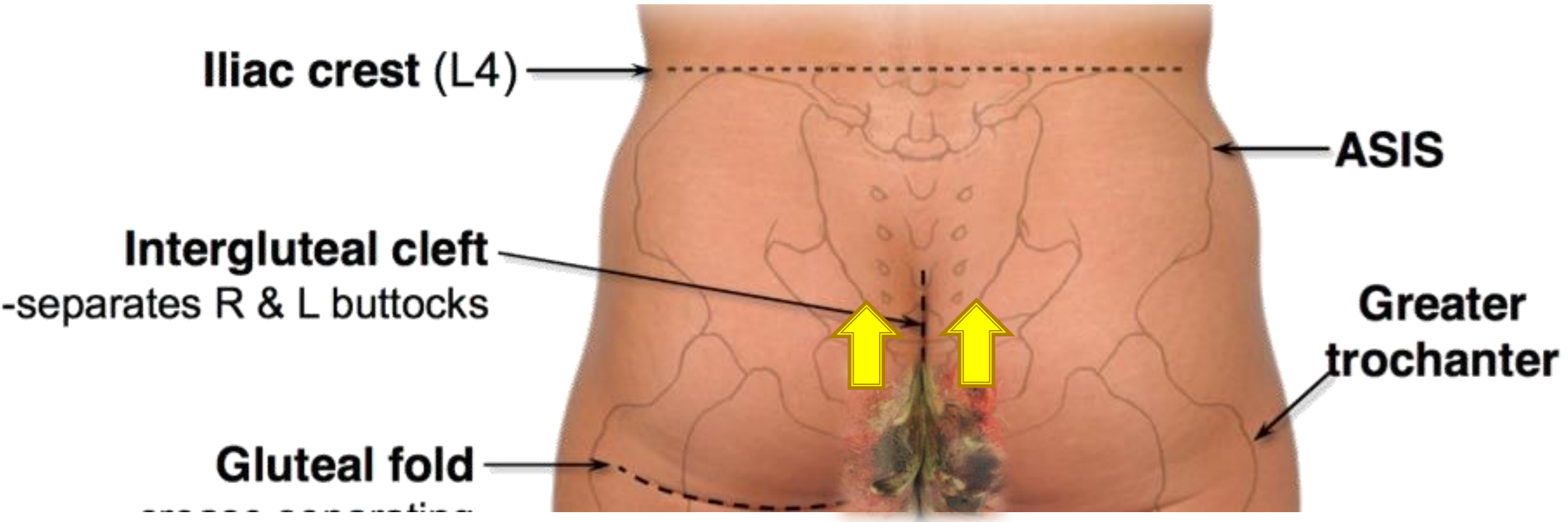


This PI was not from LOCAL pressure

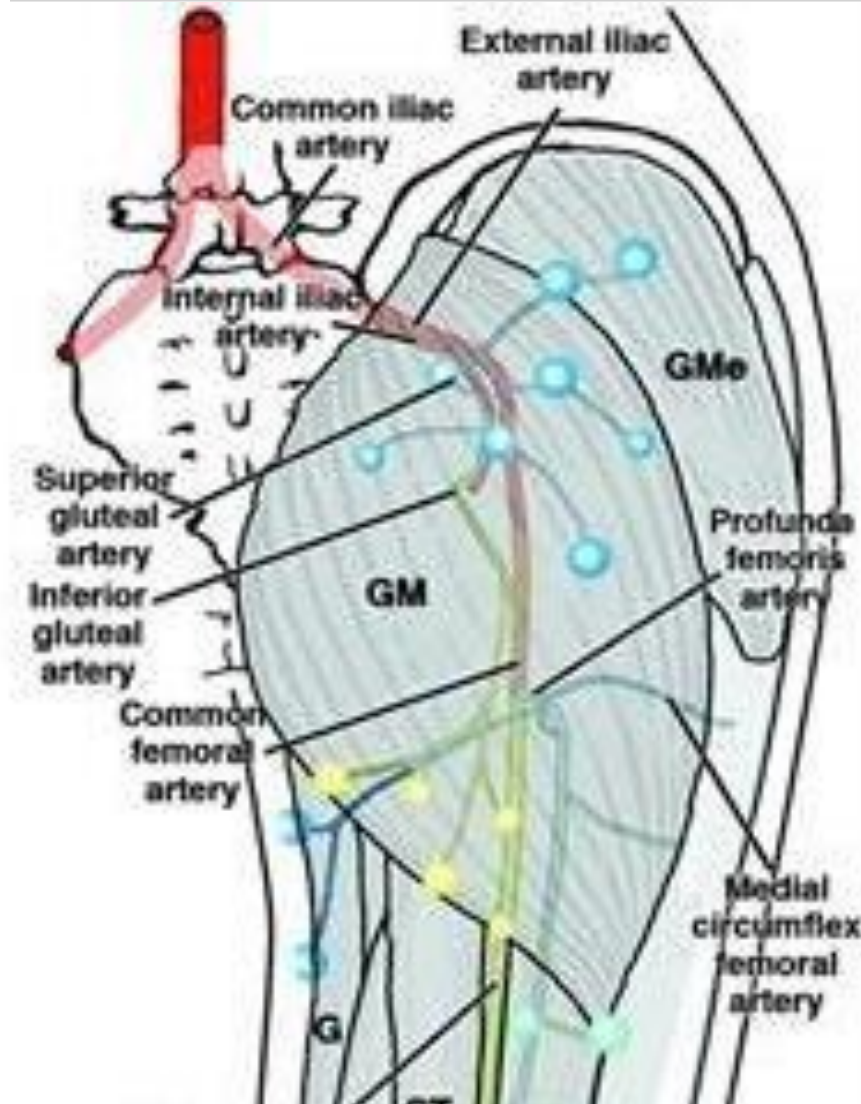
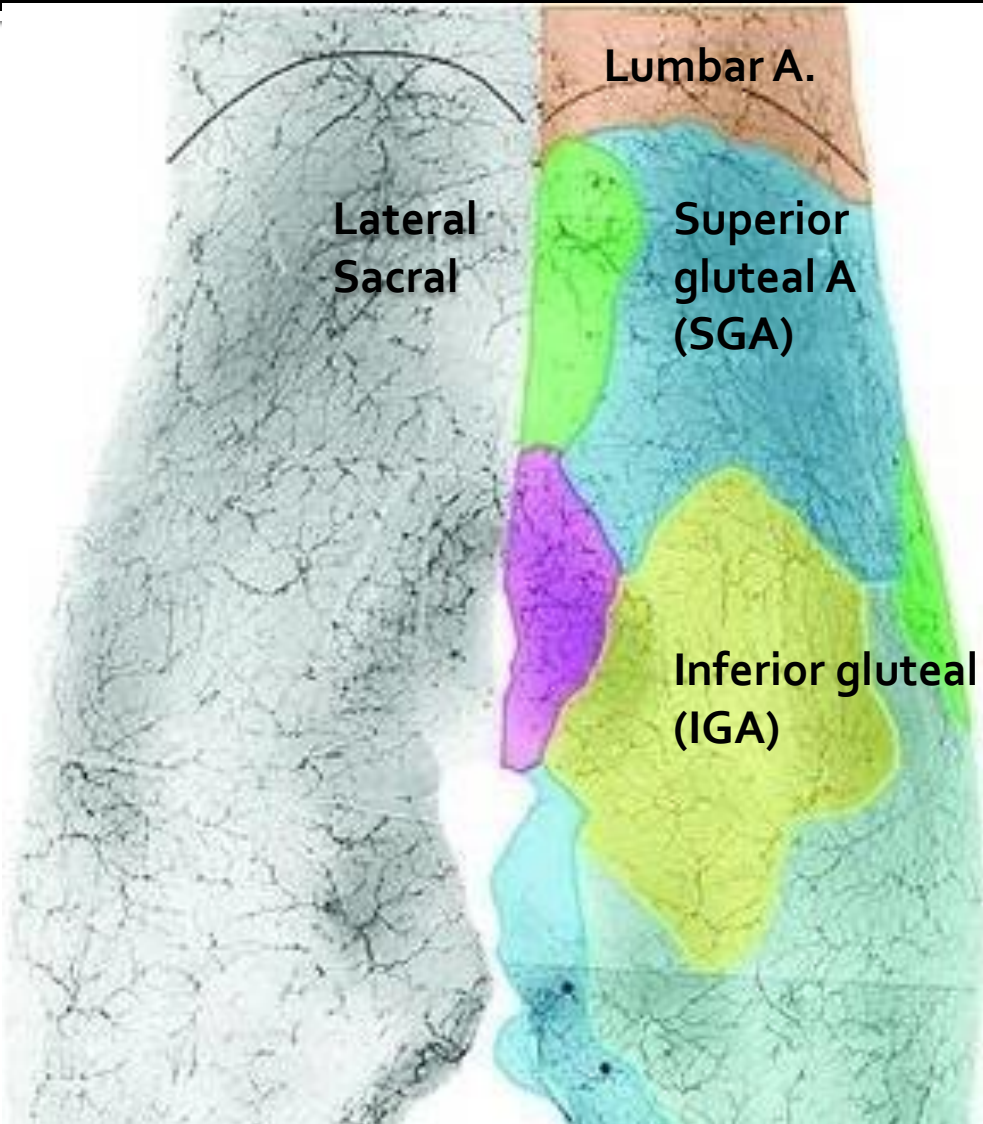


Arrows show where the vessel originates that supplies the buttock cheeks

This buttock DTI is due to compression against WHICH bones?



