SESSION 1

Anatomy
- Embryology
- Vasculature
- Segmental anatomy
- Wolff's Law

Diseases
- Auto-immunopathies
- CVD-CTD
- Hypercoagulable disorders

Math & Engineering
- Materials & mechanics
- Bioplasticity
- Soft tissue & scar mechanics
- LaPlace's law
- Flapology – free bodies
- Flapology – brownout-blackout
- Surface-volume-compliance
- Pressure
- Quickies

SESSION 2

Wounds
- General concepts
- Wound healing biology
- Chronic & pathological wounds
- Wound management
- Pressure

ARIMEDICA

THESE MATERIALS ARE ALL ON THE WEBSITE -- SO JUST ENJOY THE PRESENTATION

www.arimedica.com

37 f

Crohn’s, typical complicated history.

7 fistulas

Rx - certolizumab Cimzia® tnf-α inhibitor

Surgery when signs-symptoms subsided.

- 4 small bowel resections
- 3½ anastomoses
- Abdominal wall restoration

Uncomplicated recovery.

Active disease remains under control.

78 m

Recent onset inter-gluteal pain.

General surgery consult >> “pilonidal”

>> pilonidal surgery

>> persistent wound

>> increasing pain

Overlooked or ignored history :

Recent weakness, generalized

Recent Neurology consult

>> “autonomic neuropathy”

Recent increase dyspepsia / ugi symptoms

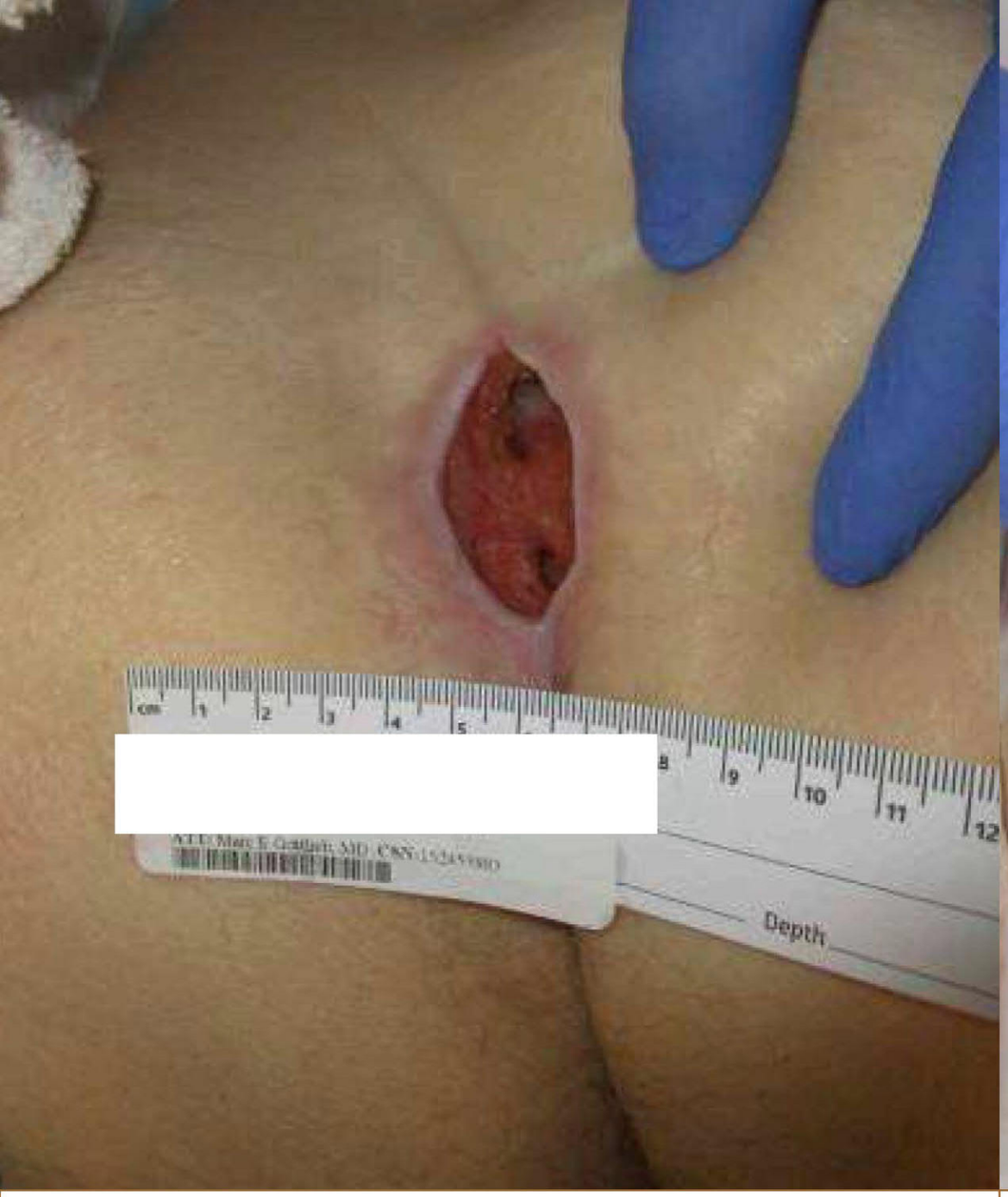
Physical exam :

Pt looks “pale as a ghost” white

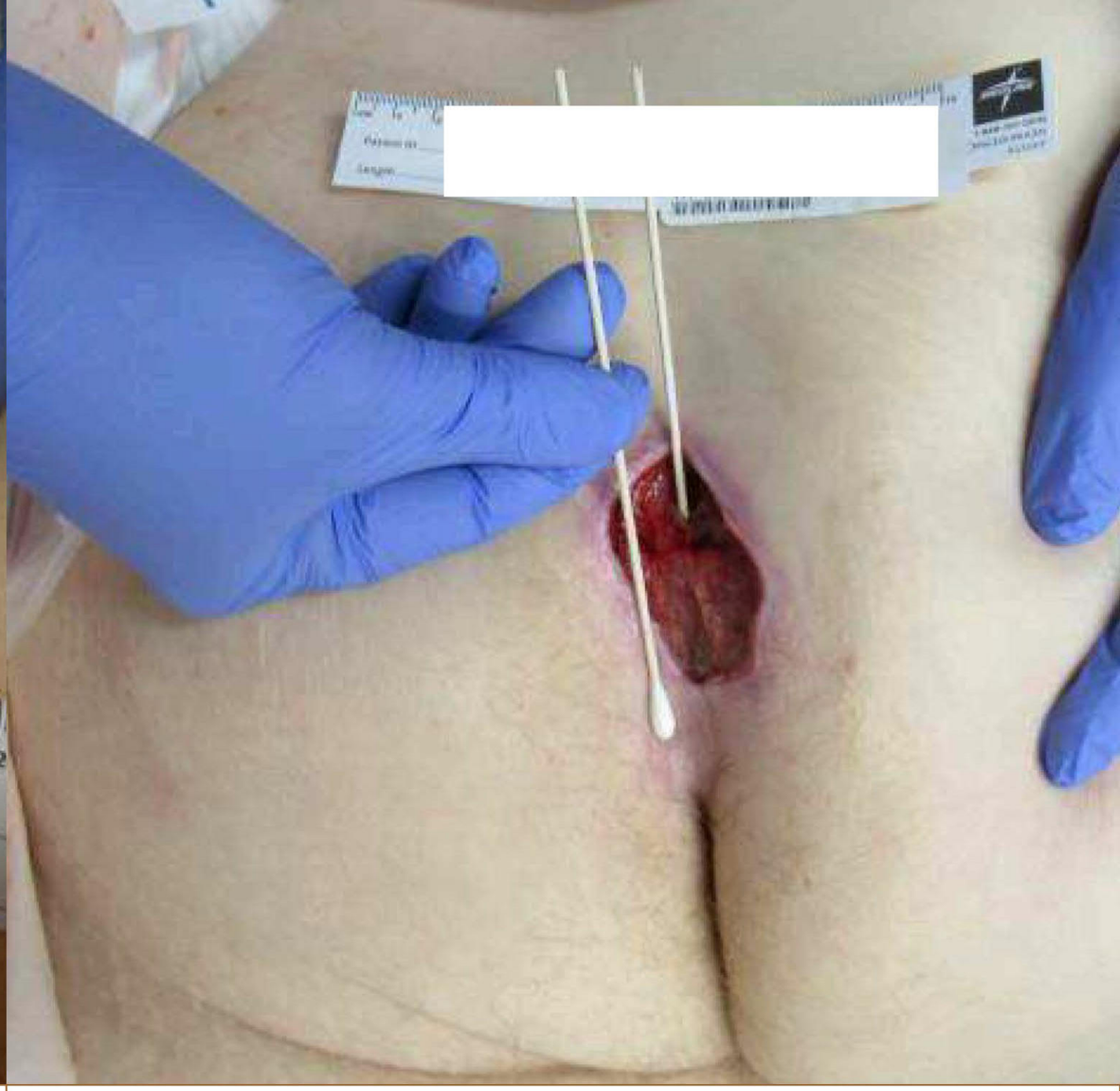
Hct 20, macrocytic, normochromic

Abdomen benign

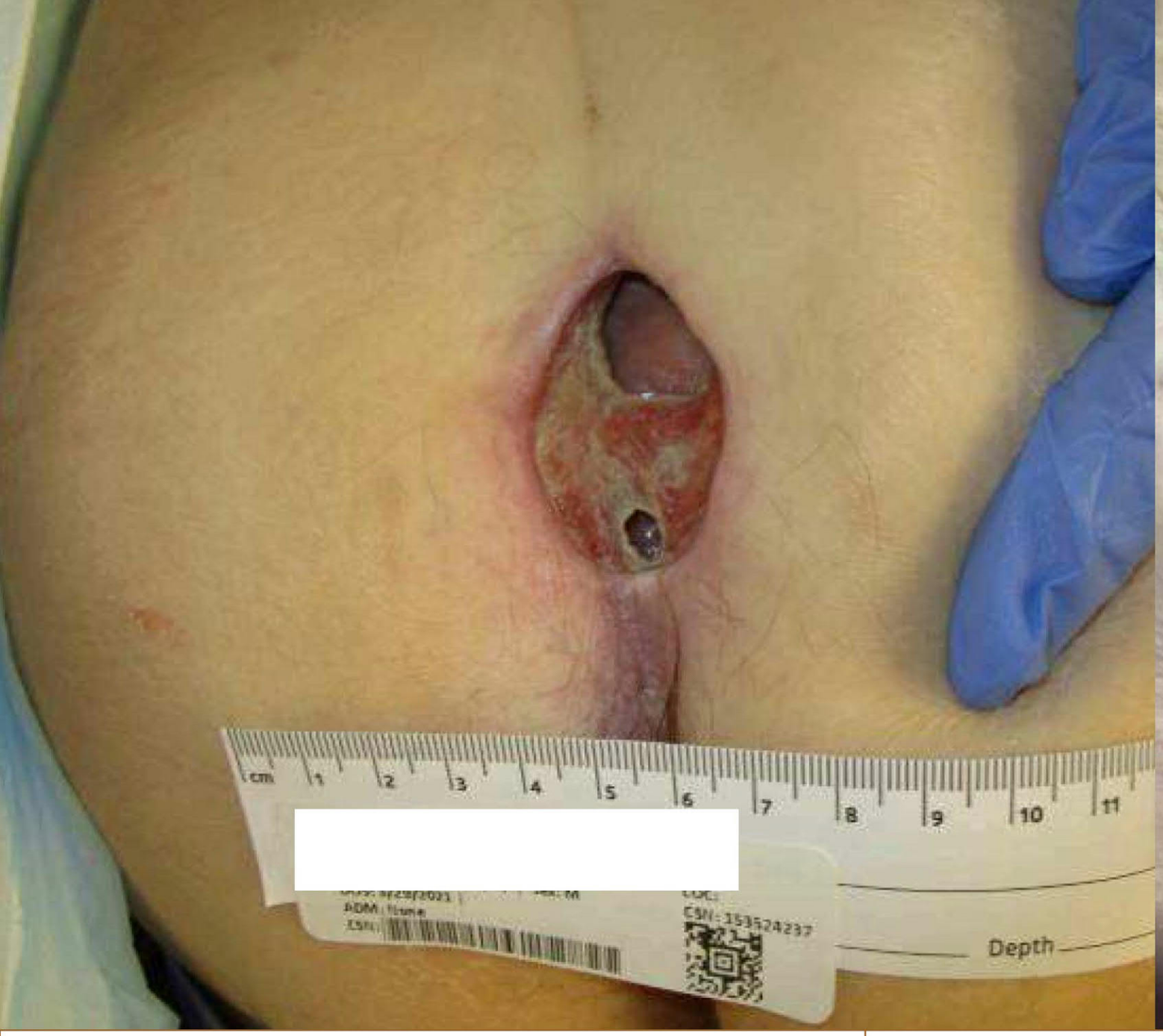
Rectal exam negative, stool guaiac negative



+ 3 weeks

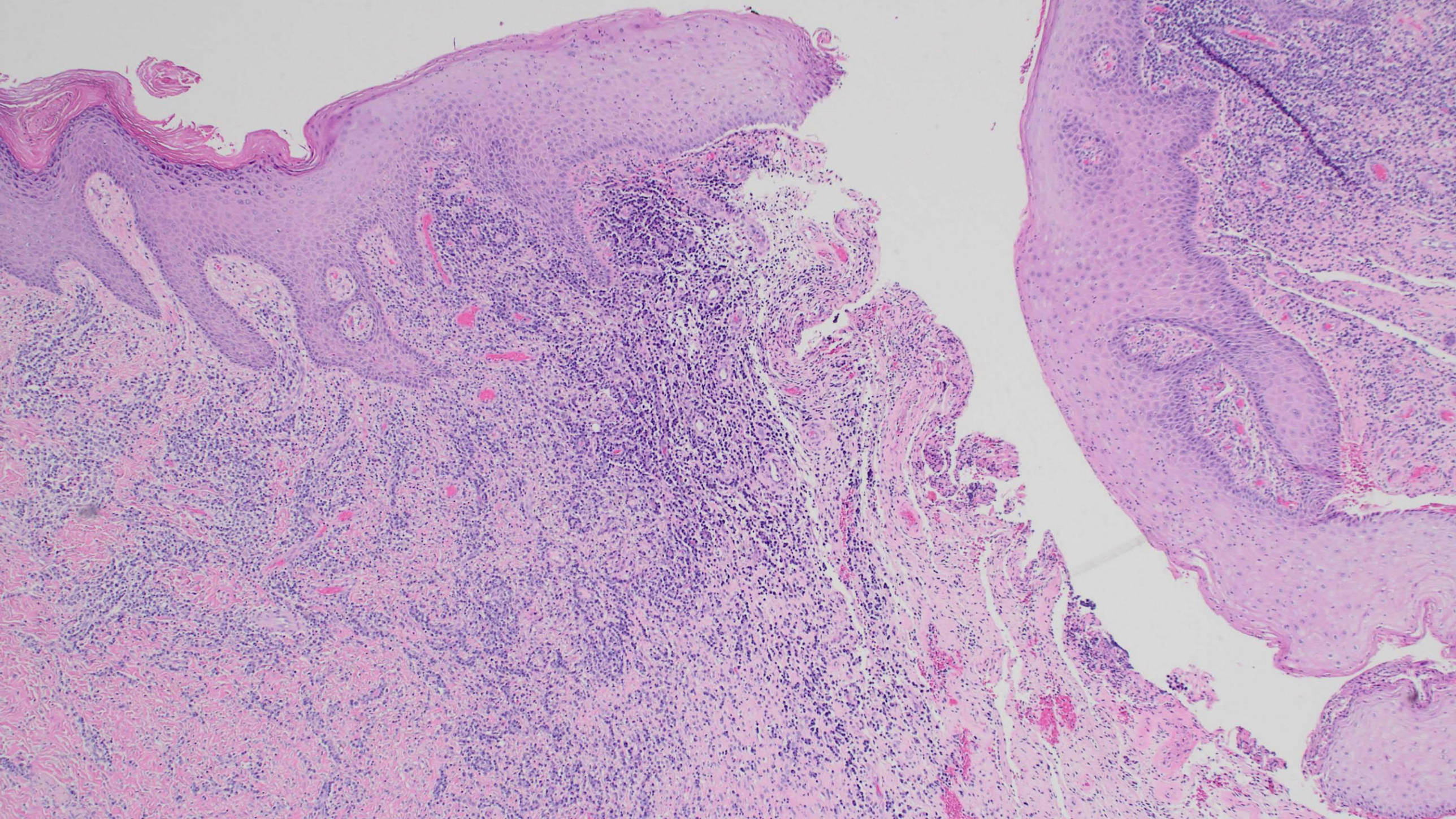


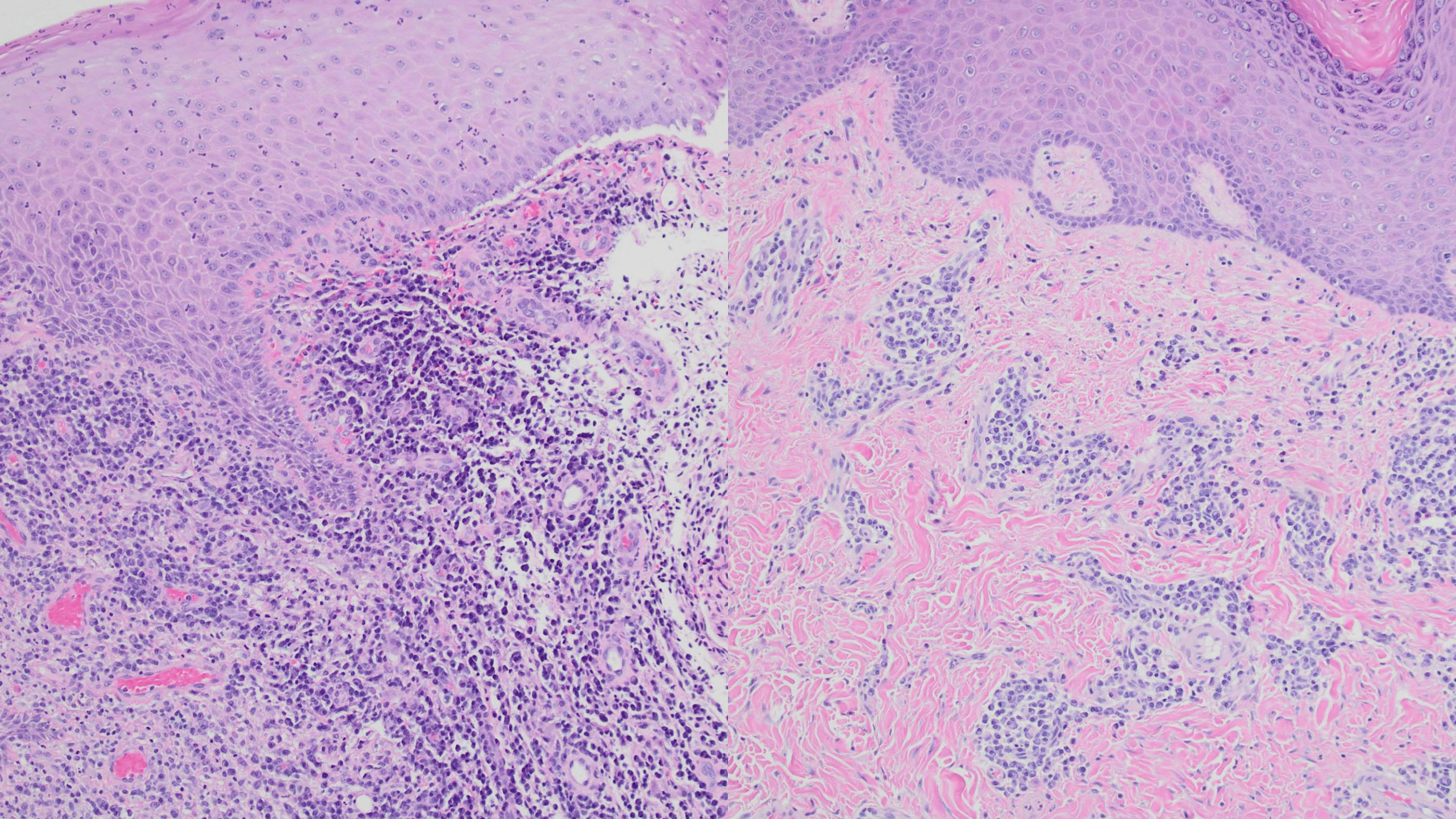
+ 3 weeks (meg #1)



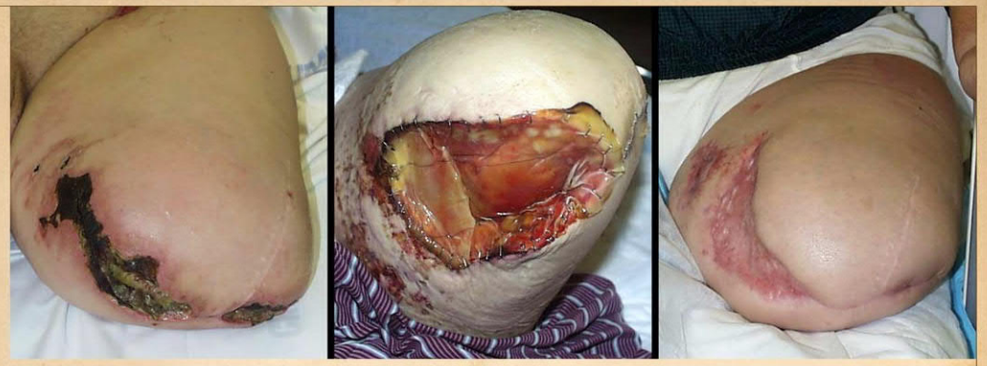
+ 2 weeks

+ 2 weeks





64 M,
aorto-iliac aso-pvod

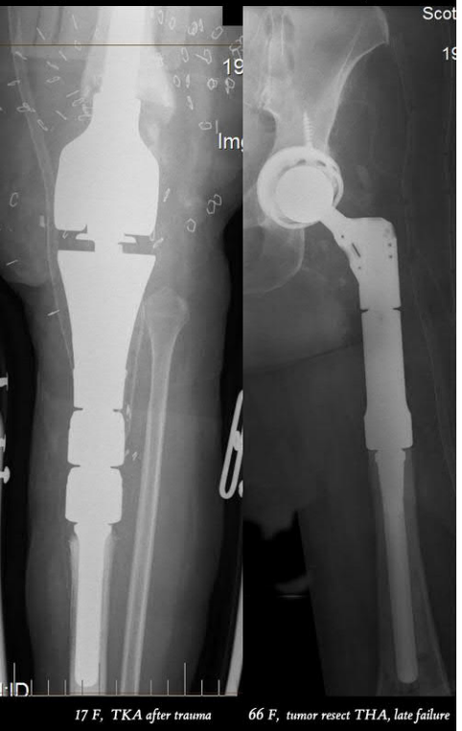


42 F,
dm, aso-pvod hands

49 M, back trauma, arthrosis, abscess

74 F, multiple hernia, abscess, e-c fistula

65 M, *osteosarcoma femur* — 56 M, *TKA after trauma*

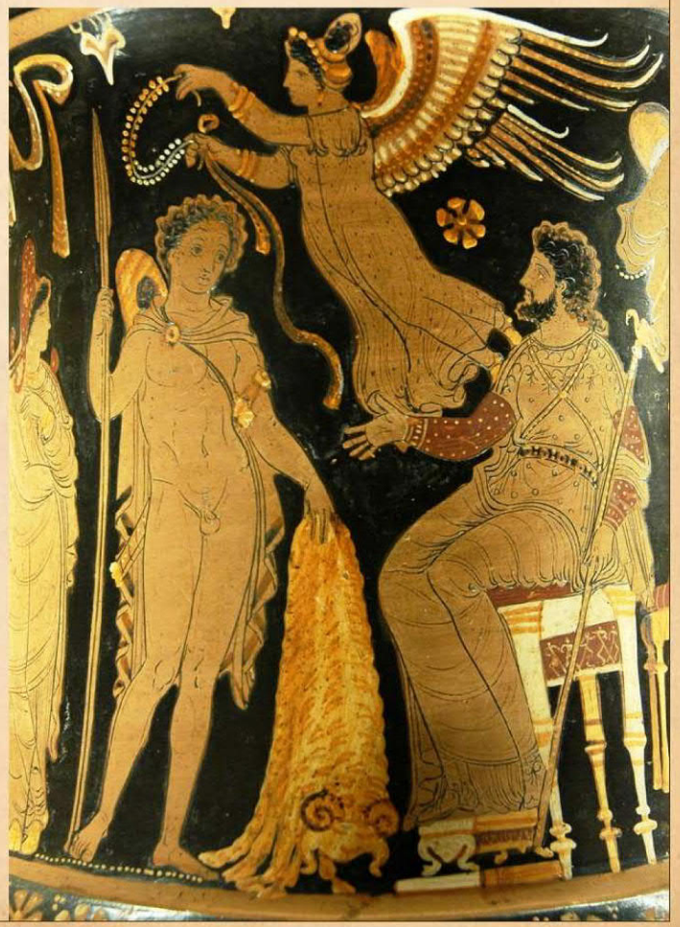


17 F, *TKA after trauma* — 66 F, *tumor resect THA, late failure*

Morai
The Three Fates
Clotho, Lachesis, and Atropos
1558
by Giorgio Ghisi
(Mantua, 1520—1582)

MAGIC STITCHES

The cotton fiber sews my britches,
And baseball weaving throws my pitches.
Synthetic blends make costume kitsches,
While fate spins yarns by mythic witches.
Floss and fabric create with stitches,
But ne'er in there are magic stitches.



Jason presenting the Golden Fleece to Pelias
c 340 - 330 BCE
Taranto, Italy.
Apulian red figure painting on calyx krater by the "Underworld Painter".

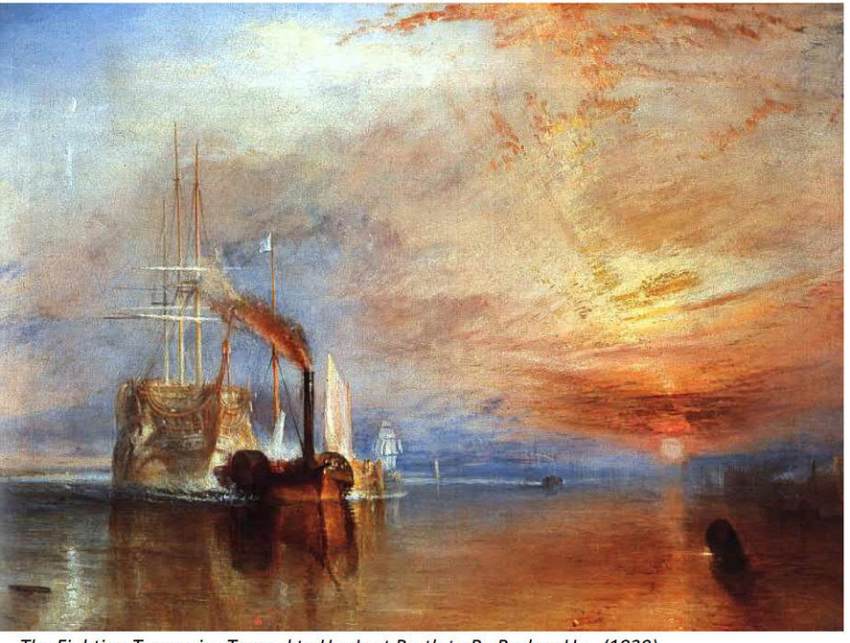
From ropes and jute to make strong hitches
That pull a truck from roadside ditches,
To feathery wisps like plumed ostriches
Of gold lamé from Argonaut riches,
Cloth and ribbon the eye bewitches,
But naught I've seen of magic stitches.

With woven gauze to dress my itches,
And fabric splints that quell my twitches,
Sutures strong for surgical niches,
Like silk and gut to fix my glitches,
These strings and threads our craft enriches,
But I do not have magic stitches.

READING LIST : arimedica.com

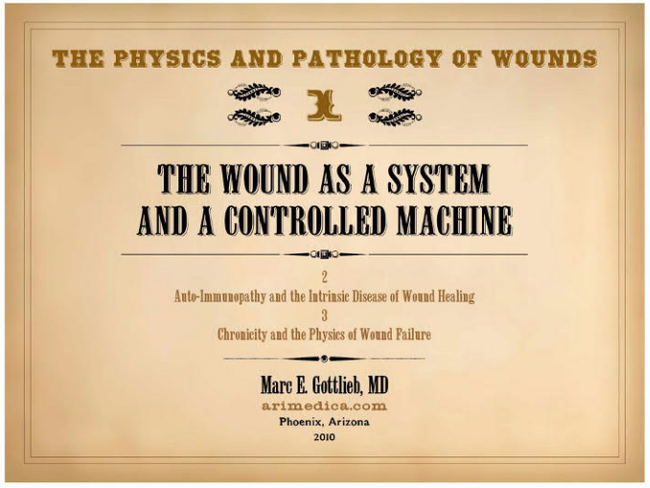
Joseph Mallord William Turner (1775 - 1851)

The Burning of the Houses of Parliament (1834)



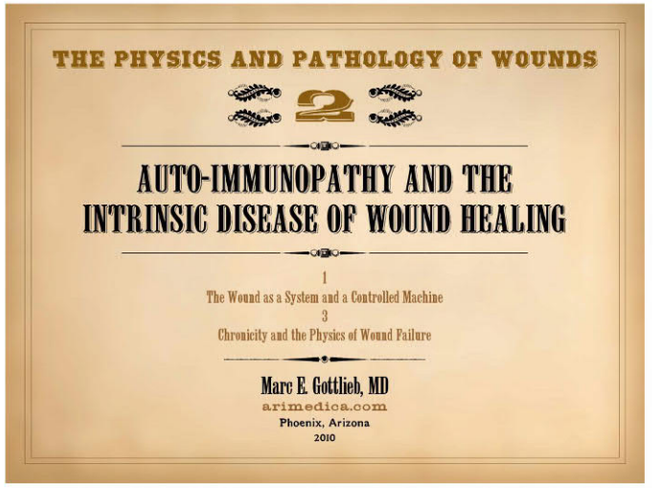
The Fighting Temeraire Tugged to Her Last Berth to Be Broken Up (1839)

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_wpp-1_wound control_gottlieb-me_maui-2010-0222_annotated.pdf
The Physics and Pathology of Wounds, Part 1
The Wound as a System and a Controlled Machine



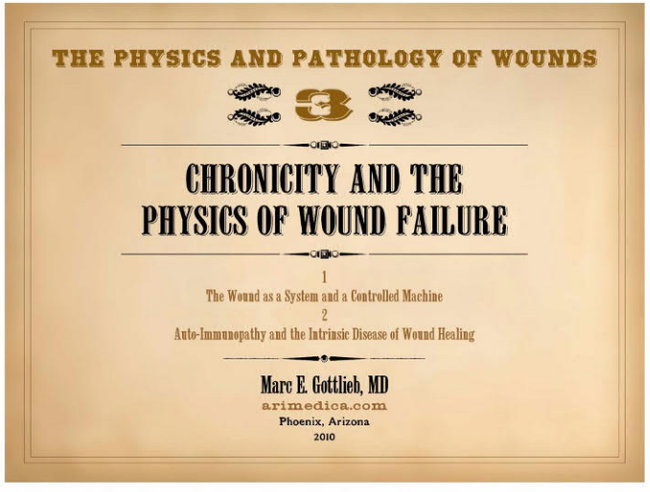
Wound physiology 1 - how the ad hoc wound organ functions, it's normal biology from a dynamical and systems point of view.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_wpp-2_autoimmune & intrinsic_gottlieb-me_maui-2010-0222_annotated.pdf
The Physics and Pathology of Wounds, Part 2
Auto-Immunopathy and the Intrinsic Disease of Wound Healing



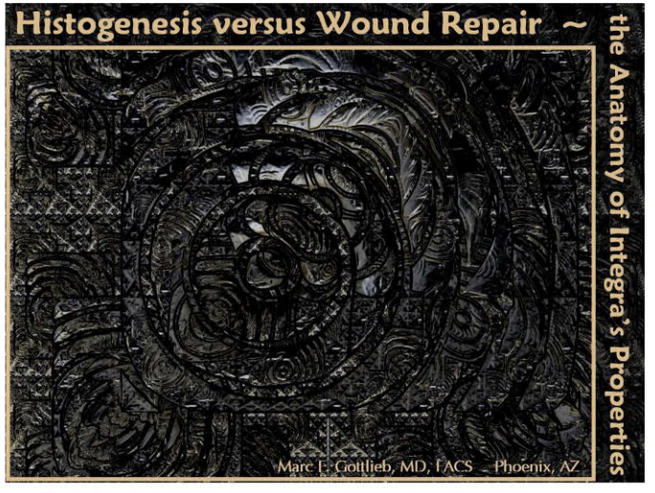
Wound physiology 2 - how wounds become intrinsically impaired by repetitive acute inflammation leading to auto-immunization.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_wpp-3_chronicity & failure_gottlieb-me_maui-2010-0222_annotated.pdf
The Physics and Pathology of Wounds, Part3
Chronicity and the Physics of Wound Failure



Wound physiology 3 - how the auto-immunized 3-population wound fails, accounting for the wound healing failure of chronic wounds.

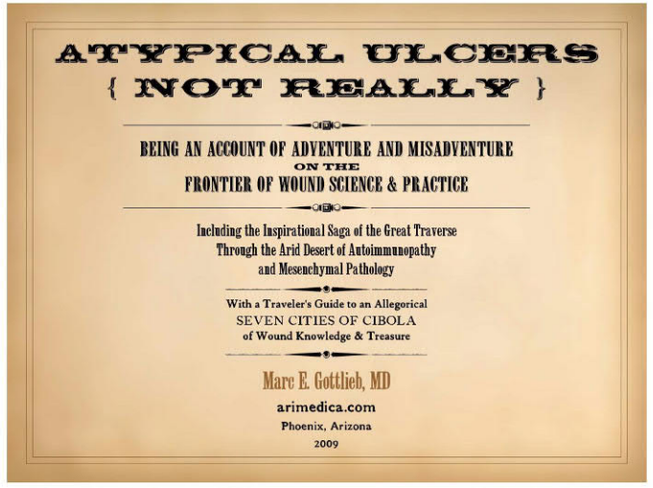
https://www.arimedica.com/subjects_integra.htm
https://www.arimedica.com/content/
arimedica_integra%20histogenesis_gottlieb-me_v2003.pdf
Histogenesis versus Wound Repair: the Anatomy of Integra's Properties



Detailed and highly illustrated look at wound healing biology, emphasis on its anatomy and time-dependent evolution.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_(not)%20atypical%20wounds_gottlieb-me_2009-0926_annotated.pdf

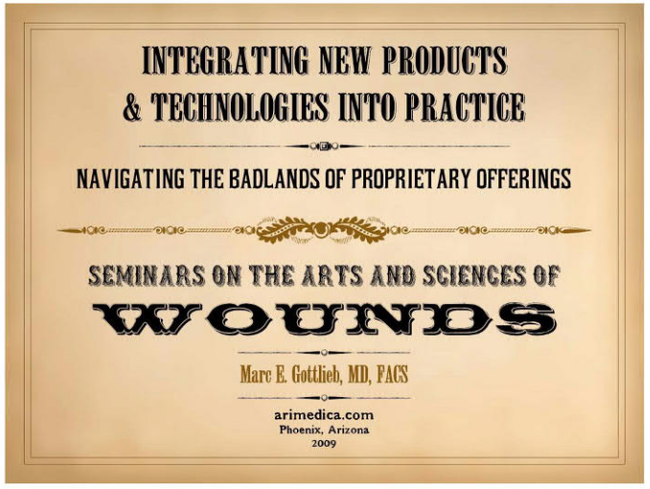
(NOT) Atypical Ulcers (Autoimmunopathy and Connective Tissue Disorders: The True Intrinsic Diseases of Wound Healing)



Wound physiology and failure - a precursor paper to the 3-part series, with a compact presentation of wound healing physiology.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_integrating new products_gottlieb-me_annotated.pdf

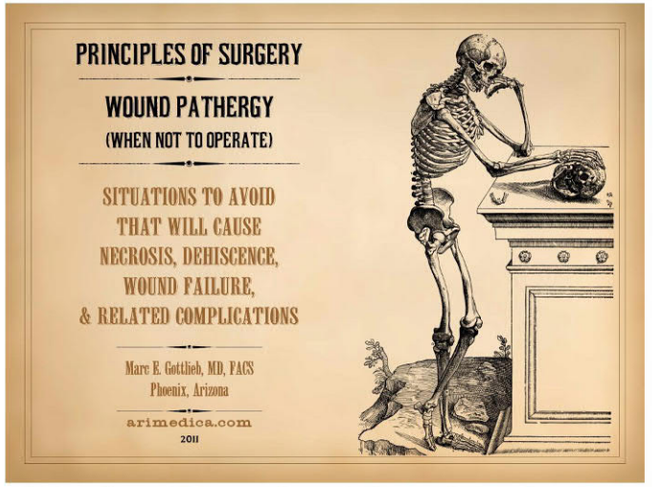
Integrating New Products & Technologies into Practice : Navigating the Badlands of Proprietary Offerings



A general discussion about evaluating and using new technologies, focus on wounds, including a survey of wound care products.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_wound pathergy_gottlieb-me_2017-1025_annotated-200.pdf

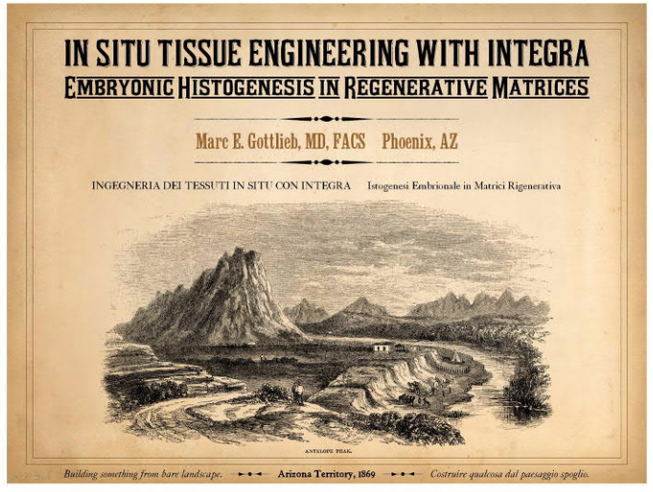
Wound Pathergy – When Not to Operate Situations to Avoid that Will Cause Necrosis, Dehiscence, Wound Failure



Biology of wound failure, and systemic diseases that interfere with wound healing and surgery, 30 cases and detailed explanation.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_integra-histogenesis_gottlieb-me_napoli_2015-0417_annotated.pdf

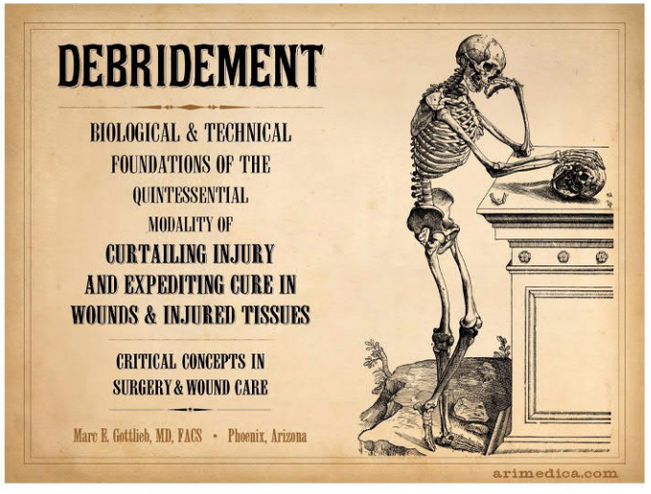
In Situ Tissue Engineering with Integra : Embryonic Histogenesis in Regenerative Matrices



Overview of wound healing biology, then a look at how tissue regeneration occurs in biomatrices.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_debridement-1_gottlieb-me_2016-0413_annotated.pdf

Debridement Biological & Technical Foundations of the Quintessential Modality ...

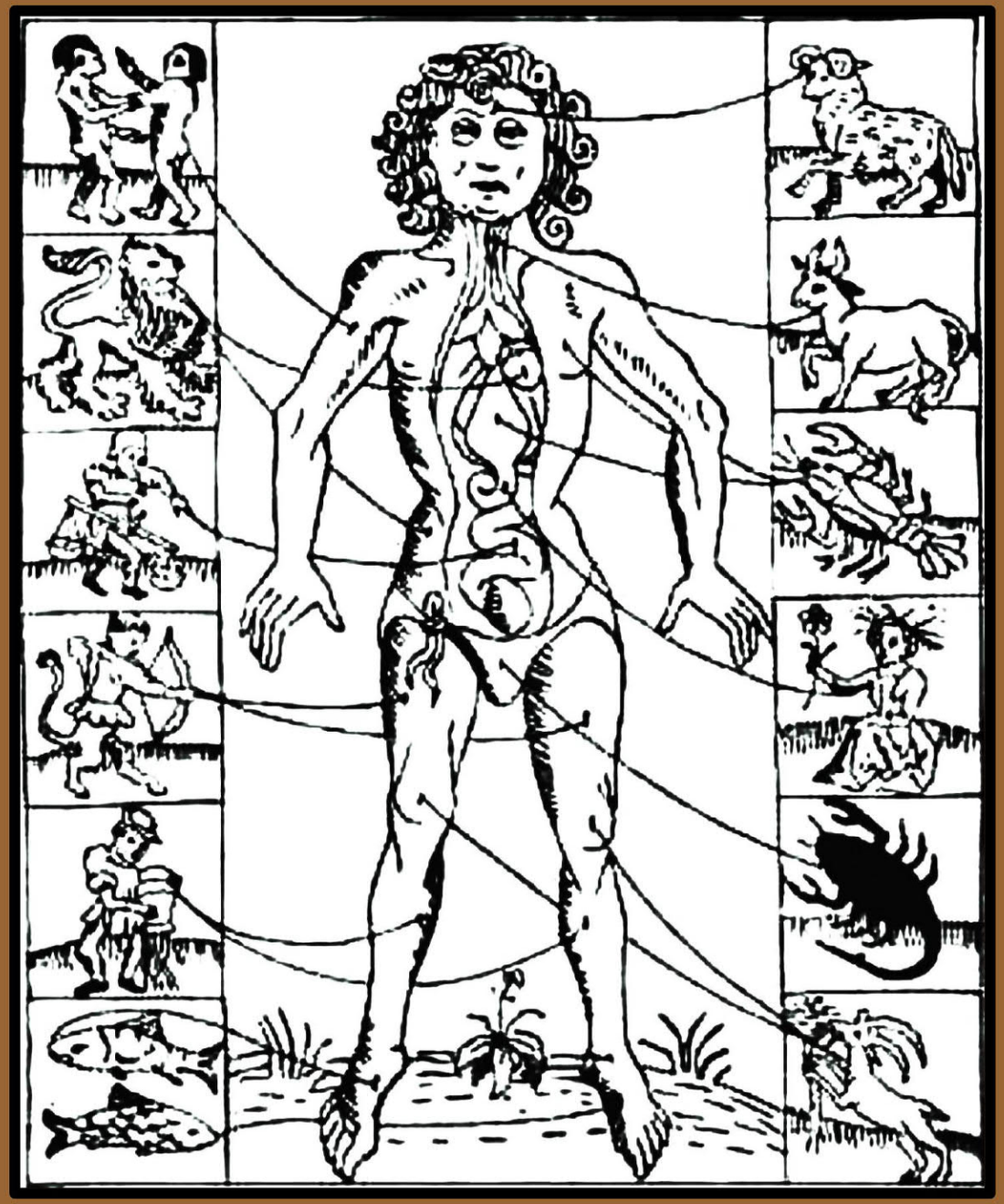


Explanation of debridement, written for non-surgeons, but with a variety of interesting information.

https://www.arimedica.com/subjects_integra.htm
https://www.arimedica.com/content/
arimedica_hypercoag-2018_gottlieb-me_2018-0920_(annotated).pdf

Hypercoagulable Disorders – Implications For Wounds & Surgery Pathophysiology, Clinical Features, Diagnosis & Treatment

Detailed text on hypercoagulable disorders and their profound significance to surgery complications and chronic wounds.



General Concepts

Wound Healing Biology

CAP
Chronic and Pathological Wounds

Wound Management

Pressure Ulcers

WOUNDS : General concepts

Wounds

- General concepts
- Wound healing biology
- Chronic & pathological wounds
- Wound management
- Pressure

General ::

"wound" - context

why it is important

wounds - vs - 6 domains of medical knowledge

wounds and doctors

wound types & context

wound diagnosis

wounds heal - when they don't, there is a reason
diagnose and fix it

management - different rules for acute vs cap wounds

wounds and surgery

See the website, read the papers there.

arimedica.com

Wounds - why it is important

patients with problems; patients with serious problems
cured patients; happy patients
intellectually challenging; professionally fulfilling
full range of medical and surgical knowledge and skills
easy to develop busy non-competitive practice

Wounds - vs - 6 Domains of medical knowledge

anatomy
physiology
pathology
diagnosis
therapeutics
management

Wounds and doctors

parasites - vs - dilettantes - vs - experts
legitimate : comprehensive - vs - focused
plastic surgery, general surgery, podiatry
vascular, primary care, medicine
nurses & therapists

Wounds types & context

acute : trauma, surgery
chronic & pathological : CAP wounds

cap wounds due to : persistent injury
underlying disease
impaired wound healing

Wound diagnosis

7 signs
complex, detailed
understand biology and state of the wound

Wounds heal - when they don't, there is a reason
diagnose and fix it

Wound management rules are different for acute vs cap wounds,
acute wound principles applied to cap wounds
makes more wound
3 phases of care

Wounds and surgery

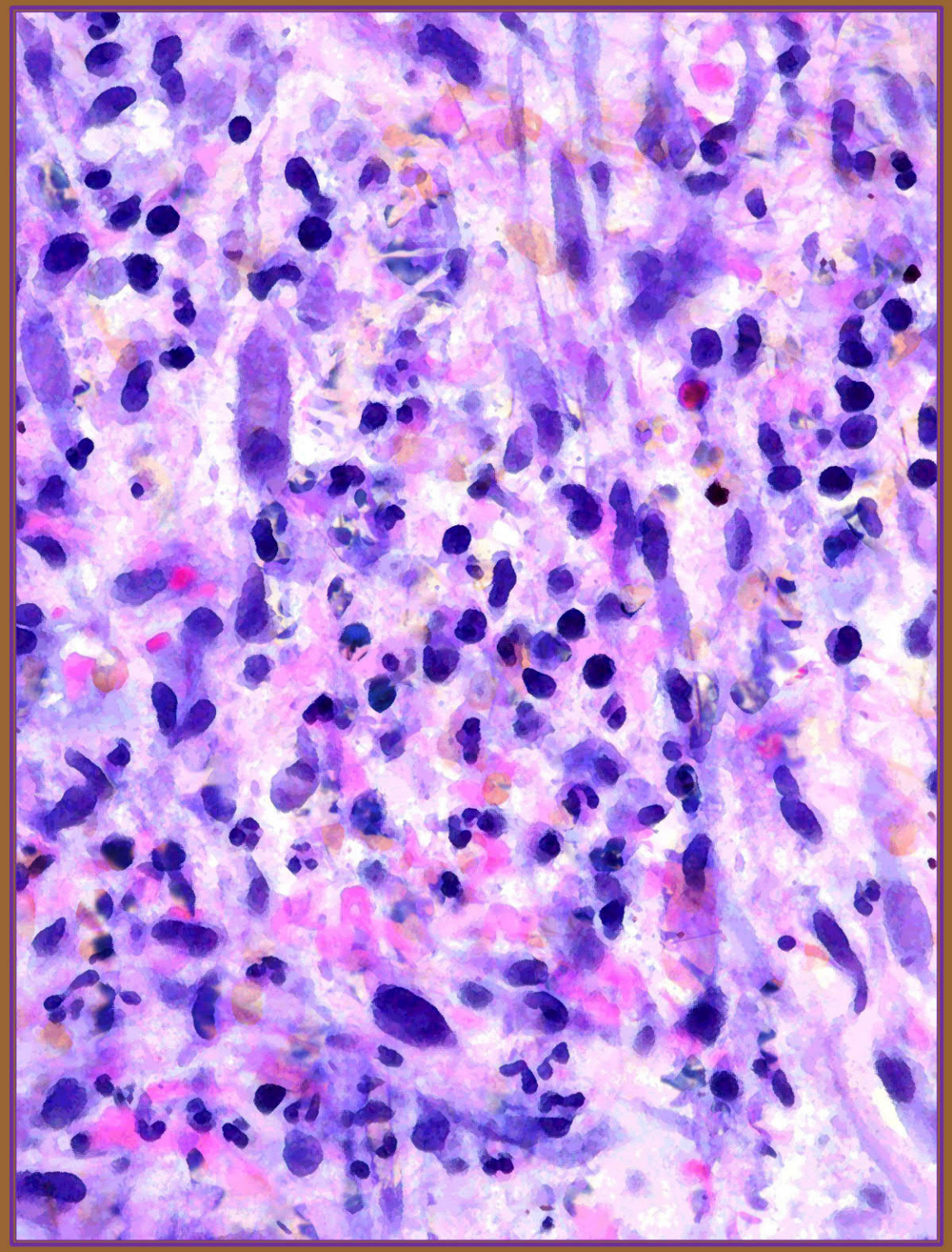
wounds heal, or you the physician restore that biology
wounds heal, so many do not need surgery
but, many wounds do need surgery
DO NOT do surgery until wound healing biology is correct
do not mess things up by doing errant surgery
unindicated, poorly timed, incorrect procedure
do not mess things up denying patients needed surgery
protect or save your patients from bad care

Plastic Surgery is the only specialty with all requisite knowledge & skills for comprehensive wound cure.

you WILL be consulted about wounds on the assumption that you will know what to do

" WOUND "

One word for many contexts.



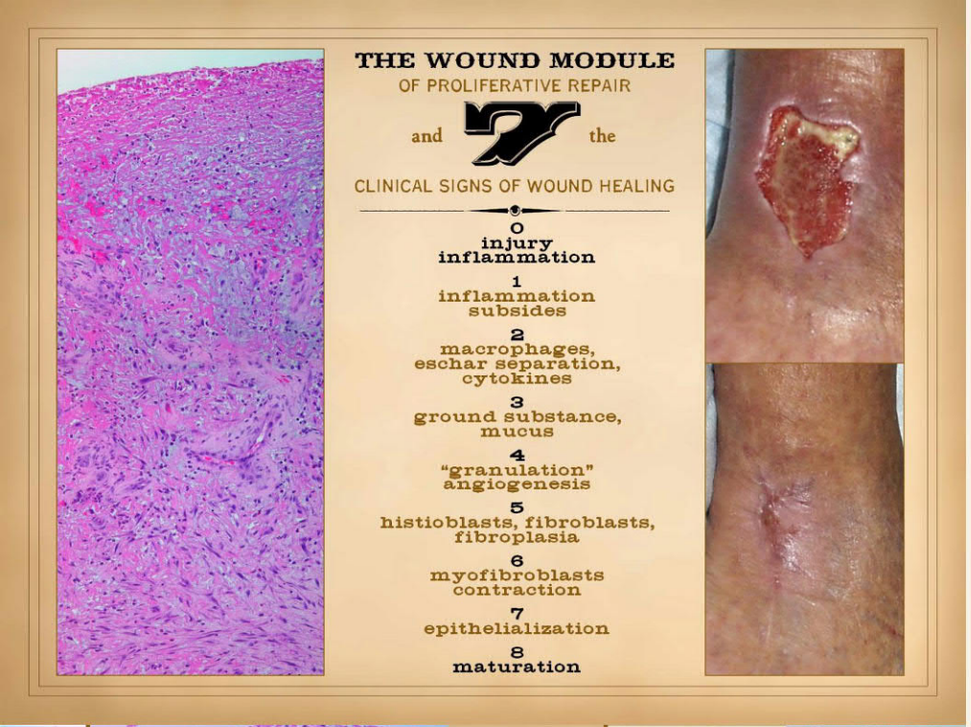
General Concepts

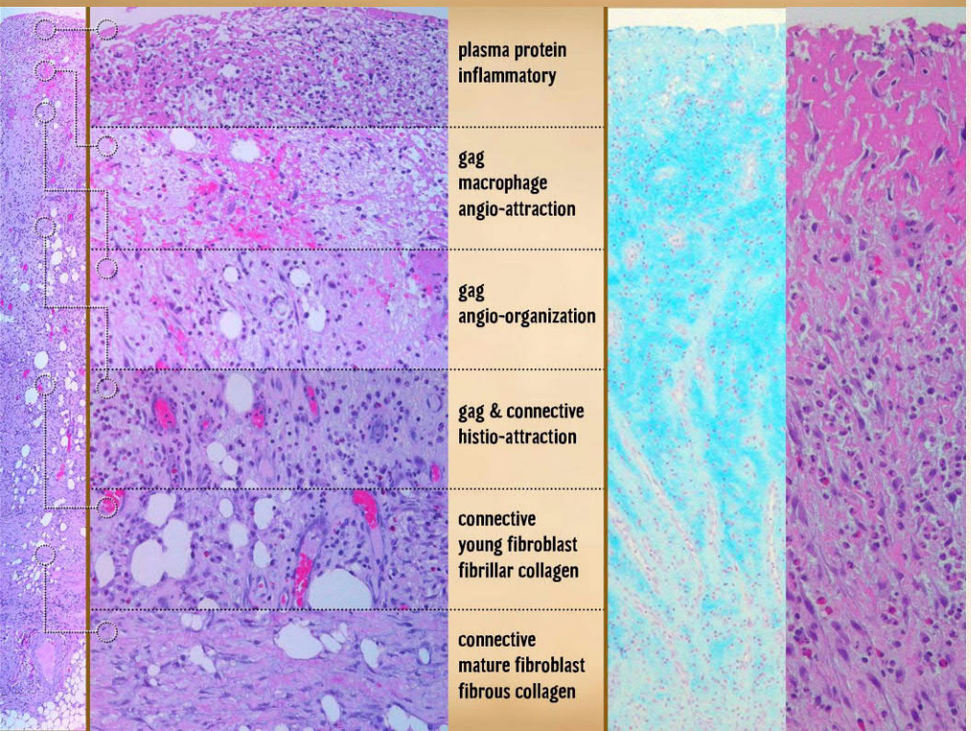
Wound Healing Biology

CAP
Chronic and Pathological
Wounds

Wound Management

Pressure Ulcers





Multi-cellular life has 2 requirements :

- skeletal framework for cells to bind and organize
- vascular distribution to share products of speciation

Basic biology of these anatomical elements was established in animals in Porifera and Cnidaria.

The framework is based on proteins - collagen and other connectives.

The [gastro-] vascular distribution is a fractal r-net, formation governed by VEGF.

Speciated cells form **parenchyma** (specialized metabolic functions) from ectoderm-endoderm.

Speciated cells form the skeletal framework and vascular net - the **stroma** - from mesoderm (also, other common services).

Only two cells make the stroma :

fibroblasts make the protein framework,

angiocytes make vascular distribution vessels.

A wound is :

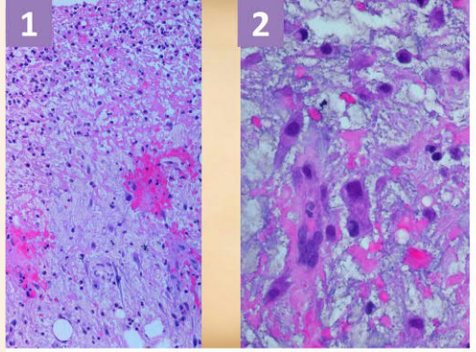
- any injury that causes a defect.
- any injury or event that triggers the process of inflammation and post-inflammatory repair.
- the ad hoc organ that arises to repair the injury.
- the biology of that ad hoc organ.

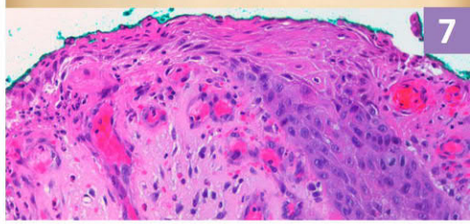
Wound healing is stroma reforming after injury.

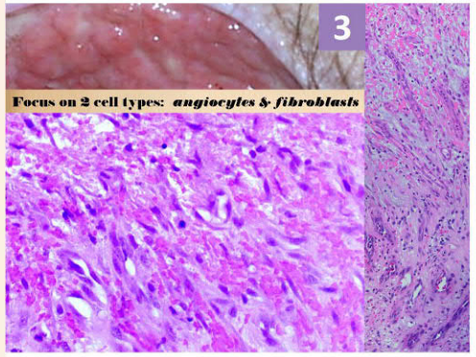
Wound healing is complete when epithelial continuity is restored, and internal mesenchyme is sequestered from the environment.

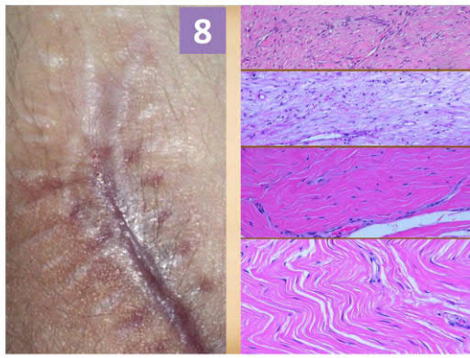
Epithelium cannot grow except on a substrate of proper stroma.

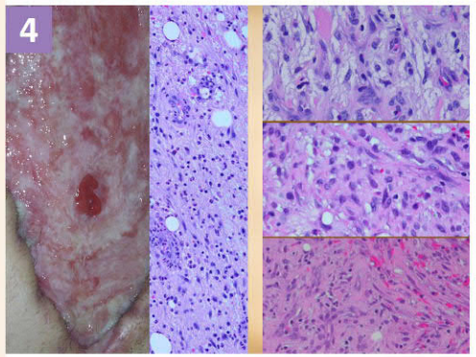
Angiocytes and fibroblasts proliferate to make new connective framework and vessels, then epithelium grows, and the wound is then healed.





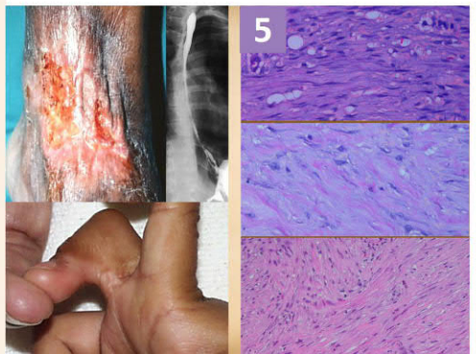


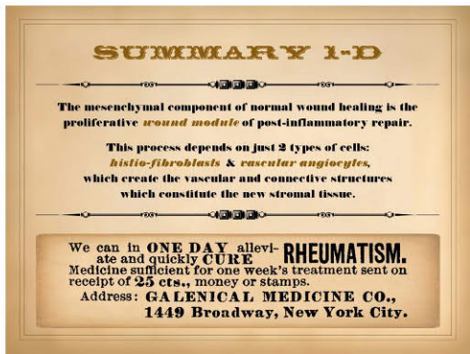


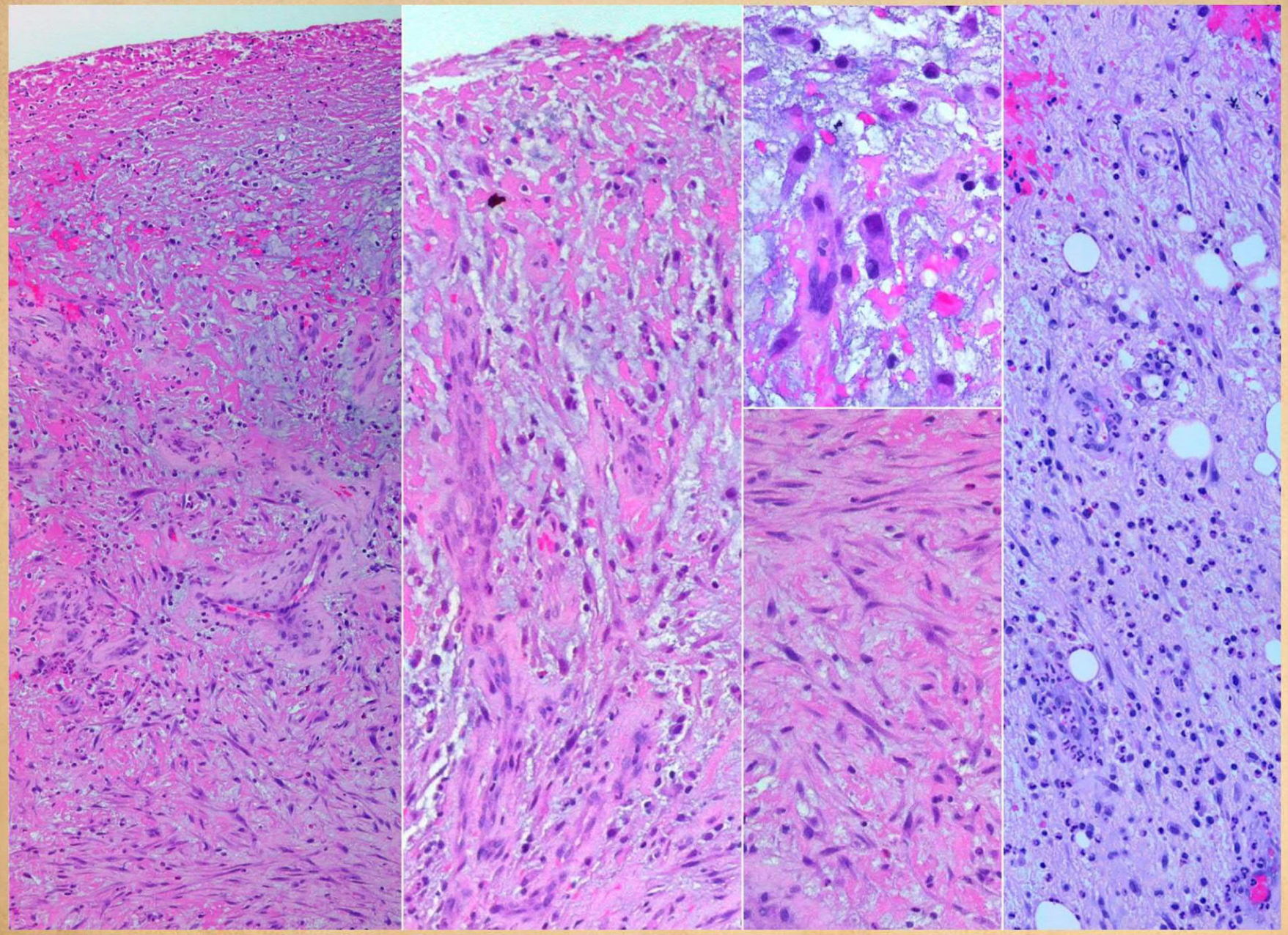


Response to injury and the reactive wound healing process have **7** observable sequential features that indicate the health and progress of the process.

Each of these 7 features correlates physical exam with cell and chemical biology as observed histologically.

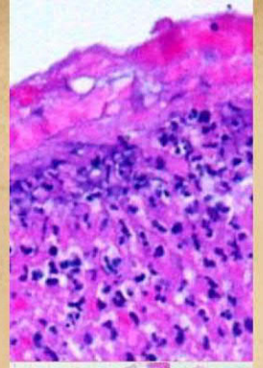






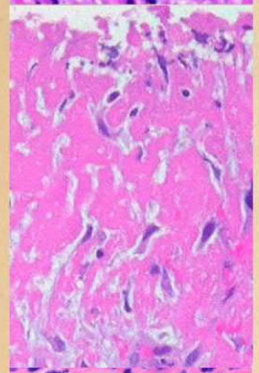
0
Injury & inflammation

Plasma proteins. Neutrophils.



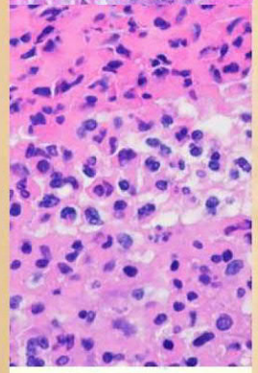
1
Inflammation Subsides

Defensive, injurious. Induces repair.



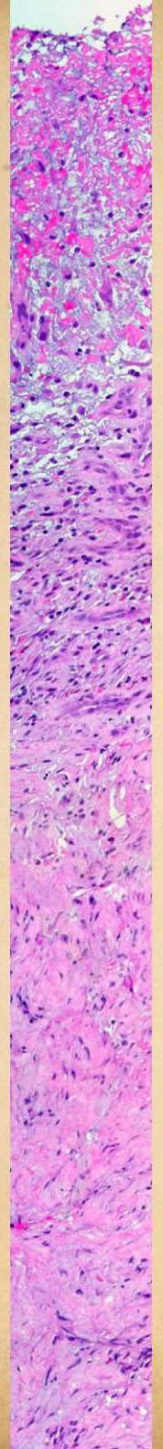
2
Monocyte - macrophages

Separate eschar. Recruit repair cells.



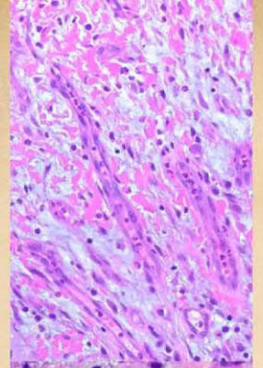
3
Aminoglycan ground substance

Environment for repair cells to create new structure.



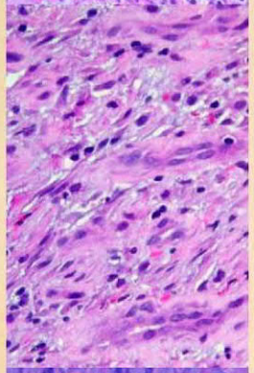
4
Angiogenesis inflammation

New blood vessels in the AG layer.



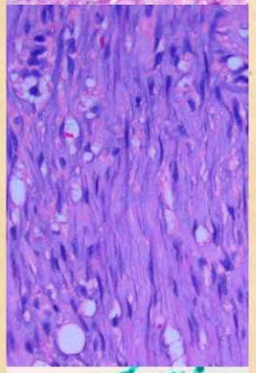
5
Fibroplasia

Connective protein tissue matrix.



6
Contraction

Reduction of wound surface.



7
Epithelial migration

Outgrowth onto the other proliferative elements.

8
Maturation

Scar remodeling back to normal dermis or fascia.

NORMAL WOUND HEALING

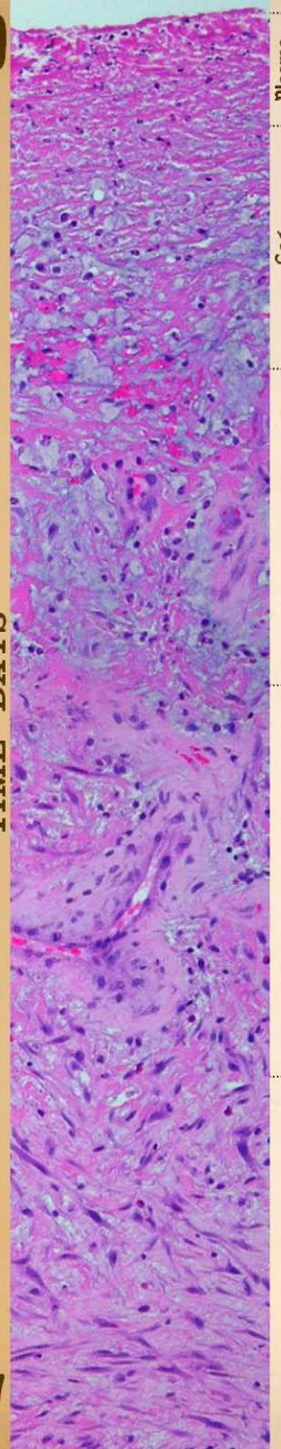
SEQUENTIAL STRUCTURES & EVENTS

TEMPORAL & SPATIAL RELATIONS

TIME - DAYS

0

7



Plasma
Acute infl

Gag
Angio - attraction

Gag - vertical migration

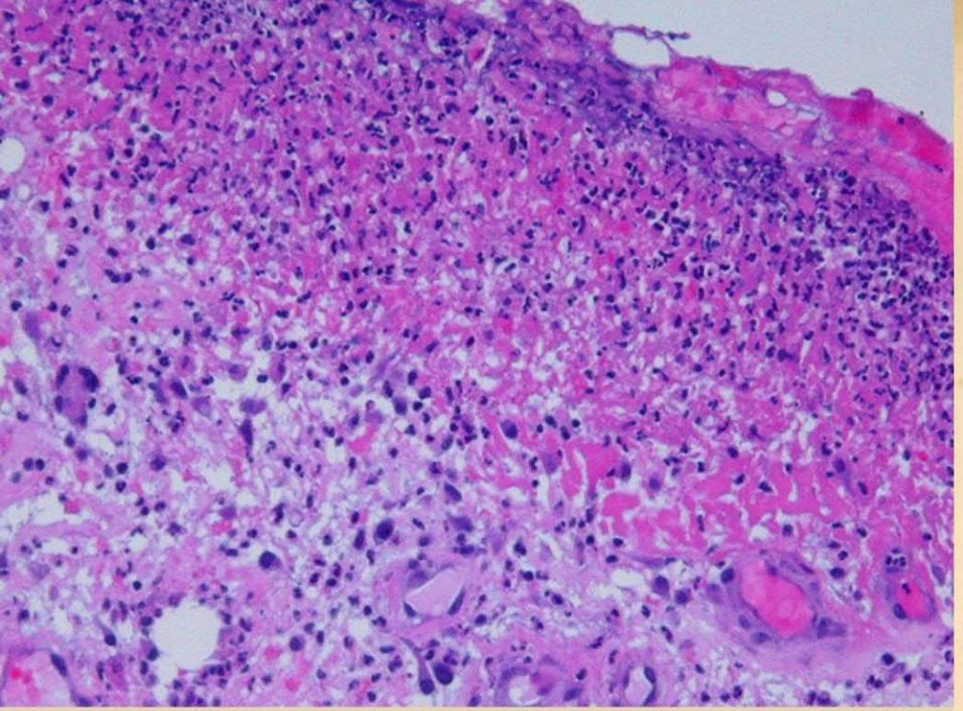
Angio - organization

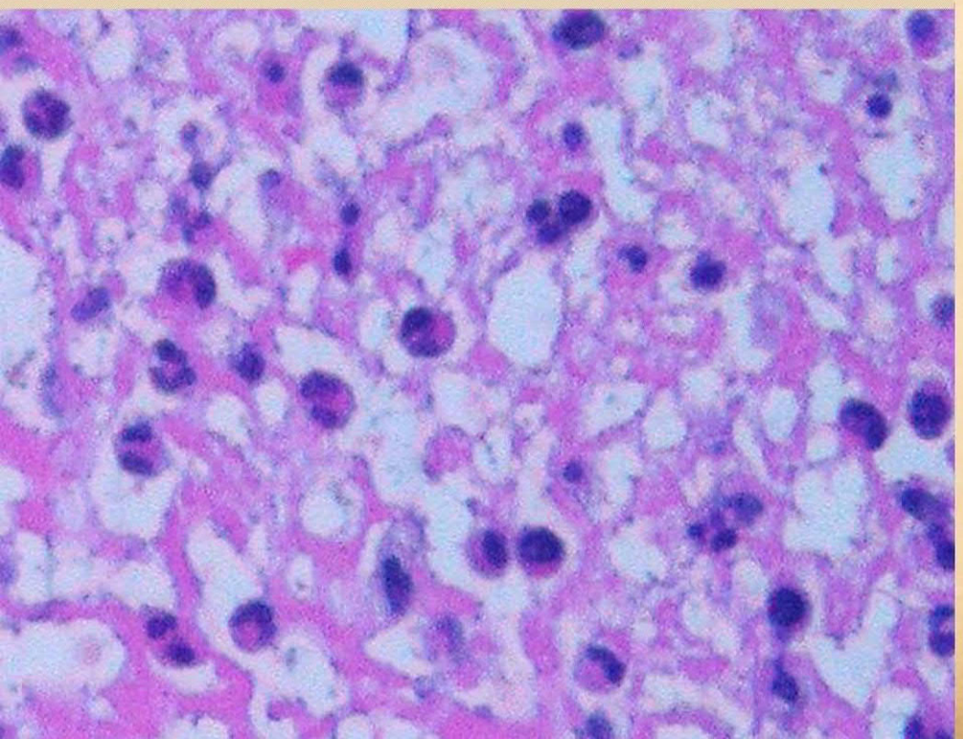
Connectives

acute inflammation

angiogenesis & granulation tissue

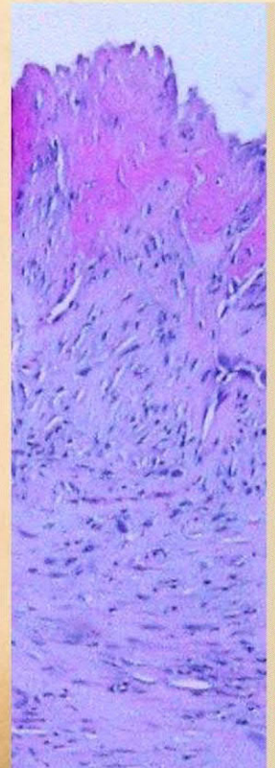
fibrogenesis & scar

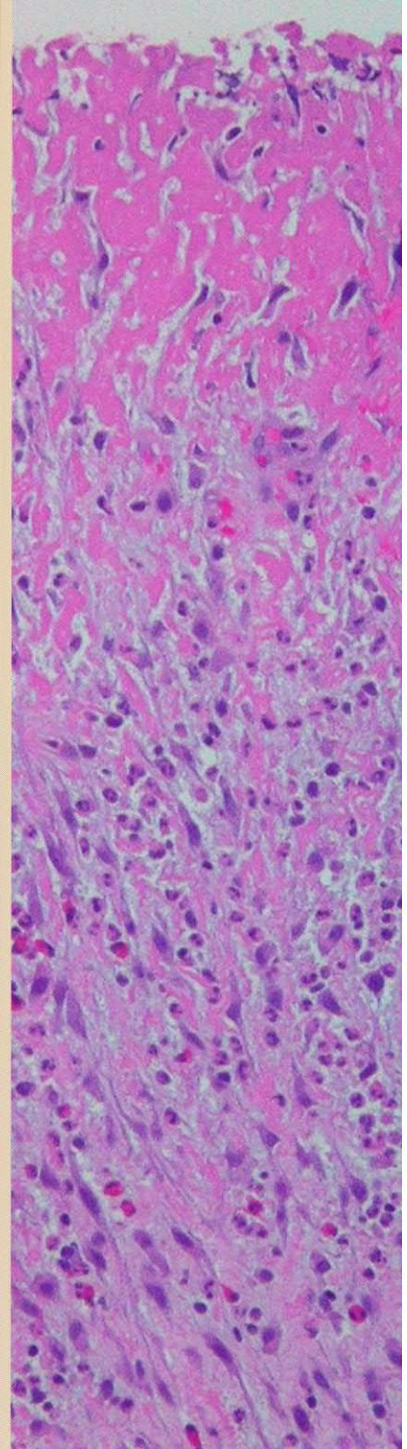




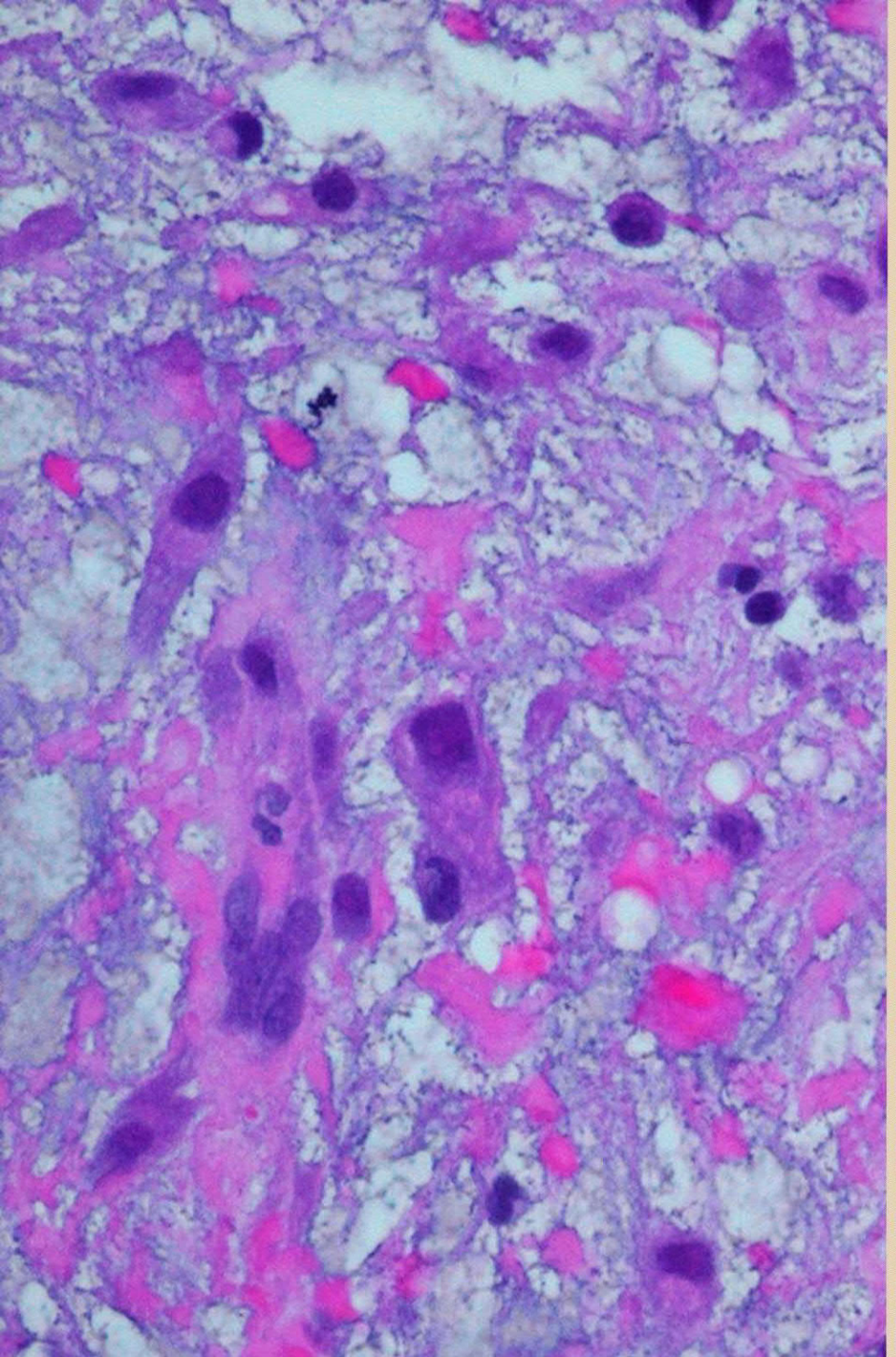
0
Inflammation
& Injury

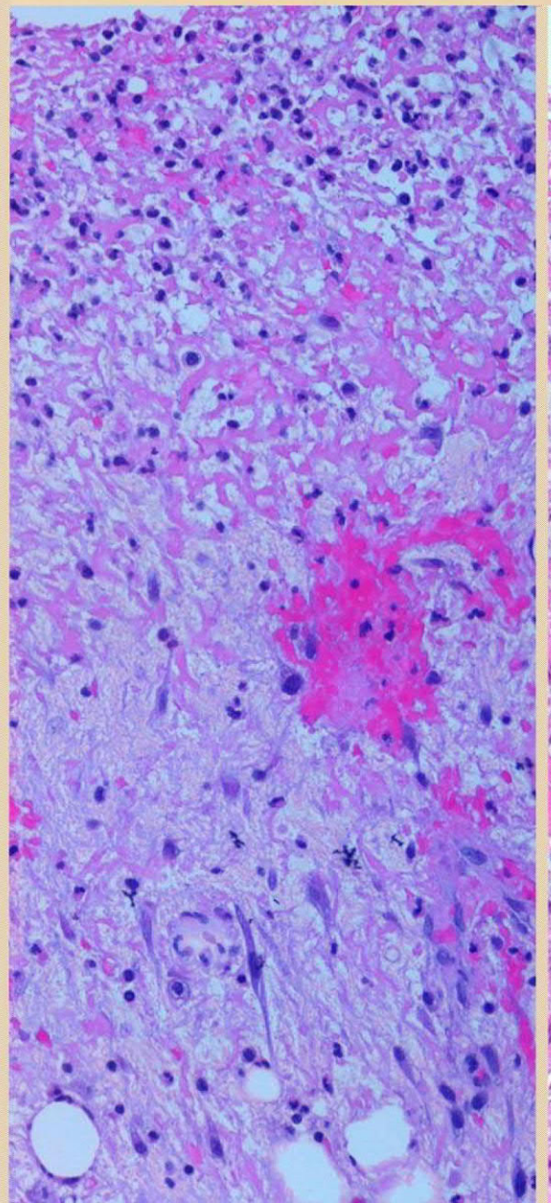
I
Inflammation
Subsides

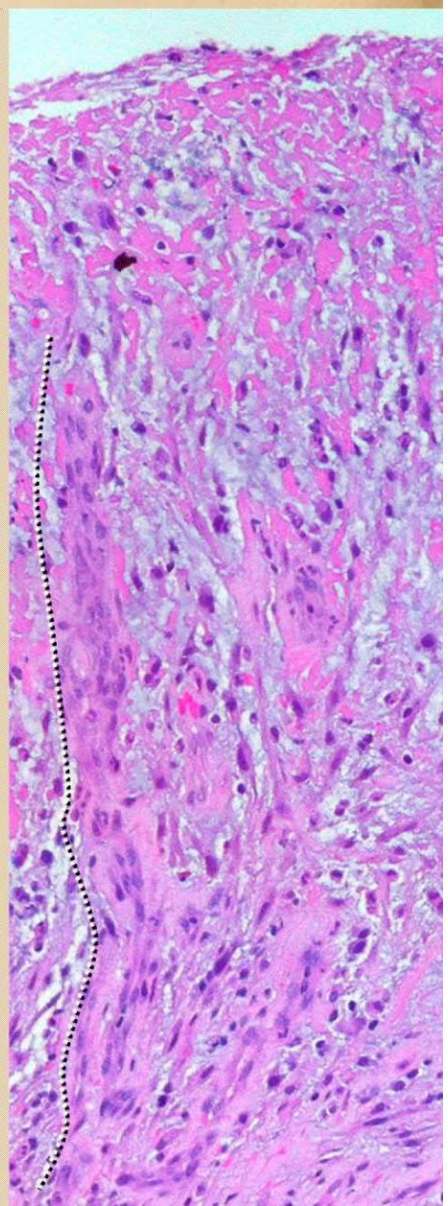




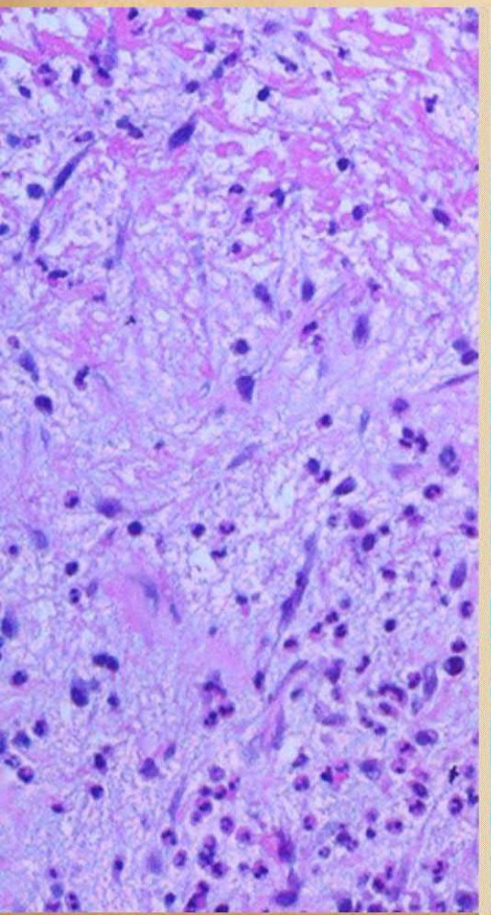
II
Macrophages & Cytokines
Initiation of Repair