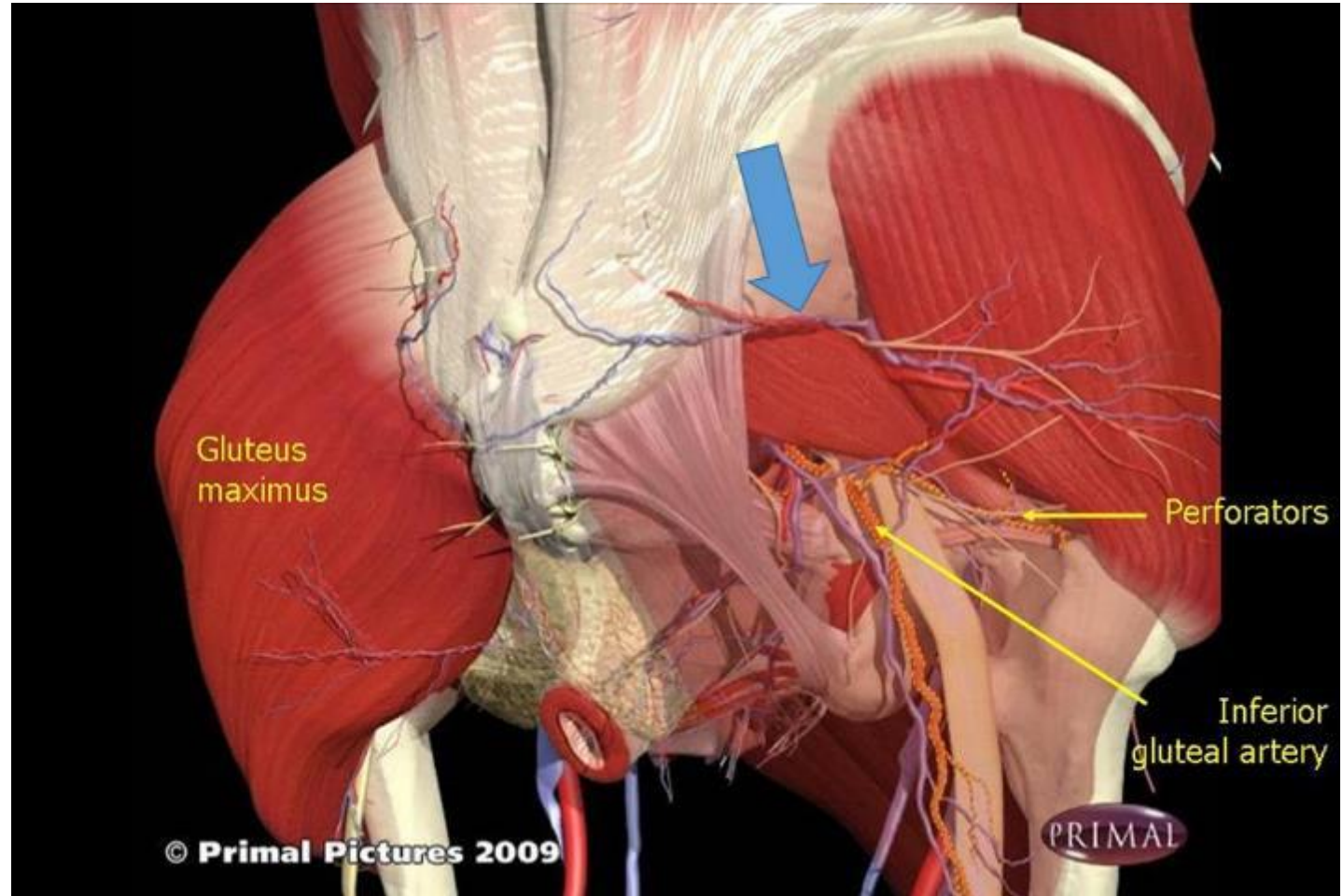
Territorial supply to the buttock and hip



The blue dots are perforators that "perforate" the muscle and fascia

Why do these vessels get occluded?

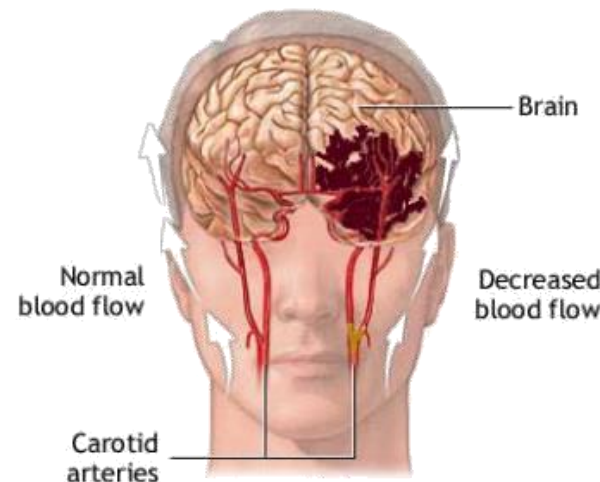
Here's where the Superior Gluteal Artery exits superior to the piriformis muscle – a really tight spot.



Factors associated with DTIs and Stage 4 PIs

- Low mean arterial pressure
- Low cardiac output
- Low albumin (Low oncotic pressure-interstitial edema?)
- Vascular disease
- Vasopressors
- Fever
- Arterial hypoxemia
- Decreased oxygen carrying capacity
- Anemia

- 48 yo morbidly obese (BMI 42) WM
- PMHx: CAD, Left ventricular dysfunction, AMI, HTN, sleep apnea, anemia, ischemic cardiomyopathy
- CABG 5 .5 hrs:
 - MAP ~ 50 mmHg
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**That sounds like
ischemia/infarction
to me . . .**

Angiosome Maps, used for >50 years

British Journal of Plastic Surgery (1987), 40, 113–141
© 1987 The Trustees of British Association of Plastic Surgeons

The vascular territories (angiosomes) of the body: experimental study and clinical applications

G. I. TAYLOR and J. H. PALMER

Department of Plastic and Reconstructive Surgery, Royal Melbourne Hospital and Department of Anatomy, University of Melbourne

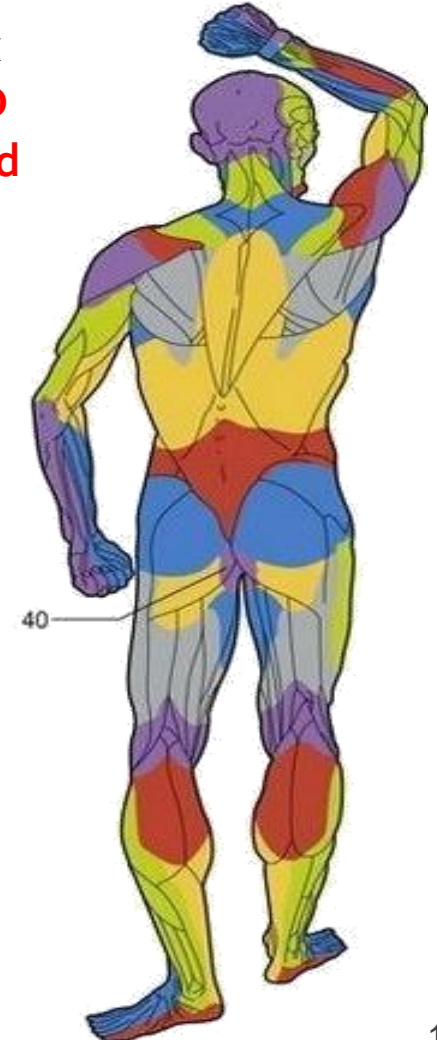
Summary—The blood supply to the skin and underlying tissues was investigated by ink injection studies, dissection, perforator mapping and radiographic analysis of fresh cadavers and isolated limbs. The results were correlated with previous regional studies done in this department.

The blood supply is shown to be a continuous three-dimensional network of vessels not only in the skin but in all tissue layers. The anatomical territory of a source artery in the skin and deep tissues was found to correspond in most cases, giving rise to the angiosome concept.

Arteries follow closely the connective tissue framework of the body. The primary supply to the skin is by direct cutaneous arteries which vary in calibre, length and density in different regions. This primary supply is reinforced by numerous small indirect vessels, which are "spent" terminal branches of arteries supplying the deep tissues.

An average of 374 major perforators was plotted in each subject, revealing that there are still many more potential skin flaps. Our arterial roadmap of the body provides the basis for the logical planning of incisions and flaps. The angiosomes defined the tissues available for composite transfer.