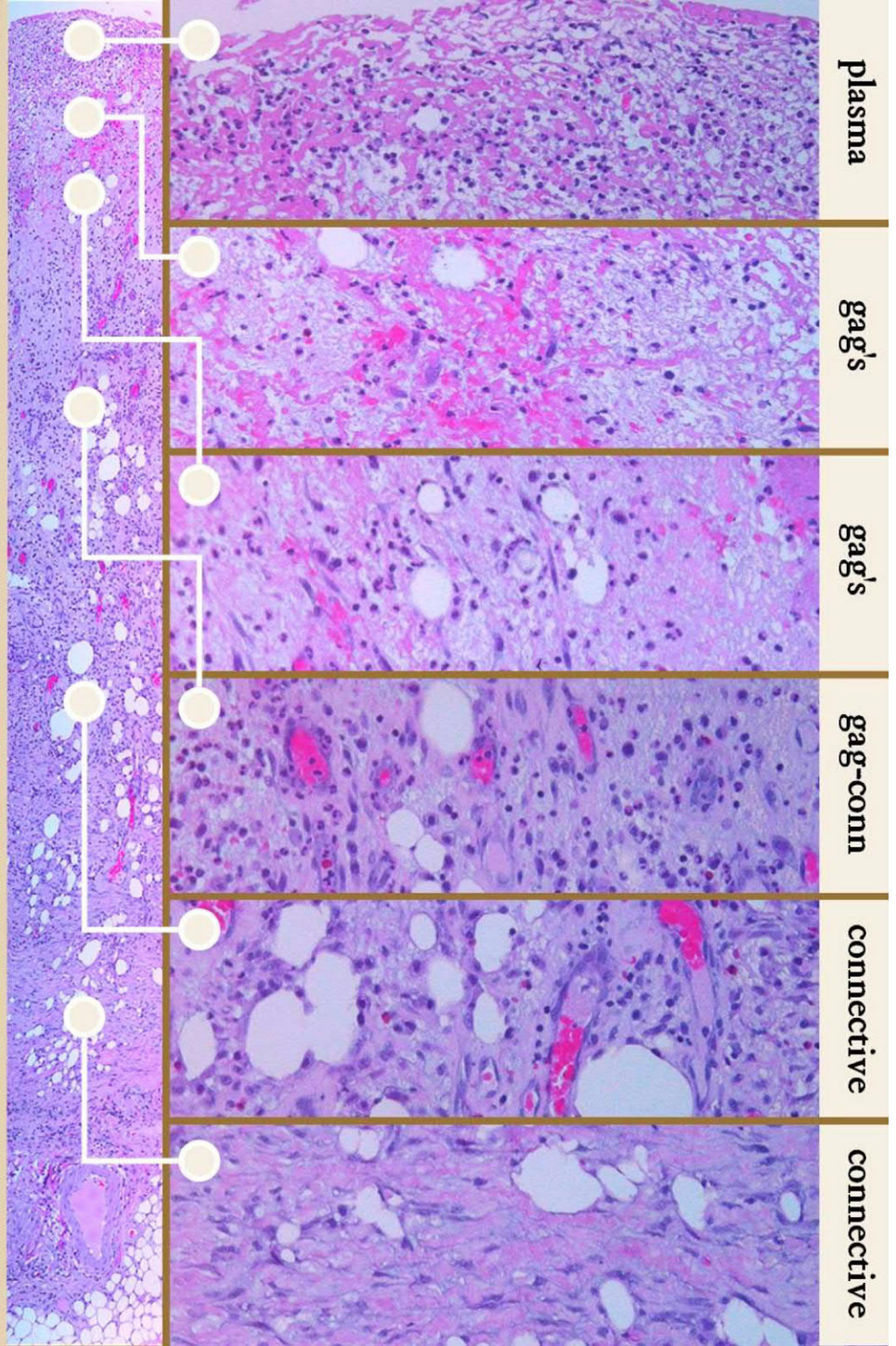






III
Aminoglycan
Ground Substance

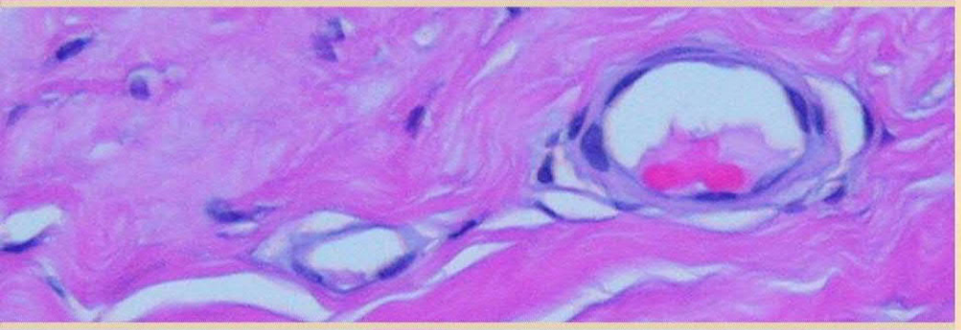


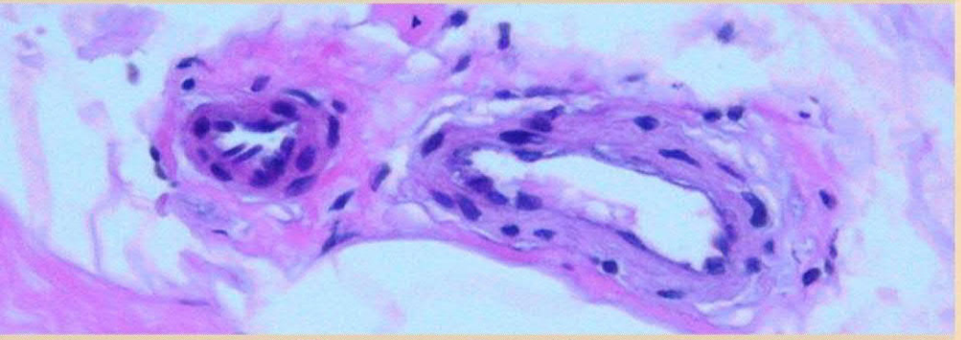


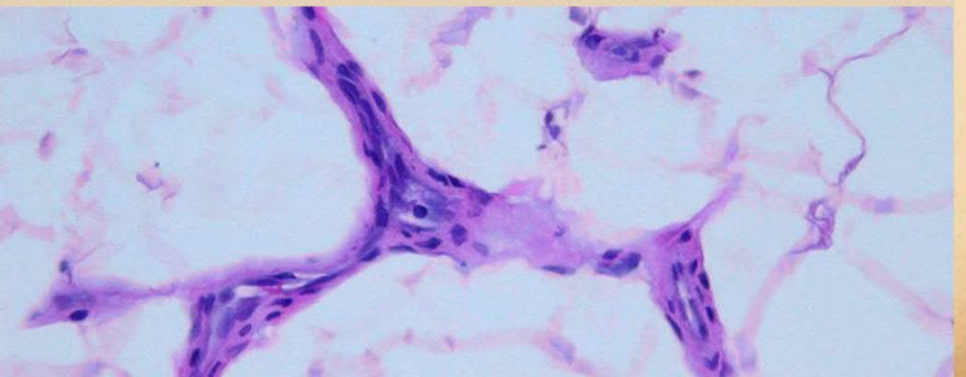
Stimulated

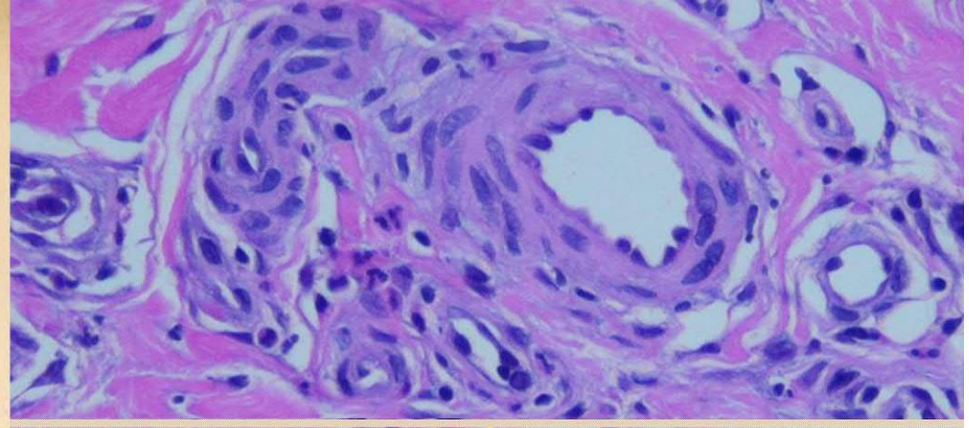
IV a
Angiocytes & Histogenetic Cells

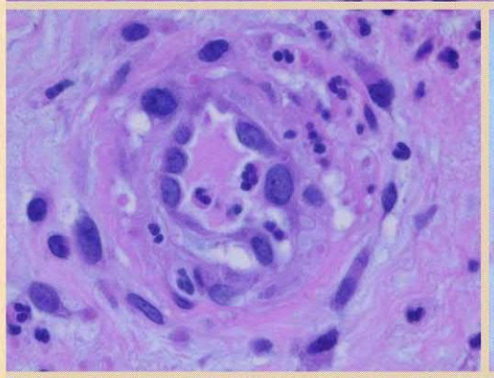
Normal

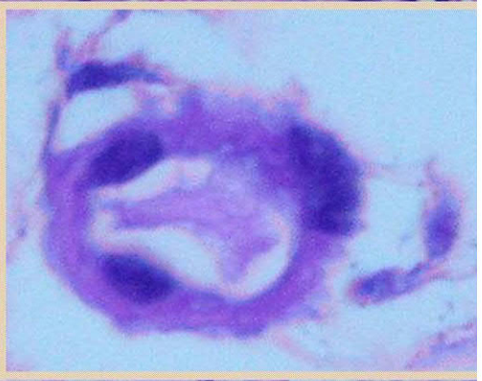


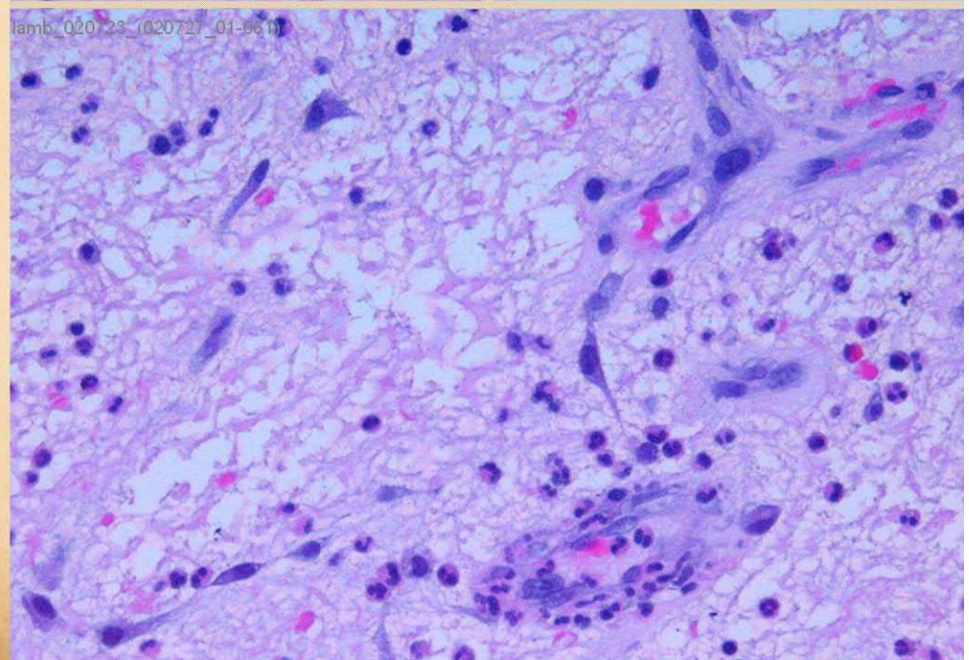




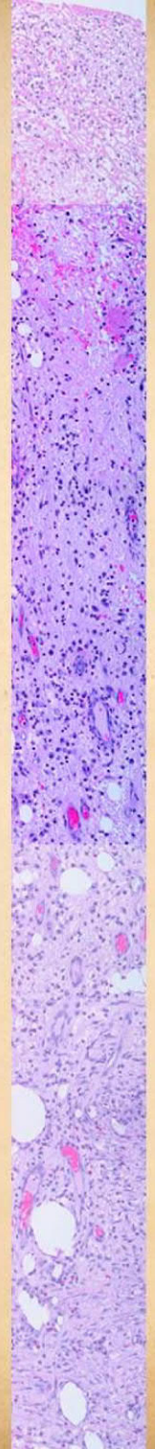


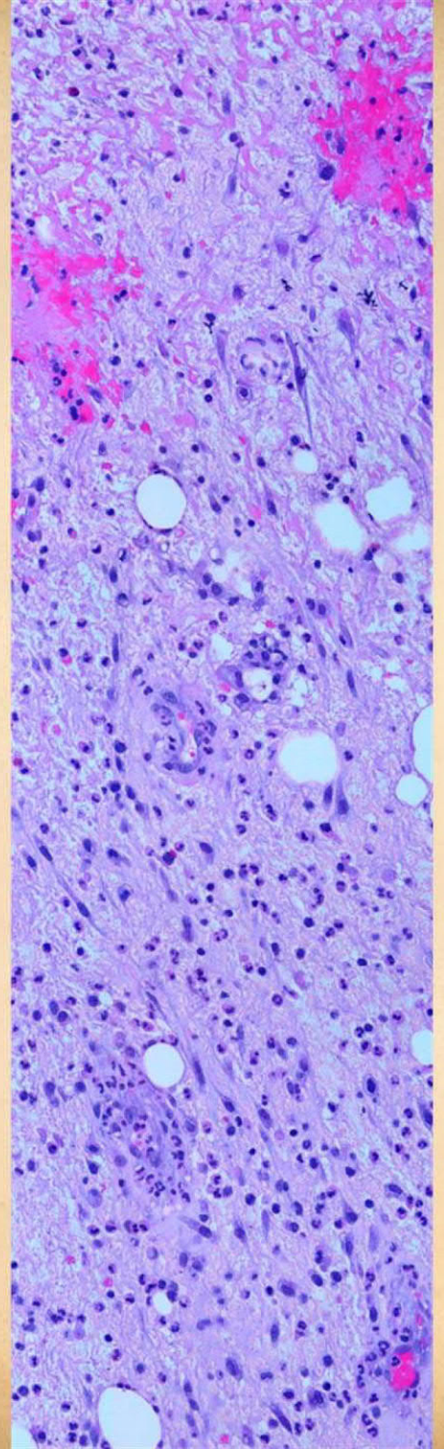


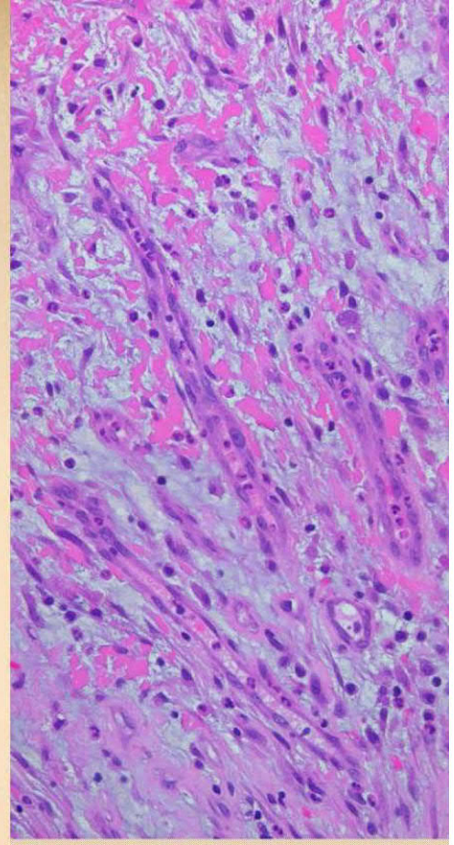


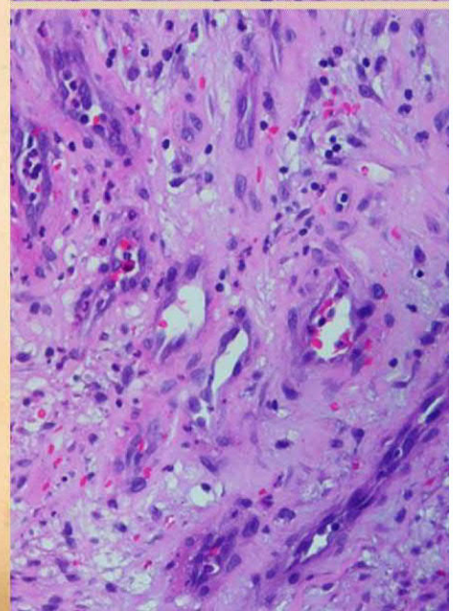


IV b
Angiogenesis & Granulation Tissue

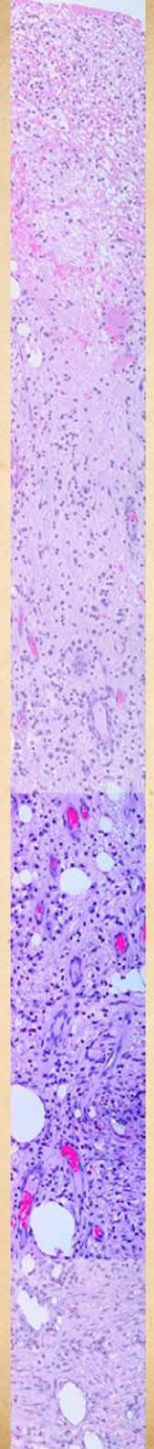


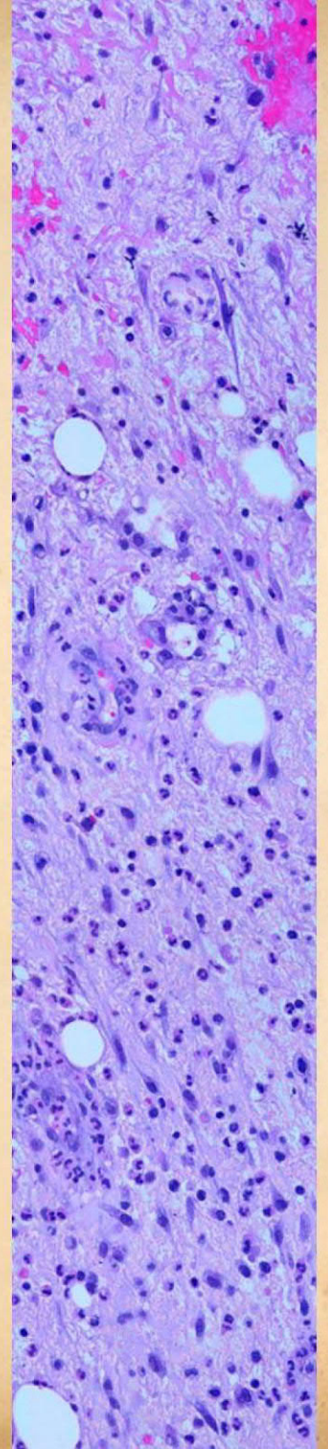


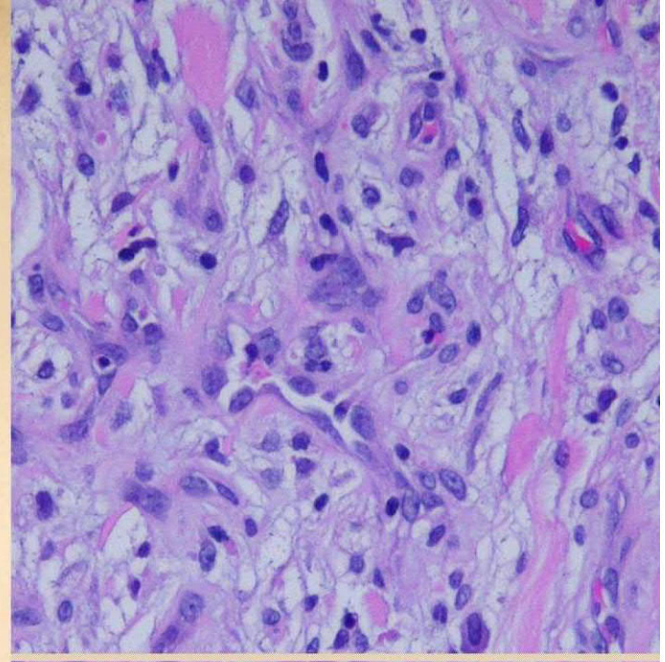


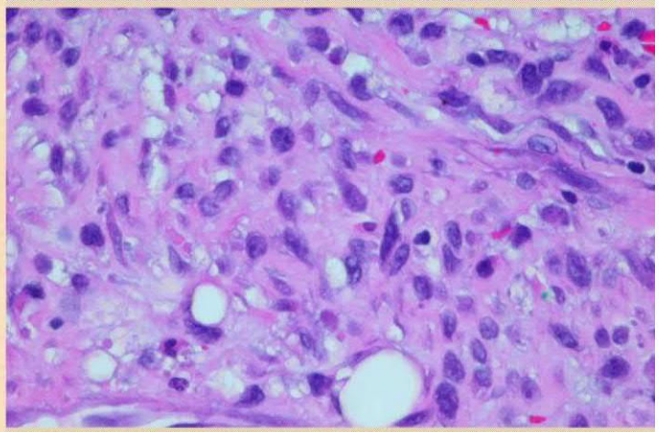


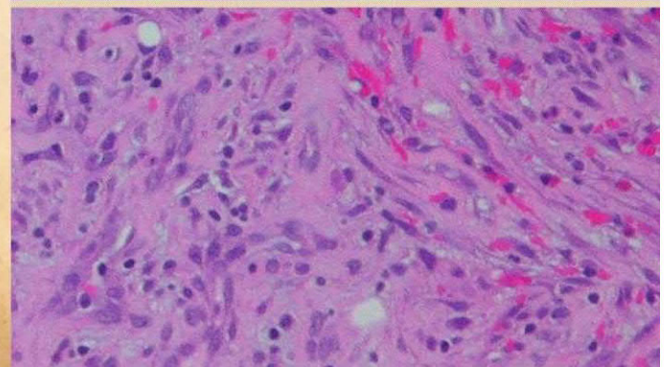
V a
Fibroblasts & Connective Matrix





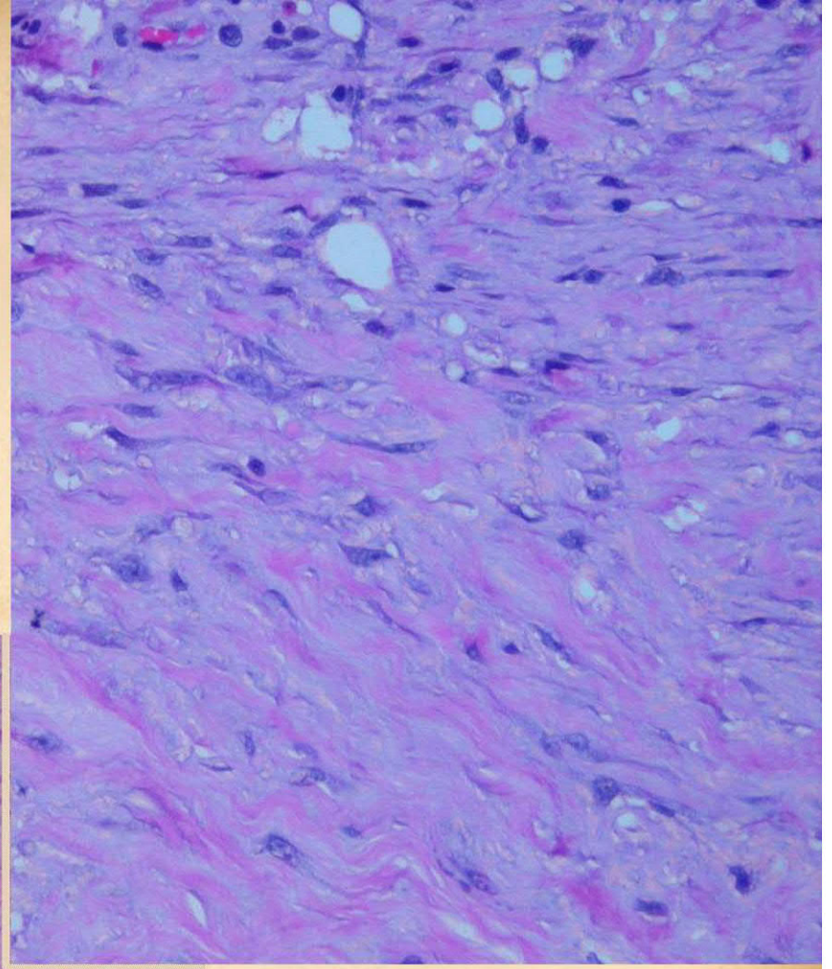


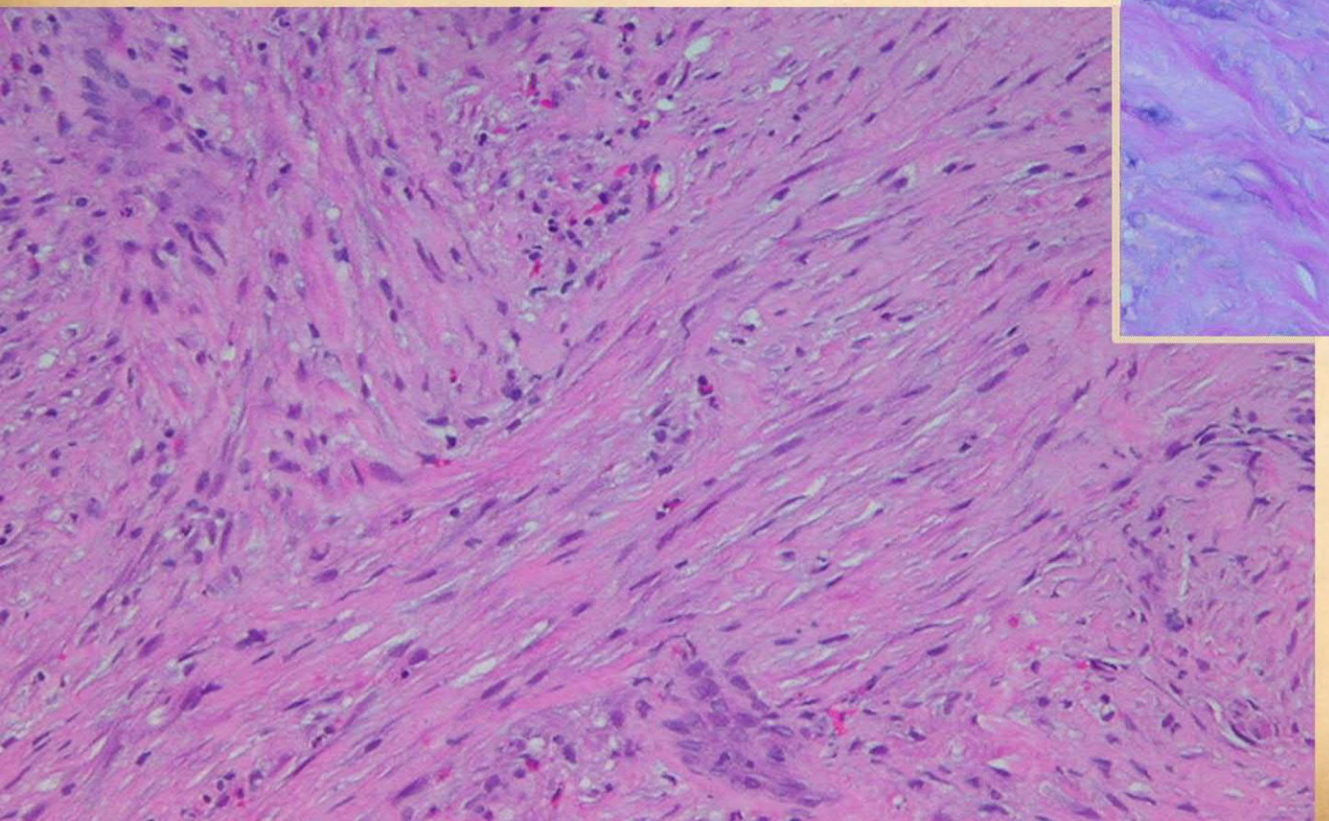


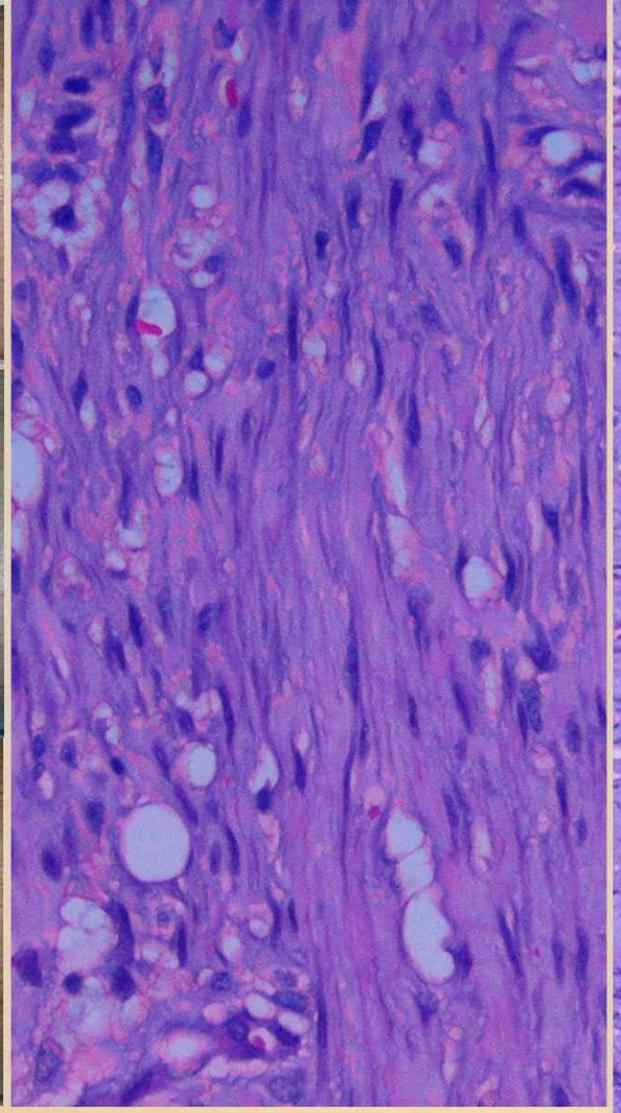


V b

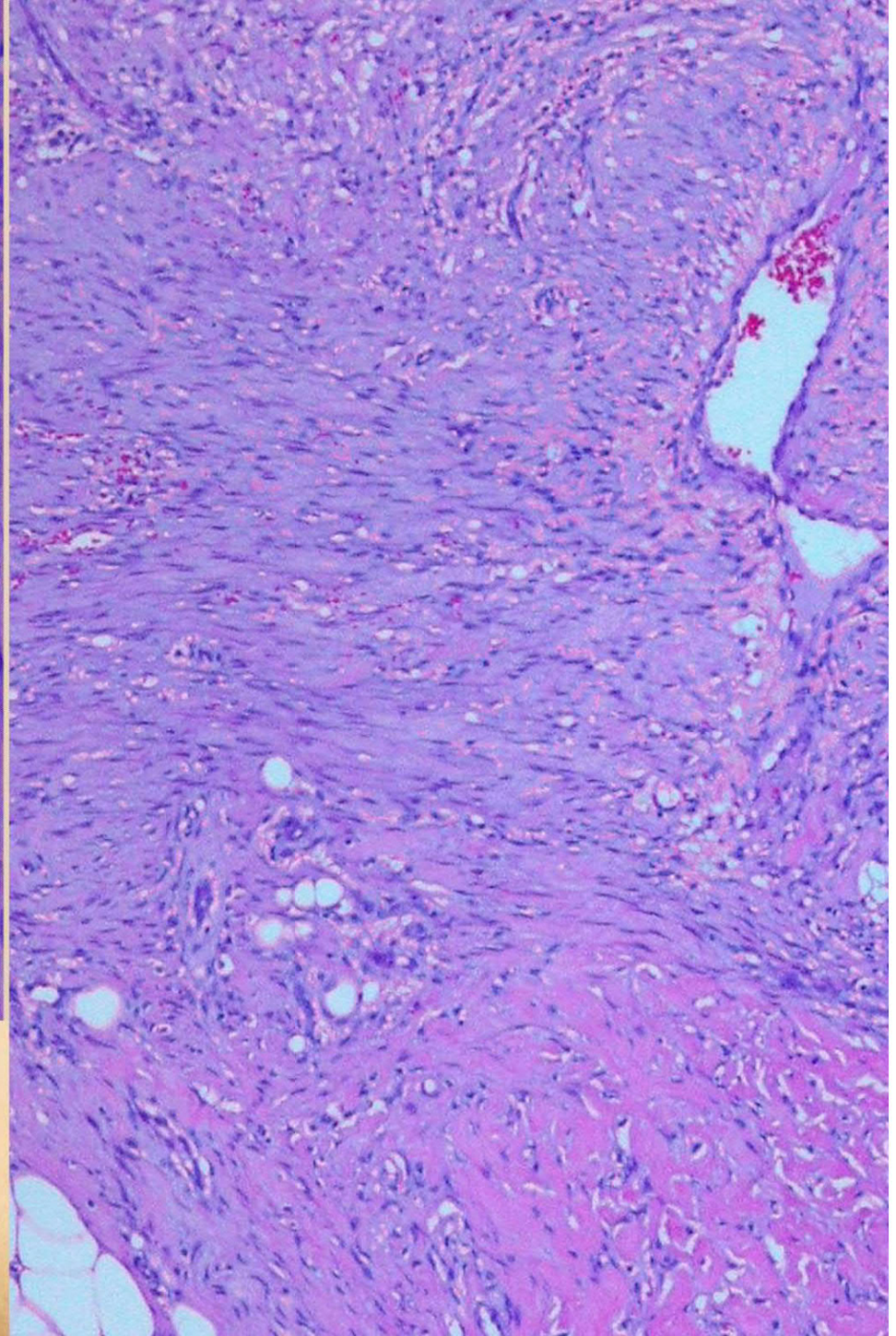
Fibroplasia & Scar



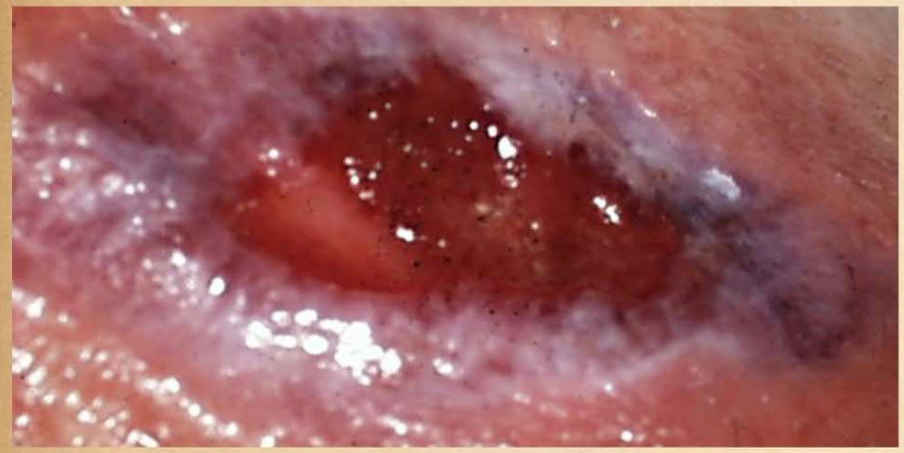


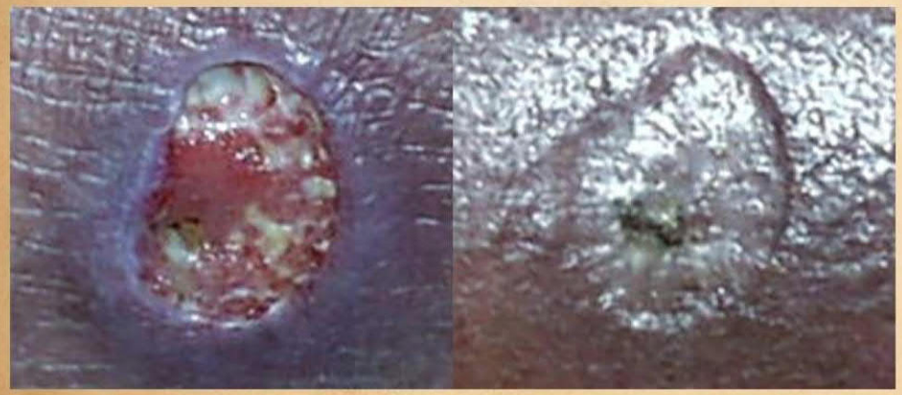


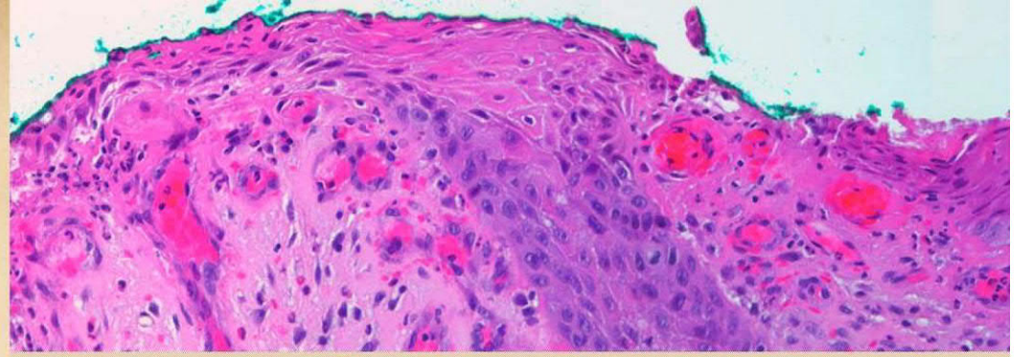
VI
Myofibroblasts & Contraction

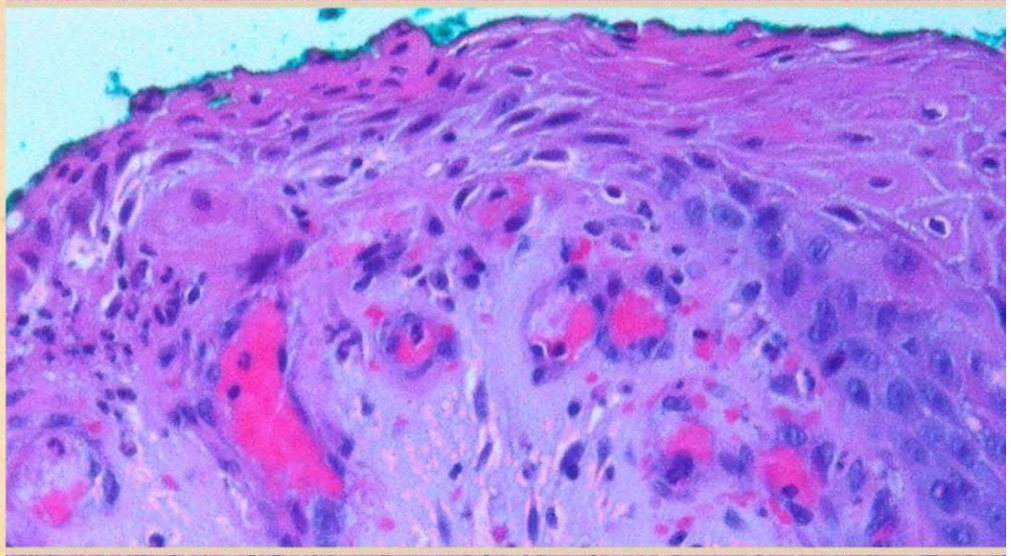


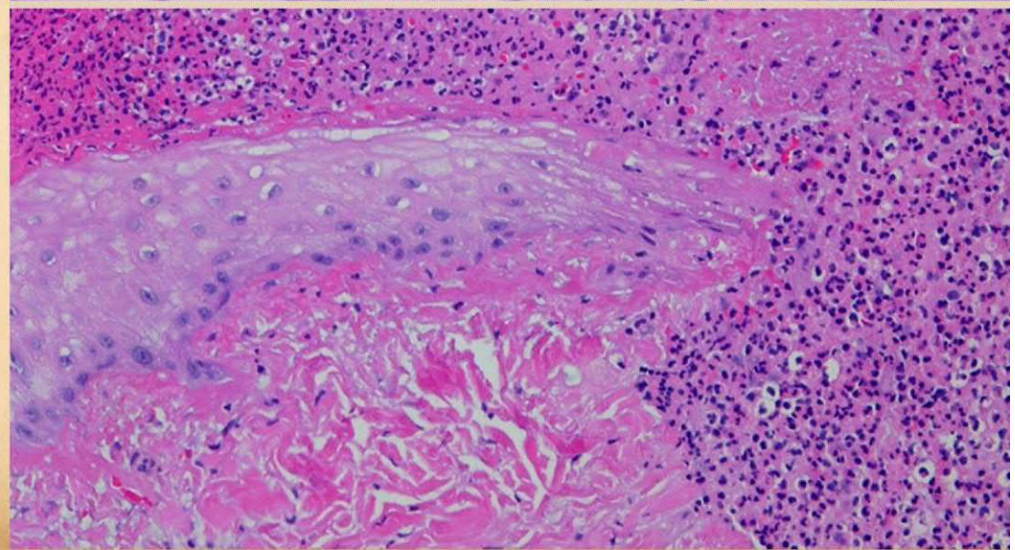
VII
Epithelialization & Closure



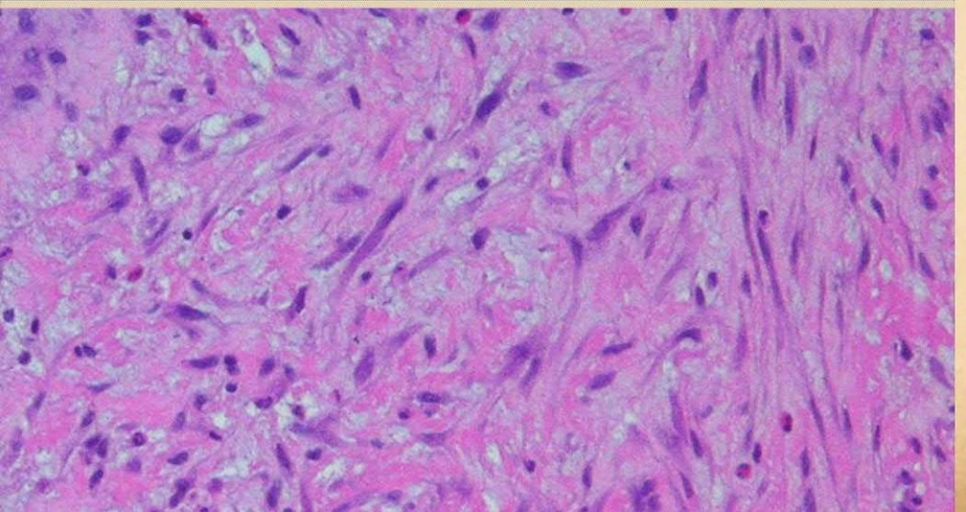


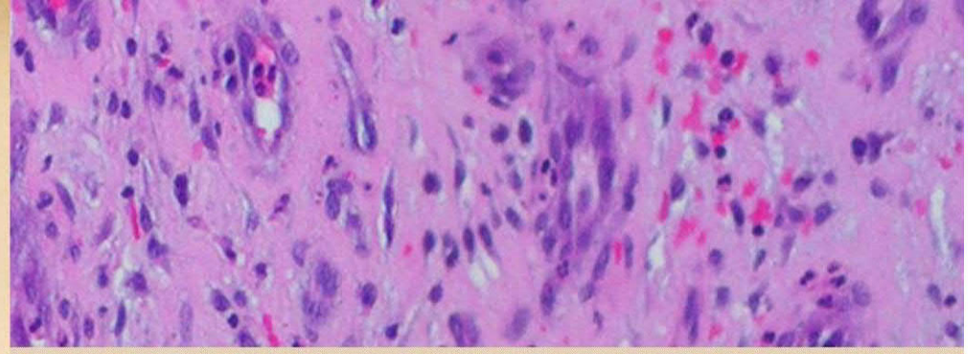


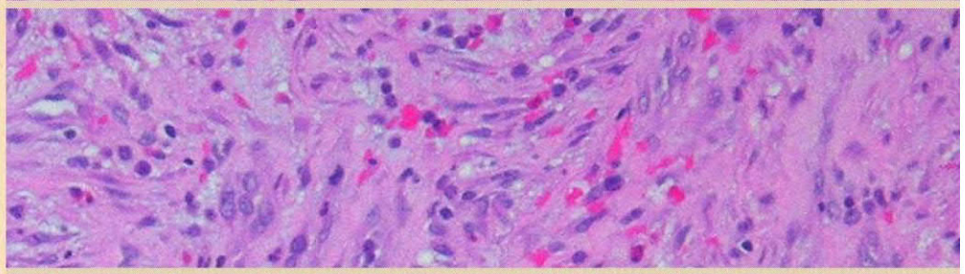


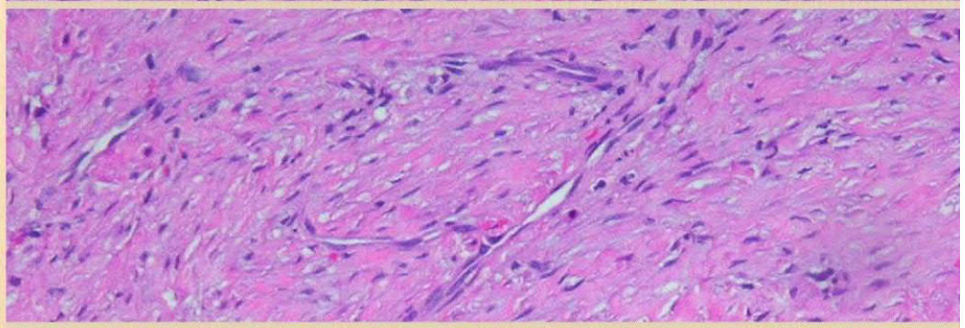


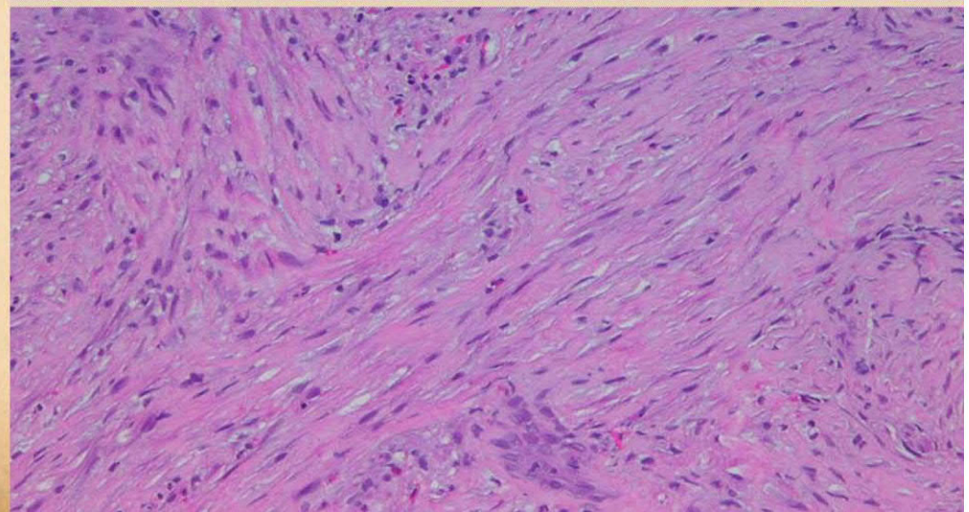
VIII a
Maturation
Consolidation of fibrous scar



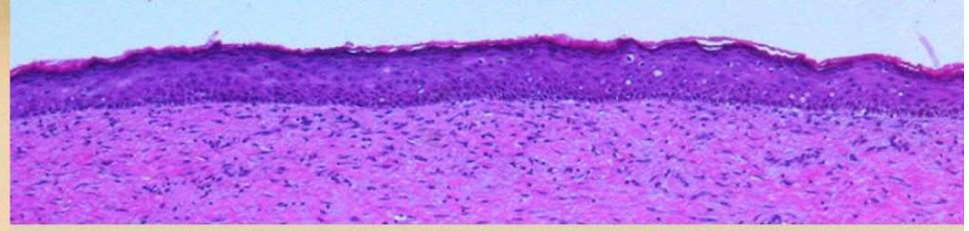


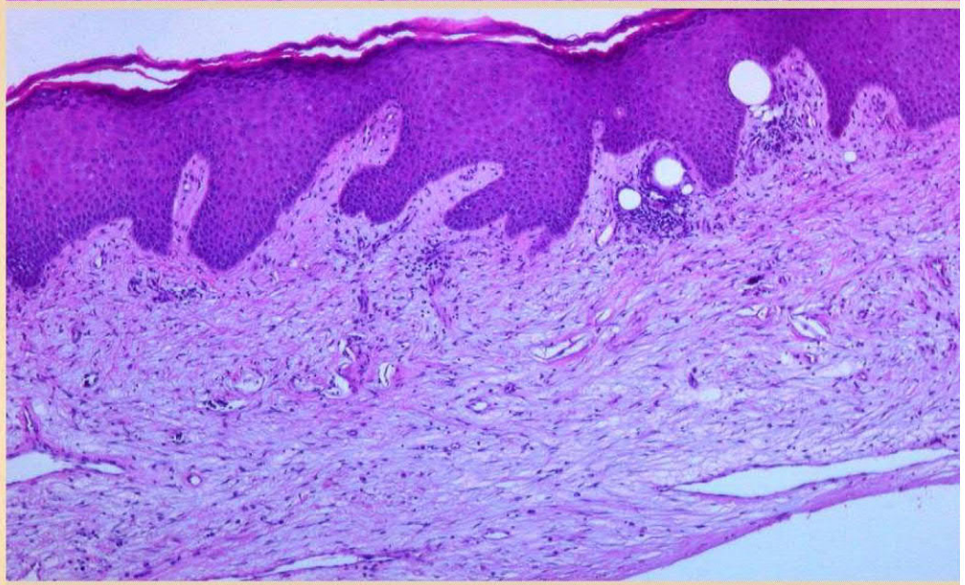


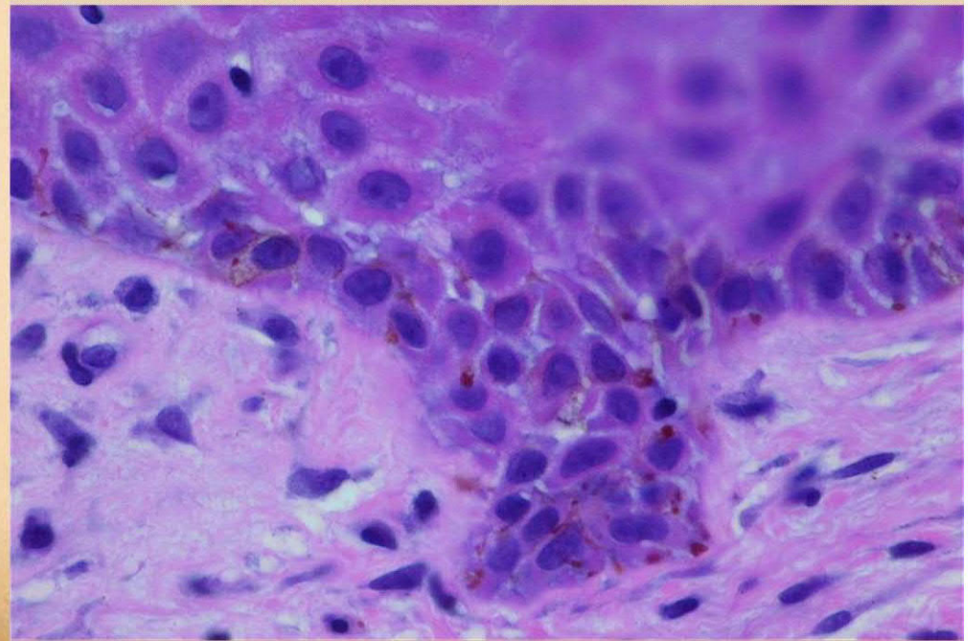




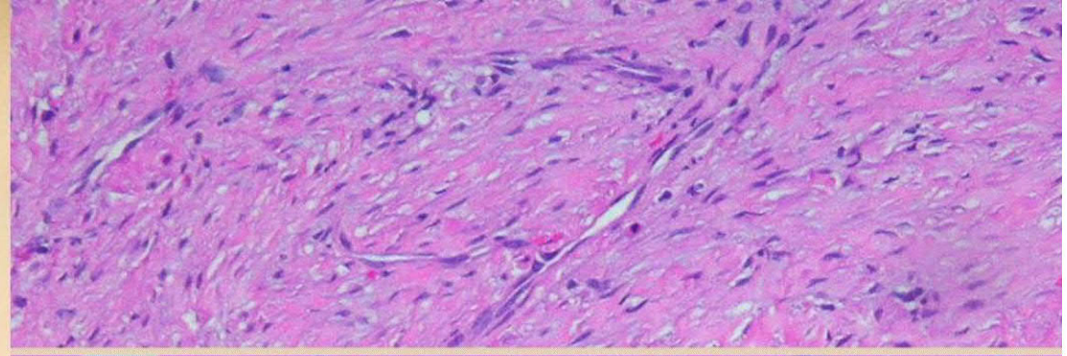
VIII b
Maturation
Epidermal papillation

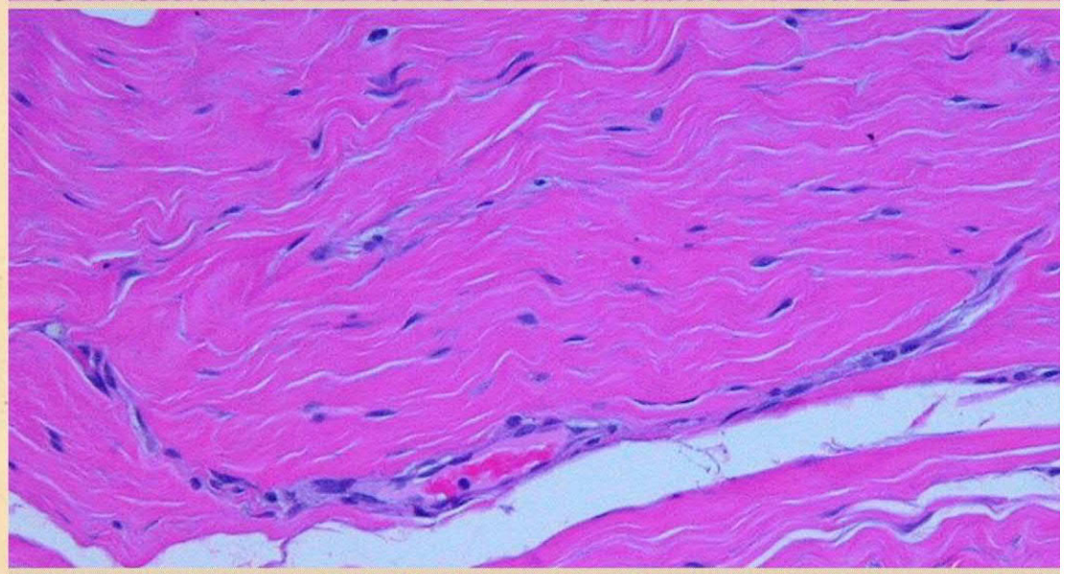


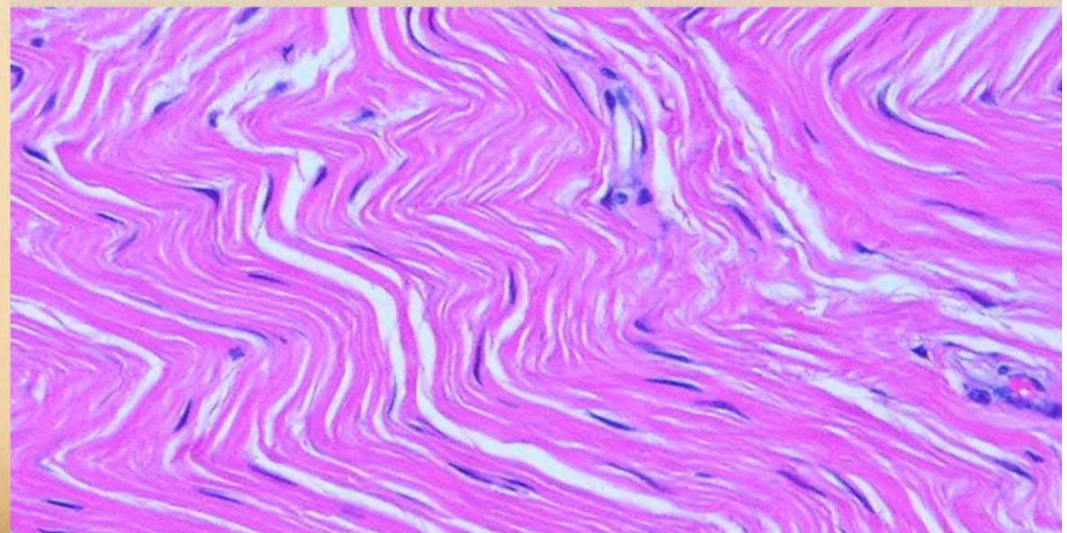




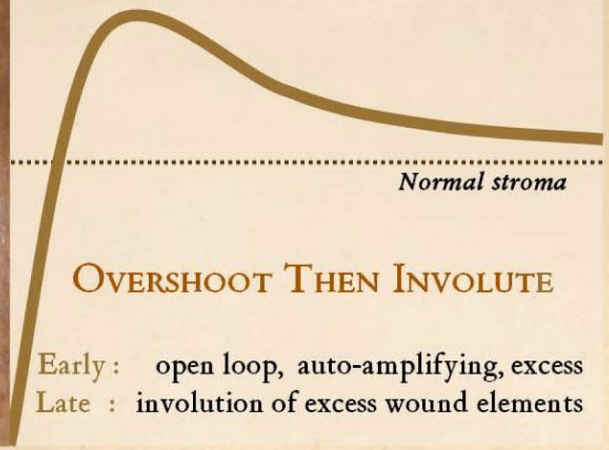
VIII c
Maturation
Involution





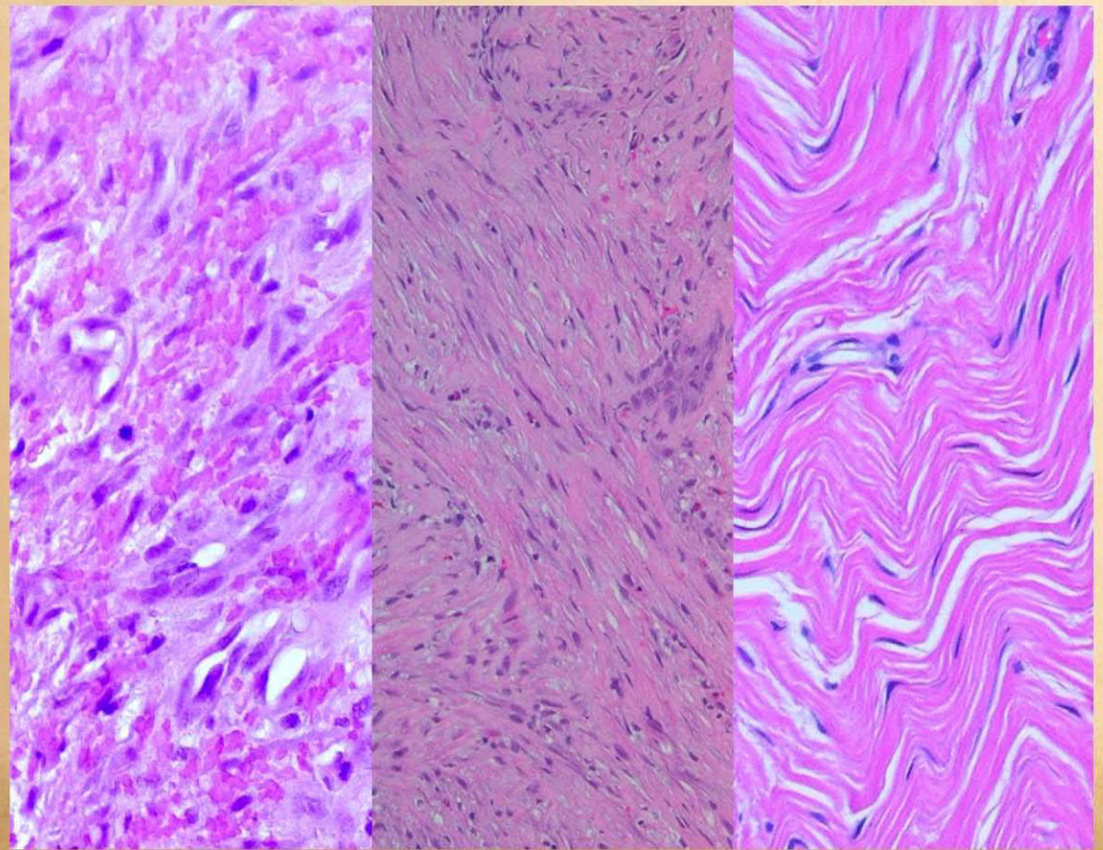


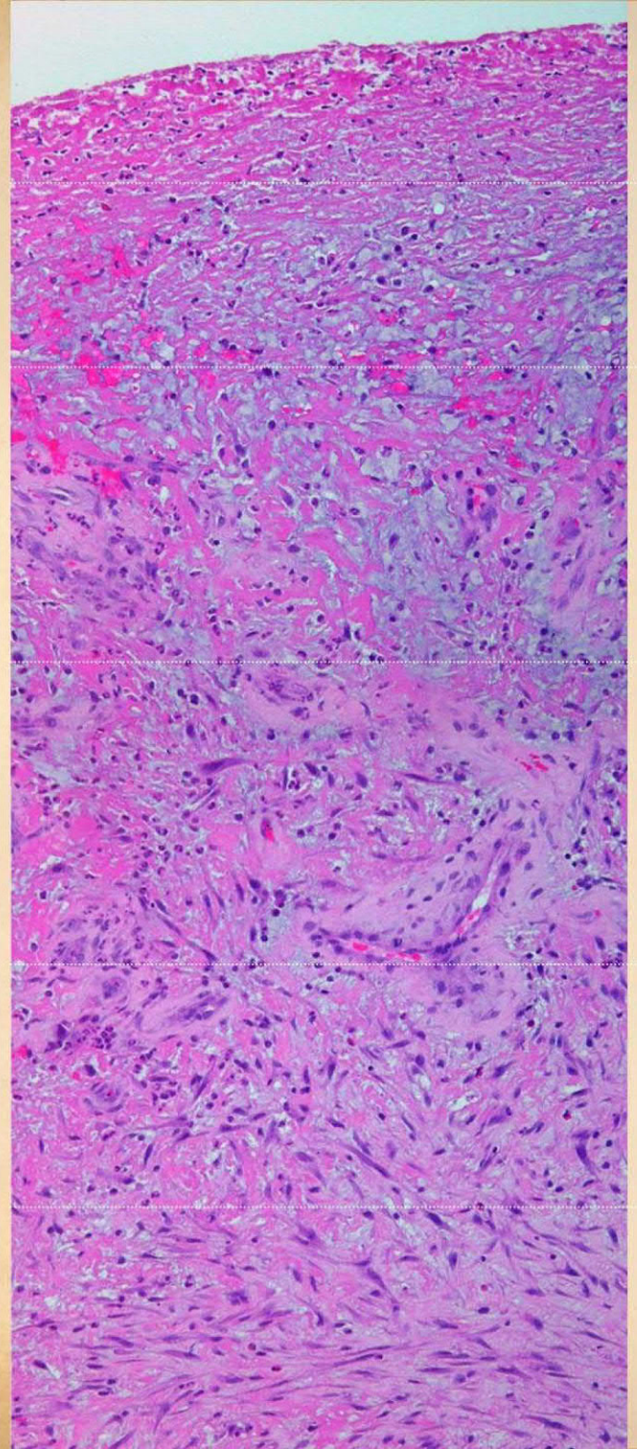
Inflammatory Wound Healing



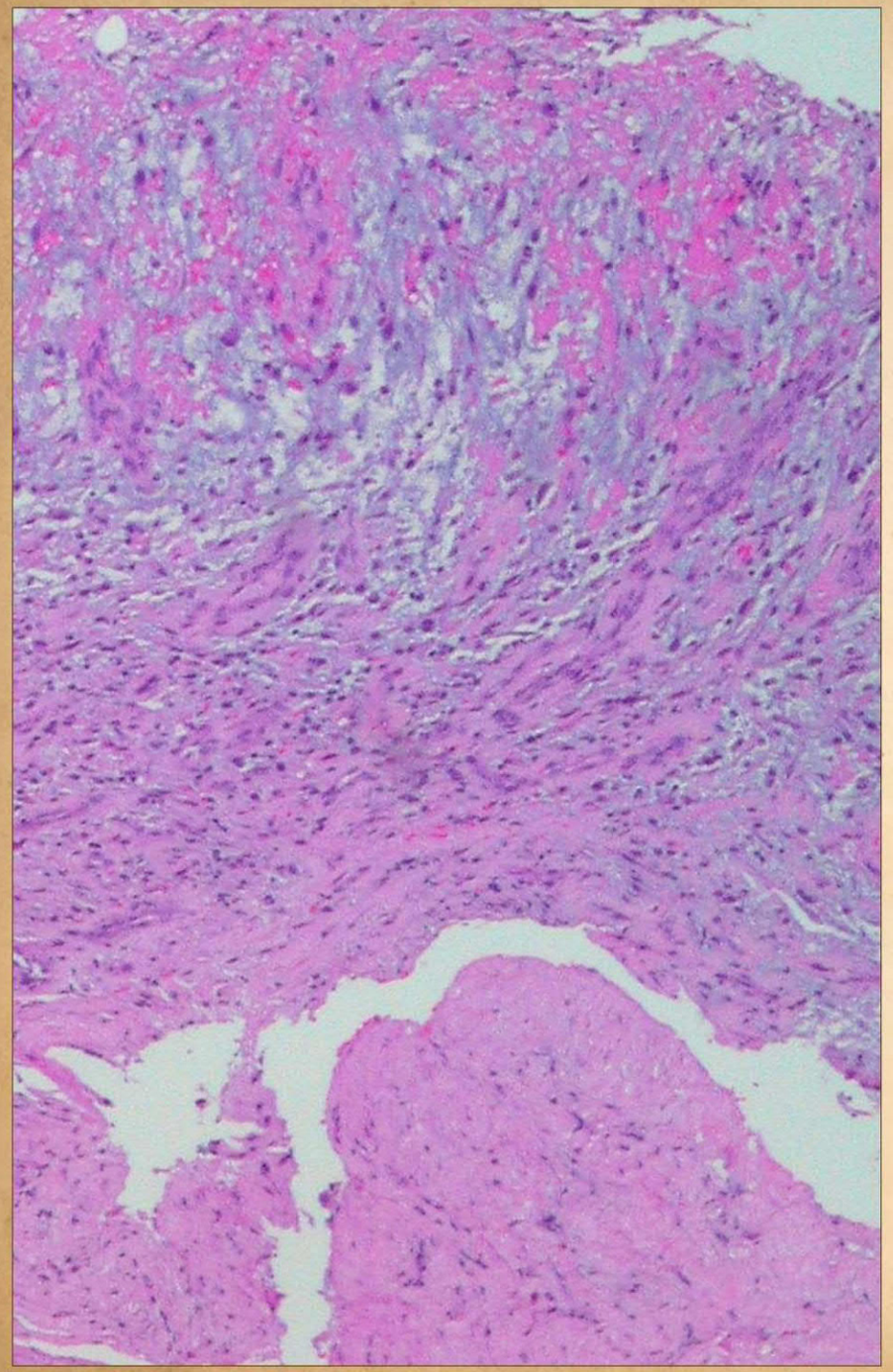
Overshoot Then Involute

Early : open loop, auto-amplifying, excess
Late : involution of excess wound elements



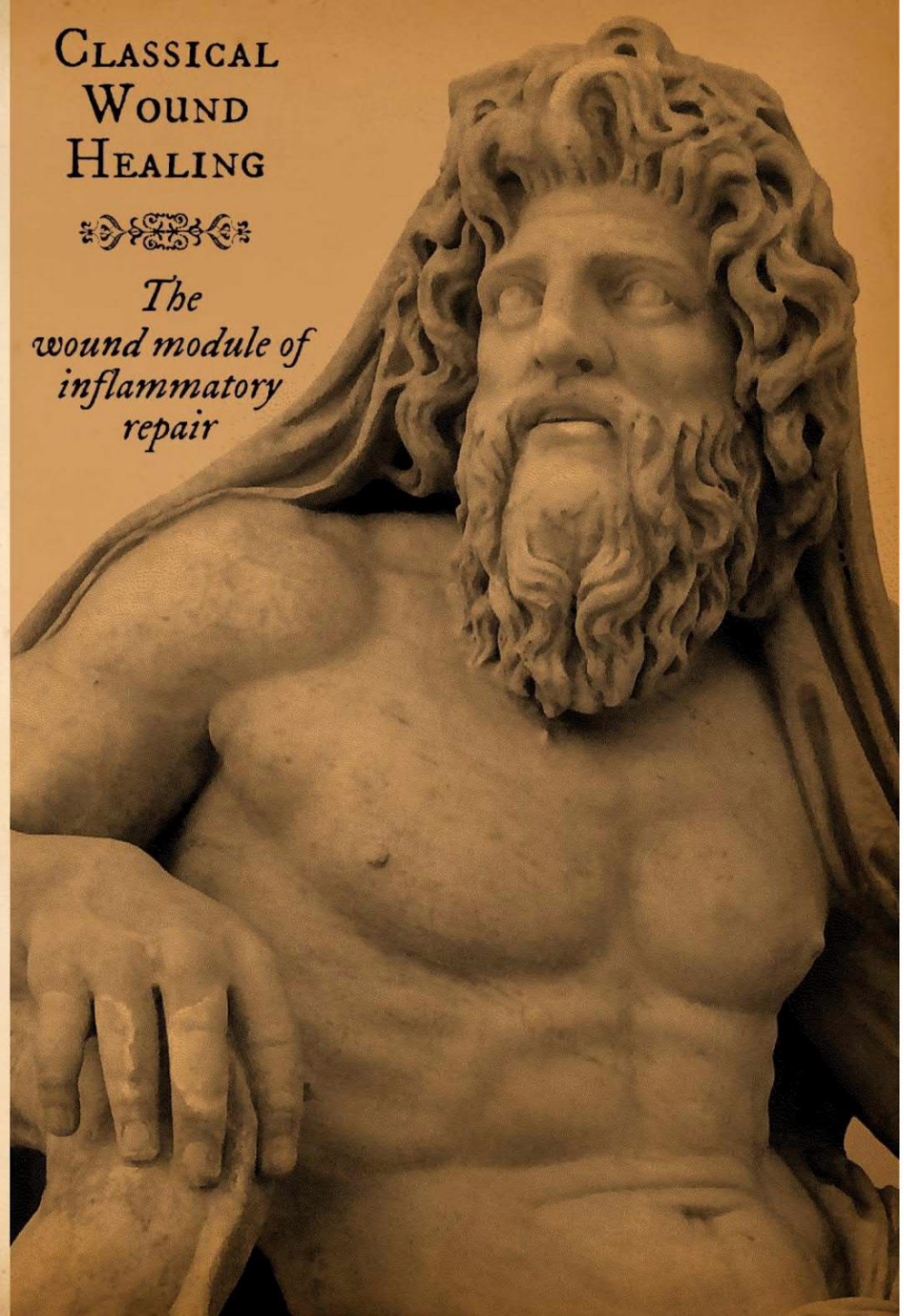


plasma
gag's
gag's
gag-conn
connective
connective



Classical Wound Healing

The wound module of inflammatory repair



General Concepts

Wound Healing Biology

CAP
Chronic and Pathological
Wounds

Wound Management

Pressure Ulcers

ACTIVE ULCERATION
NON-HEALING WOUNDS
CHRONIC WOUNDS

CAUSES

Wound healing (stromal restoration), depends on two quintessential "perfect", cells fibroblasts and angiocytes.
It is an ancient biological "subroutine", robust, fully "debugged", highly conserved.

There are virtually NO INTRINSIC diseases of wound healing.
When wounds are actively ulcerating, or they are not healing, it is because :

ANATOMICAL - PATHOLOGICAL

Thrombo-infarctive pattern of ulceration

Inflammatory-lytic pattern of ulceration

Each has its own causes (or mixed).
Make the diagnosis in order to treat successfully.

Persistent disease or injury - vs - impaired healing

DYNAMICAL

Disorders of privation or deprivation

Disorders of predation or depredation

Disorders of disorganization (dysdynamia)

Each has its own causes (or mixed).
Make the diagnosis in order to treat successfully.

NECROSIS & ULCERATION - TWO GENERAL PATHOLOGIES & PATTERNS

THROMBO-INFARCTIVE

Macro-occlusive
Micro-occlusive
Micro-angiopathies
Hemopathologies
Hypercoagulable / Coagulopathic

INFLAMMATORY-LYTIC

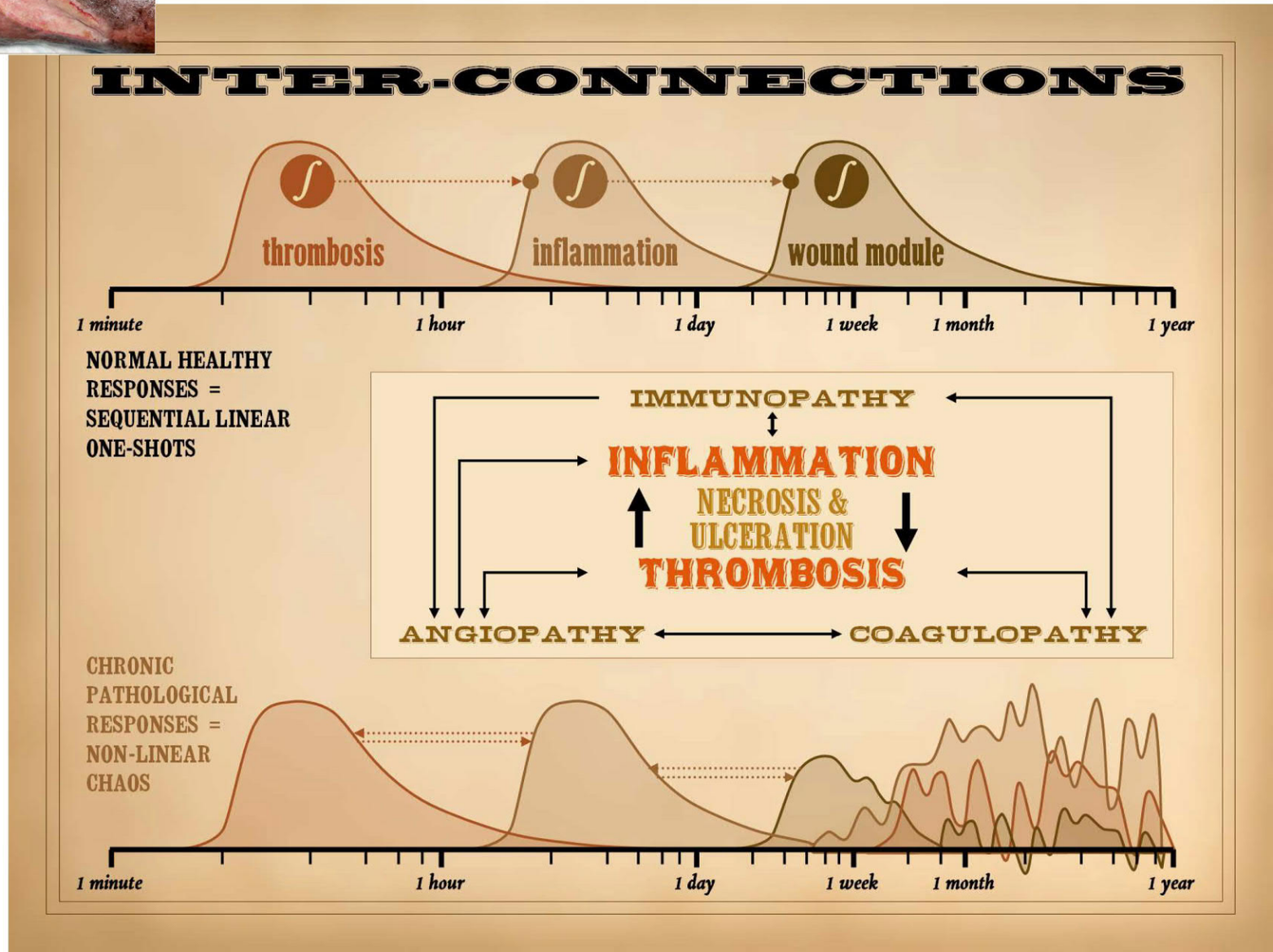
Inflammatory
Autoimmune
Atopic, Suppurative
Connective Tissue Disorders
Lymphoreticular / Reticuloendothelial





18 months
Rheumatoid, pyoderma


18 months
Sickle disease


INTER-CONNECTIONS
thrombosis
inflammation
wound module
1 minute
1 hour
1 day
1 week
1 month
1 year
NORMAL HEALTHY RESPONSES = SEQUENTIAL LINEAR ONE-SHOTS
IMMUNOPATHY
INFLAMMATION
NECROSIS & ULCERATION
THROMBOSIS
ANGIOPATHY
COAGULOPATHY
CHRONIC PATHOLOGICAL RESPONSES = NON-LINEAR CHAOS
1 minute
1 hour
1 day
1 week
1 month
1 year

WOUND DIAGNOSIS

THE CAUSE

risks

injuries

disease

THE STATE

anatomical

dynamical

functional

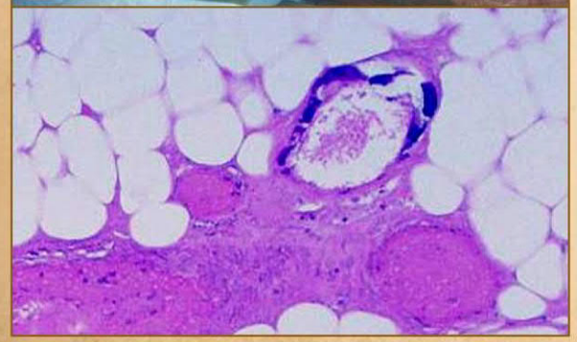
ARTERIAL

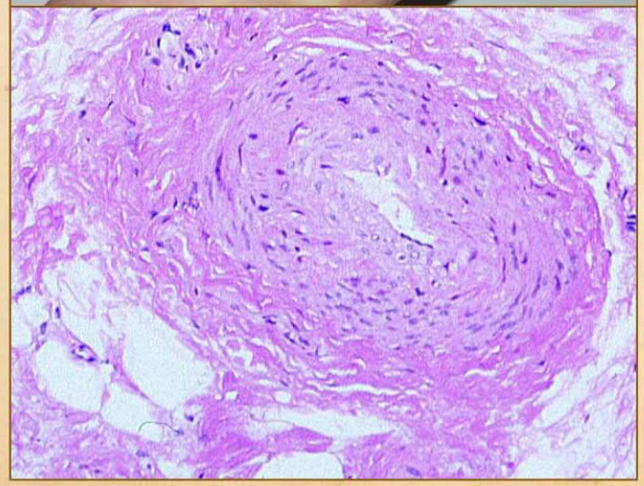
VENOUS

DIABETES

PRESSURE

ARTERIAL DISEASES

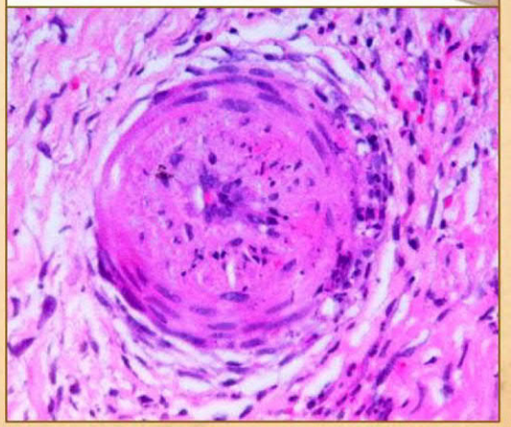




MICRO-OCCLUSSIVE DISORDERS

Angiopathies

Hemopathologies



Hemoglobinopathies

sickle cell disease
thalassemias
other hemolytic anemias

Dys- & cryoproteinemias

cryoglobulinemia
cryfibrinogenemia
macroglobulinemia & myeloma

Hematocytes & platelets

polycythemia rubra vera
hereditary spherocytosis
thromb. thrombocytopenic purpura
myeloproliferative disorders
leukemias

Hypercoagulable & prethrombotic disorders




IMMUNOPATHY – CVD - CTD

DERMATOSES AND PANNICULOPATHIES

TOXIC & METABOLIC

hydrofluoric
arsenicals
caustics
misc industrial

chemotherapy
antimetabolites

corticosteroids

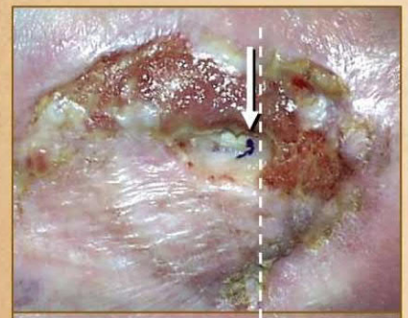
actinic exposure
radioactives
radiotherapy

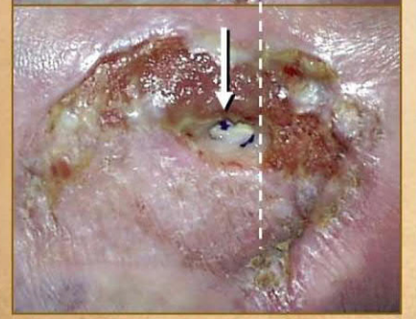
sarsaparilla

infliximab
rapamycin

DIABETES & NEUROPATHY

MECHANICAL



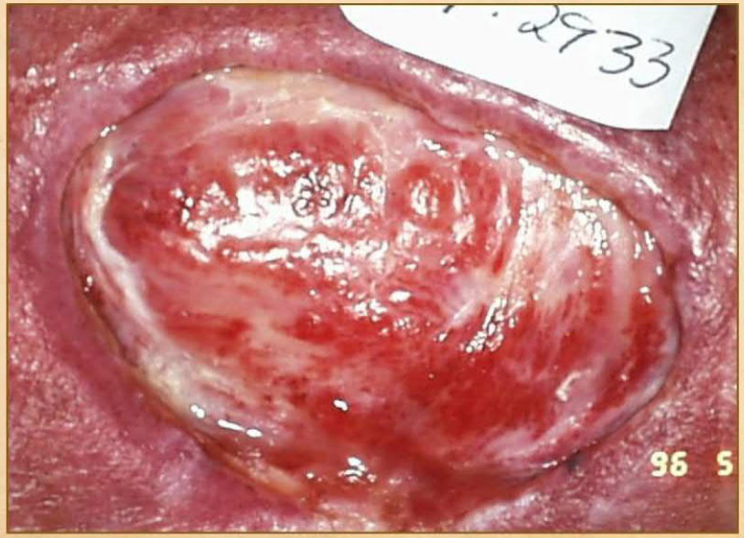


INFECTIOUS

CANCER

FACTITIOUS & IATROGENIC

MIXED DIAGNOSES



WOUND DIAGNOSIS

THE CAUSE

risks

injuries

disease

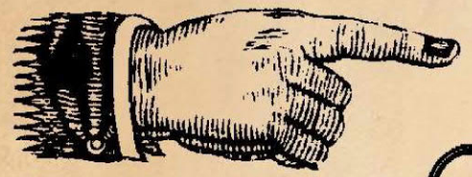
THE STATE

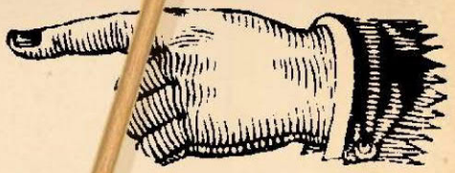
anatomical

dynamical

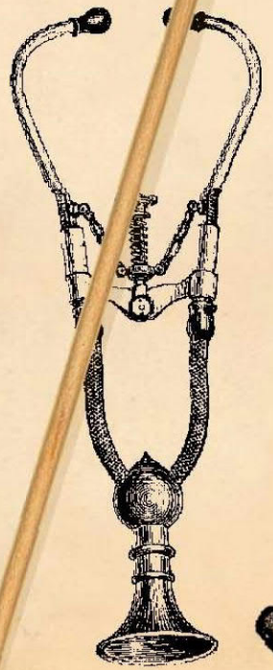
functional

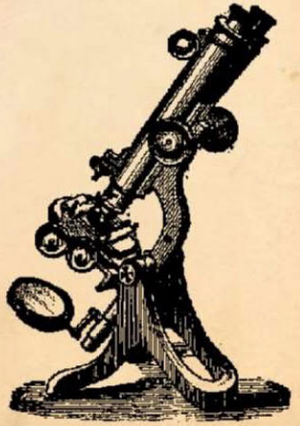
DIAGNOSTIC TOOLS





Books / Knowledge
Ears
Eyes
Hands
Q-tip
Curette / Scissors
First 2 weeks of care
Laboratory





HISTORY

IMMUNOPATHY

malaise
fevers
myalgias
arthralgias
joint areas
symmetry
morning stiffness
rashes
purpura
nodules
allergies
drug reactions
photosensitivity
oral ulcers
pleurisy
renal disorders
ocular problems
pulmonary disorders
neurologic disorders
hematologic disorders
genitourinary problems
vascular symptoms
sicca
calcinosis
skin lesions
problem wounds
pathergy
thrombosis
complications of trauma

VASCULAR

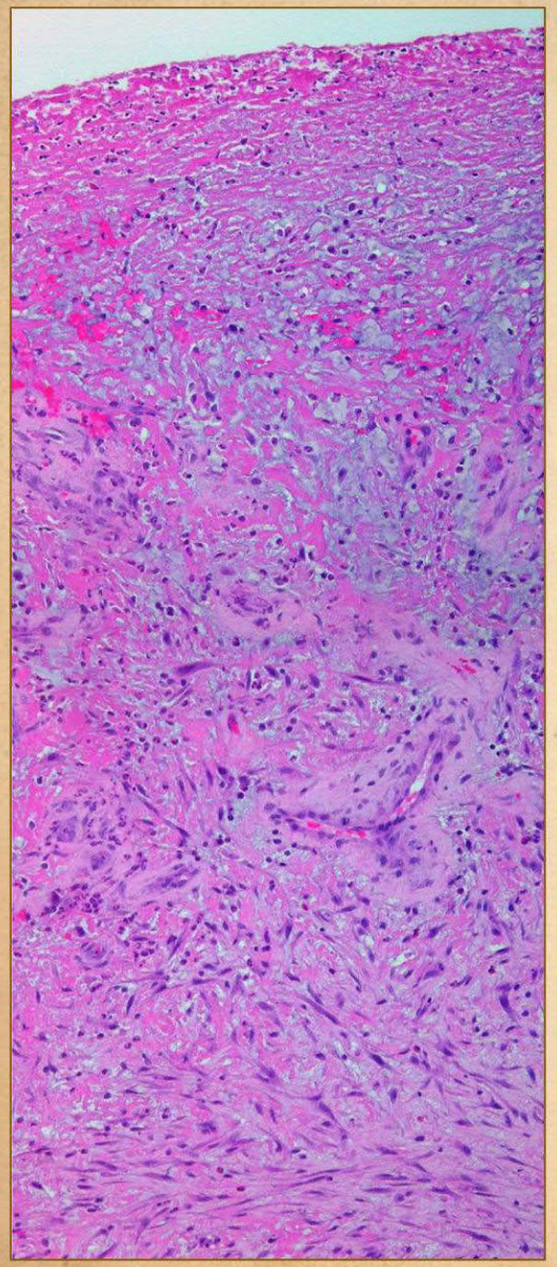
rest pain
claudication
postural pain
postural edema
non-postural edema
thrombosis, embolism
blindness, amaurosis
stroke, tia
renal disease
pulmonary disease
cardiovascular disease
metabolic disorders
dietary disorders
immunopathies
coagulopathies
hypertension
diabetes
smoking
activities
medications
surgical history
non-healing wounds

PRESSURE

circumstances
timewise history
surgical history
spinal level
dysreflexia
spasticity
domicile
family status
independence
economic security
neurological status
physical adaptation
psychological status
psychiatric history
ulcer history
bladder care
bowel care
posture
seating
bedding
footwear
hygiene

COAGULOPATHY

arterial thrombosis
venous thrombosis
thrombosis syndromes
graft & valve thrombosis
pulmonary thrombosis
heart attack, angina
visceral apoplexy
nephropathy
stroke, tia
embolism
blindness
skin ulcers
pathergy
complications of surgery
complications of trauma
non-healing wounds
venous disorders
immunopathies
blood disorders
cancer
chronicity
recurrence
recalcitrance
warfarin necrosis
warfarin resistance
oral contraceptives
miscarriage
age spectrum
family history



THE WOUND MODULE

OF PROLIFERATIVE REPAIR

and the

CLINICAL SIGNS OF WOUND HEALING

0
injury
inflammation

1
inflammation
subsides

2
macrophages,
eschar separation,
cytokines

3
ground substance,
mucus

4
"granulation"
angiogenesis

5
histioblasts, fibroblasts,
fibroplasia

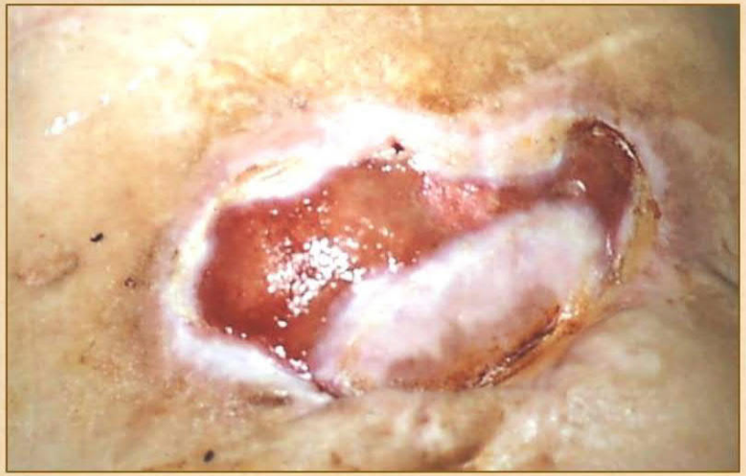
6
myofibroblasts
contraction

7
epithelialization

8
maturation

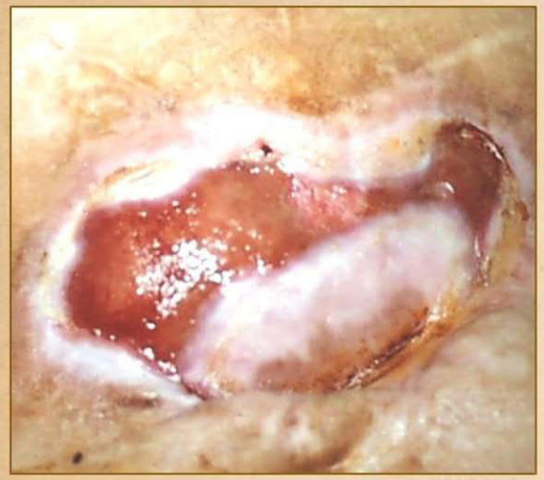
Physiological Diagnosis of Wounds

Is it healing or not?



Physiological Diagnosis of Wounds

Wound Healing Competent or Not?



Events, Signs

0 - injury, inflammation

1 - inflammation subsides

2 - macrophages, cytokines, eschar separation

3 - ground substance, mucus

4 - angiogenesis, granulations

5 - fibroblasts, fibroplasia

6 - myofibroblasts, contraction

7 - epithelialization

8 - maturation

Physiological Diagnosis of Wounds

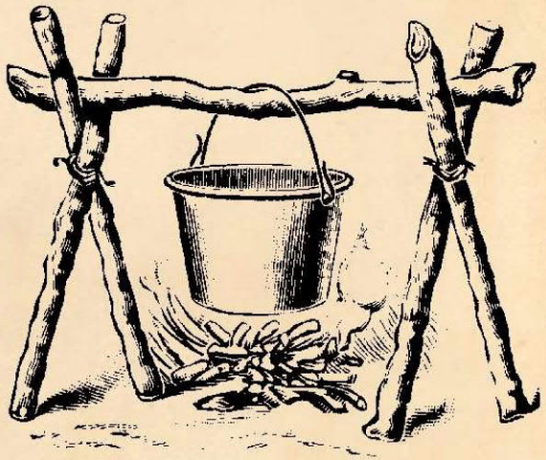
The Importance of Sequential Observation

The State of the Wound: Dynamics & Kinetics

healing
chaotic or stalled
not healing
progressive ulceration

wound healing competent
wound healing retarded
wound healing incompetent

active disease and injury
impaired wound healing



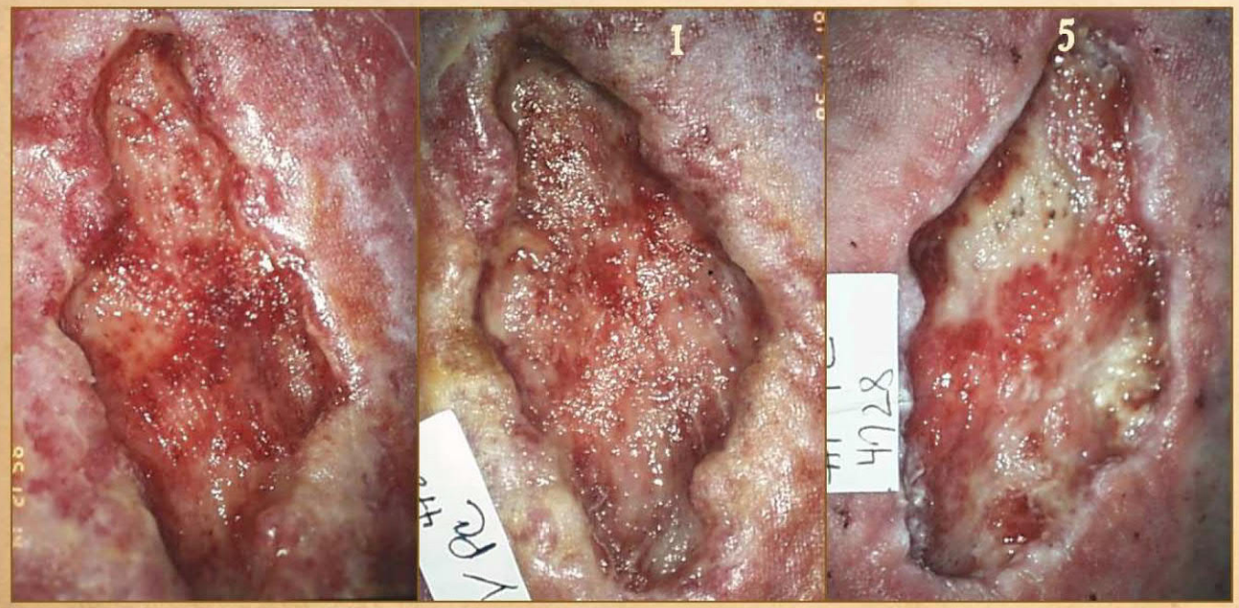
"A watched pot never boils."