The body is a patchwork quilt of angiosomes- **3-D blocks of tissue supplied by a named vessel.**



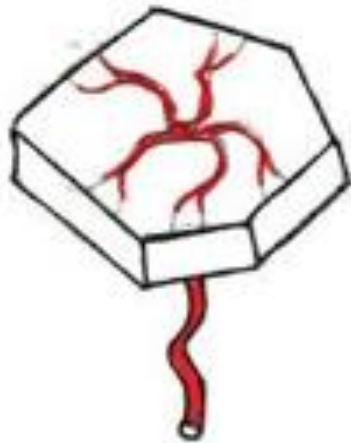
<https://plasticsurgerykey.com/vascular-territories/>

<https://ajops.com/index.php/ajops/article/download/38/197>

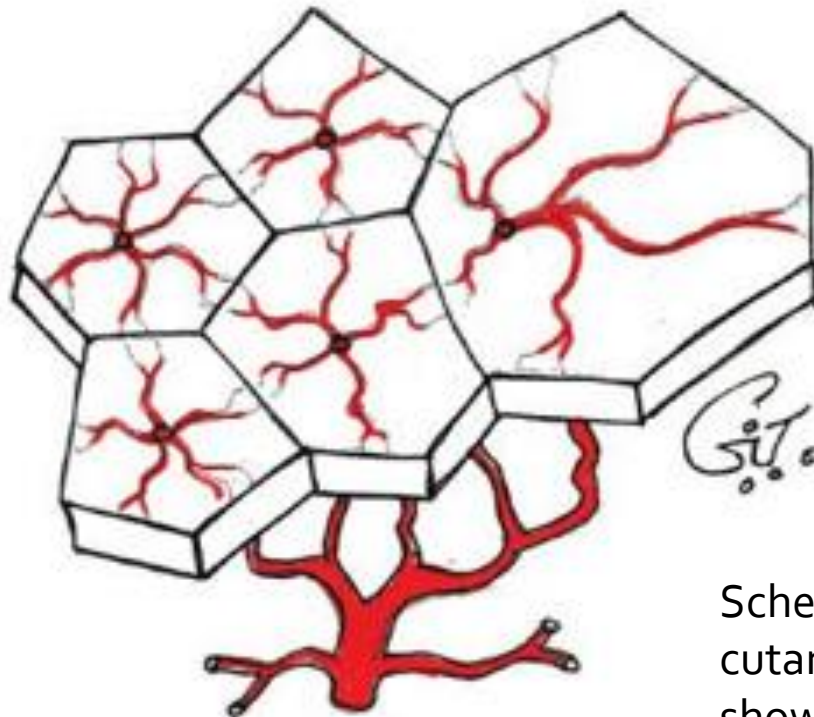
3-D block of tissue – supplied by a named vessel



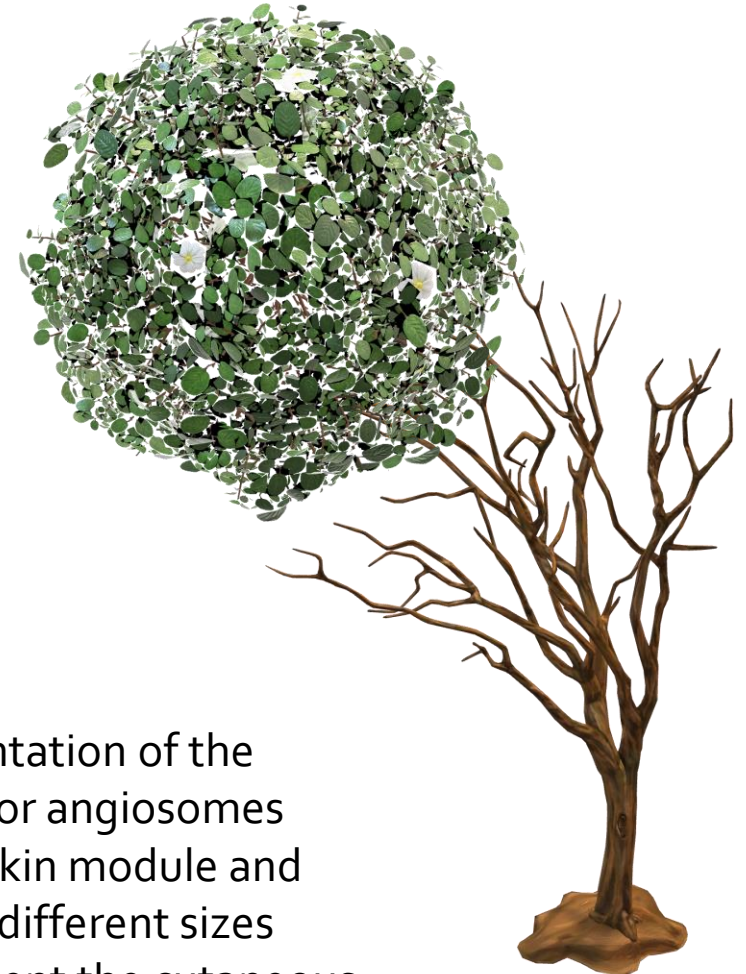
3-D block of tissue – supplied by a named vessel



Angiosome territory
of a
cutaneous perforator



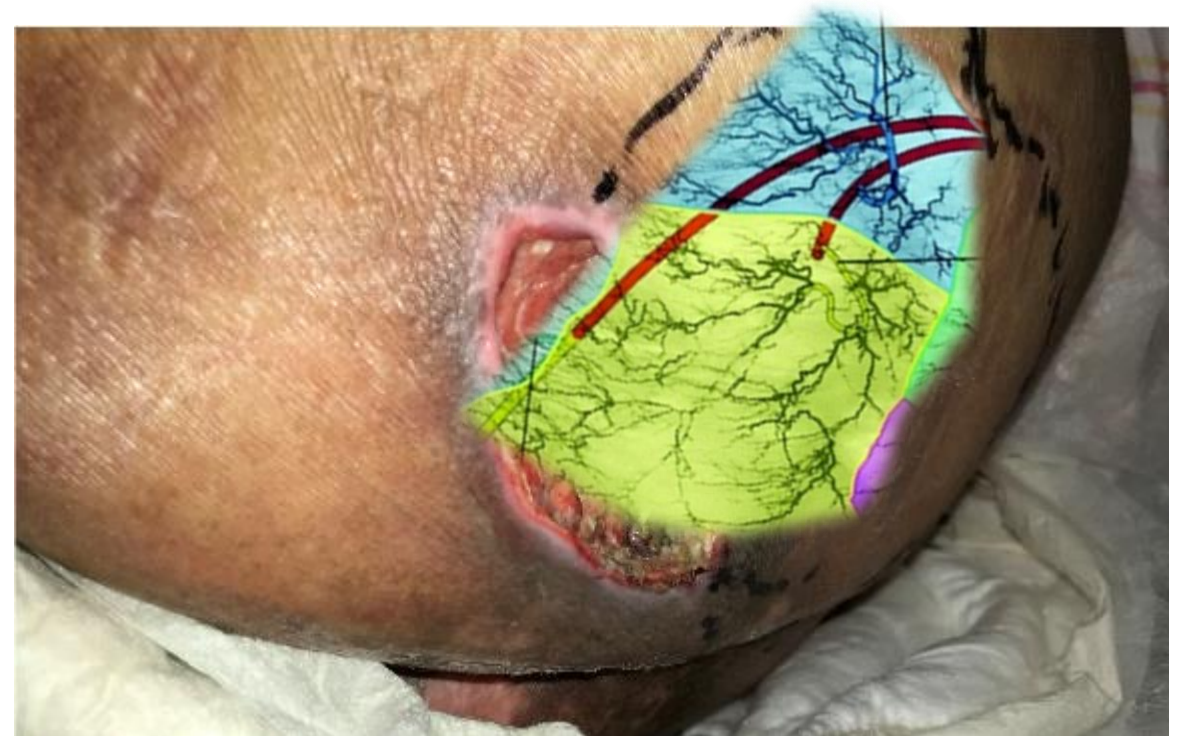
Angiosome skin territory
of a
source artery



Schematic representation of the cutaneous perforator angiosomes showing the basic skin module and several modules of different sizes combined to represent the cutaneous territory of a source artery (*right*).