THE STATE OF THE WOUND: DYNAMICS & KINETICS

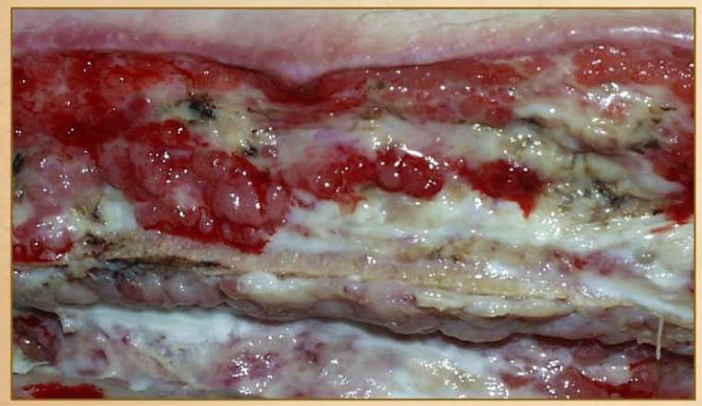
healing

chaotic or stalled

progressive ulceration



THE STATE OF THE WOUND: DYNAMICS & KINETICS



wound healing competent
wound healing retarded
wound healing incompetent

THE STATE OF THE WOUND: DYNAMICS & KINETICS

active disease and injury

impaired wound healing

active pathology

wound failure, intrinsic

sustained injury

wound failure, extrinsic

THE MISDIAGNOSIS OF WOUNDS

1

NOT KNOWING THE SPECTRUM OF COMMON DISEASES AND DISORDERS

1

THE MISDIAGNOSIS OF WOUNDS

2

NOT KNOWING BASIC FACTUAL INFORMATION OR THE CURRENT BODY OF KNOWLEDGE

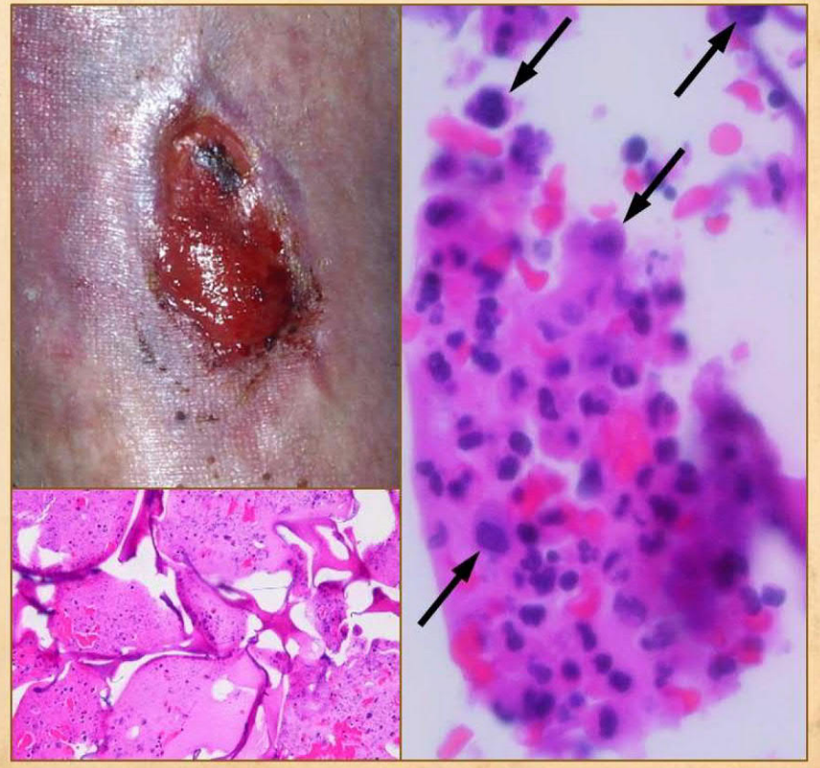
2

THE MISDIAGNOSIS OF WOUNDS

3

NOT UNDERSTANDING THE PHYSIOLOGY, PATHOLOGY, AND MECHANISMS OF DISEASE OF A GIVEN WOUND OR PATIENT

3



THE MISDIAGNOSIS OF WOUNDS

4

NOT PROPERLY ASSESSING THE WOUND AND PATIENT

4

RHEUMATOID

(4 of 7)

morning stiffness
3 or more joint areas
hand joints
symmetric arthritis
rheumatoid nodules
serum rheumatoid factor
radiographic changes

LUPUS

(4 of 11)

malar rash
discoid rash
photosensitivity
oral ulcers
arthritis
serositis
renal disorder
neurologic disorder
hematologic disorder
ant-DNA or LE prep
ANA

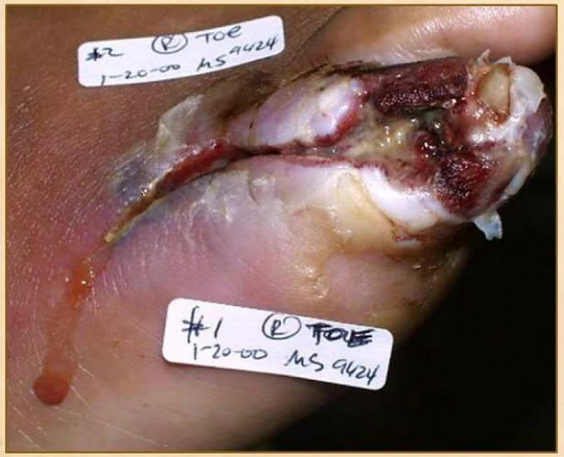
malaise myopathy pain uveitis sicca vasculitis angiopathy pathergy
dermatoses skin lesions ulceration panniculitis synovitis mucosal ulcers
calcinosis abdominal sx pneumonitis hepatitis nephritis urethritis atopy

THE MISDIAGNOSIS OF WOUNDS

5

IMPRECISE EXAM, ANALYSIS, KNOWLEDGE, VOCABULARY

5

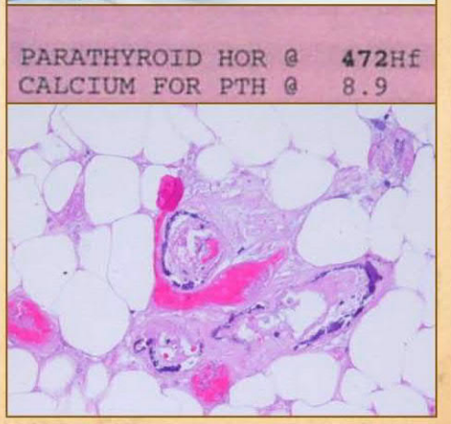


THE MISDIAGNOSIS OF WOUNDS

6

NOT KNOWING THE SPECTRUM OF MORE RECENTLY APPRECIATED DISORDERS

6



THE MISDIAGNOSIS OF WOUNDS

7

IGNORING THE INTRICACIES OF COMPLEX DISEASE

7






THE MISDIAGNOSIS OF WOUNDS

8

NOT KNOWING BASIC INFORMATION, COROLLARY: DEFAULT TO ERRONEOUS PRE-CONCEPTIONS

8

pressure
diabetic
venous
infection











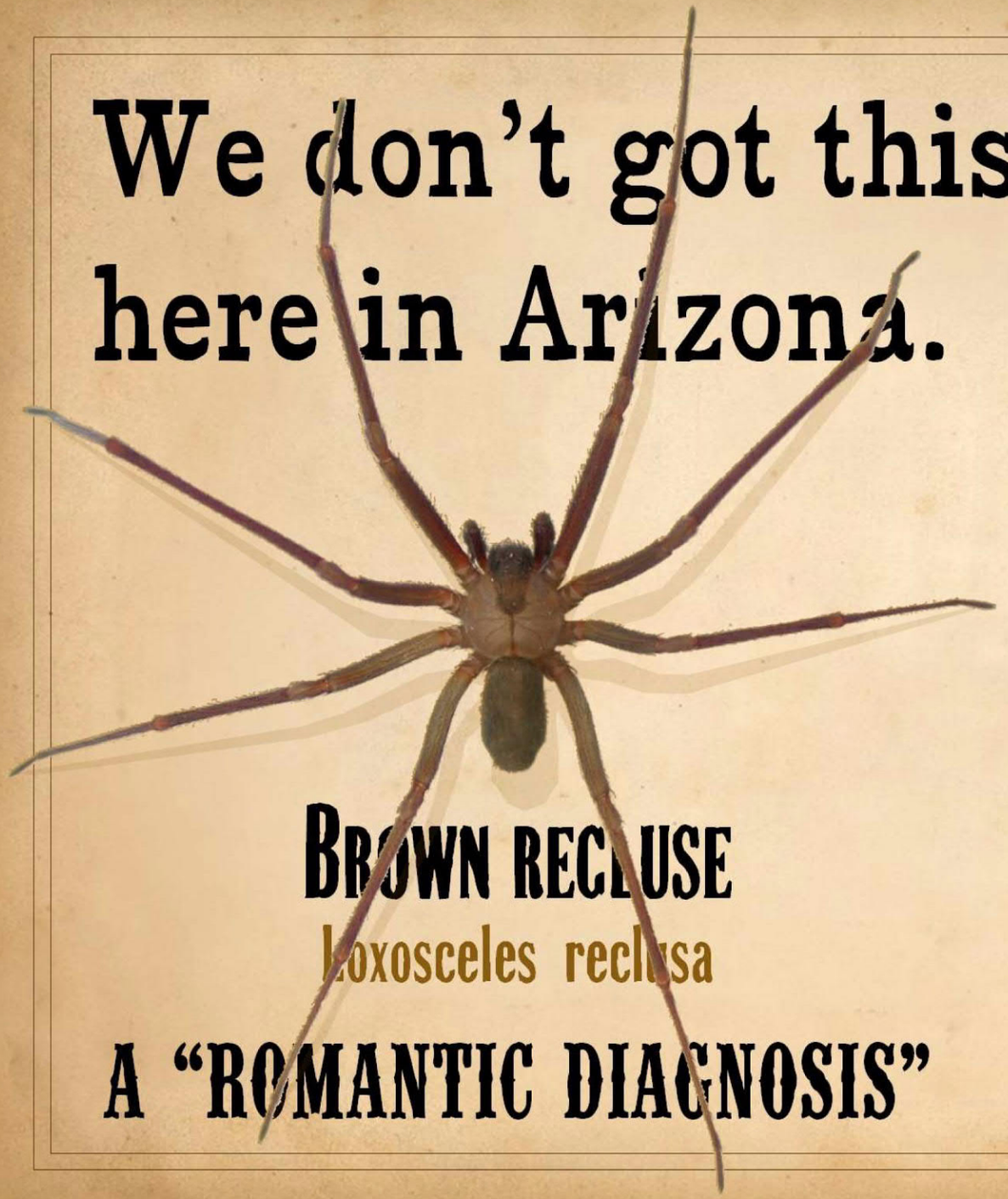


THE MOST OVERUSED INCORRECT DIAGNOSES

Cellulitis
Osteomyelitis
Any infection
Patient's fault
Bad circulation

Diabetes
Decubitus
Vasculitis
Venous stasis
Spider bite

We don't got this varmint here in Arizona.



Arizona recluse
Loxosceles arizonica

Apache Recluse
Loxosceles apachea

Desert Recluse
Loxosceles deserta

Tucson Recluse
Loxosceles sabina

Grand Canyon Recluse
Loxosceles kaiba

BROWN RECLUSE
Loxosceles reclusa

A "ROMANTIC DIAGNOSIS"

Vetter RS, Myth: idiopathic wounds are often due to brown recluse or other spider bites throughout the United States. Western Journal of Medicine 173:357-358, 2000.

Physical Examination

THE CAUSE

THE STATE

Injury -vs- Repair

Healing -vs- Not Healing
-vs- Active Ulceration

W.H. Competent -vs- Not

EVENTS, SIGNS

0 - injury, inflammation

1 - inflammation subsides

2 - macrophages, cytokines, eschar separation

3 - ground substance, mucus

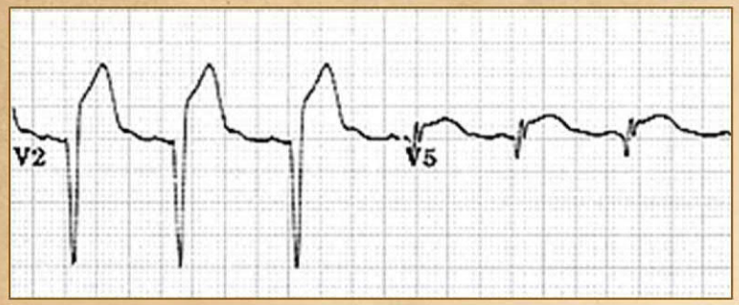
4 - angiogenesis, granulations

5 - fibroblasts, fibroplasia

6 - myofibroblasts, contraction

7 - epithelialization

8 - maturation



Anterior MI

Rheumatoid

Venous

RBBB

Anticardiolipin

Basal call ca

WPW

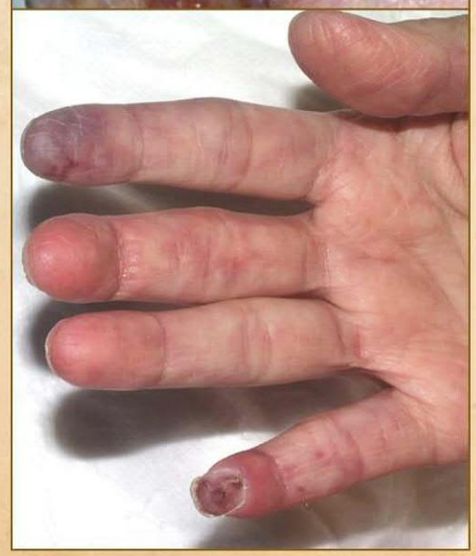
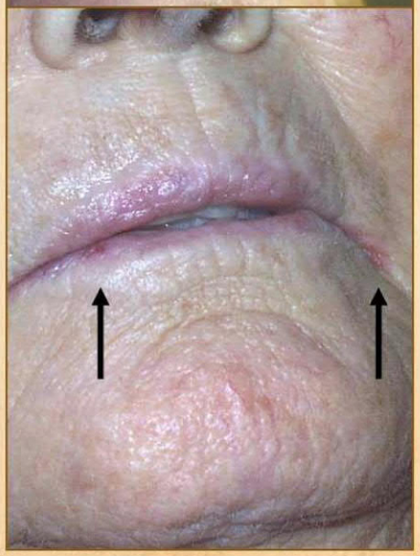
Osteomyelitis

Postural edema








THE PROPER DIAGNOSIS OF WOUNDS

DIFFERENTIAL DIAGNOSIS

THE PROPER DIAGNOSIS OF WOUNDS

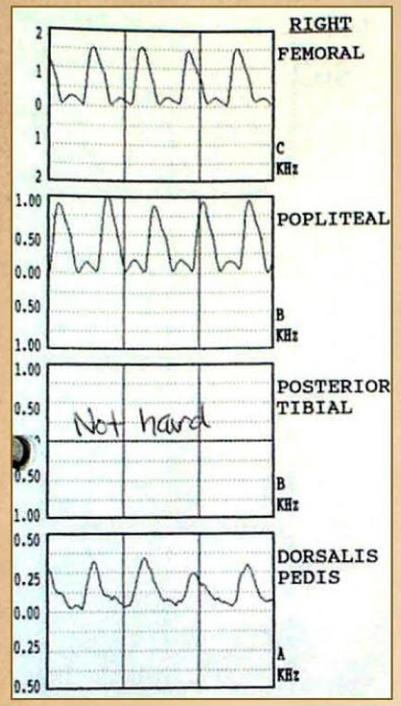
ANCILLARY EVALUATION & PRELIMINARY CARE

2 - 4 week intervals

Activated Protein C Resistance

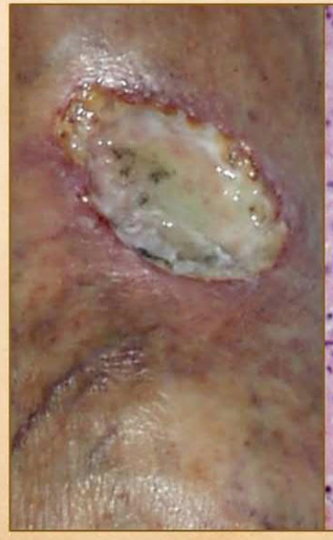
Act.Prt.C Resist. 1.9 **Low**

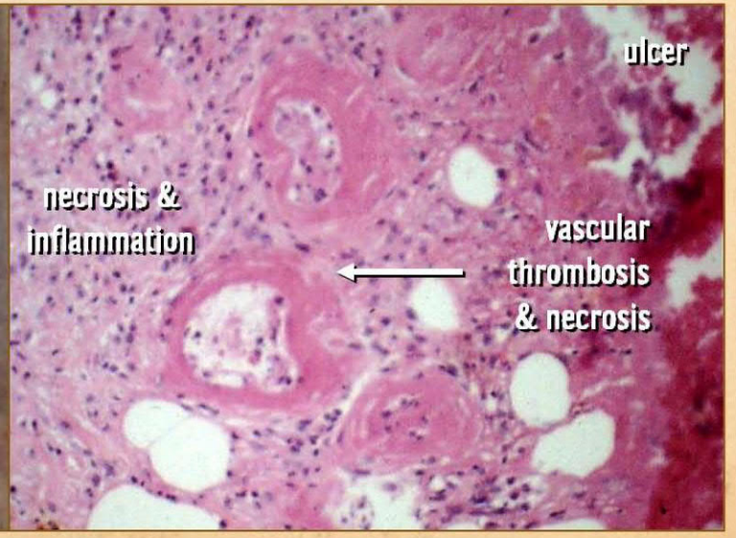
Low results have been considered an important parameter in the risk of thromboembolic disorder.



TOMBSTONE CLINICAL LABORATORY

	VALUE	NORMALS
sed rate	56	<30
C-reactive protein	7.4	< 0.5
ANA	1:1280	<1:40
fibrinogen	477	150 - 350
plasminogen	> 150	70 - 130
protein S	58	72 - 157
cardiolipin IgM	134	>80 high pos





THE PROPER DIAGNOSIS OF WOUNDS

PROGRESS & TRAJECTORY - AD HOC RE-EVALUATION

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Focus on Five Common Problems

GOOD RESULTS BASED ON PROPER MEDICAL PRACTICE

VENOUS
PRESSURE
DERMATOSES
IMMUNOPATHY
HYPERCOAGULOPATHY

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Five Common Problems — Misunderstood & Misdiagnosed

VENOUS

VENOUS DISEASE

IMPORTANT PATHOLOGY

Venous incompetence / reflux
Venous insufficiency
Venous hypertension
Venous stasis

Leukocyte & platelet activation
Venous vasculitis
Dermatitis - eczematous
Dermatitis – nodosum pattern
Panniculitis
Atopy

Edema
Liposclerosis
Autoimmune
Post-phlebitic
Hypercoagulable

1835
-
2005

"Sores in the upper parts of the body, however produced,–the result of injury, abscess, or sloughing,–in consequence of the circulation being more readily carried on, heal kindly, and it is, accordingly, in the lower limbs that obstinate ulcers (leaving out of consideration those of a specific nature) are generally encountered. [. . .] A great many of the indolent ulcers are complicated with varix, and are occaisioned or kept up by this state of the venous circulation. The causes of varicose enlargement are various, and the changes in the vessels inportant, but I dare not digress into this subject now. When the branches are dilated to a certain extent, the valves fail in answering the end of sustaining the column of blood. Hence enlargement of the extreme branches, thickening of the integument and eruption, effusion into the cellular tissue, breaking up of that tissue, abscess, and ulcer; hence also the difficulty, whilst other causes disturbing the course of the blood are in operation, in bringing about a healthy action and a cicatrization of the sore."

"On the Treatment of Ulcers", Robert Liston, Esq., Surgeon to the Hospital, and Professor of Clinical Surgery in the University of London. **The Lancet,** Vol. II, May 16, 1835.

VENOUS DISEASE

PATHOLOGY	TREATMENT
Hypertension	Elevation
Stasis	Compression
Reflux	Surgery
Insufficiency	
Post-phlebitic	
Activation	NSAIDs
Vasculitis	Steroids
Dermatitis	Topical
Panniculitis	Systemic
Autoimmune	Anti-immune
Atopy	Careful with Rx
Hypercoagulable	Anticoagulation
Liposclerosis	Surgery
Edema	Compression

VENOUS DISEASE

DON'T

Admit the patient
Give antibiotics
Talk about amputation
Ignore the problem
Forget all those things to the right

DO

Compress and elevate
Use suitable topical products
Give anti-inflammatory drugs
Consider coagulopathy
Consider immunopathy
Consider surgery

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Five Common Problems — Misunderstood & Misdiagnosed

PRESSURE

See Full Pressure Presentation Below

PRESSURE AND RELATED

Pressure and related ulcers are Trauma, not Disease.

They are due to complex physical-adaptive & psycho-socio-economic factors.

They cannot be cured if the causes are not treated.

DIAGNOSIS MEANS

Be discriminating about anatomy and terms
Ascertain the physical limits of each ulcer
Ascertain the biomechanics of each ulcer
Analyze & deduce the means of injury
Understand posture
Understand the timeline
Understand illnesses which mimic or affect

Understand the patient's psyche
Understand the patient's life & lifestyle
Understand spasticity & dysreflexia
Understand individual physical needs
Don't overlook anatomical complications
Don't confuse pressure with other problems
Understand that treatment is detail oriented

BE EDUCATED. BE PROFESSIONAL. DON'T BE AN ALARMIST.

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Five Common Problems — Misunderstood & Misdiagnosed

DERMATOSES AND PANNICULOPATHIES



15 g NDC 51672-1270-1

Desoximetasone Cream USP, 0.25%

FOR EXTERNAL USE ONLY.
NOT FOR OPHTHALMIC USE.

CAUTION: Federal law prohibits dispensing without prescription.
Keep this and all medication out of the reach of children.

Directions for puncturing tube seal: Remove cap. Turn cap upside down and place puncture tip onto tube. Push cap until tube end is punctured. Screw cap back on to reseal tube.
Mfd. by: TARO Pharmaceuticals Inc.
Brampton, Ontario, Canada L6T 1C1
Dist. by: TARO Pharmaceuticals U.S.A., Inc.
Hawthorne, NY 10532

3 51672 1270 1 5

ULCERATIVE DERMATOSES AND PANNICULOPATHIES

ULCERATIVE DISORDERS and WOUNDS

SKIN DISEASES and DERMATOLOGICAL PRACTICE

Inflammatory, suppurative, necrotizing, & ulcerative disorders of the skin and adipose fascias, mostly immunopathic in origin.

Dermatoses
Eczema
Pyoderma
Pemphigus
Pemphigoid
Sweet's

Panniculitis
Weber-Christian
Erythema nodosum
Necrobiosis lipoidica
Eosinophilic

CTD-CVD
Lupus
Poly-dermatomysositis
RA / granuloma annulare
Scleroderma / CRST
Behcet's
Crohn's

Vasculitis
Leukocytoclastic
Polyarteritis

Miscellaneous
Uncategorized
Drug eruptions

. . . AND MANY MORE . . .

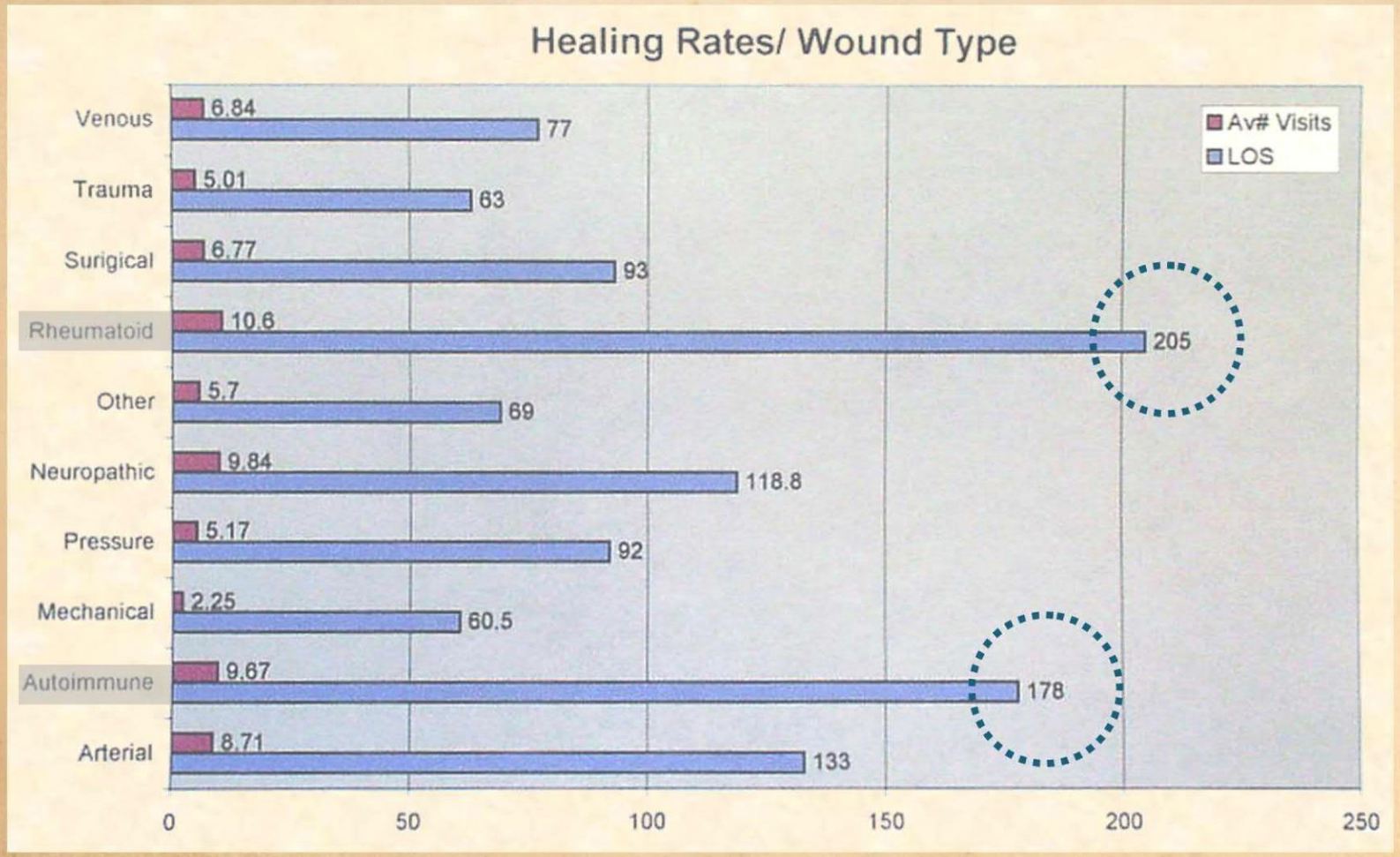
Spectrum of severity
Steroid responsive
Anti-inflammatory rx
Anti-immune rx

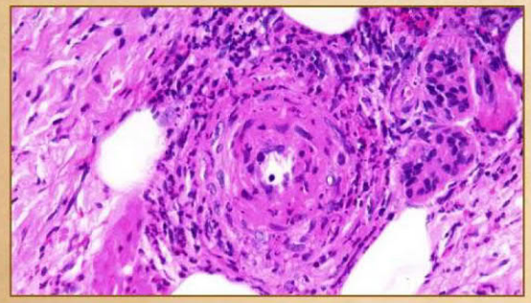
THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Five Common Problems — Misunderstood & Misdiagnosed

IMMUNOPATHY

SIGNIFICANCE OF IMMUNOPATHIC ULCERATION AND IMPAIRED WOUND HEALING





Immunopathies, Spectrum of Disease

Connective Tissue Disorders

rheumatoid
lupus
sjögren's
scleroderma
polymyositis
mctd (mixed)
uctd (undiff'ed)
ank. spondylitis
behçet's
wegener's
sarcoidosis
fam. med. fever

Vasculitides

polyarteritis nod.
autoimmune
giant cell
hypersensitivity
thromboangiitis

Fasciitis & Panniculitis

weber-christian
nodular fasciitis
erythema nodosum
necrobiosis lipoidica

Miscellaneous

crohn's
ulcerative colitis
others

Inflammatory Dermatoses

eczema
pyoderma gangrenosum
erythema nodosum
pemphigus / -goid

Immunopathies, Features and Findings

General and Common Findings	Findings by System	Distinctive and Unique Findings
arthropathies rashes ulcers neurological abnormal serology	musculoskeletal renal & pulmonary cardiac & vascular blood & immune cns & eye	crst sicca pathergy necrotizing synovitis necrotizing vasculitis
Findings by Disease	**Disease Associations**	**Other Tip-Offs**
rheumatoid lupus scleroderma sjogren's polymyositis	hypercoagulability venous arterial disease neuro-psych many misdiagnoses	multiple allergies drug hypersensitivity photosensitivity malar rash nasal perforation

Immunopathic Ulceration . . . Physical Findings

Pre-Ulcerative

inflammation
edema
dermatitis
panniculitis

vascular stasis
congestion
infarction

systemic sx
malaise
athralgias, etc.
pain

distribution
focal
multifocal

Immunopathic Ulceration . . . Physical Findings

Acute & Early Ulcers

necrosis & ulceration

focal vs. multifocal

immune lysis vs. microthrombotic infarction

inflammed vs. bland

vasculitis & synovitis

general

inflammation
dermatitis
panniculitus
vascular stasis
systemic sx
pain

Immunopathic Ulceration . . . Physical Findings

Late & Chronic Ulcers

persistent inflammation

progressive ulceration

retarded wound module

chaotic behavior

pain & symptoms

Immunopathic Ulceration . . . Physical Findings

Other Distinctive Findings

- ulceration along tendons
- unstable scars lysis & ulceration
- not just gaiter not just leg
- skin atrophy skin sclerosis
- post-op & -injury pathergy
- features of each disease

Immunopathic Ulceration . . . Physical Findings

What is NOT There

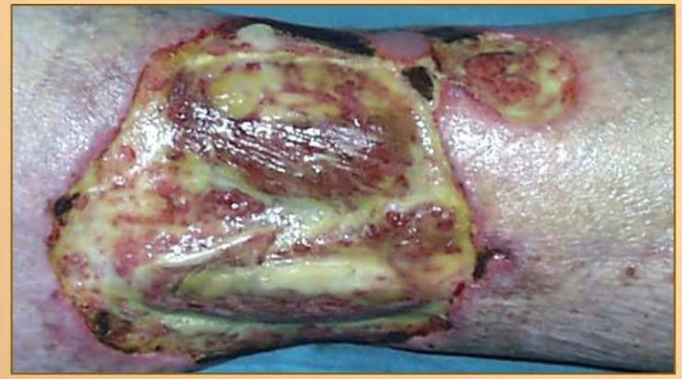
no venous

no arterial

no eschar

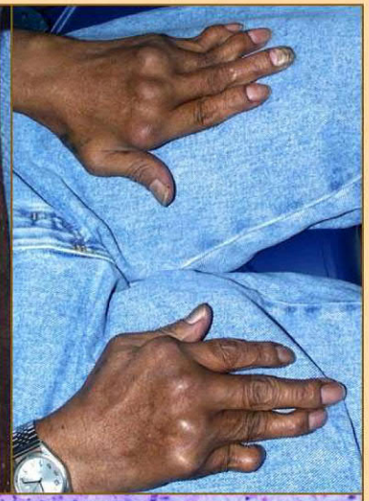
no wound module

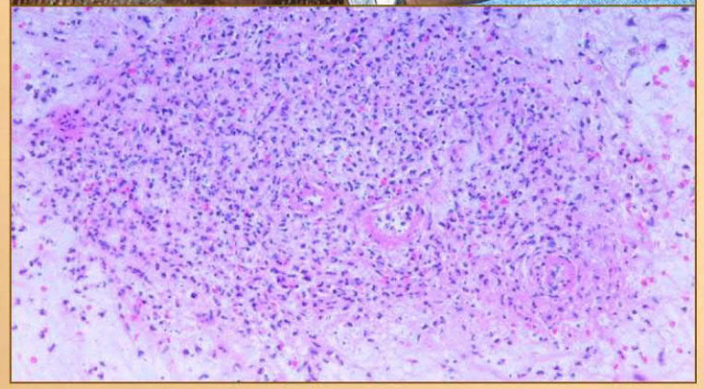
age & risks



Immunopathic Ulceration . . . Physical Findings

General Exam & History





general physical exam

common signs of immunopathy

inflammatory state

arthropathies

neurolepsy & neurological

malaise & systemic

laboratory histology

history & system review

disease history
family hx
Rx history

steroids lowered

complications of trauma or surgery

disease associations

vasculopathies
hypercoagulopathy
neurological
renal
pulmonary

Immunopathic Ulceration . . . Physical Findings

Treatment History

no response
failed Rx
multiple failed Rx
surgery failures

adverse repsonse
disease flare-up
surg. complication
atopic dermatitis
atopic vasculitis

correct response
steroids
anti-immune
anti-inflammatory

contrary response
cytokines

IMMUNOPATHIES

Collagen-Vascular Diseases AND Connective-Tissue Disorders

These are in essence the intrinsic wound healing diseases.

DON'T

Forget or overlook these diagnoses
Confuse pathological inflamm. with infection
Forget about atopy
Give antibiotics
Withdraw patient's drugs sudenly
Ignore the problem
Forget to look for associated disorders
Forget all those things to the right

DO

Learn how to recognize & manage them
Respect their destructive disabling nature
Respect their potential lethality
Learn to read the signs of active disease
Give anti-inflammatory drugs
Work to keep disease quiet
Intervene before ulceration occurs
Recall that immune ulcers heal slowly

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Five Common Problems — Misunderstood & Misdiagnosed

HYPERCOAGULOPATHY

HYPERCOAGULOPATHY

NOMENCLATURE OF THROMBO- & MICRO-OCCLUSIVE DISORDERS

hemodynamic disorders	vessels, blood, & coagulation normal **fluid dynamics abnormal**	Examples: arteriovenous malformations vascular compression, atrial fibrillation
endo-vasculopathies	blood & coagulation normal **vessels abnormal**	Examples: small vessel atherosclerosis thromboangiitis, alloplastic implants
exo-vasculopathies	blood & coagulation normal **vessels abnormal**	Examples: calcium-phosphate disorders, immunopathies & connective tissue disorders
non-hypercoag hemopathologies	vessels & coagulation normal **blood abnormal**	Examples: red cell & platelet abnormalities, hemoglobinopathies, dys- & cryoproteinemias
hypercoagulability	vessels & blood normal **coagulation abnormal**	disorders of the coagulation system intrinsic: the prethrombotic disorders extrinsic: examples - estrogens, cancer

Key Syndromic Features

thrombotic - embolic events • miscarriages • wound pathergy

connective tissue disorder • family history

Prethrombotic Disorders

factor V Leiden
other f.V mutations
prothrombin mutation
antithrombin III
protein C
protein S
fibrinogen
plasminogen
warfarin

Related Disorders

antiphospholipid antibodies
anticardiolipin
lupus anticoagulant
homocysteine disorders
estrogens, pregnancy

Disease Associations

inflammation
connective tissue disorders
acute & chronic venous
cancer (Trousseau)
parox. noct. hemoglobinuria

Macrothrombosis

Acute Large Vessel

overt life-and-limb threatening events

cava-tibial venous thrombosis
aorto-tibial arterial thrombosis
other peripheral thrombosis
coronary artery thrombosis
cerebrovascular thrombosis
pulmonary embolism
intracardiac thrombosis
graft and valve thrombosis
subclavian v. (paget-schroeder)
hepatic veins (budd-chiari)
pituitary apoplexy (sheehan)
retinal artery & vein occlusion
intracranial sinus thrombosis
spinal apoplexy
visceral apoplexy
(renal, adrenal, bowel)

Microthrombosis

Subacute, Chronic, Recurring

perplexing refractory problems of non-obvious origin

vascular occlusion not overt
secondary clinical events
underlying causes elusive

miscarriage
complications of trauma & surgery
non-healing ulcers
non-immune glomerulonephritis
primary pulmonary thrombosis
warfarin necrosis
complications of contraceptives

chronic, recurring
refractory to Rx
long history of failed Rx
young age
family history
warfarin resistance

HYPERCOAGULOPATHY RECOGNITION & DIAGNOSIS

HYPERCOAGULABLE ULCERS HAVE NO PATHOGNOMONIC FEATURES, BUT THEY DO HAVE A DISTINCTIVE APPEARANCE.

APPEARANCE

ischemic infarction
periwound stasis
active ulceration
edema absent
inflammation absent
mixed wound module

good pulses
no signs of other dx

RESPONSE TO WRONG RX

pathergy
necrosis
dehiscence
failed response

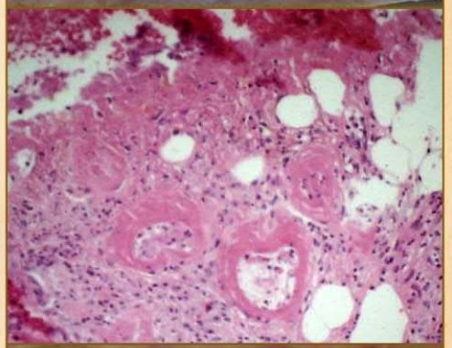
DYNAMICAL BEHAVIOR

impaired wound
behavior characteristic
of severe ischemia

recalcitrant
continuously pathological
persistent active:
necrosis
pathergy
active ulceration

misbehavior over time

rapid evolution
slow resolution



HYPERCOAGULABLE STUDIES

Factor V Leiden
prothrombin mutation
antithrombin III
protein C
protein S
fibrinogen
DIC screen
plasminogen
homocysteine
lupus anticoagulant
anticardiolipin
cryoglobulins
cryofibrinogen

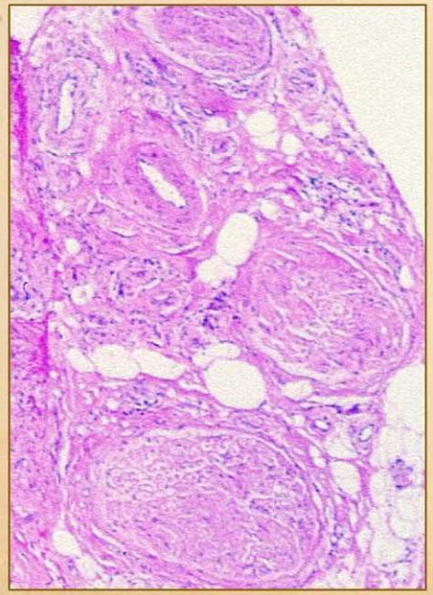
SCREEN FOR CONNECTIVE TISSUE DISORDERS

sedimentation rate
CRP
ANA
anti-DNA
rheumatoid factor

OTHER STUDIES

TcPO2
laser doppler

Biopsy and Histology
microthrombi
aggregates
minimum inflammation
microvasculopathies
vascular fibrosis
stenosis
vasculitis

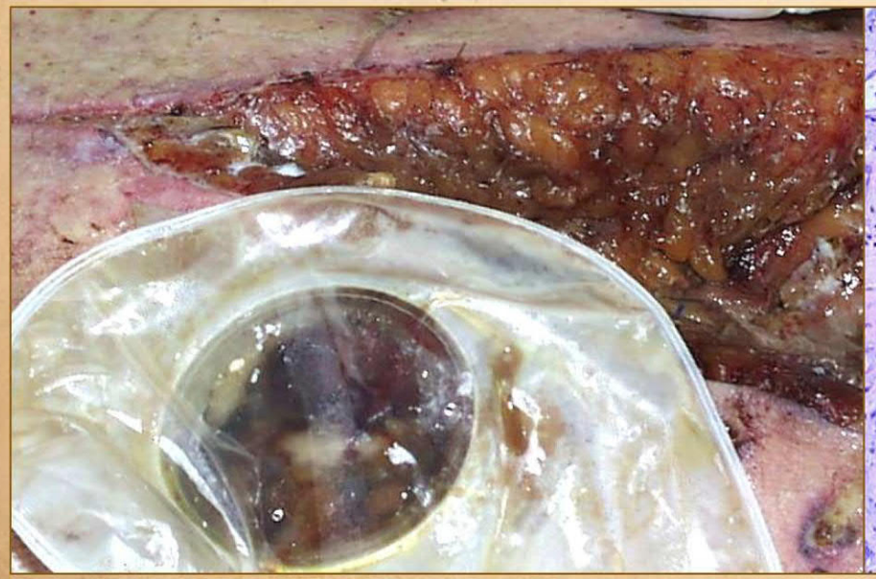


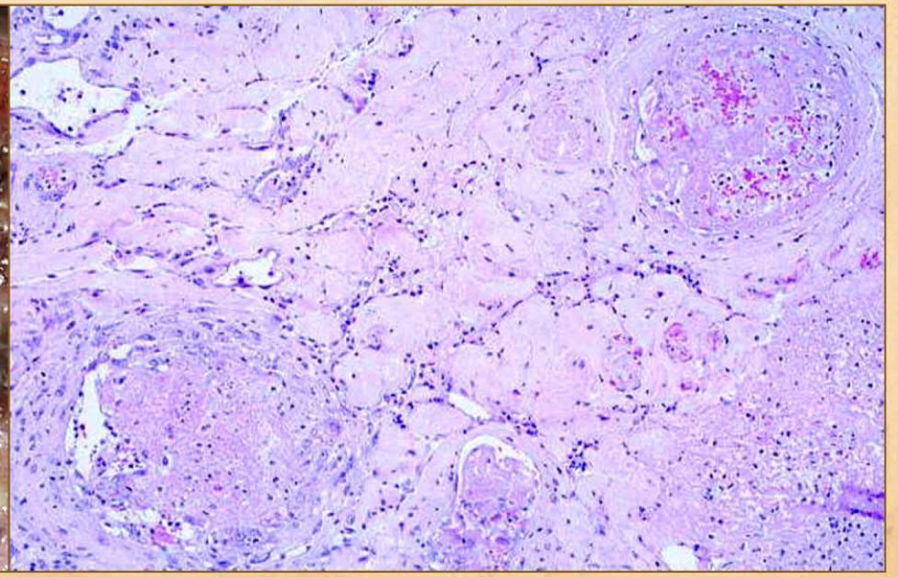
RECOGNITION & DIAGNOSIS - LABORATORY -

Hypercoagulable ulcers are NOT diagnoses of exclusion.

These diagnoses can be made on specific criteria.

HYPERCOAGULOPATHY
- BAD OUTCOMES -





HYPERCOAGULOPATHY
- GOOD OUTCOMES -

DO NOT CONFUSE HYPERCOAGULABILITY WITH

pyoderma
immunopathies
other vascular and thrombotic disorders

AND VICE VERSA

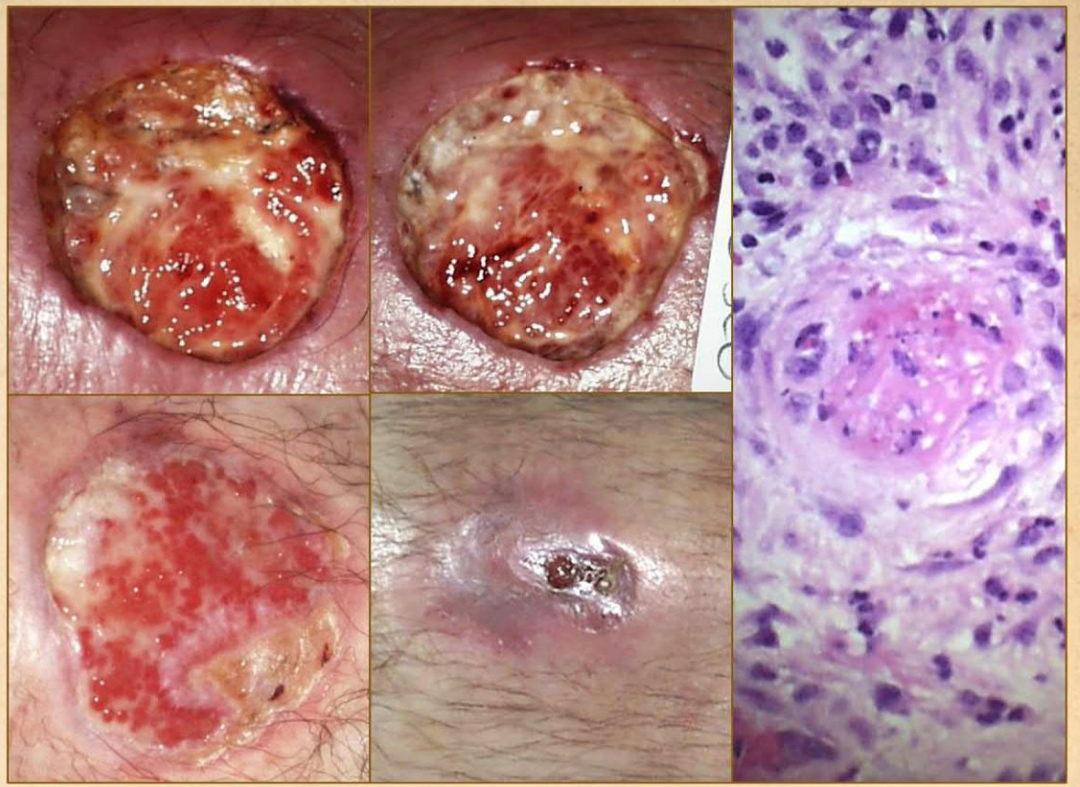
CORRECT DIAGNOSIS

THE DIAGNOSIS AND MISDIAGNOSIS OF WOUNDS

Common Problems — Misunderstood & Misdiagnosed

CONNECTIONS

ANGIOPATHY - IMMUNOPATHY
- COAGULOPATHY -
ISCHEMIA - INFLAMMATION
NECROSIS - ULCERATION



TOMBSTONE CLINICAL LABORATORY

sed rate	56	+
C-reactive protein	7.4	+
ANA	1:1280	++
cardiolipin IgM	134	++
fibrinogen	477	+
plasminogen	> 150	+
protein S	58	-

34M, lupus, trauma wounds

pathergy, multiple wound failure: hand, groin, leg

multiple failed operations, refractory to all care

antiphospholipid antibodies

healed with warfarin

54M **No prior diagnosis**

FactorV Leiden	heterozyg	+
ANA	1:80-sp	+
lupus anticoag	pos	+
cardiolipin IgA	15	+
cardiolipin IgG	>150	+++
cardiolipin IgM	20	+
protein C	60	-
protein S	56	-
homocysteine	14.6	+

72F **Polycythemia Vera**

ANA	1:160	+
cardiolipin IgM	80	++
protein S	53	-

75M **Anemia / Cythemia**

rheumatoid factor	2780	++
cardiolipin IgM	70	+
protein C	65	-
cryoglobulin	pos	+

69F **Rheumatoid Arthritis**

FactorV Leiden	heterozyg	+
protein C	51	-
protein S	52	-

81F **Leg ulcer**

rheumatoid factor	27	+
ANA	1:1280-hm	++
lupus anticoag	pos	+
cardiolipin IgM	51	+
protein C	142	+
fibrinogen	429	+
homocysteine	19.3	+

66F **Scleroderma / MCTD**

rheumatoid factor	35	+
ANA	1:1280-cn	++
protein S	62	-
fibrinogen	499	+

78F **Sjögren's**

protein C	60	-
fibrinogen	565	++

67F **Rheumatoid Arthritis**

F.V Leiden	heterozyg	+
protein C	136	+
plasminogen	135	+
fibrinogen	640	+

67 & 176

1 & 3

$TcPO_2$

air & O_2

57M **Cirrhosis**

Bili	2.1	+
AlkPhos	160	+
RF	44	+
ANA	1:80	+
AT-III	47	-
ProtC	35	-
ProtS	55	-

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Marc E. Gottlieb, MD, FACS

Phoenix, AZ

https://www.arimedica.com/presentations.htm
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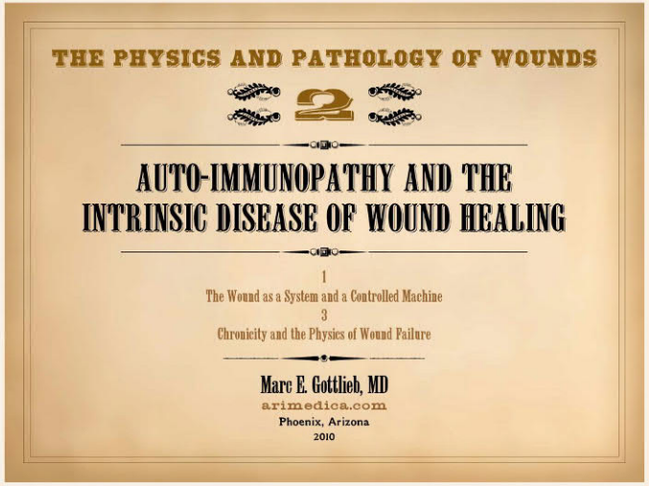
arimedica_wpp-2_autoimmune & intrinsic_gottlieb-me_maui-2010-0222_annotated.pdf

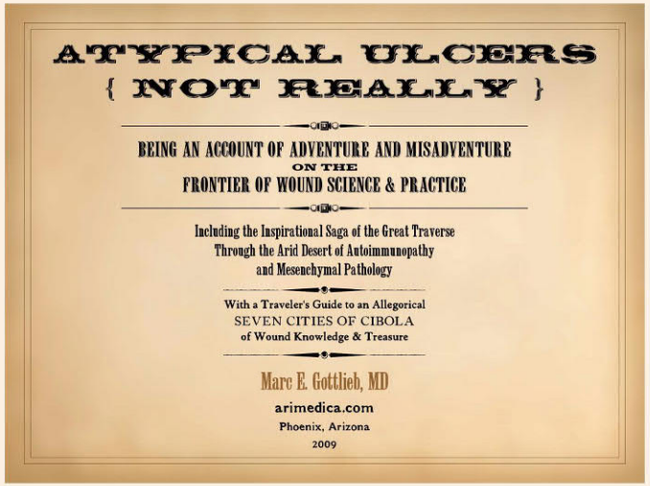
The Physics and Pathology of Wounds, Part 2
Auto-Immunopathy and the Intrinsic Disease of Wound Healing



Wound physiology 2 - how wounds become intrinsically impaired by repetitive acute inflammation leading to auto-immunization.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_(not)%20atypical%20wounds_gottlieb-me_2009-0926_annotated.pdf

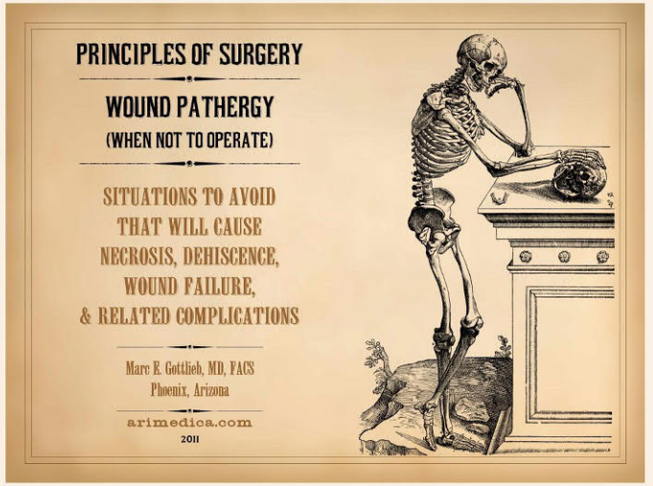
(NOT) Atypical Ulcers (Autoimmunopathy and Connective Tissue Disorders: The True Intrinsic Diseases of Wound Healing)



Wound physiology and failure - a precursor paper to the 3-part series, with a compact presentation of wound healing physiology.

https://www.arimedica.com/presentations.htm
https://www.arimedica.com/content/
arimedica_wound pathergy_gottlieb-me_2017-1025_annotated-200.pdf

Wound Pathergy – When Not to Operate
Situations to Avoid that Will Cause Necrosis, Dehiscence, Wound Failure



Biology of wound failure, and systemic diseases that interfere with wound healing and surgery, 30 cases and detailed explanation.

https://www.arimedica.com/subjects_integra.htm
https://www.arimedica.com/content/
arimedica_hypercoag-2018_gottlieb-me_2018-0920_(annotated).pdf

Hypercoagulable Disorders – Implications For Wounds & Surgery
Pathophysiology, Clinical Features, Diagnosis & Treatment

Detailed text on hypercoagulable disorders and their profound significance to surgery complications and chronic wounds.

General Concepts

Wound Healing Biology

CAP
Chronic and Pathological Wounds

Wound Management

Pressure Ulcers

THE 5 D'S OF MEDICINE : *Life, Liberty, and the Pursuit of Happiness*

Disease

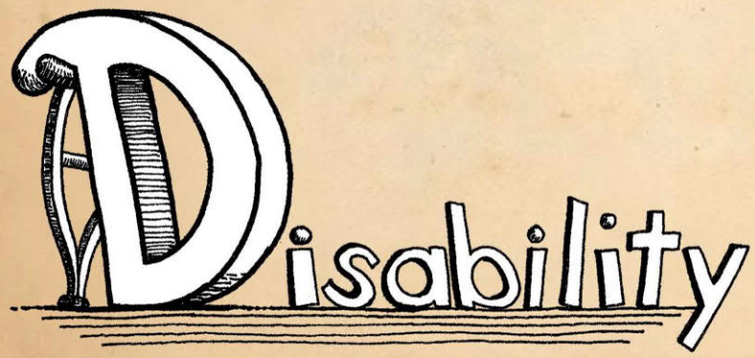
CURE

SAVE LIVES

Curing disease and saving lives is not enough.

The goal is never just to fix the nerve, the bone, the vessel, nor heal the wound,

— rather — *to restore people to function, lifestyle, ADL, vocation, avocation, social acceptability, and economic independence, symptom-free.*



REHABILITATION

Deficiency & Deformity

RECONSTRUCTION

PROFILES OF DISEASE AND TREATMENT

Types of medical problems	**Short term acute medical problems**	**Long term chronic medical problems**	**Intermediate terminable medical problems**
	diabetic ketoacidosis bowel obstruction phlebitis	diabetes inflam. bowel disease venous hypertension	diabetic plantar ulcer perineal fistulas venous ulcers
Types of therapeutics	**Short term therapeutics**	**Long term therapeutics**	**Intermediate term therapeutics**
	emergency medicine general surgery	internal medicine geriatrics	reconstructive surgery rehabilitation medicine
Agents of treatment	**Physician as agent of therapeutics**	**Patient as agent of therapeutics**	**Allied professionals as agent of therapeutics**
	surgery angioplasty endoscopy	pills insulin inhalers	dialysis musculoskeletal rehab wound care

WOUND MANAGEMENT - THE BASIC SCHEMA

PHASE 1: MANDATORY RX

Wound Control

Control disease and turn the wound into a healthy, non-threatening, asymptomatic condition.

PHASE 2: DISCRETIONARY RX

Definitive Care and Resolution

Establish and implement realistic treatment goals for closure, palliation, or other specific resolution.

PHASE 3: FOLLOW-UP RX

Maintenance Care

Maintain control to prevent flare-ups and recurrence.

WOUND MANAGEMENT - THE BASIC SCHEMA

PHASE 1
MANDATORY RX
Wound Control

PHASE 2
DISCRETIONARY RX
Definitive Care and Resolution

PHASE 3
FOLLOW-UP RX
Maintenance Care

Control disease and make the wound healthy, non-threatening, asymptomatic.

identify causative disease and risks

refine and reconcile rx

establish good wound hygiene

control topical risks

debride the wound

control edema

treat pathological inflammation

treat contributing disorders
e.g. revascularize, offload,
anti-immune & anti-coagulant therapy

WOUND MANAGEMENT - THE BASIC SCHEMA

PHASE 1
MANDATORY RX
Wound Control

PHASE 2
DISCRETIONARY RX
Definitive Care and Resolution

PHASE 3
FOLLOW-UP RX
Maintenance Care

Plan & implement realistic treatment goals: closure, palliation, or other resolution.

expert evaluation and treatment for related disorders

correct intrinsic wound module deficits

ancillary modalities as indicated e.g. hyperbaric O_2, devices

surgery if indicated

modify or accelerate wound repair

wound stimulatory therapies

devices & drugs

WOUND MANAGEMENT - THE BASIC SCHEMA

PHASE 1
MANDATORY RX
Wound Control

PHASE 2
DISCRETIONARY RX
Definitive Care and Resolution

PHASE 3
FOLLOW-UP RX
Maintenance Care

Maintain control to prevent flare-ups & recurrence.

keep underlying diseases controlled

long term control of edema and inflammation

skin care

rehabilitation

maintain orthotics

periodic surveillance

rapid intervention for flare-ups

"Palliative" or "Maintenance" care for those who cannot be cured, applying the same principles of chronic preventive management, maintaining "control" at all times, never losing control.

Wound Therapies: Basic and Advanced

1 Basic Rx: Fundamentals of management and preventive care 3

Wound control and maintenance

Hygiene - Topicals - Dressings - Skin Care

Edema & Inflammation Control

Disease & Injury Control

2 Advanced Rx: Technology in support of new paradigms of care 2

Environment regulating therapies

VAC, Promogran & Prisma

Wound stimulatory therapies

PDGF - Becaplermin, Apligraf

Regenerative therapies

Integra

https://www.arimedica.com/presentations.htm

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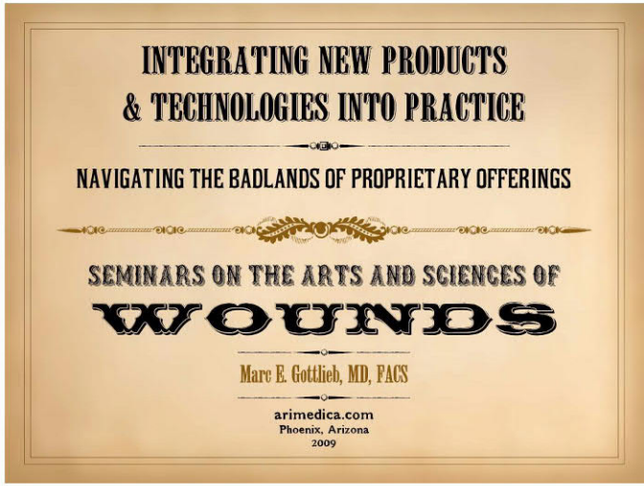
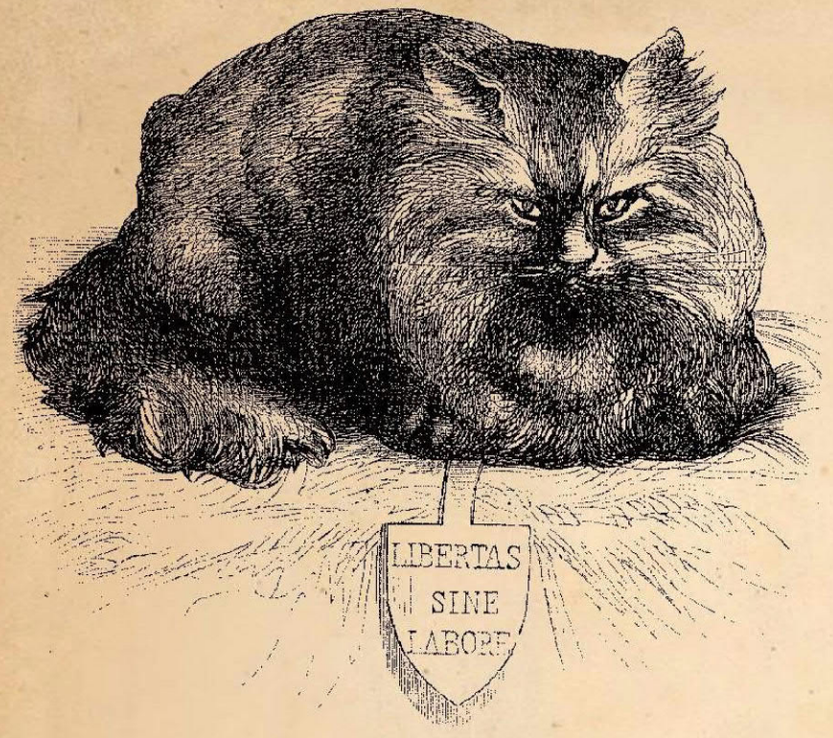
Integrating New Products & Technologies into Practice :
Navigating the Badlands of Proprietary Offerings



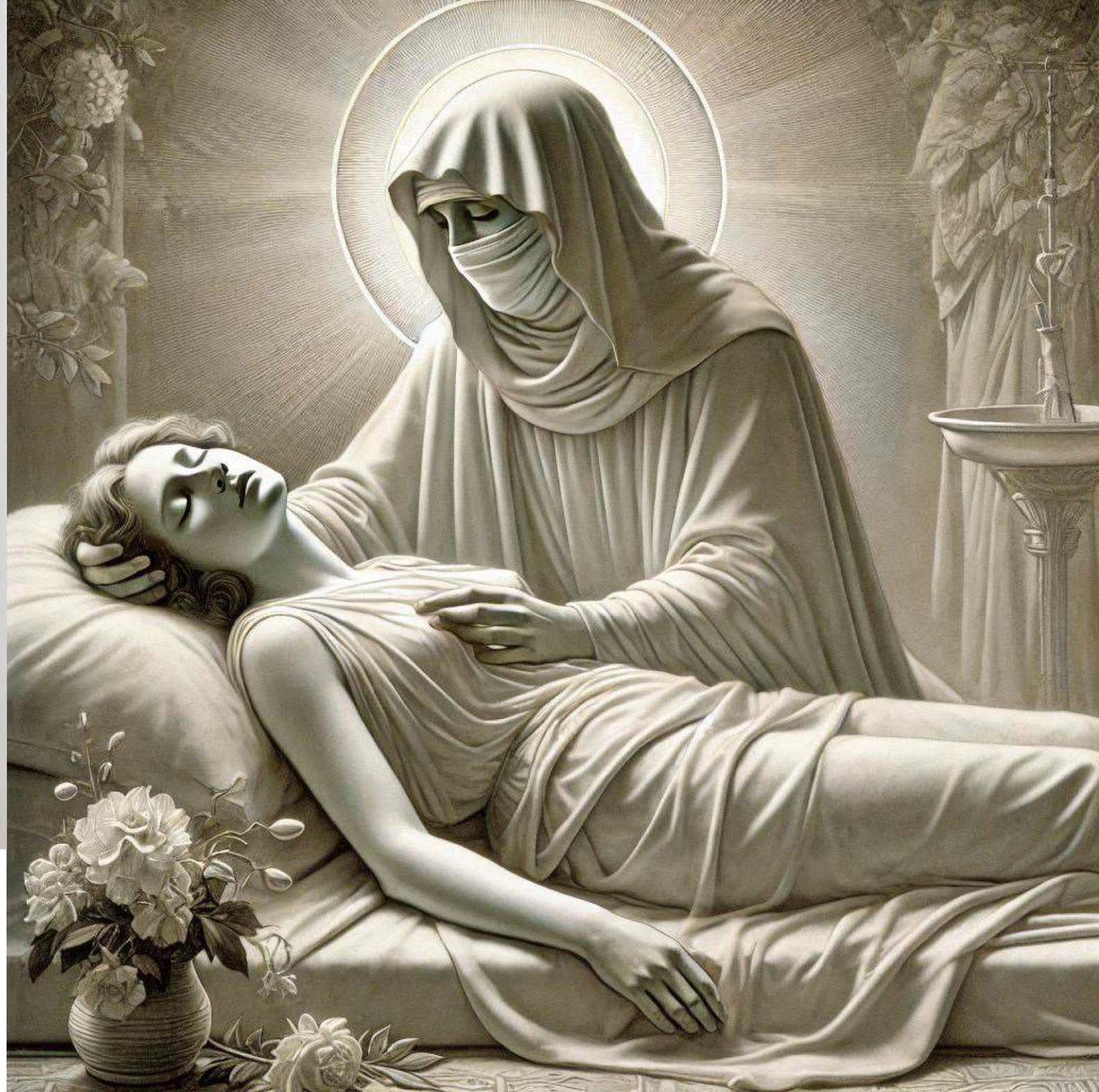
A general discussion about evaluating and using new technologies, focus on wounds, including a survey of wound care products.

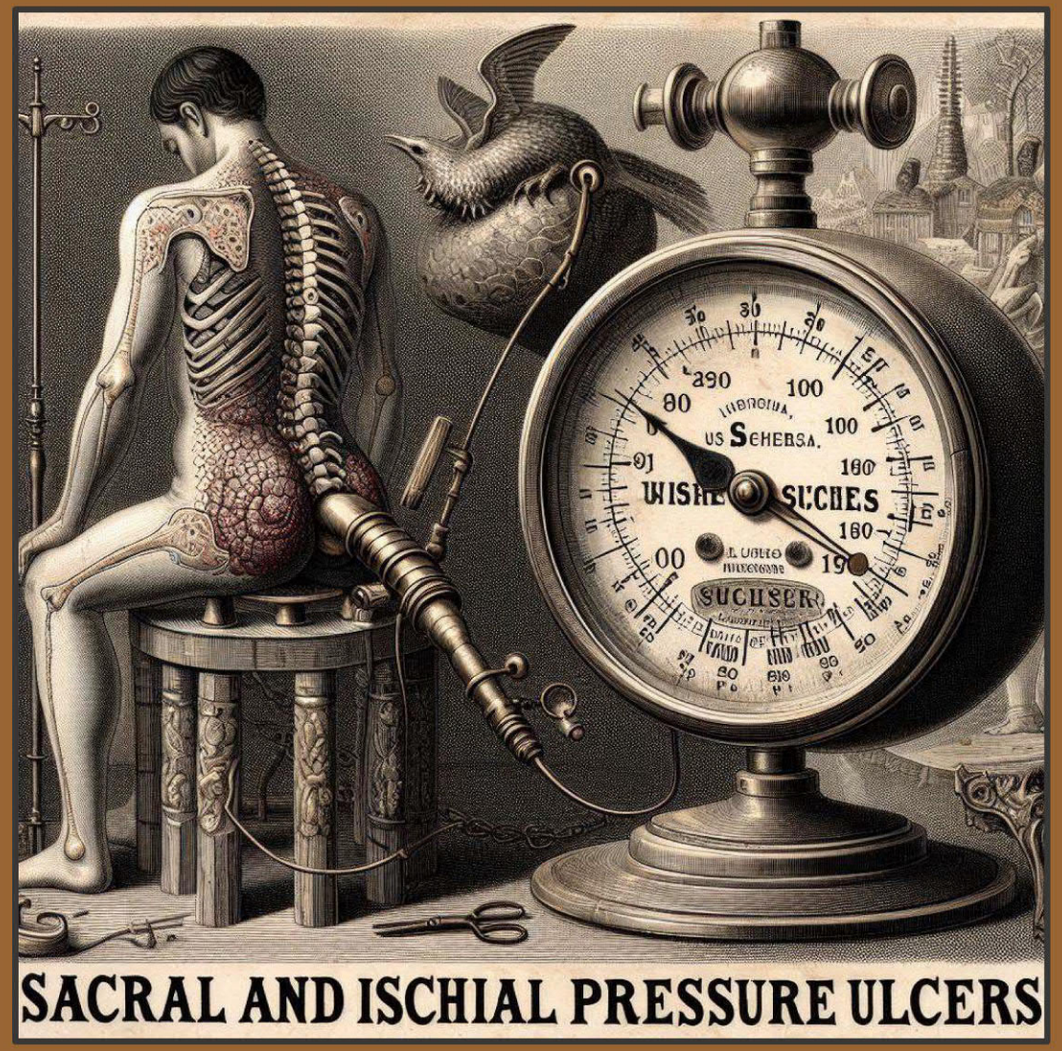


CHRONIC ILLNESS

Teaching people to live with and manage their problem.

No obsessive fixation on getting it “healed”.





General Concepts

Wound Healing Biology

CAP
Chronic and Pathological Wounds

Wound Management

Pressure Ulcers

WOUNDS AND ULCERS IN GERIATRIC PATIENTS

5

Common Causes and Diagnoses

5

PRESSURE & CONTACT

PHYSICAL

Afferent neuropathy (sensation)
Efferent neuropathy (palsy)
Acute & chronic disabilities
Psycho-socioeconomic factors

Pressure
Tension - shear
Motion
Musculoskeletal anatomy
Skin injury
Dermatoses
Anatomical pathology
Posture biomechanics

CLINICAL

Pressure ulcer
Shear ulcer
Closed pressure bursa
Contact ulcer
Burns and injury
Intertriginous ulcer

Urinary fistula
GI fistula
Spinal abscess
Pelvic abscess
Inflammatory bowel disease
Hidradenitis & pilonidal

BEDSORES SADDLE SORES
OTHER PERINEAL ULCERS

PRESSURE AND RELATED

They are due to complex physical-adaptive & psycho-socio-economic factors.
Pressure and related ulcers are trauma, not disease, and largely benign.

BE EDUCATED. BE PROFESSIONAL. DON'T BE AN ALARMIST.

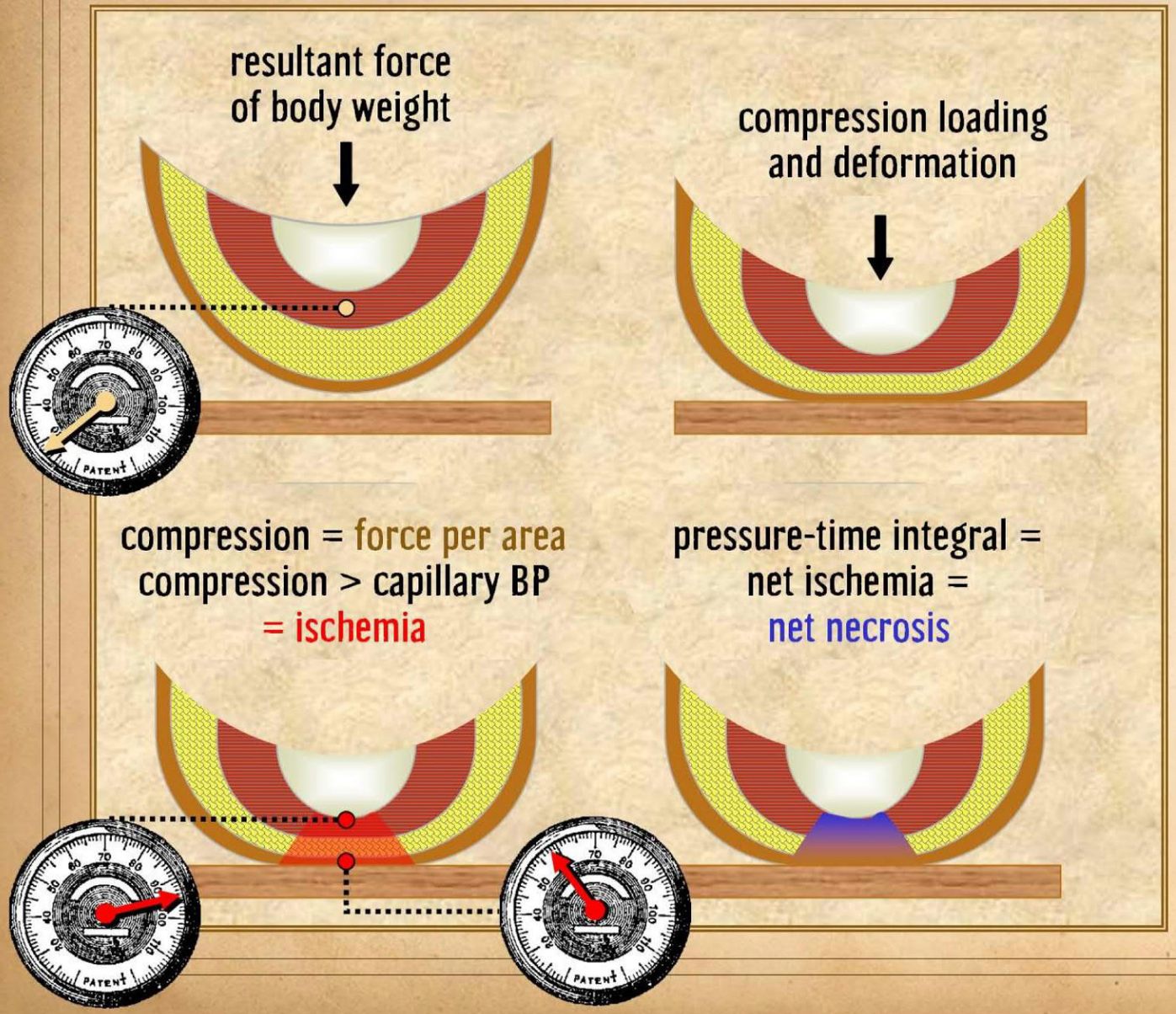
Be discriminating about anatomy and terms
Make correct diagnosis & means of injury
Ascertain the biomechanics of each ulcer
Understand pressure vs. shear vs. contact

Understand psychological and mental status
Understand physical capacity and needs
Understand family, support, lifestyle
Formulate a comprehensive plan of care

DO FORMULATE AN INTELLIGENT STRATEGIC PLAN OF CARE.

The causes must be relieved, but do not kill the patient with concern.
All must be managed - but - NOT all can nor SHOULD be closed and cured.

PRESSURE ULCERATION



Efferent
neuro deficits
can't move

Afferent
neuro deficits
can't feel

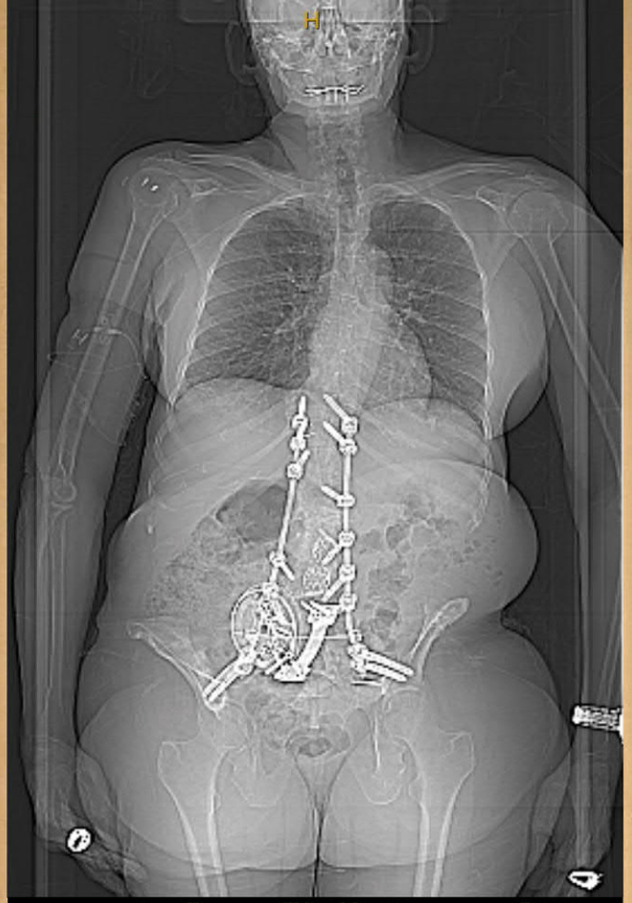
Psychiatric deficits
no motivation

Social deficits
no resources

Adverse
biomechanics
stress-strain

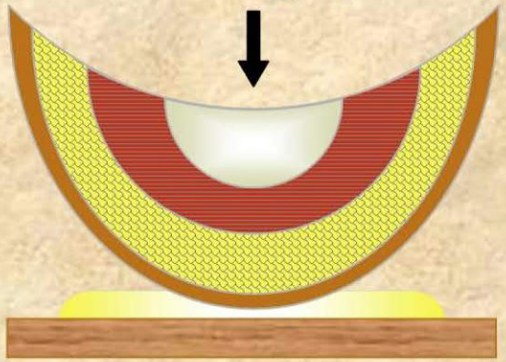
Repetitive injury

Normal
wound healing



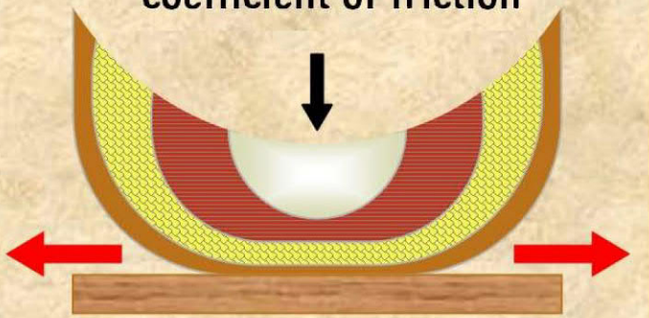
SHEAR AND CONTACT ULCERATION

maceration, inflammation,
skin fragility

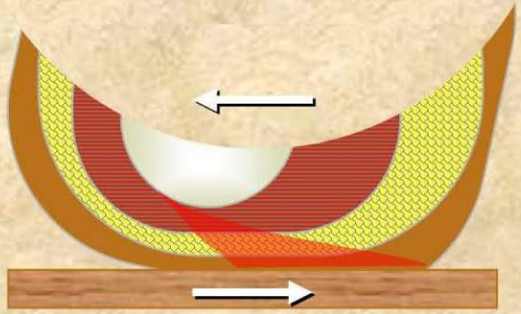


tangential force & motion, friction

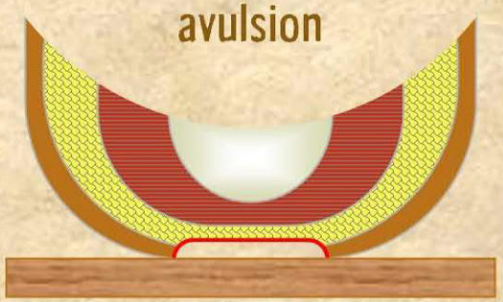
compression loading
coefficient of friction



shear stress
shear strain



injury due to shear
blisters
desquamation
avulsion

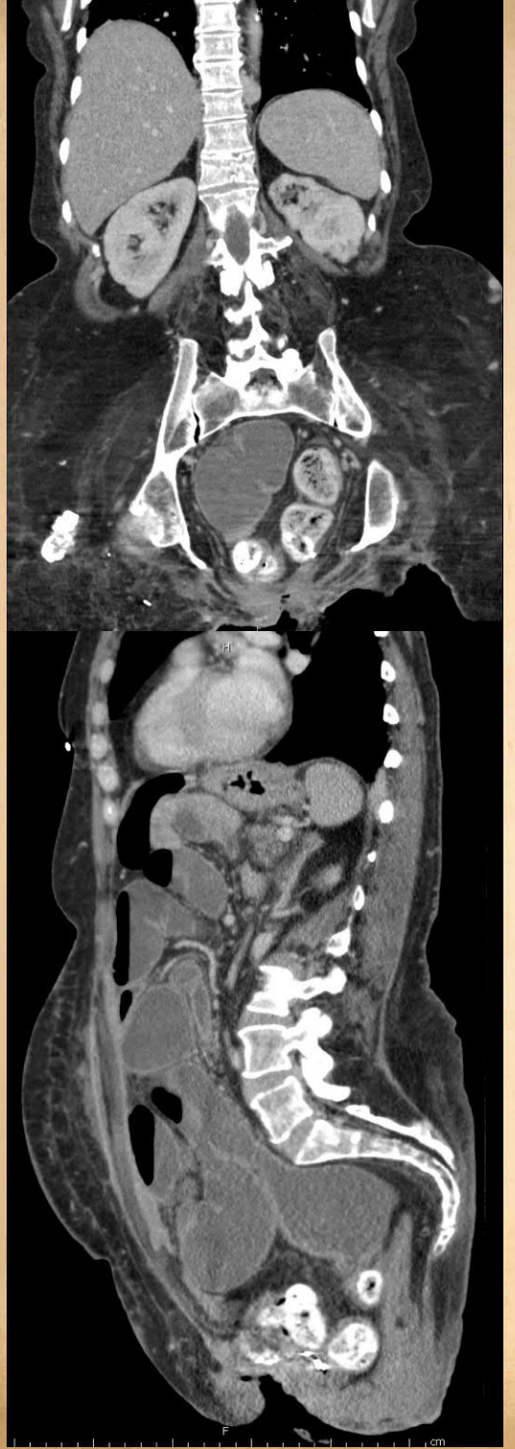


PROPER CONCEPTS *and* TERMINOLOGY

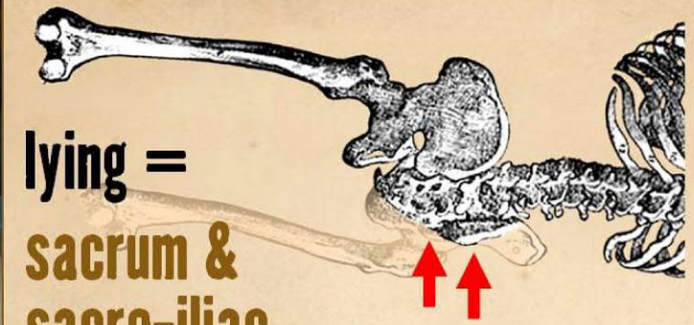
Pressure ulcer
closed bursa
shear ulcer
contact ulcer
dermatitis
intertriginous ulcer

"Non-pressure"
burns and injury
vascular infarction
iliac aso/pvod
calciphylaxis
coincidental events

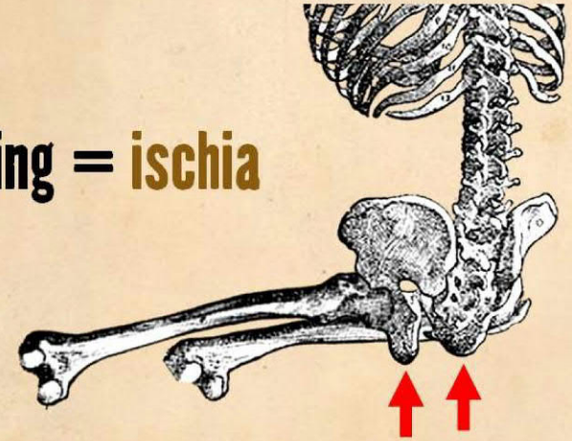
Decubitus
bedsores
other postures



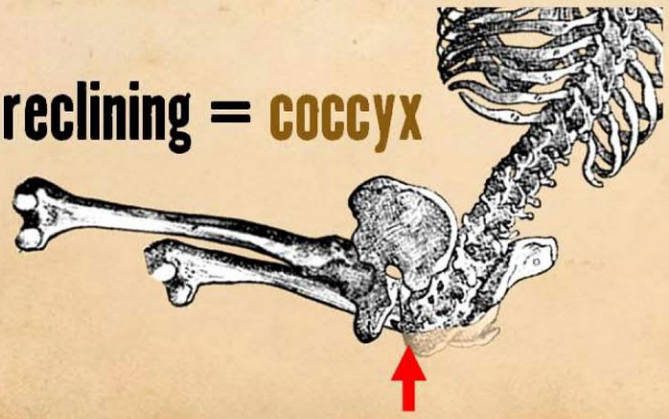
lying =
sacrum &
sacro-iliac



sitting = ischia



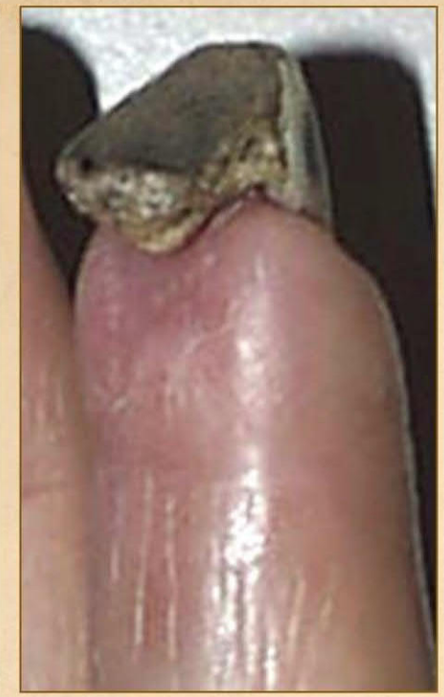
reclining = coccyx

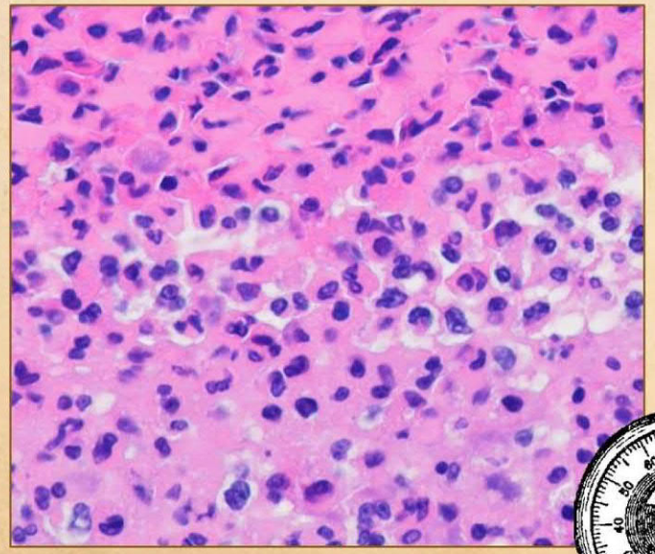




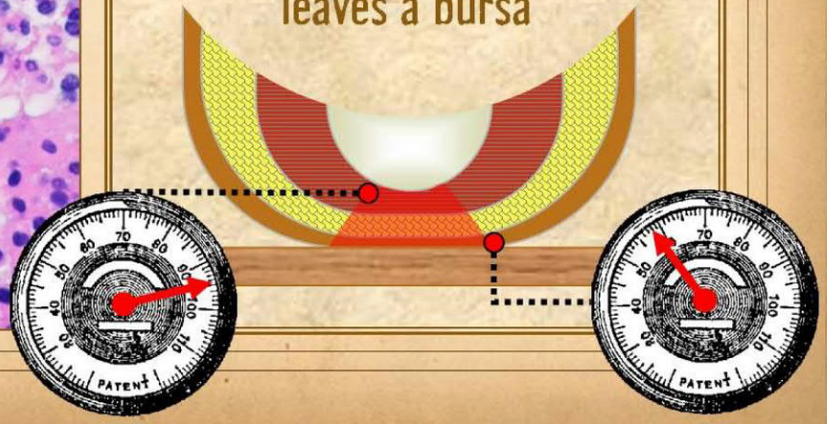








pressure necrosis is
volumetric
full thickness
leaves a bursa



WHERE-THE-SUN-DON'T-SHINE THEATER

PRESENTS

SADDLE SORE TALES

FAREWELL ESCHAR, GO

Presented at a Snail's Pace

Starring

Larry Leadbottom, *the Gimpalong Cowboy*
Billy "Beef Jerky" Buttwound, *his sidekick*

with

Penelope Pemican, *nurse ingenue*
Stinky McOdor, *the alarmist*
Mack Roefage, *gold miner*
Auntie Biotic, *gold digger*
Doc Rongeur, *problem solver*
Dudley Dowrong, *ne'er do well*
Betty Bursa & Sally Sinus, *the chorus*
and as the Narrator
"Snake Eyes" Slimy Sluggo

REMEMBER

Pressure necrosis is full thickness, from skin to bone.

Understand the biology of pressure injury & evolution.

DO NOT CONFUSE

pressure necrosis with other causes of ulceration.

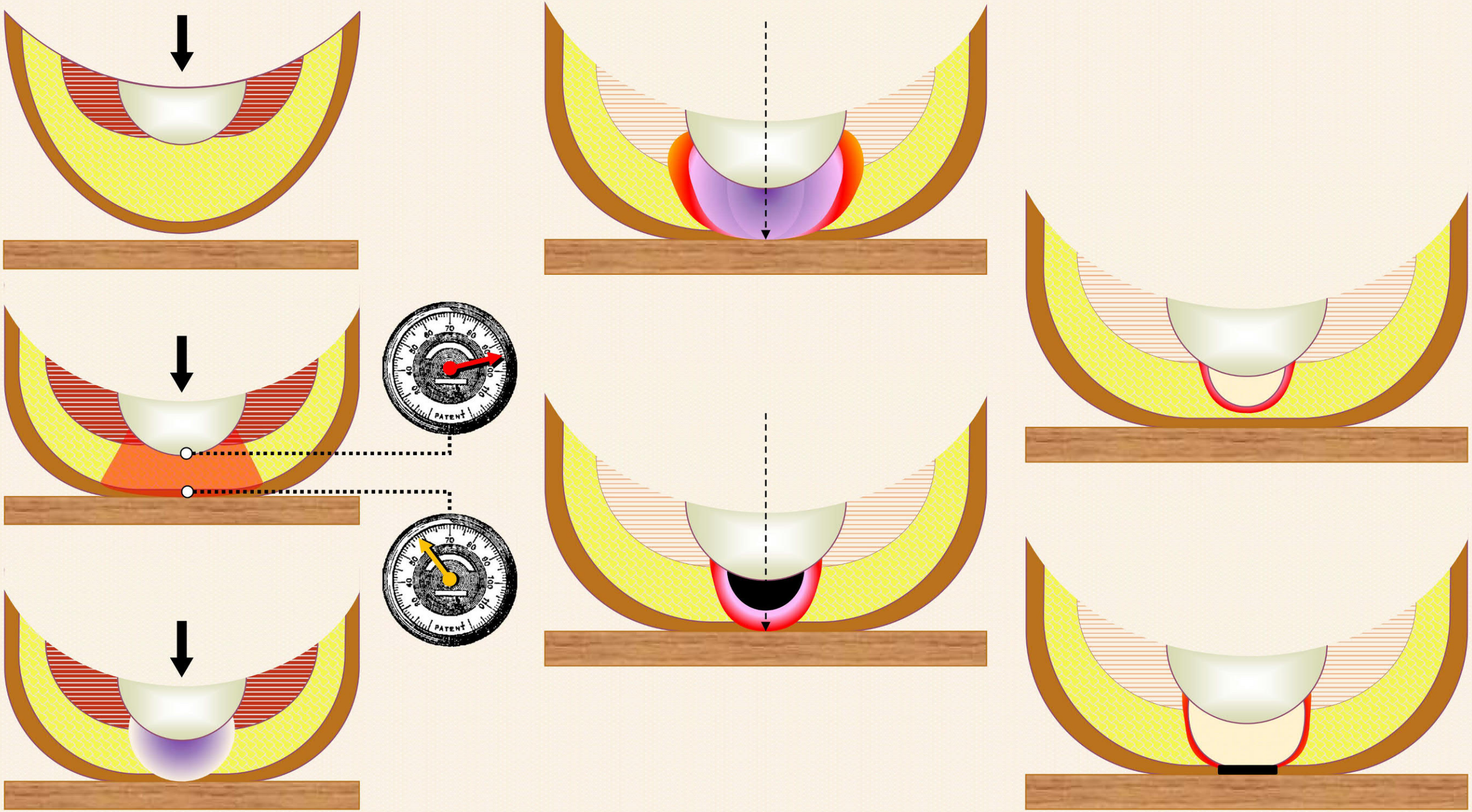
MANY PATIENTS WITH PRESSURE ULCERS

have months or years of their lives, and their residual good health, sometimes life itself, robbed from them by doctoring with those with no knowledge of the subject, zero training or education on the subject, zero experience with proper care, and no shame treating these people anyway, mostly wrongly.