Angiosome infarction explains these defects

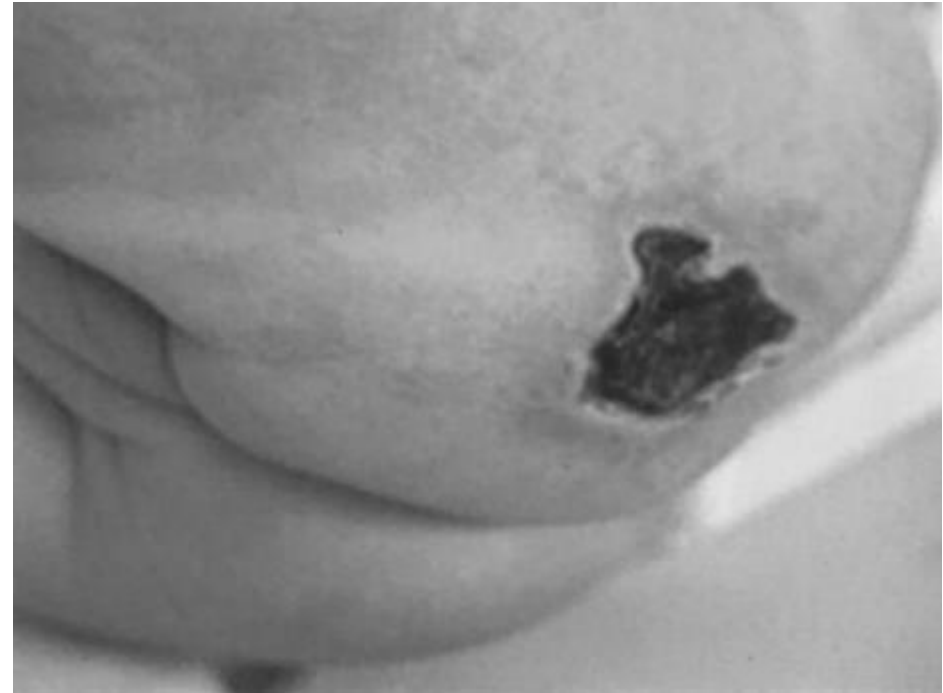
There is no possible way that local pressure could result in a tissue defect of this magnitude. This massive lesion is due to the loss of a very large muscle group and the associated soft tissue and skin – all supplied by the inferior gluteal artery.



It's been right in front of us all along



I started reviewing all my Stage 4 Pressure Ulcer photos . . . This is an Inferior gluteal artery infarction.



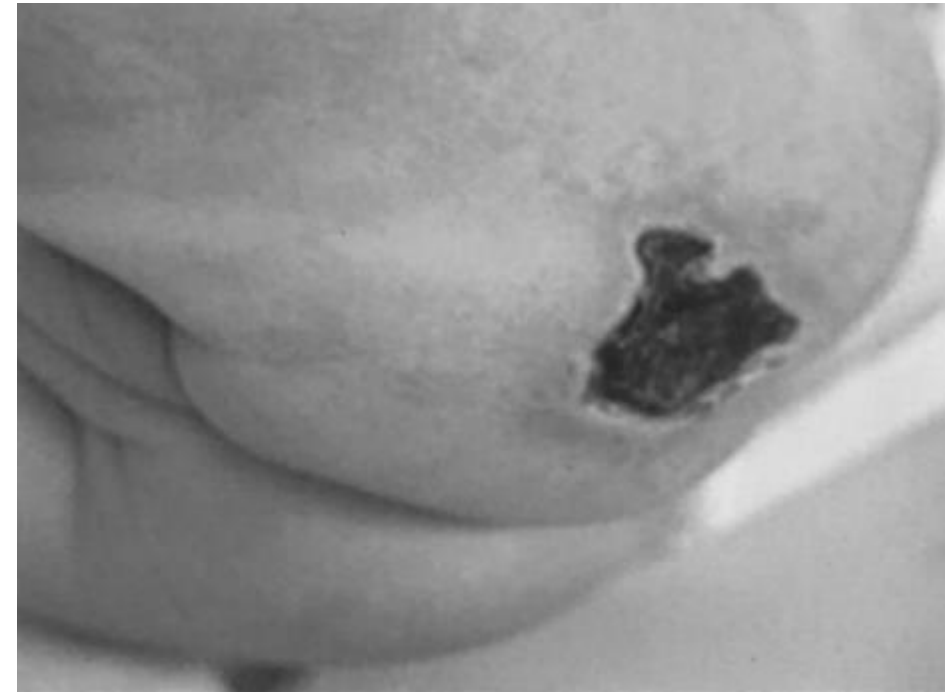
This is an Inferior gluteal artery infarction in a neonate after umbilical artery catheterization.

It's been right in front of us all along

- Full term infant that underwent umbilical artery catheterization while being treated for respiratory distress.
- Initially the catheter tip was displaced into the inferior gluteal artery which supplies the lower aspect of the gluteus.

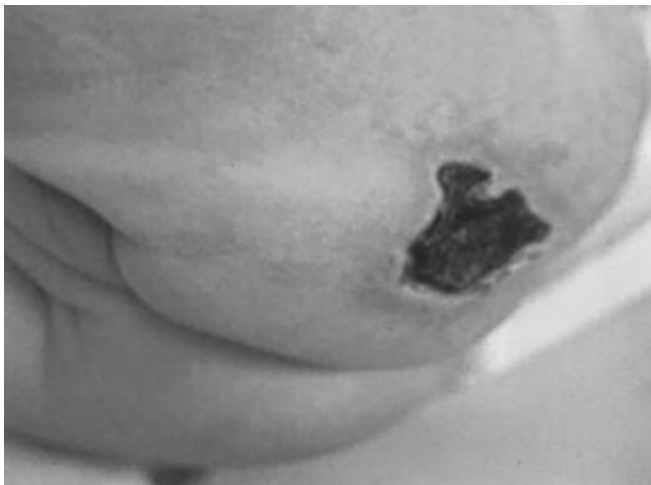
“Three days after umbilical arterial catheterization, **bruising** was observed over the left gluteal region. . . .the bruised skin and underlying tissues . . .developed central necrosis surrounded by a rim of dark red skin, which in turn was sharply demarcated from normal skin by a narrow pale zone.”

- The authors postulated that the gangrenous changes were probably caused by vascular occlusion resulting from catheter-induced vasospasm of the inferior gluteal artery.

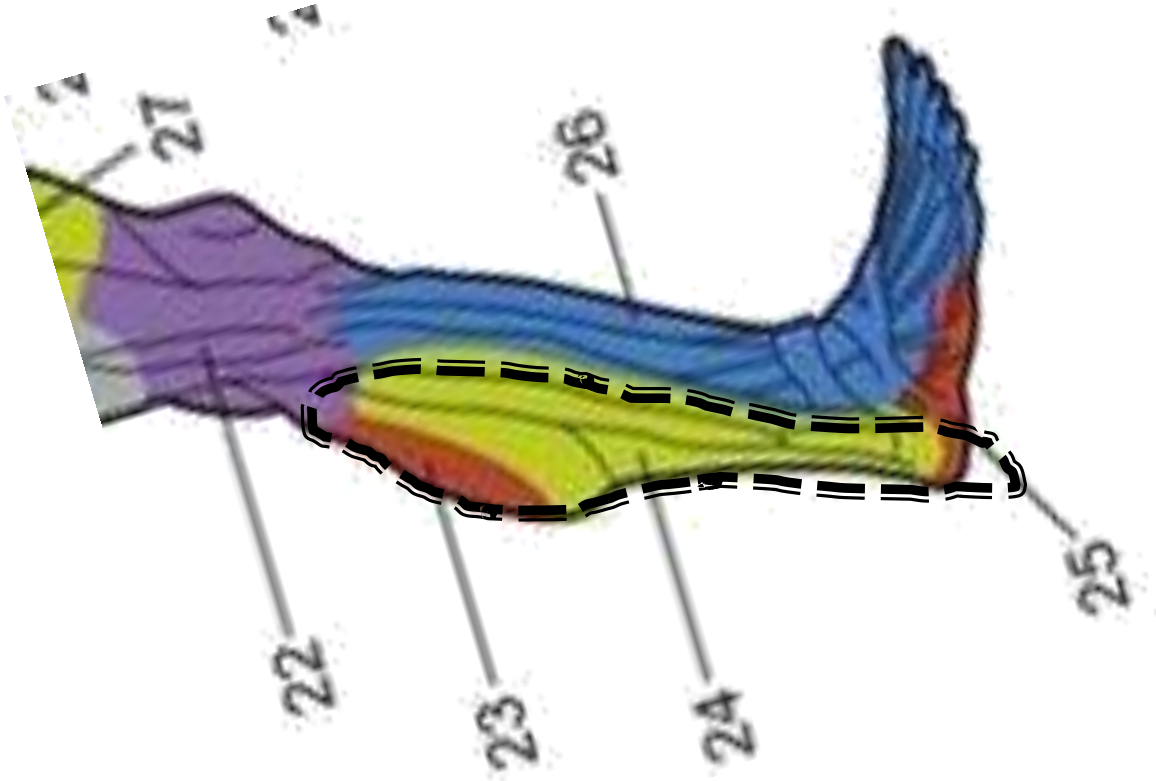


It's been right in front of us all along

“Three days after umbilical arterial catheterization, **bruising** was observed over the left gluteal region. . . .the bruised skin and underlying tissues . . .developed central necrosis surrounded by a rim of dark red skin, which in turn was sharply demarcated from normal skin by a narrow pale zone.”