(There are also doctors who know what to do and do it well, and YOU need to find a circle of colleagues who fit with this.)

Don't be THAT doctor.

Protect your patients from them and from a broken and corrupt system that makes money off of their misery without returning good results.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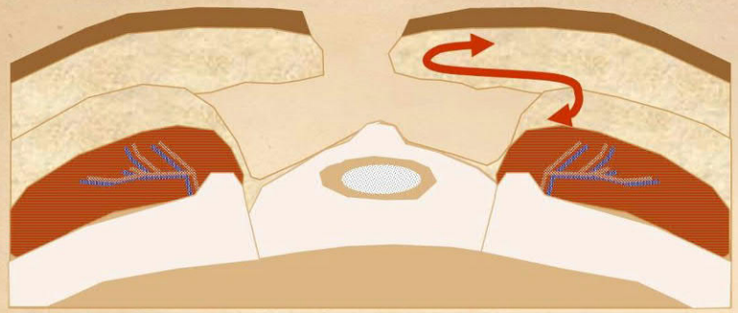
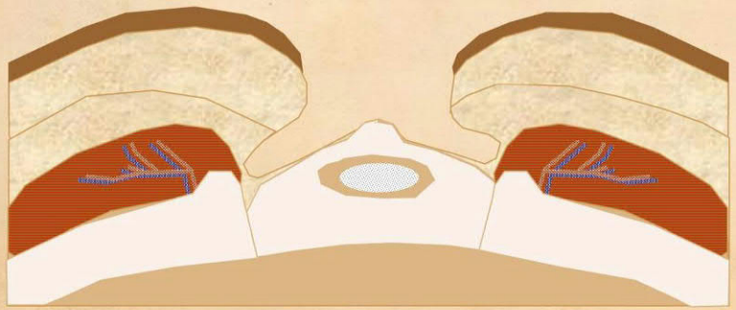
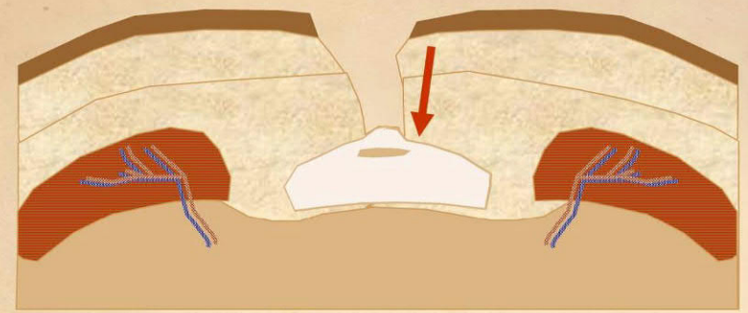


BURSA





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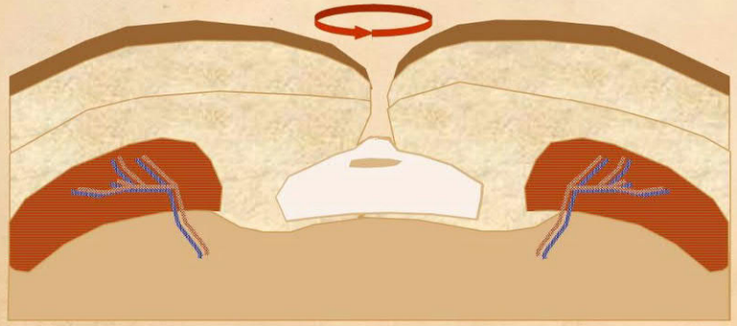
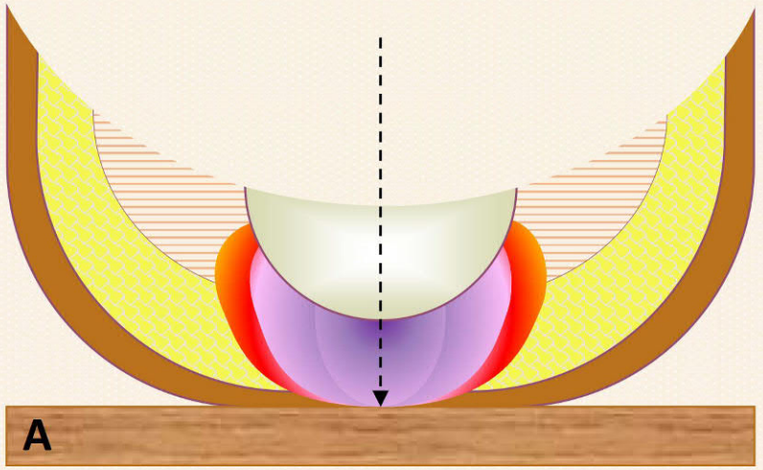
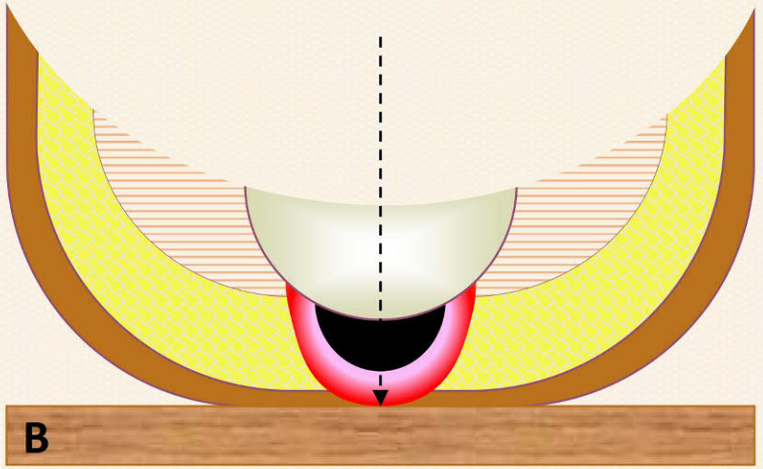






crucial concepts

pressure mechanics , wound geometry
trauma , not pathology
necrosis occurs at point of highest pressure (over bone)

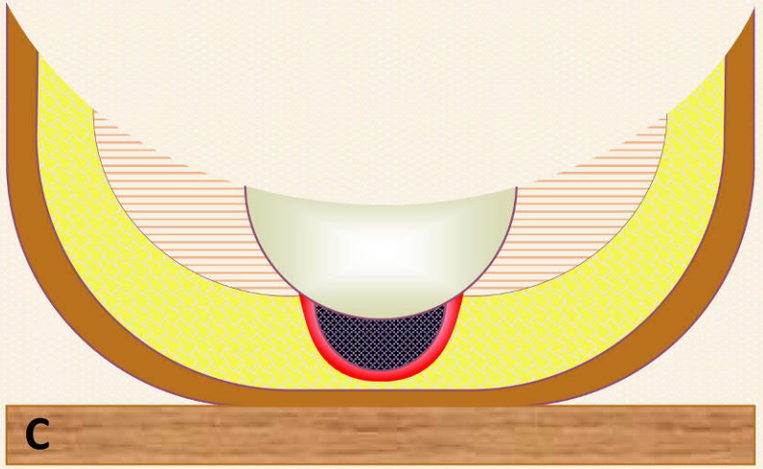
wound healing, eschar separation, contraction
cannot fully heal because bone does not contract
thus, surgery required

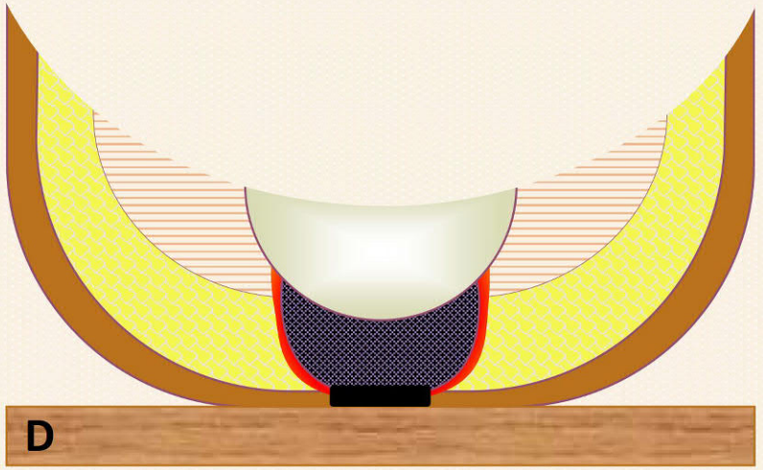
crucial concepts

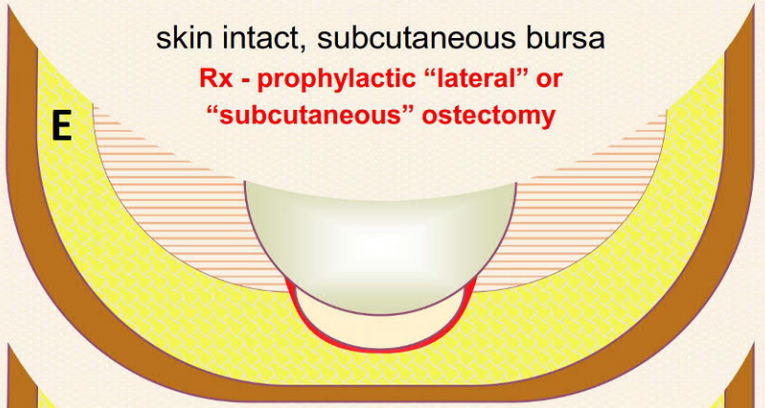
no bone pathology (except with neglect)
bone must be remodeled (geometry, mechanics, Wollf-Davis)

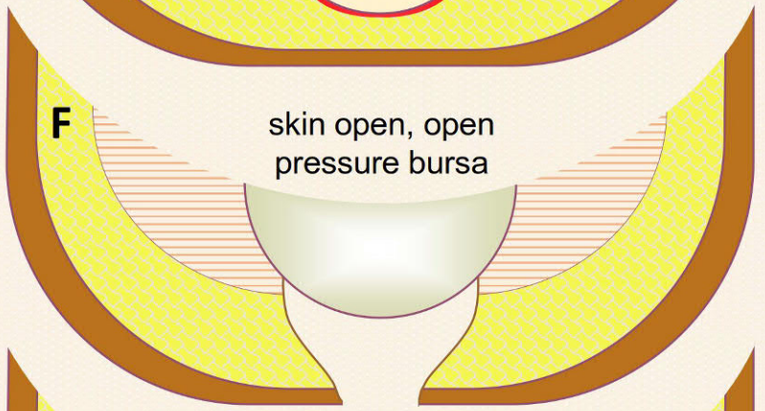
all para's have **E** (ischial subcutaneous pressure bursa),
* * * don't let it get to **F** * * *

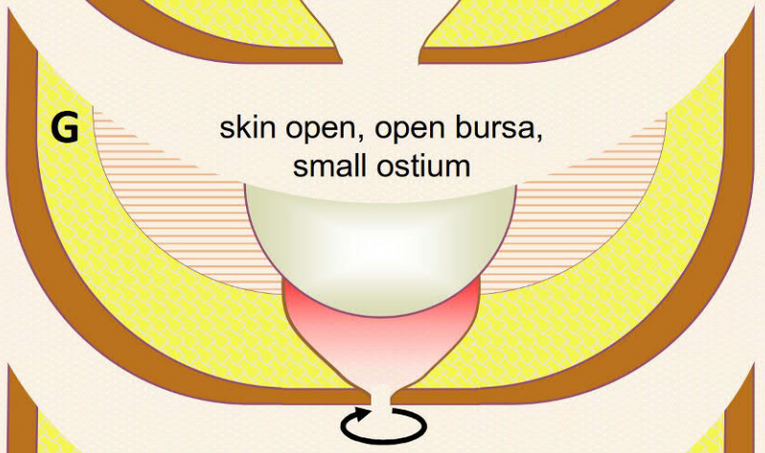
once the skin is open over the pressure point,
you and the patient have misery and surgery

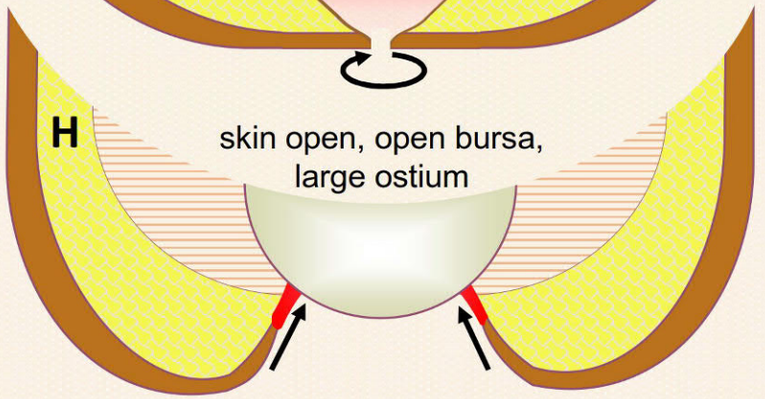


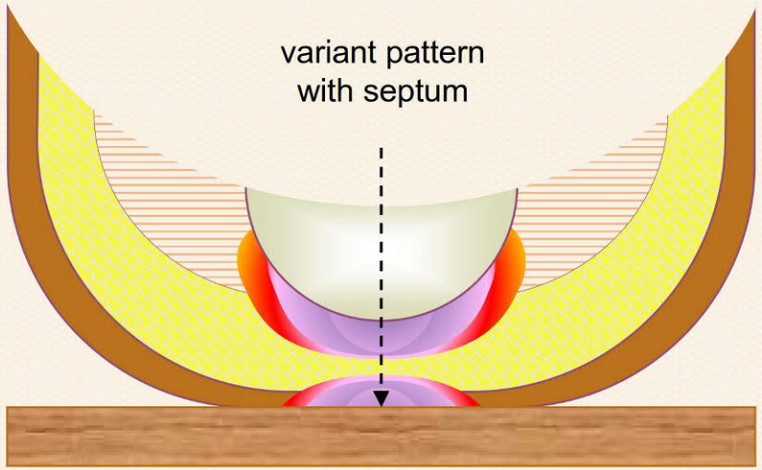


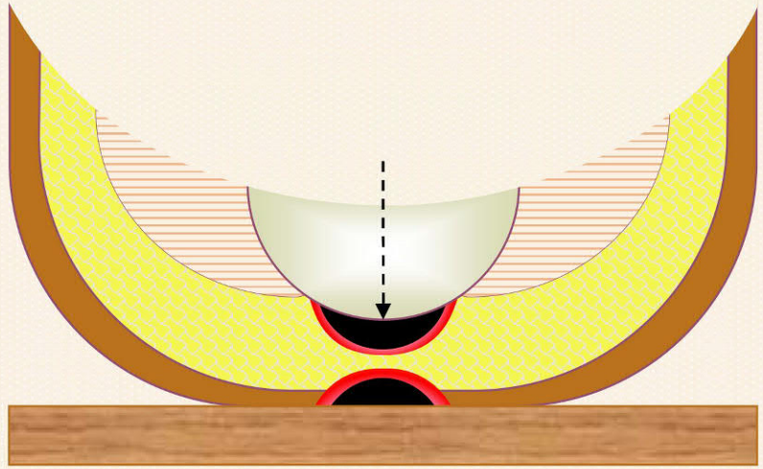


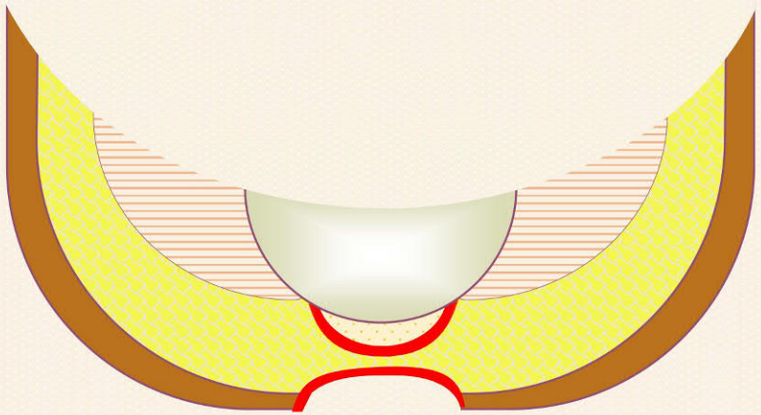


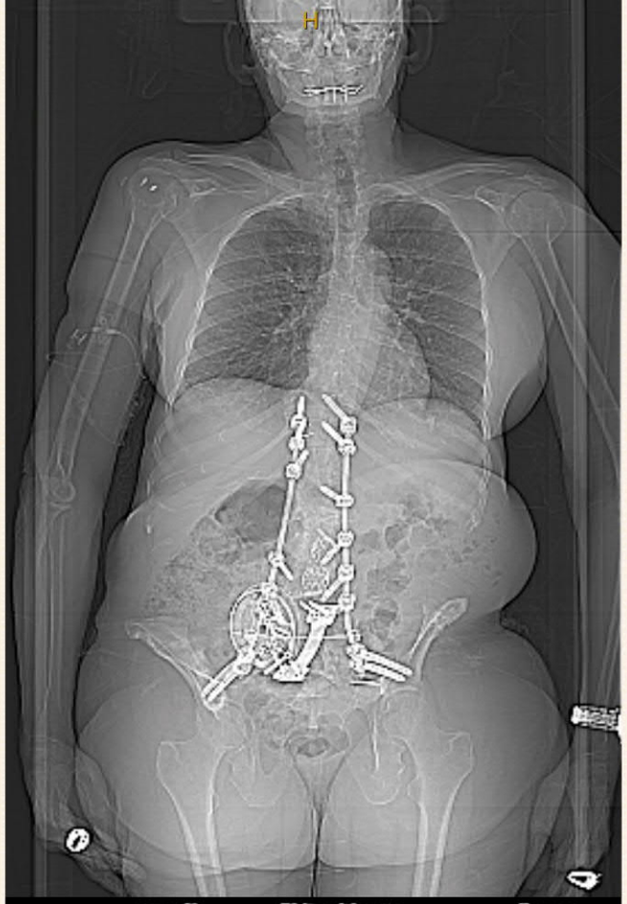


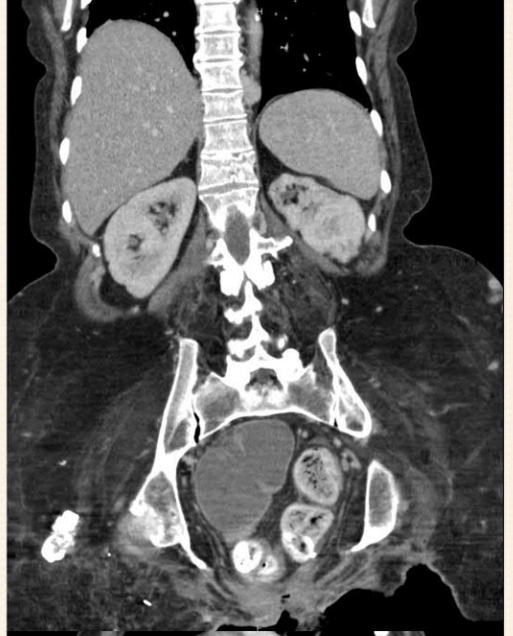












axial anatomy & mechanics
spine fracture plasticity

pelvic anatomy & geometry
e.g. pubic / pseudo-ischial p.s.

charcot hip and necrosis
proximal femur resection

WOUNDS : Pressure

General Principles ::

except for some incidental trauma conditions,
pressure ulcers are a consequence of neuropathy
no neuropathy, no pressure ulcer

common scenarios :

- senile disabilities
- bed or chair confinement, hospital acquired
- paraplegia
- quadriplegia
- ms, als, misc, etc.
- pediatric - cp, spina bifida, etc.
- syndromic profiles, e.g. geriatric, “angry young man”
- psycho-socio-economic stress

pressure anatomy, biomechanics, pathology, pathogenesis :

essential to understand this and know the details

obligations :

- manage the wound
- close the wound if and when eligible
  - ** pressure ulcers rarely close without surgery
  - no rush - patients must earn their surgery
- manage pressure exposure
  - seating, bedding, age dependencies
- prevent more wounds
- manage the patient and neuropathy
- manage the sequelae of the primary disorder
  - **urinary**, bowel, neurologic, msk, lungs
  - neurogenic bowel-bladder : flaccid vs spastic
  - orthotics, therapies, functional integration
  - autonomic dysreflexia ** the good, & the bad
  - spasticity
- protect the patient from [the abundant] inexpert care
- incidental peculiarities
  - hyperhidrosis
  - urinary calculus
  - heterotopic ossification, h.o. / m.o.
  - syrinx, tethered cord
  - chronic pain
  - vascular disease & time-pressure
  - pubic / pseudo-ischial
  - urethral fistulas
  - non-pressure wounds in same areas
    - exposure-shear, dermatoses, cancers
    - internal iliac aso or infarcts, ano-rectal pathology
  - misc

Cheat sheets for hospitalized pressure sore patients.

You did not admit them, don’t know them yet, you were consulted :

- stop antibiotics
- fluids - LR or similar 125 cc/ hr or 3 liters daily
- urinary antiseptics
- proper wound care
- pressure relief surface
- take advantage of being there to evaluate anything required:
  - pelvis-spine-hips, x-ray & ct, for biomechanics & h.o.
- urinary eval
- get patient out of hospital as expeditiously as possible
  - to prevent unnecessary problems, and
  - get them into correct program of care
- long term f/u in your office / clinic, or that of your colleagues

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- if possible, pre-admit by 2-3 days for pre-op prep
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- urinary antiseptics : mandelamine hippurate, 1 gm bid (or nitrofurantoin)
  - vit C, 500 - 2000 mg bid
- pulmonary care : not needed for para’s
  - mandatory for quad’s :
    - svn’s, pos.press modalities, +/- bronch, baseline cxr, oximetries, abg’s
- bowel care : pt’s routines, laxatives, enemas, dig stim
- pressure relief surface : prone care when eligible and possible
- wound care
- rx sweating for occasional pts : glycopyrrolate, 1 mg h.s.
- rx dysreflexia for select pt’s :
  - β-blocker, α-blocker, ca-channel blocker
- rx spasticity : benzo’s, baclofen, dantrolene, misc others
- pain : narcotics, nsaid’s etc., Li_2CO_3 , mexiletine, tricyclics, misc
- evaluate biomechanics (imaging), and make plans
- make body casts in advance for select pt’s
- young trauma para’s : infrequent medical problems needing rx
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- psych and social rx
- wheelchair, bedding, d/c planning

KAFU - “All psych, all the time.”

Pressure sore surgery - 0 - Principles

see Session 1 - EDSMI, segmental, pressure math

Pressure sore surgery - 1 - Preliminaries

- debridement & wound prep in OR : ONCE if at all
- resect pathological bone : ONCE if at all
  - (these have usually been done in office / clinic)
- resect heterotopic bone (select patients)
  - *staged as needed for blood loss, safety, wound quality*
- hip or proximal femur resection (select patients)
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- tendonotomies, neurectomies, casts, etc. (select patients)

Pressure sore surgery - 2 - Flaps and closure

ONLY WHEN READY

- excise wound
- ostectomy - level bones and balance pressure
  - (essential to understand their spine-pelvis-hips)
- flaps - one or more as required for the circumstances
  - (limited times when direct closure is eligible)
- staged recon if h.o. or hip resection is needed
- multiple wounds - concurrent or staged (circumstances)
- flap choices (don’t believe everything in the books)
  - anatomy, biomechanics, wound closure principles
- technicalities
  - one flap per wound; multi flaps per wound;
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  - ** know your pedicle and vascular anatomy,
  - “read the waves” and have a “lifeboat”
- post-op care
  - flotation bed superior
  - plan 25 days bed confine for sacral, ischial, troch.;
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WOUNDS : **Pressure**

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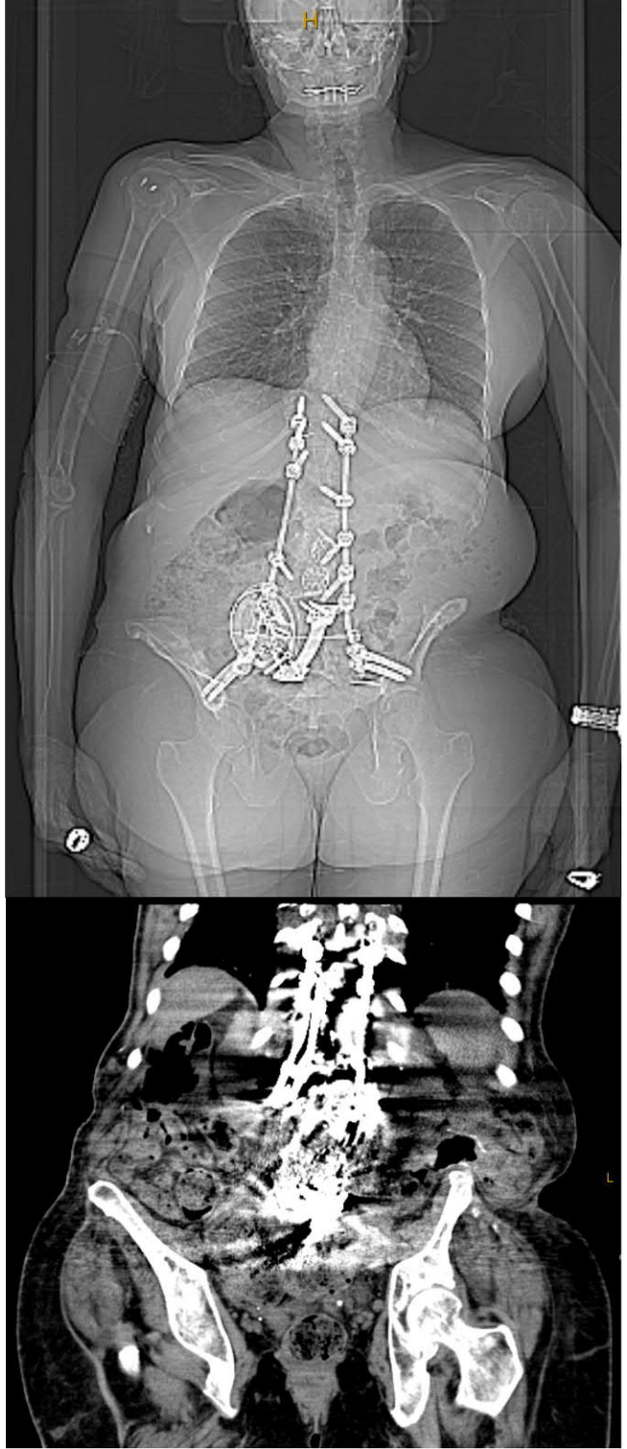

PRESSURE INJURY & ULCERATION ARE COMPLICATIONS OF NEUROPATHY

Pressure injury is almost **EXCLUSIVELY** an affliction of the neurologically impaired.

IMPAIRED
sensation
mobility
reflexes
biomechanics

SHORT TERM
coma
sedation
stroke
etc

LONG TERM
spine injury
periph nerve
neuropathies
myopathies
palsies
spasticities
ms
cp
etc

WHY PEDIATRIC PRESSURE ULCERS TYPICALLY OCCUR AROUND PUBERTY

NEUROLOGICAL PREDISPOSITIONS

Spina bifida
Cerebral palsy
Muscular dystrophies
Other neuromuscular disorders

BIOMECHANICS OF GROWTH

Capillary perfusion pressure
Area-to-volume scaling ratios

PRE-TEEN & TEENAGE BEHAVIORS

Rebellion
Attention deficit or distraction
Mental deficits
Social & peer pressures
Shift to adult responsibilities
"Caught unawares"
Lack of preparedness for this transitional period (patient & family)
Ageing family

length
$x\ 2^1$
$= 2$

area
$x\ 2^2$
$=4$

volume
$x\ 2^3$
$= 8$

Pressure = force / area
L = linear scaling factor

$P_n / P_0 =$

$(f_n/a_n) / (f_0/a_0) =$

$(f_0 \cdot L^3 / a_0 \cdot L^2) / (f_0 / a_0) =$

$L^3 / L^2 = L$

Pressure change = linear scaling

Teenage growth spurt equals bad news for butt sores.

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staged as needed for blood loss, safety, wound quality

hip or proximal femur resection (select patients)
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incidental bowel and bladder procedures (select patients)

tendonotomies, neurectomies, casts, etc. (select patients)

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WOUNDS : Pressure

General Principles ::

except for some incidental trauma conditions,
pressure ulcers are a consequence of neuropathy
no neuropathy, no pressure ulcer

common scenarios :

- senile disabilities
- bed or chair confinement, hospital acquired
- paraplegia
- quadriplegia
- ms, als, misc, etc.
- pediatric - cp, spina bifida, etc.
- syndromic profiles, e.g. geriatric, "angry young man"
- psycho-socio-economic stress

pressure anatomy, biomechanics, pathology, pathogenesis :

- essential to understand this and know the details

obligations :

- manage the wound
- close the wound if and when eligible
  - ** pressure ulcers rarely close without surgery
  - no rush - patients must earn their surgery
- manage pressure exposure
  - seating, bedding, age dependencies
- prevent more wounds
- manage the patient and neuropathy
- manage the sequelae of the primary disorder
  - **urinary**, bowel, neurologic, msk, lungs
  - neurogenic bowel-bladder : flaccid vs spastic
  - orthotics, therapies, functional integration
  - autonomic dysreflexia ** the good, & the bad
  - spasticity
- protect the patient from [the abundant] inexpert care
- incidental peculiarities
  - hyperhidrosis
  - urinary calculus
  - heterotopic ossification, h.o. / m.o.
  - syrinx, tethered cord
  - chronic pain
  - vascular disease & time-pressure
  - pubic / pseudo-ischial
  - urethral fistulas
  - non-pressure wounds in same areas
    - exposure-shear, dermatoses, cancers
    - internal iliac aso or infarcts, ano-rectal pathology
  - misc

Cheat sheets for hospitalized pressure sore patients.

You did not admit them, don't know them yet, you were consulted :

- stop antibiotics
- fluids - LR or similar 125 cc/ hr or 3 liters daily
- urinary antiseptics
- proper wound care
- pressure relief surface
- take advantage of being there to evaluate anything required:
  - pelvis-spine-hips, x-ray & ct, for biomechanics & h.o.
- urinary eval
- get patient out of hospital as expeditiously as possible
  - to prevent unnecessary problems, and
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- if possible, pre-admit by 2-3 days for pre-op prep
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- urinary antiseptics : mandelamine hippurate, 1 gm bid (or nitrofurantoin)
  - vit C, 500 - 2000 mg bid
- pulmonary care : not needed for para's
  - mandatory for quad's :
    - svn's, pos.press modalities, +/- bronch, baseline cxr, oximetries, abg's
- bowel care : pt's routines, laxatives, enemas, dig stim
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Pressure sore surgery - 0 - Principles

see Session 1 - EDSMI, segmental, pressure math

Pressure sore surgery - 1 - Preliminaries

- debridement & wound prep in OR : ONCE if at all
- resect pathological bone : ONCE if at all
  - (these have usually been done in office / clinic)
- resect heterotopic bone (select patients)
  - *staged as needed for blood loss, safety, wound quality*
- hip or proximal femur resection (select patients)
  - *staged as needed for blood loss, safety, wound quality*
- incidental bowel and bladder procedures (select patients)
- tendonotomies, neurectomies, casts, etc. (select patients)

Pressure sore surgery - 2 - Flaps and closure

- ONLY WHEN READY
- excise wound
- ostectomy - level bones and balance pressure
  - (essential to understand their spine-pelvis-hips)
- flaps - one or more as required for the circumstances
  - (limited times when direct closure is eligible)
- staged recon if h.o. or hip resection is needed
- multiple wounds - concurrent or staged (circumstances)
- flap choices (don't believe everything in the books)
  - anatomy, biomechanics, wound closure principles
- technicalities
  - one flap per wound; multi flaps per wound;
  - multi wounds per flap; multi-pedicle flaps;
  - ** know your pedicle and vascular anatomy,
  - "read the waves" and have a "lifeboat"
- post-op care
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resect pathological bone : ONCE if at all
(these have usually been done in office / clinic)

resect heterotopic bone (select patients)
staged as needed for blood loss, safety, wound quality

hip or proximal femur resection (select patients)
staged as needed for blood loss, safety, wound quality

incidental bowel and bladder procedures (select patients)

tendonotomies, neurectomies, casts, etc. (select patients)

Pressure sore surgery - 2 - Flaps and closure

ONLY WHEN READY

excise wound

ostectomy - level bones and balance pressure
(essential to understand their spine-pelvis-hips)

flaps - one or more as required for the circumstances
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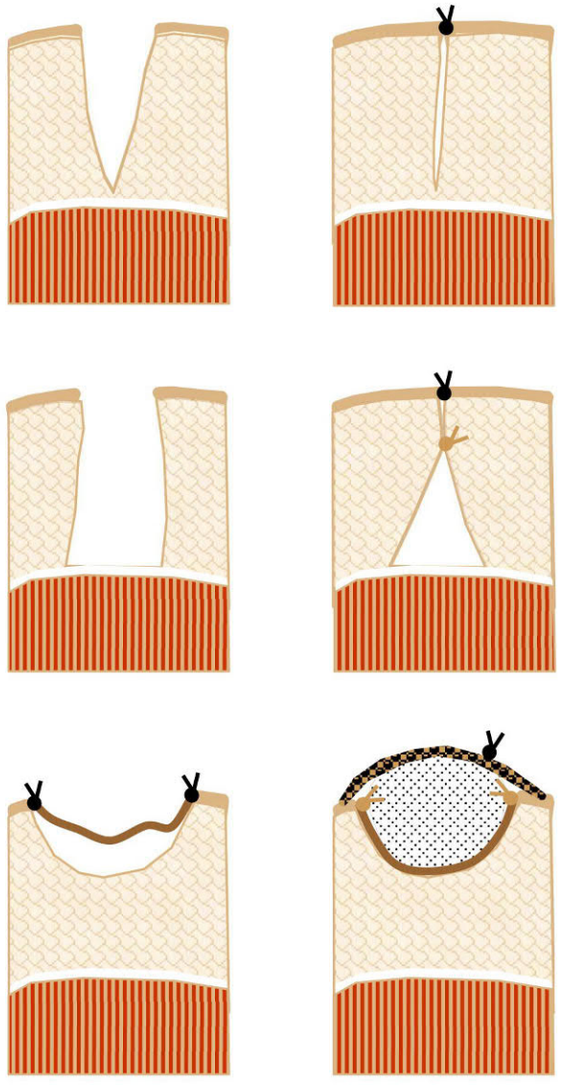
DEAD SPACE

MOTION - INFLAMMATION



MANDATORY
for successful repair:

Tissue-to-Tissue Coaptation

Tissue Interface Stability Without Shear or Distraction

DEAD SPACE CONTROL

1 - Positive pressure from without.

2 - Negative pressure from within.

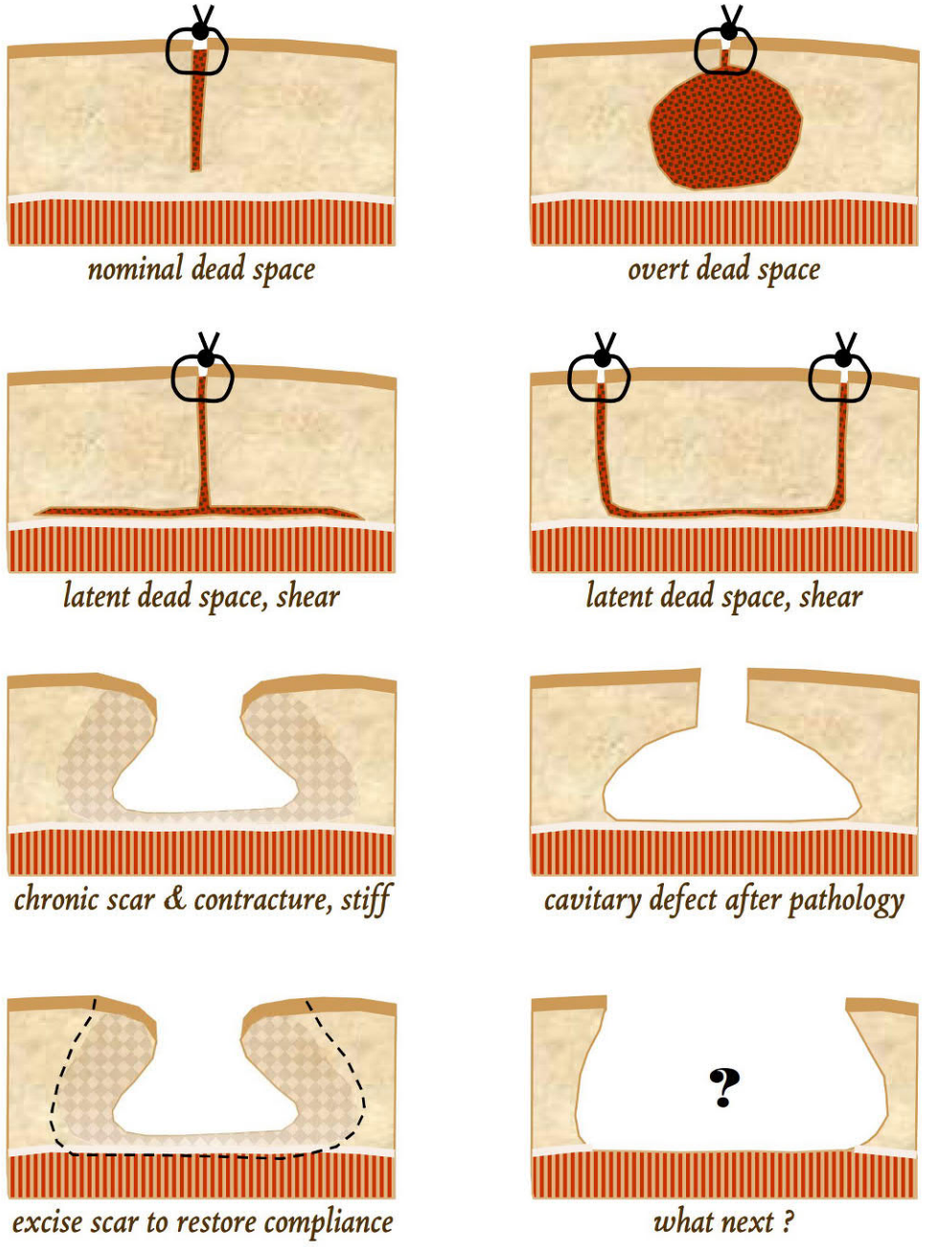
3 - Gather and bind space.

4 - Fill the space.

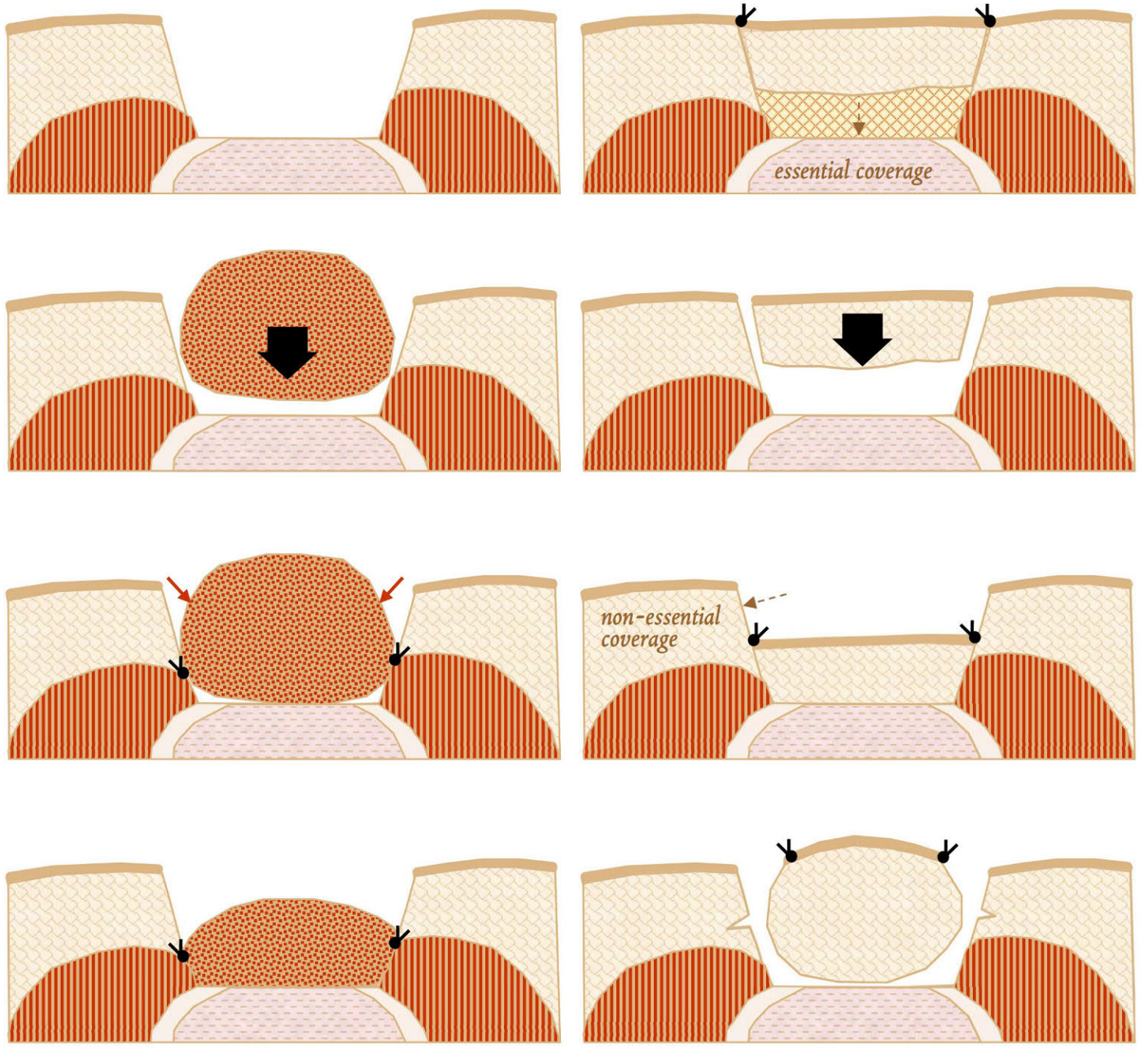
(flaps, biomatrices, cement)

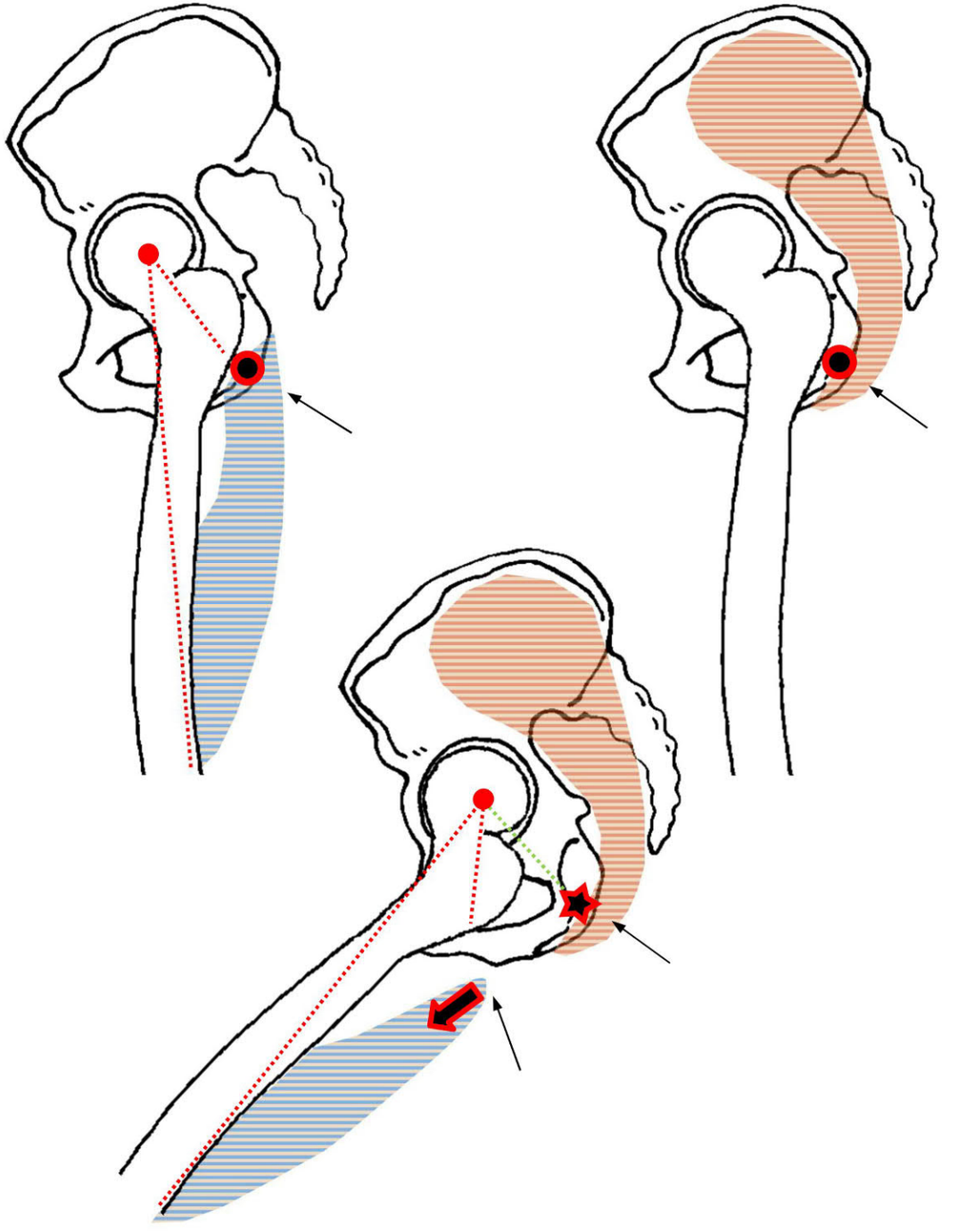
Wound remodeling.
Proper dissection &
preemptive avoidance.