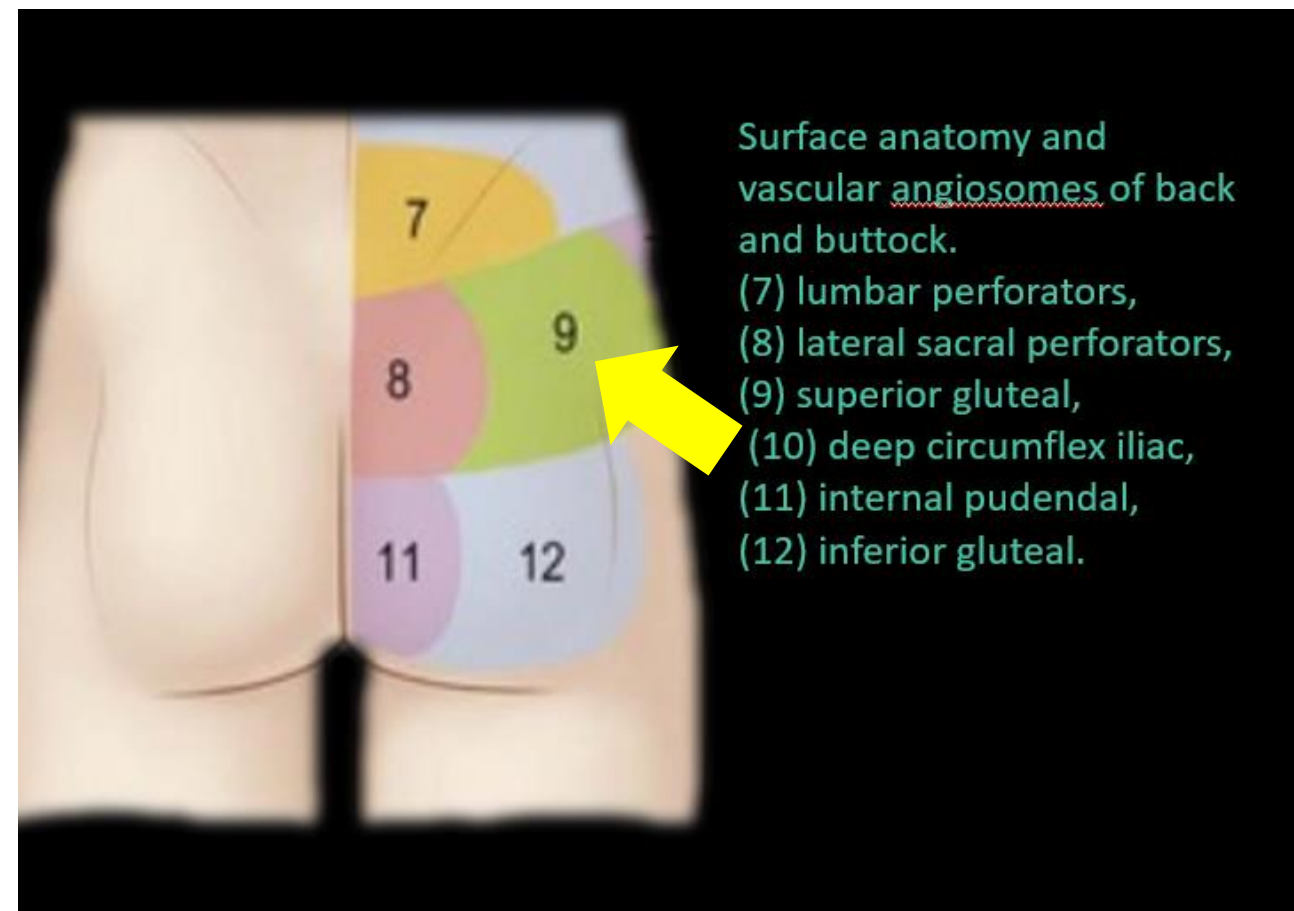
My photographic challenge to you- find a DTI or Stage 4 that is NOT a vascular territory



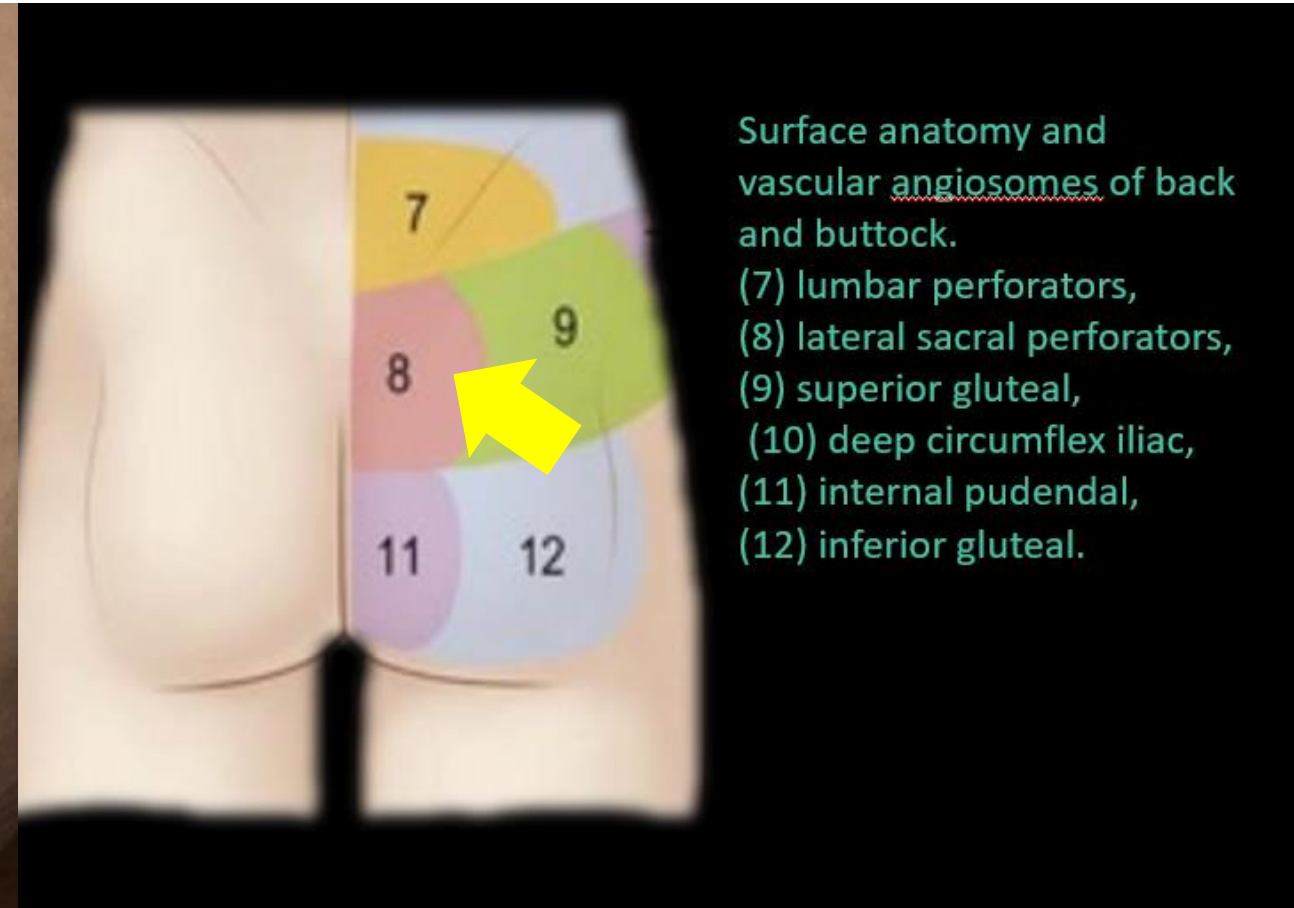
This is the angiosome of the superior gluteal a.



Superior Gluteal artery perforators



This is the angiosome of the lateral sacral a.



Surface anatomy and vascular angiosomes of back and buttock.

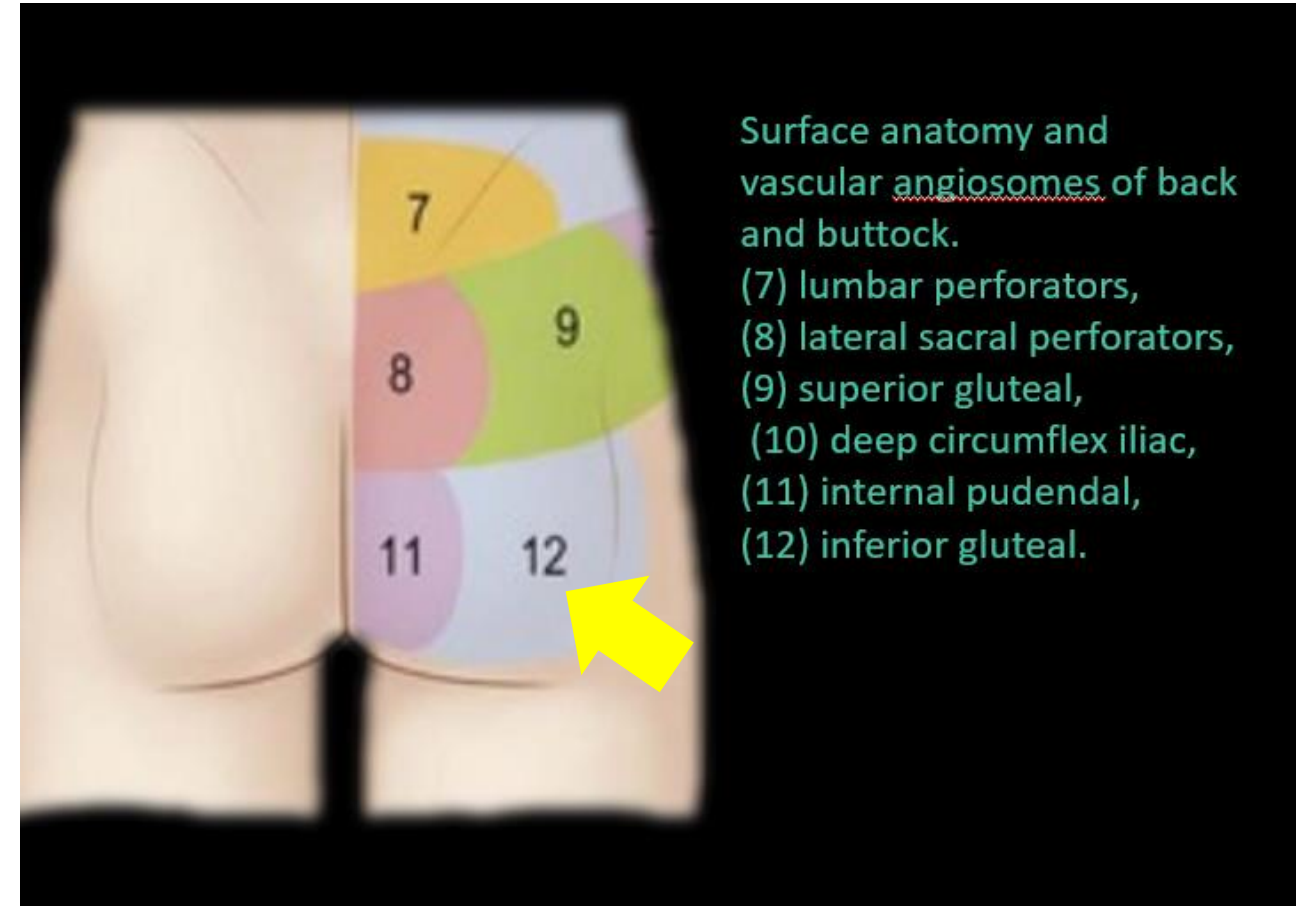
(7) lumbar perforators,
(8) lateral sacral perforators,
(9) superior gluteal,
(10) deep circumflex iliac,
(11) internal pudendal,
(12) inferior gluteal.

The perforators of the lateral sacral artery

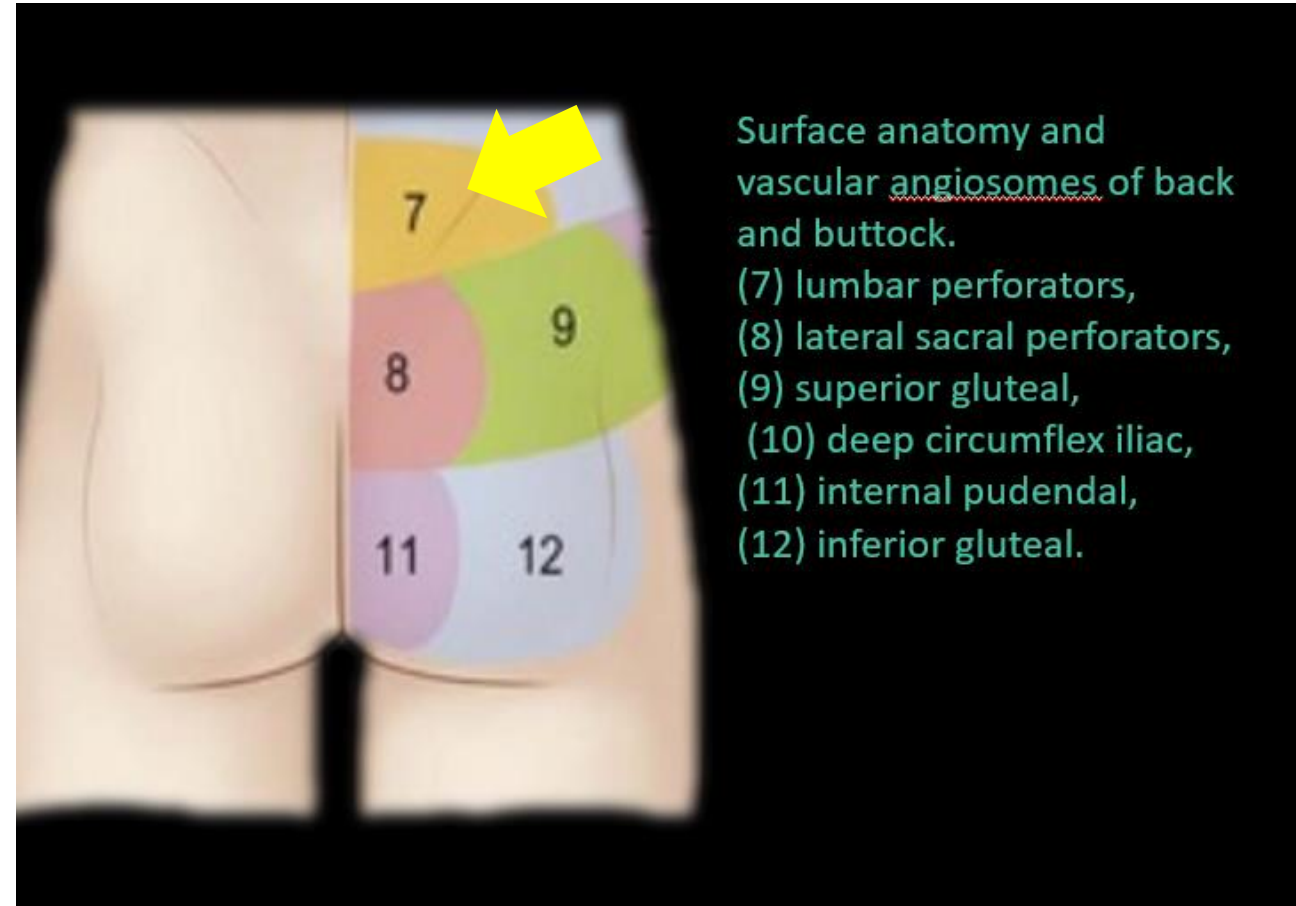
This is the angiosome of the inferior gluteal a.



The perforators of the inferior gluteal a.



This is the angiosome of the lumbar a.

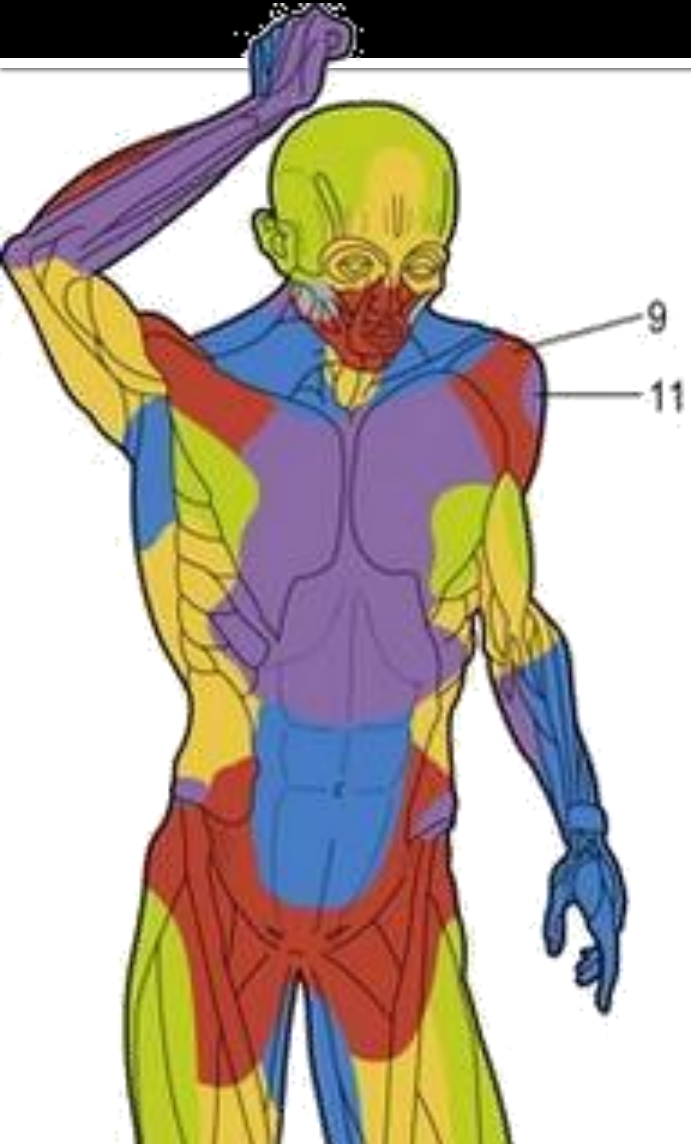


Surface anatomy and vascular angiosomes of back and buttock.

(7) lumbar perforators,
(8) lateral sacral perforators,
(9) superior gluteal,
(10) deep circumflex iliac,
(11) internal pudendal,
(12) inferior gluteal.

A lumbar perforators pressure injury

This is an angiosome



This is an angiosome



THIS is an Angiosomal infarction . . .



Dora the Explorer has a Stage 2: Due to friction and sheer



7 y.o. girl with spina bifida who gets wounds on her knees from crawling without her knee pads



These are "pothole" –
OUTSIDE to inside injuries.

Are these Stage 4 PIs the same process or a different mechanism?



84 y.o. woman who develops pressure injuries after one night on a hard mattress. These are better explained as INSIDE to outside injuries of a vascular territory (sinkholes).

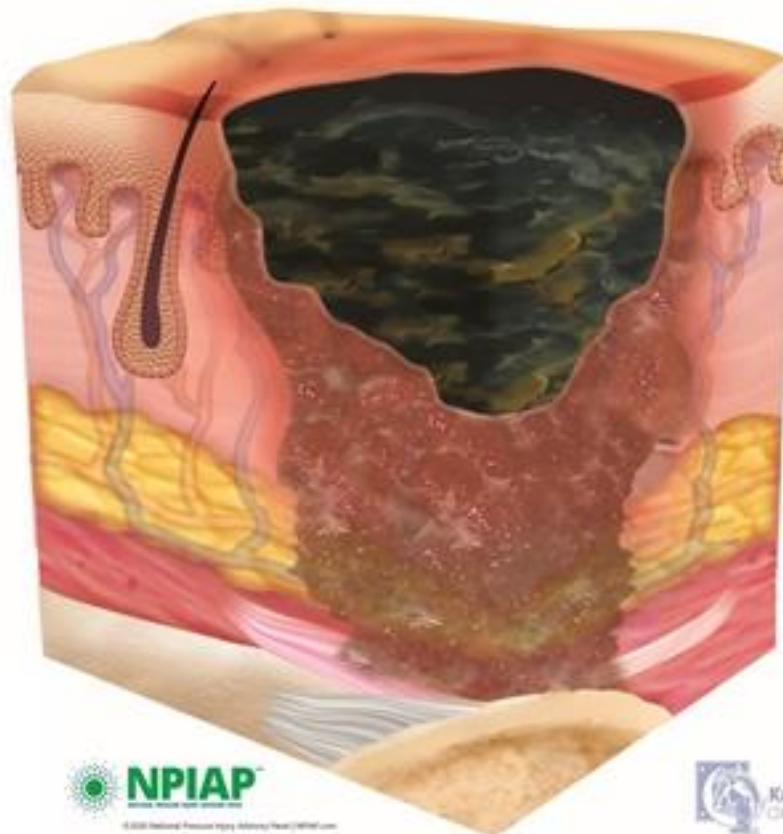
Pressure Ulcer/Injury Stages . . .

. . .classify them according to the ***amount of visible tissue loss based on visual inspection.***

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https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/position_statements/npuap-position-statement-on-.pdf

Stage 4 Pressure Injury



“The numerical staging system does NOT imply linear progression of pressure injuries from Stage 1 through Stage 4 . . .”

(If you think you saw this PI progress through the stages from the OUTSIDE to the inside, you must be mistaken.)

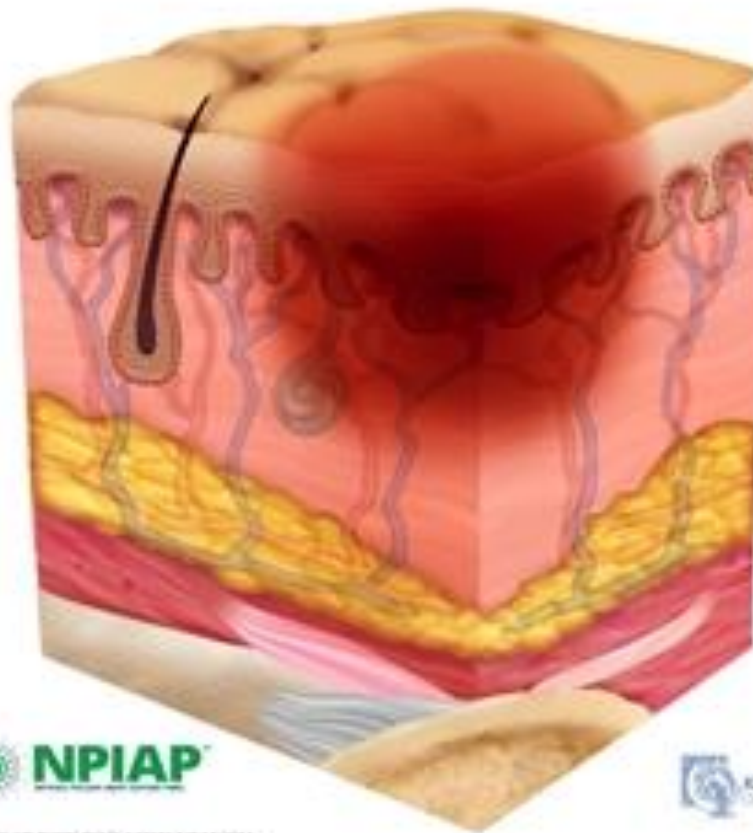
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Stage 1 Pressure Injury - Lightly Pigmented

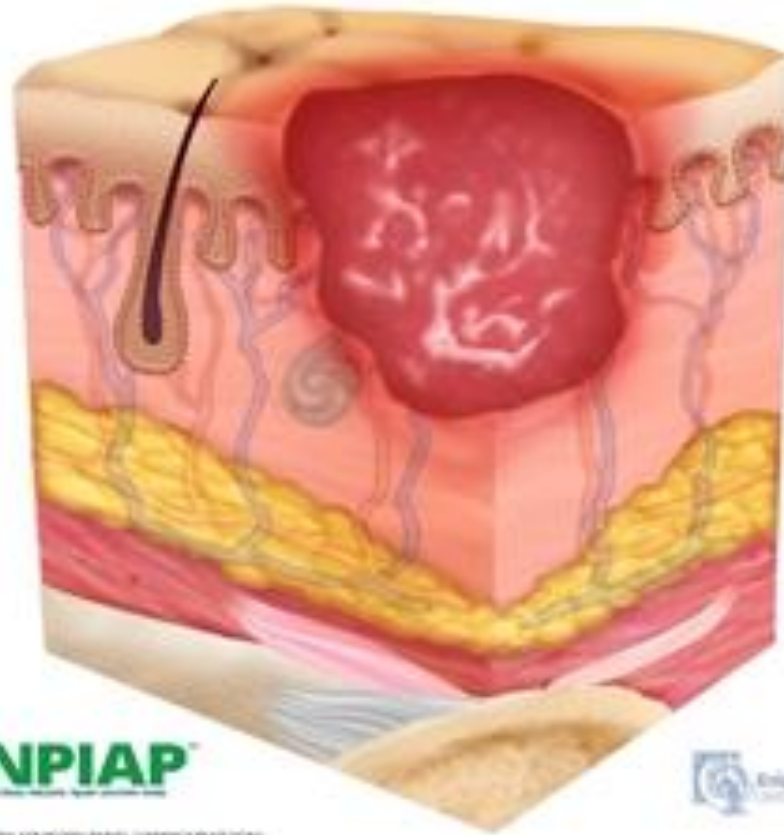


“The numerical staging system does NOT imply linear progression of pressure injuries from Stage 1 through Stage 4 . . .”

(If you think you saw this PI progress through the stages from the OUTSIDE to the inside, you must be mistaken.)

Pressure Ulcer/Injury Stages . . .

Stage 2 Pressure Injury



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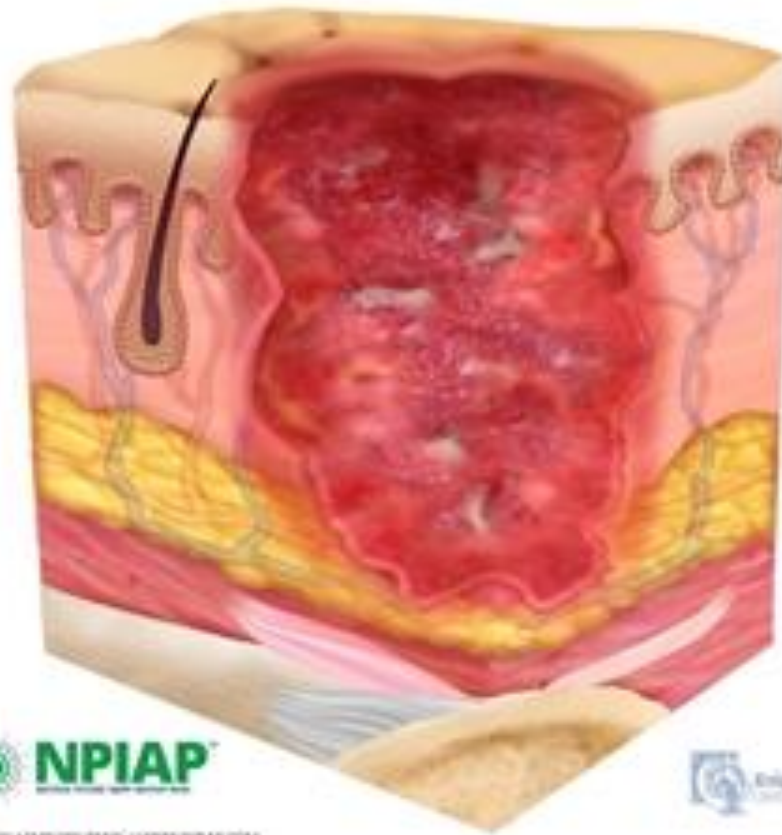


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Pressure Ulcer/Injury Stages . . .

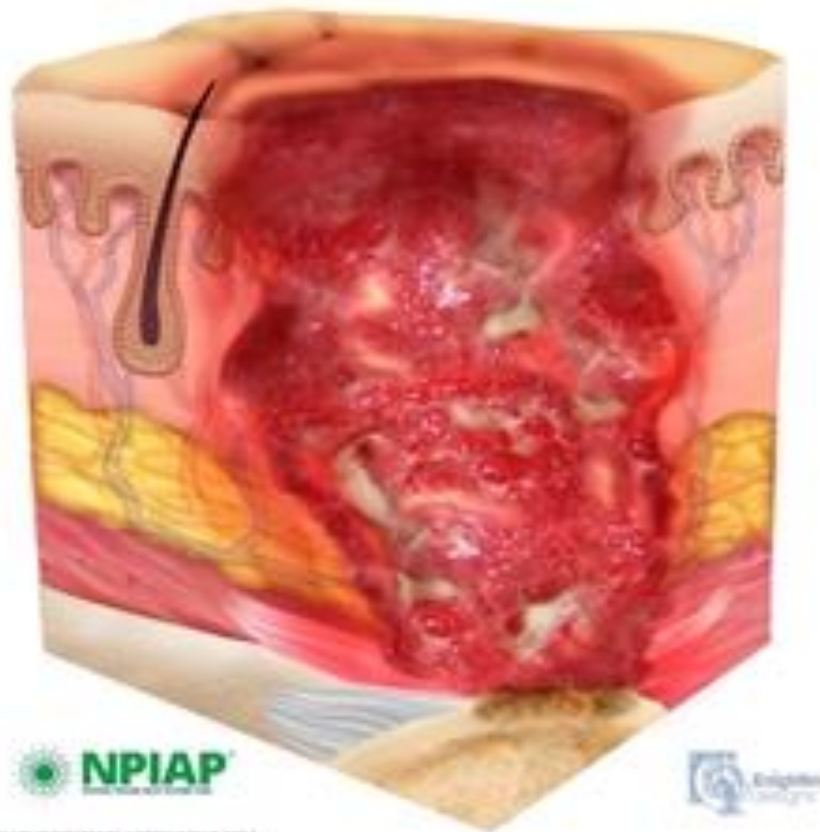
Stage 3 Pressure Injury



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Pressure Ulcer/Injury Stages . . .

Stage 4 Pressure Injury



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Pressure Ulcer/Injury Stages . . .

Stage 4 Pressure Injury

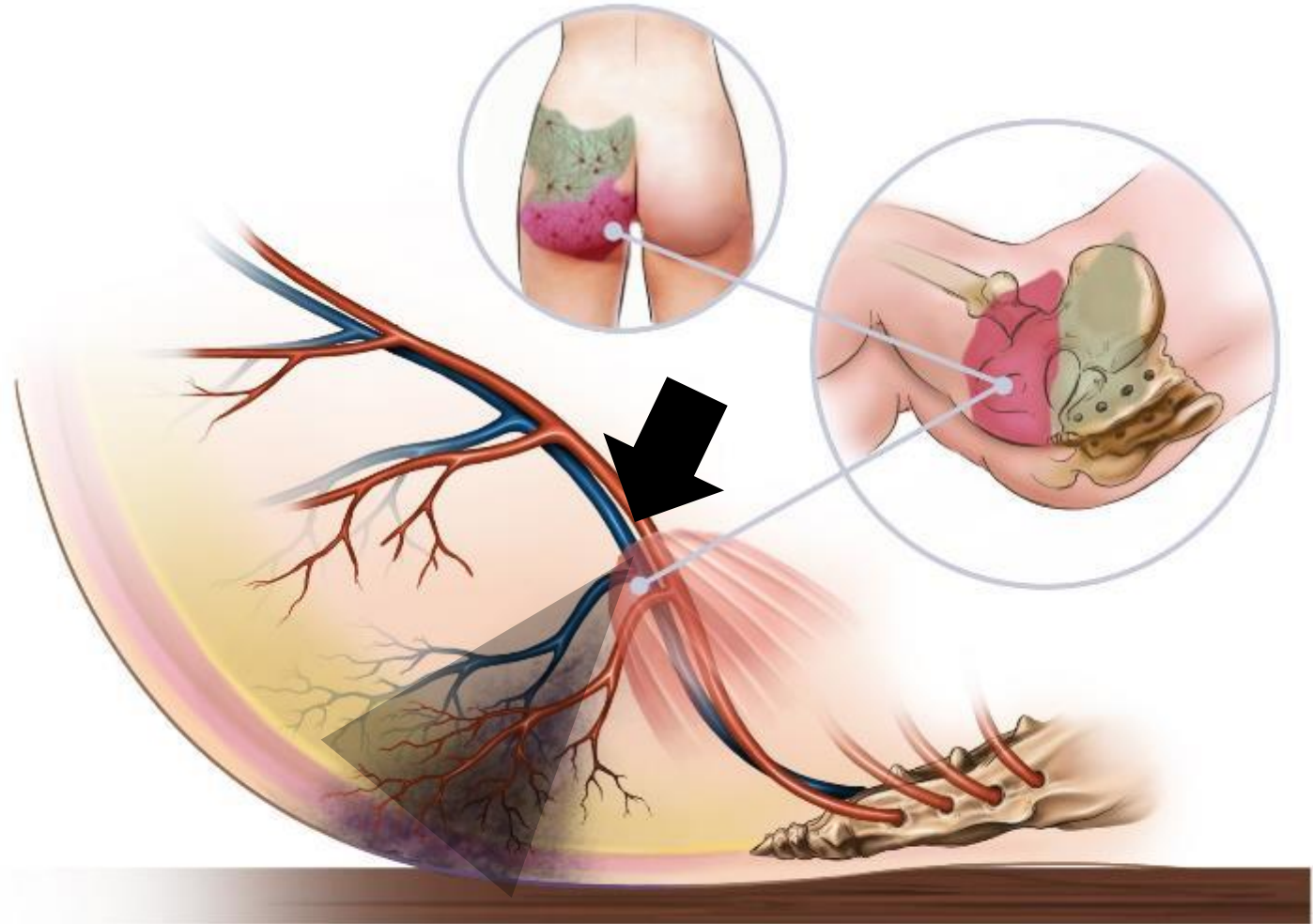


If you think you saw this PI progress through the stages from the OUTSIDE to the inside, you must be mistaken.

https://cdn.ymaws.com/npuap.site-ym.com/resource/resmgr/position_statements/npuap_position_on_staging-fi.pdf

If DTIs and Stage 4 PIs are vascular infarctions, they would form **INSIDE** to outside

- This would explain DTIs over buttock cheeks and their incredible relationship to hemodynamic parameters.
- They form from the **INSIDE** to the outside, at the origin of the named vessel that supplies the angiosome.



YES, they ARE “injuries!” Ischemic injuries

- When your understanding of a pathological process is wrong, your interventions will be misdirected.
 - We didn't create aseptic technique until the germ theory.
- **If full thickness pressure injuries (DTI, Stage 4) are “inside out” tissue infarctions/ischemic events of a named vessel, then our current “prevention protocols” are misdirected and that’s why they don’t work.**
 - **We may need “prevention” protocols that involve increased FIO₂, Increased Hgb, maintaining a specific MAP**
- At last I am on board with the term “injury” – these are *ischemic injuries*
 - Stage 1 are ischemia reperfusion injuries
 - DTI and Stage 4 are infarctions



A pink foam pad over the heel is not going to prevent a proximal vascular ischemia reperfusion injury.

Watch Pressure Injuries form from the Inside Out on my You Tube channel

- <https://www.youtube.com/watch?v=vsiuD7kbvnY&t=13s>
- <https://www.youtube.com/watch?v=CpbXx3zaS10&t=55s>
- https://www.youtube.com/watch?v=h87n2R_5q4A
- https://www.youtube.com/watch?v=_BVJrwgEFxE