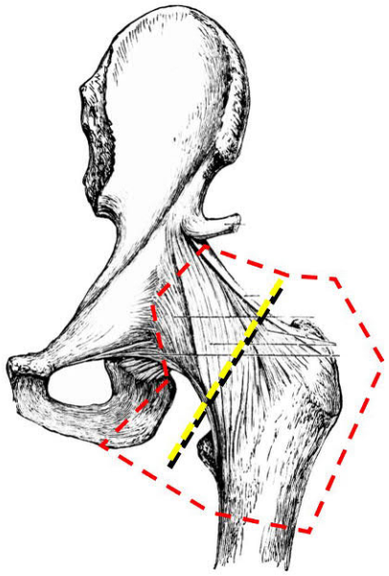
WOUND GEOMETRY & MECHANICS

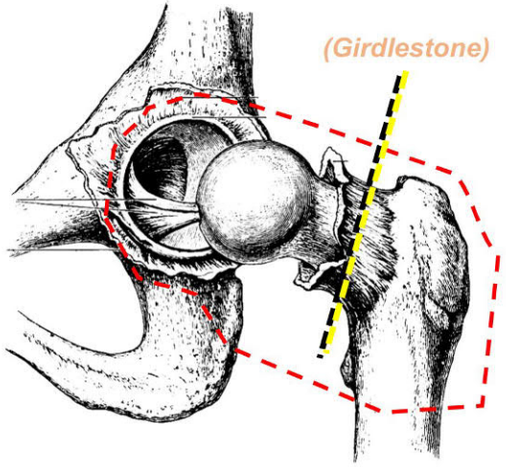


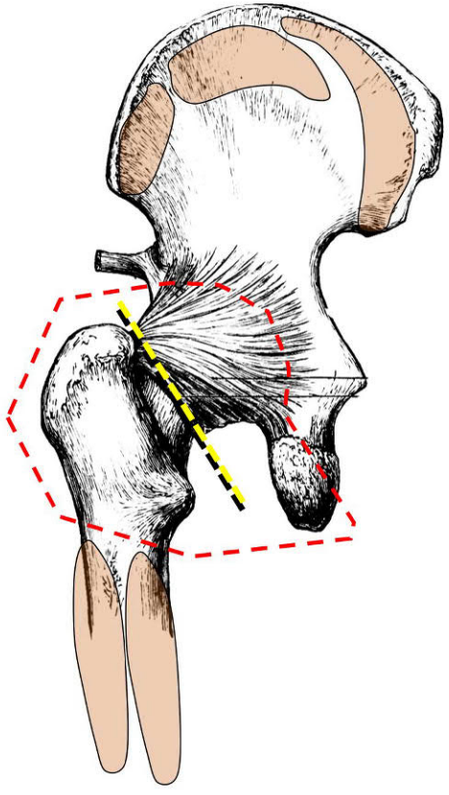
Filling Space with Flaps :: Technicalities & Pitfalls

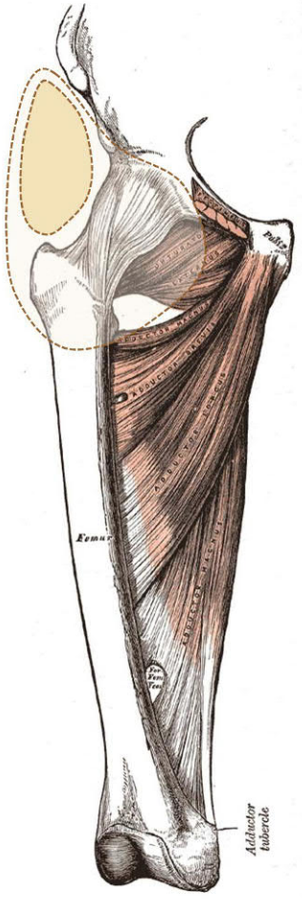


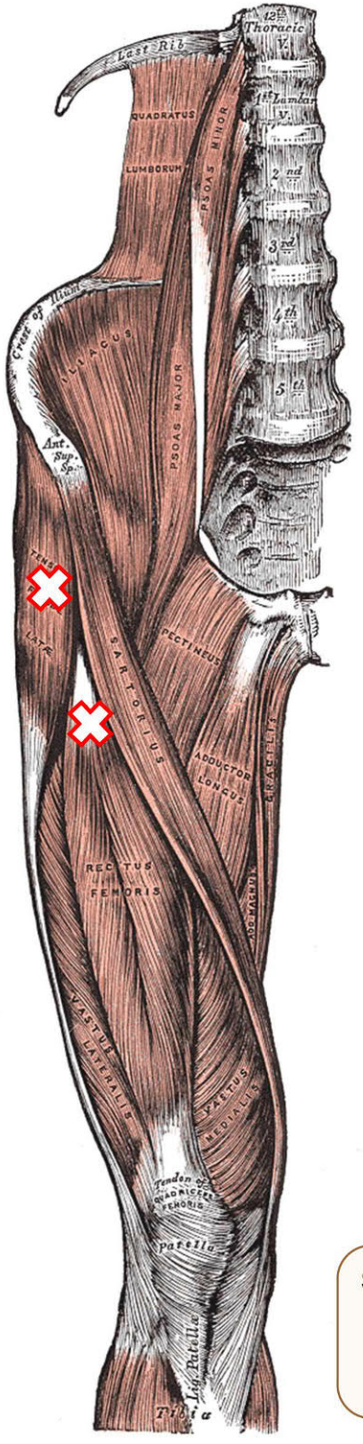


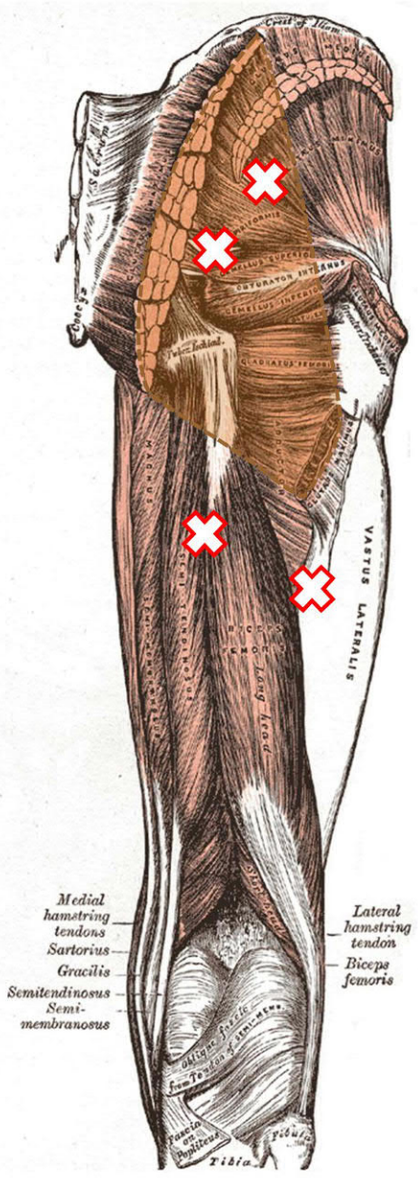


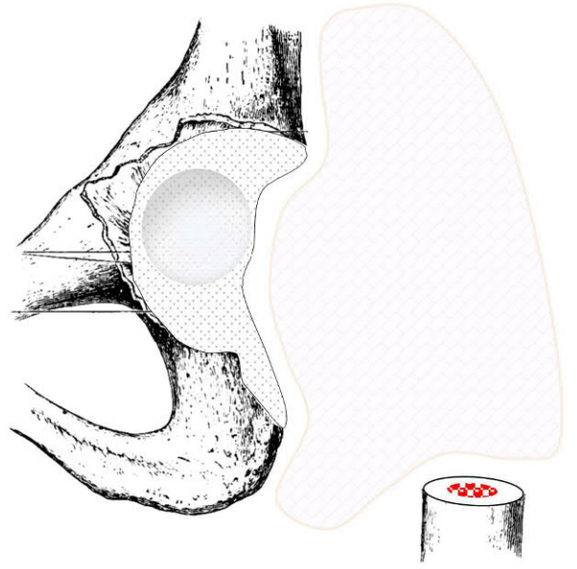


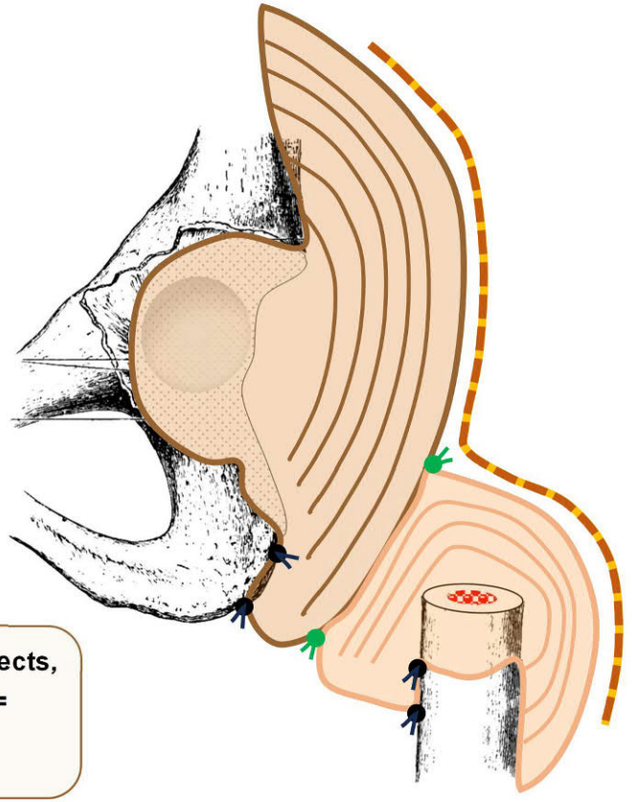












Segmental anatomy, large or complex defects, wounds with motion and dead space =

Multiple Flaps

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Technical ::

Ischial

- seating
- pressure bursa
- occasionally heal with exquisite care (6-24 months)
- hip spica or body splint
- pelvis-to-pelvis flaps
- skiotog - skin island on tail of gluteus
- lateral / subcutaneous ostectomy

Trochanter

- natural bursa
- rarely heal with exquisite care (6-24 months or more)

Sacrum

- technical challenges to flaps
- post-op bedding crucial

Coccyx

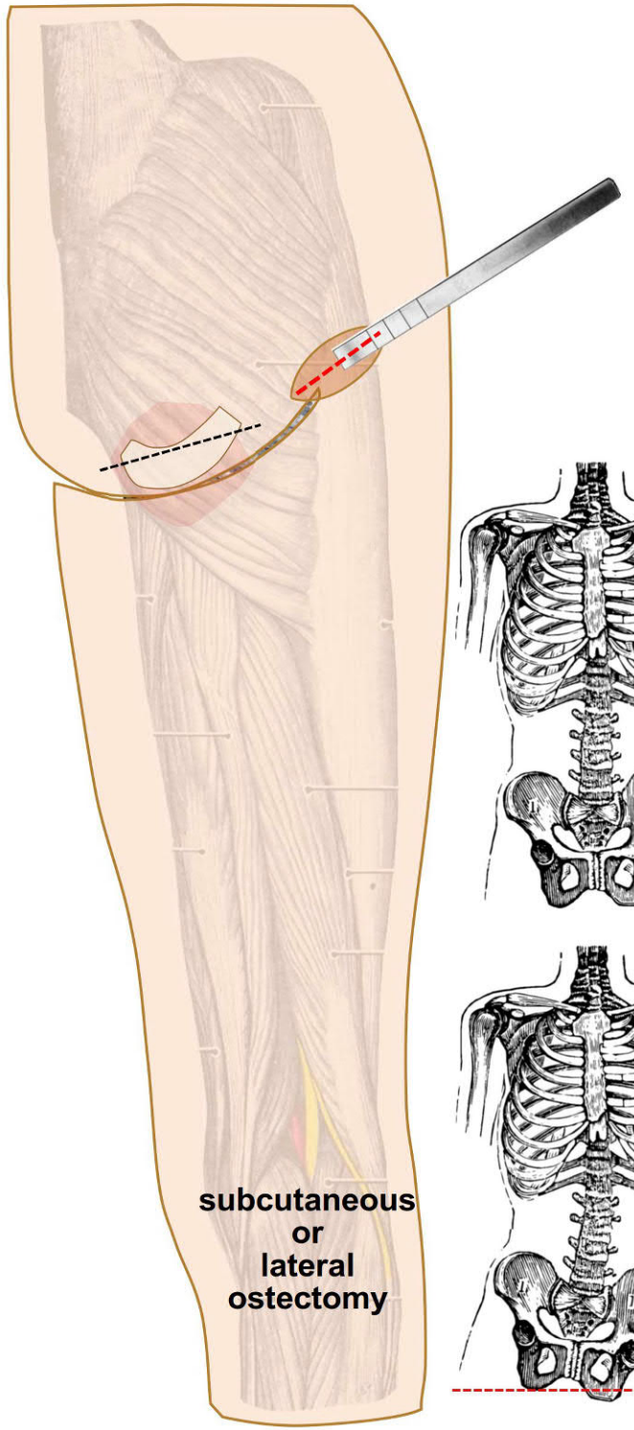
- do not overlook rectal pathology
- leave tip of coccyx

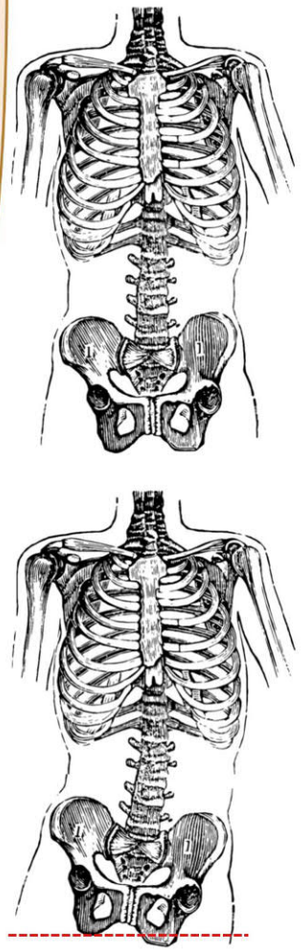
Pubis

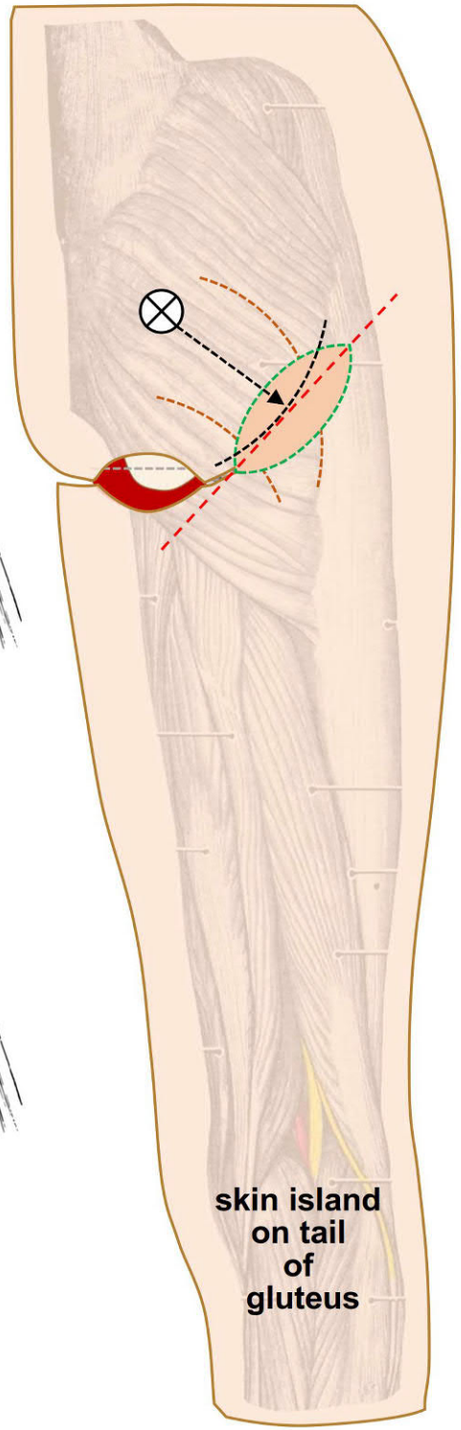
- evaluate spine
- complex orthotics & seating

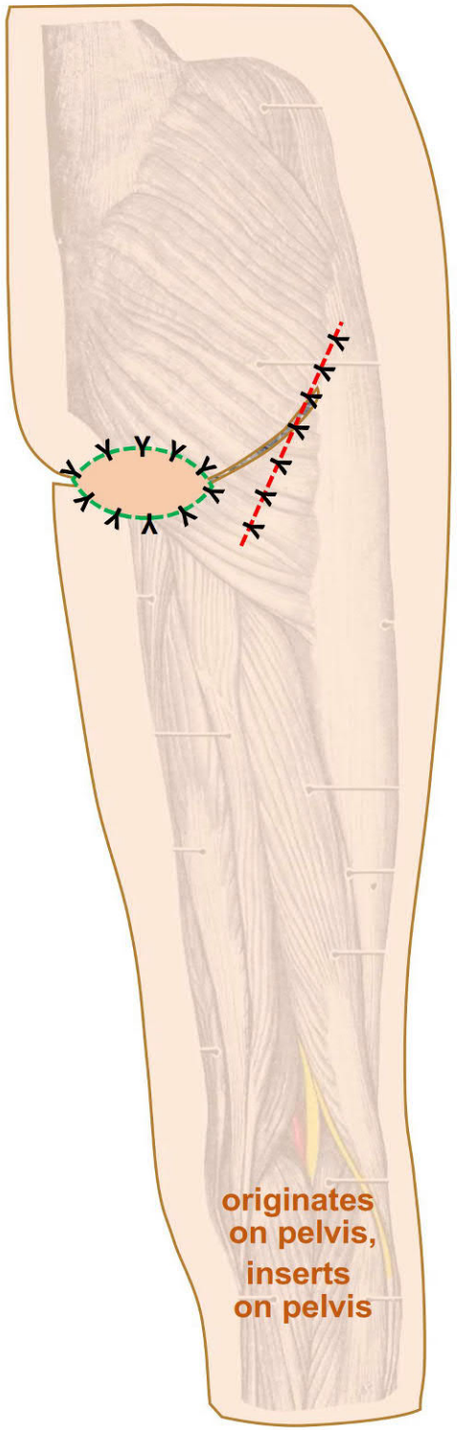
Leg and foot

- heel - calcaneus
- malleoli
- tibial ridge
- bandaging
- vascular





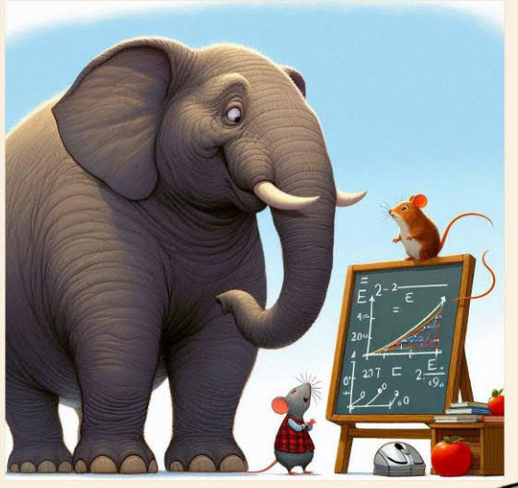


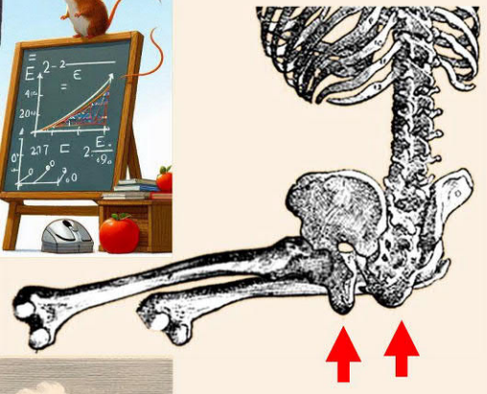


Quickies 2

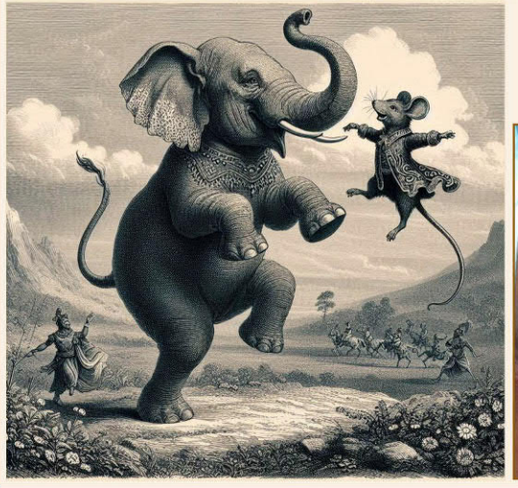
Why don't little kids get ischial pressure sores ?

Spina bifida, CP, etc. – only at puberty.

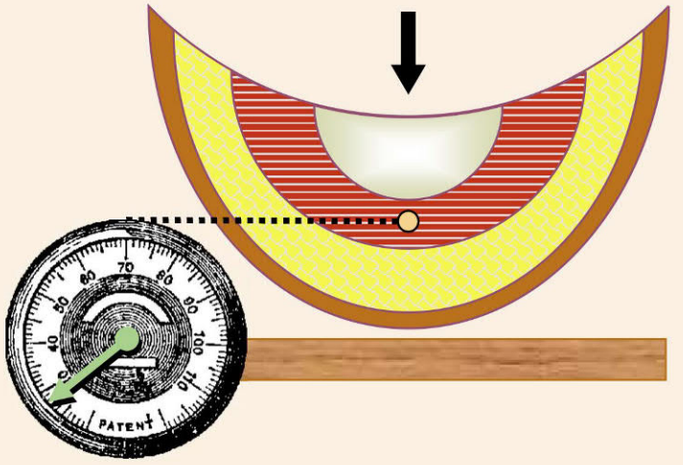




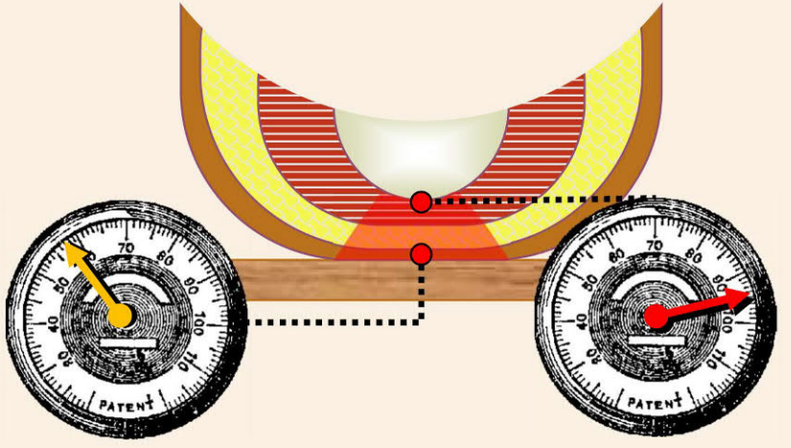
sitting = ischia



resultant force of body weight



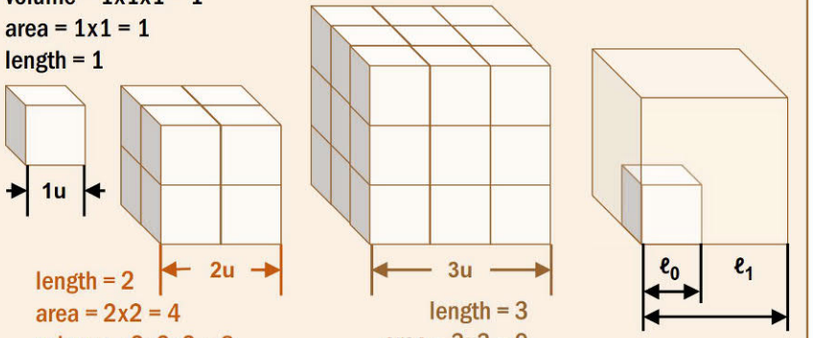
compression = force per area
compression > capillary BP
= ischemia



Why don't pre-pubertal kids get ischial pressure sores ?

The Physics of Scale.

volume = 1x1x1 = 1
area = 1x1 = 1
length = 1



length = 2
area = 2x2 = 4
volume = 2x2x2 = 8

length = 3
area = 3x3 = 9
volume = 3x3x3 = 27

$\ell_1 = S \cdot \ell_0$

S = L_1 / L_0 = linear scaling ratio.

Pressure = force / area = mass x g / area

For pt in wheelchair, assume upper body = 2/3's total mass; Approx. values :

age	height cm	pelvis+upper body, kg	w.c. contact area, cm²	bi-ischial area, cm²	skin press. torr	bi-ischial press., torr
10	140	15	400	4x1 x 2	28	1400
12	145	18	430	4.5x1 x 2	31	1500
14	160	25	530	5x1.1 x 2	35	1700

For growth with a linear increase of factor **S** :
any surface or cross section (area) scales by S^2,
any volume scales by S^3.
Pressure is force / area, P = F / A .
Assuming consistent density, force ≡ mass ≡ volume.

So, $\delta P \equiv \delta F / \delta A \equiv S^3 / S^2 = S$. **$\delta P = S$** .

Contact pressures increase proportional to linear scaling S.

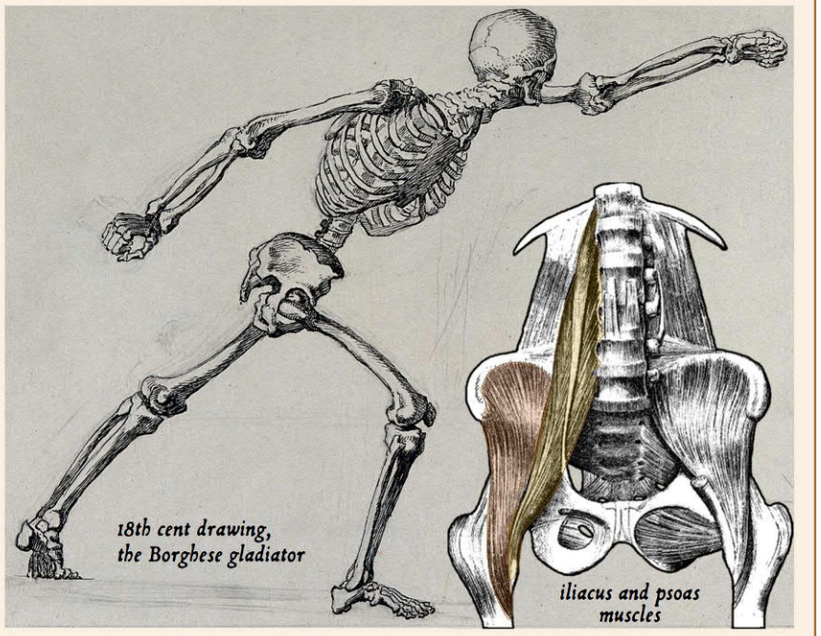
Before puberty, contact pressures on wheelchair, under ischia, are approximately arteriolar-capillary.

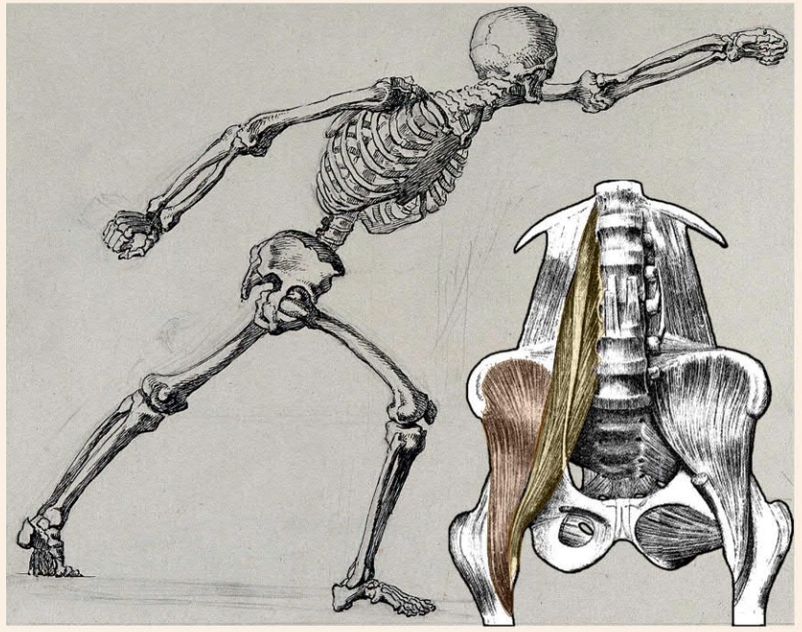
After puberty and growth spurt, they exceed those pressures.

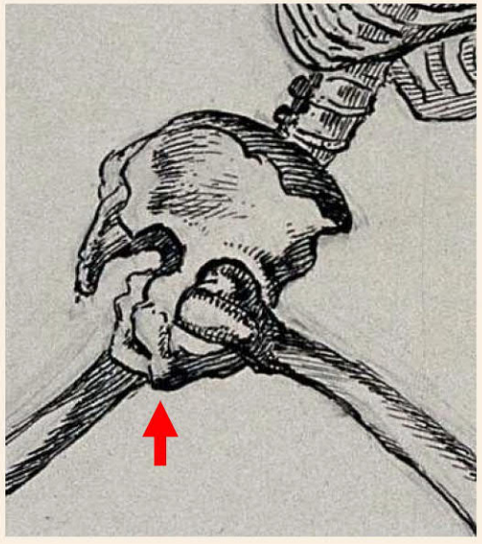
Quickies 3

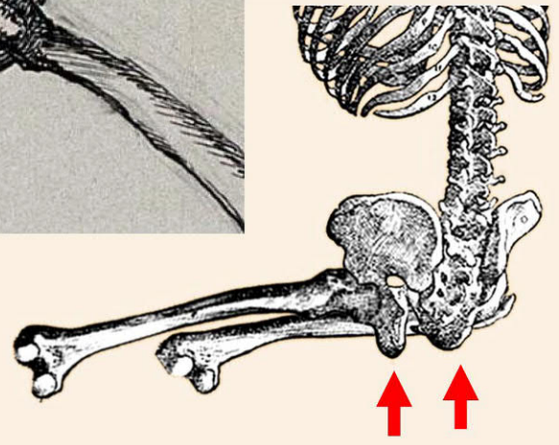
Why do AKA patients get ischial pressure sores ?

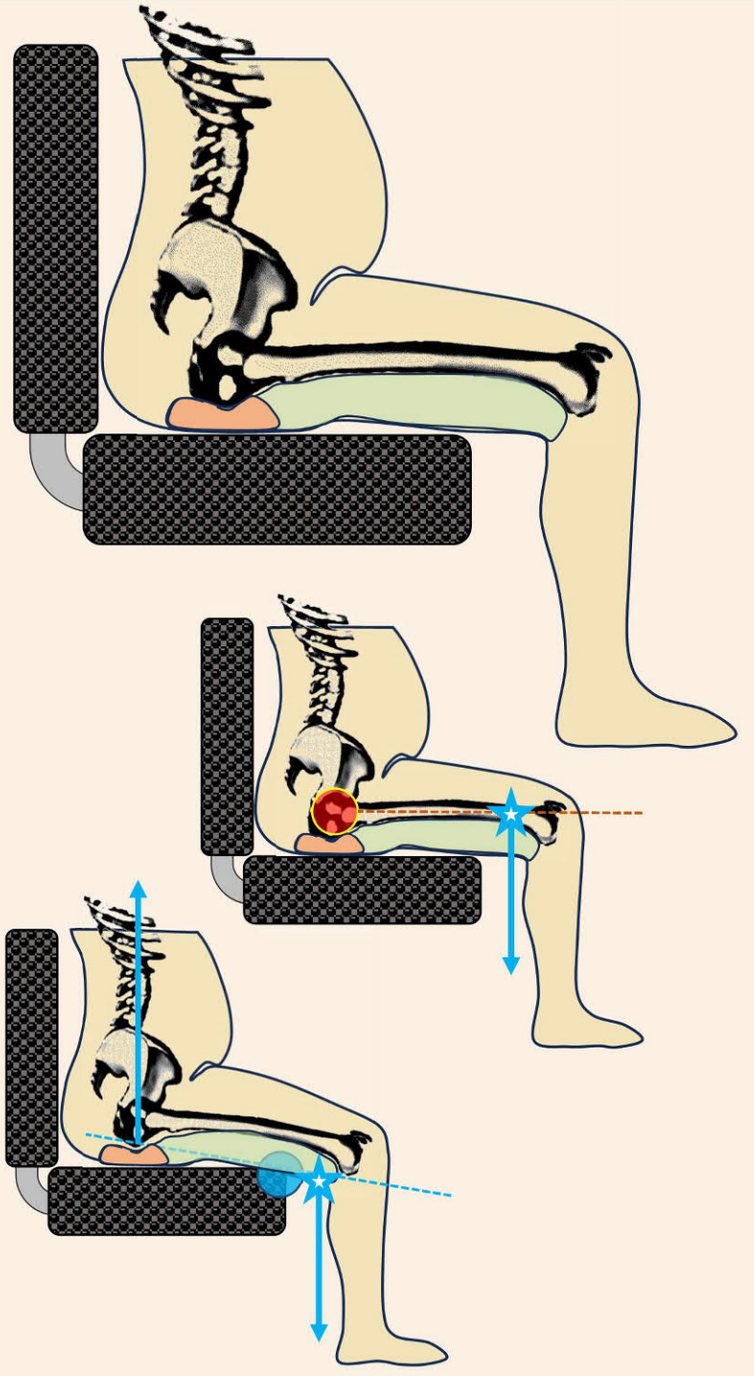
Especially old, infirm, and arteriopaths.











Why do AKA patients get ischial pressure sores ?

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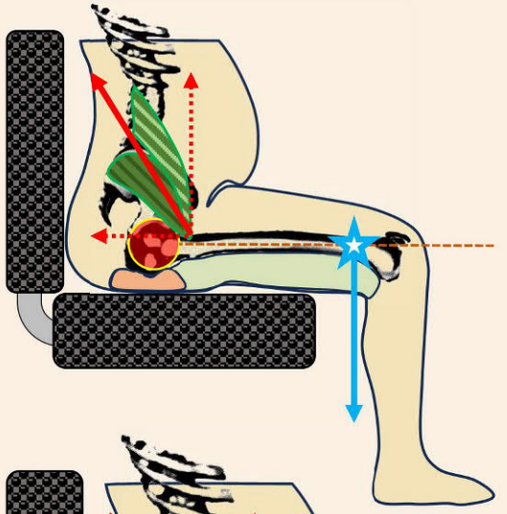
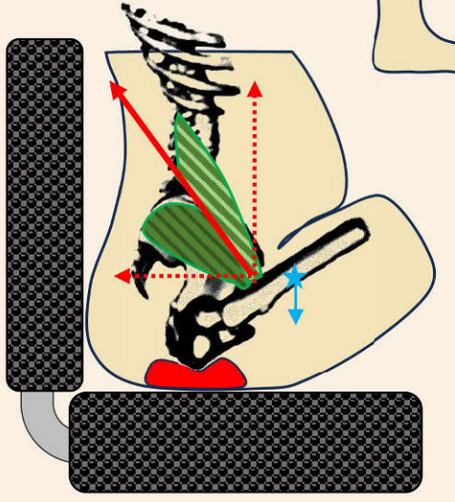


With a full lower extremity, limb center-of-gravity creates extension torque around hip to balance flexion of iliopsoas muscles.



Thigh contributes to body-mass load-bearing and dissipation of sitting pressures.

With lower extremity removed, mass is reduced, center-of-gravity moves far proximal, extension torque drops.

Flexion torque from iliopsoas is unopposed, thigh swings up, and contact surface disappears except focus under ischium = pressure ulcer.

WOUNDS : **Pressure**

Incidentals ::

almost all "99%" of pressure ulcers need surgery to close
that is a simple fact of wound anatomy and physiology

but, not all patients need or should have surgery, not all pressure ulcers need to be closed
e.g., geriatric wounds in patient with good nursing

but, ALL patients need proper overall care and management
living with the wounds if no surgery planned
living with them if surgery is pending for future
preparing wound and patient for surgery

integrated care
wounds, nursing
urine, bowel, dysreflexia
seating (w.c. & cushion), bedding
connect pt to high quality primary care, urology, PM&R
lifelong care
"an ounce of prevention is worth a pound of cure"

patients must prove their eligibility for surgery
surgery without proper longitudinal planning fails
ad hoc butt sore closure in new inpt consults fails

for surgery :
no surgery until wounds are ready
no surgery until patient is ready
do not waste a flap
do not compromise care due to 3rd party factors

do not overlook opportunities for lateral ostectomies
treat bad hips without intimidation
understand each patients' spine, pelvis, hips

diagnosis
do not overlook vascular disease influences
do not confuse pressure wounds with other wounds in contact areas, or on the pelvis or hips
hardware complications
iliac columns vs sacrum - influences surgery done

others
dural and csf fistulas
technically, pressure ulceration starts first at most rigid point in system
no colostomies for para's (unless explicit indications)

WOUNDS : **Pressure**

Incidentals :: **diagnostics & lab**

limited necessary studies
plain x-ray, spine, pelvis hips (scoliosis study ideal)
ct same areas often helpful (but often redundant)

cbc, cmp, u/a

vascular studies on at risk patients

pressure mapping, seating evaluation

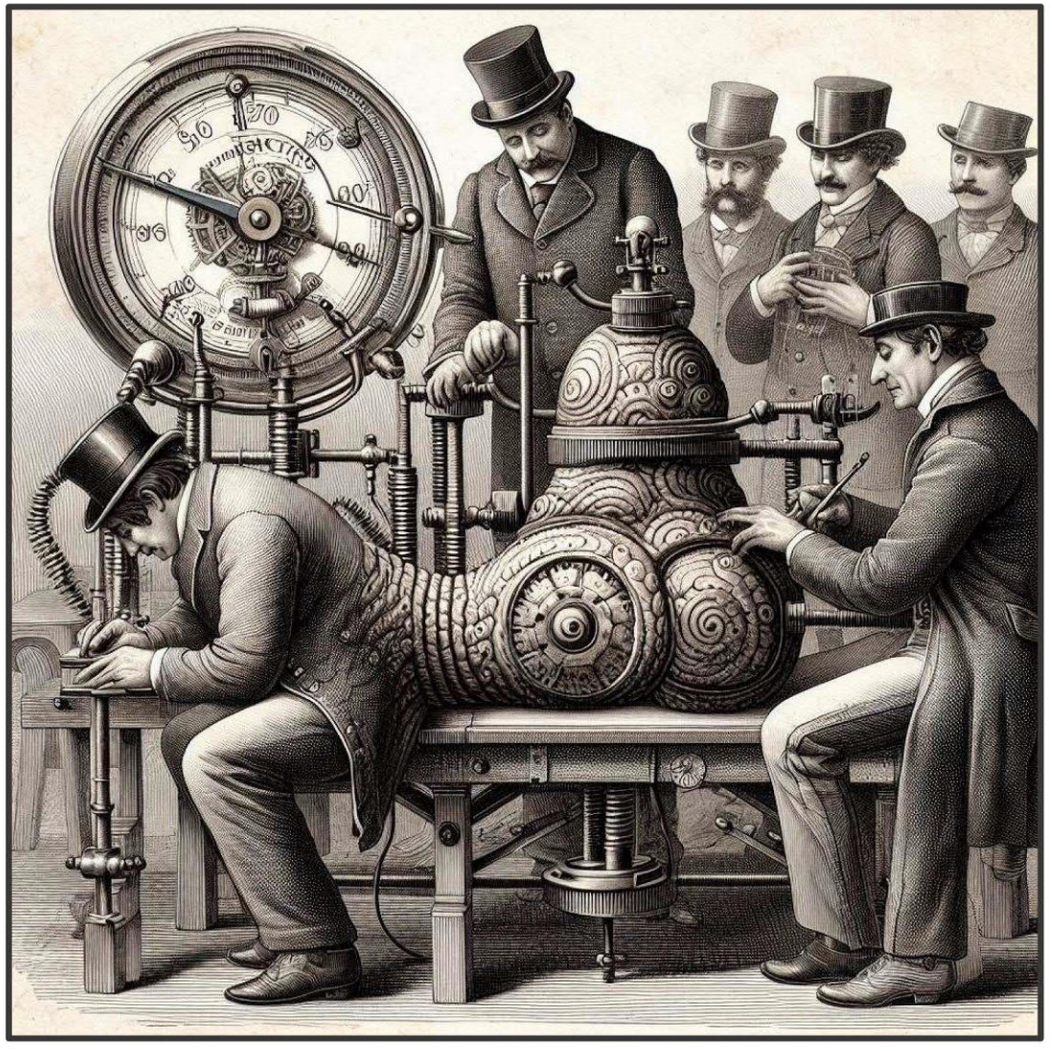
histo excised material - pro forma, sometimes useful

do not order tests as per bad habits of hospital based non-experts

no mri or other 3rd party make-money tests

no cultures and microbiology - inaccurate, misleading, simply unnecessary dangerous to patients in hands of amateurs

Most tests are ordered by doctors with no knowledge of this subject. They are not needed, contribute nothing to care and cure, and risk serious unnecessary complications and morbidity by leading to expensive errant care. **Don't be THAT doctor.**



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Marc E. Gottlieb, MD, FACS
Phoenix, AZ

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HYPERCOAGULABLE DISORDERS – IMPLICATIONS FOR WOUNDS & SURGERY

PATHOPHYSIOLOGY, CLINICAL FEATURES, DIAGNOSIS & TREATMENT

AND

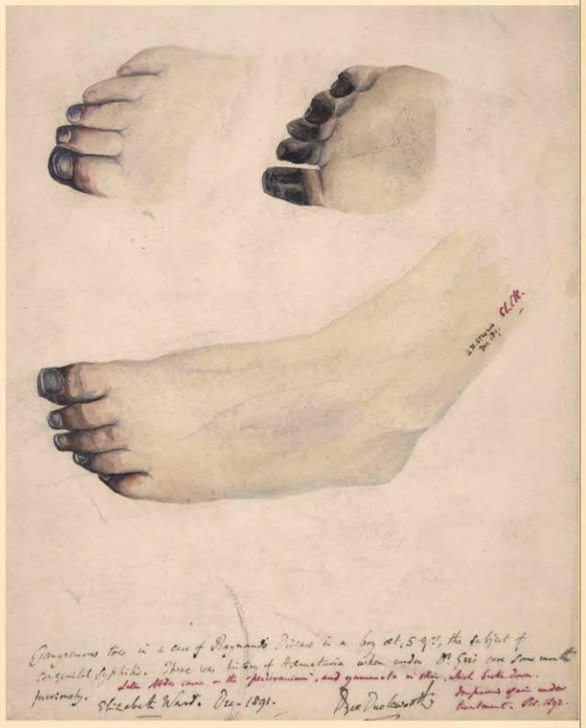
Insights About the Historical Understanding of this Subject and Why These Problems Remain Perpetually Under Appreciated, Under Recognized, and Under Treated.

Marc E. Gottlieb, MD, FACS September, 2018 Phoenix, Arizona Arimedica.com

Mortification, gangrene of the toes. Robert Carswell, London, 1837.

Dry gangrene after ligature of popliteal aneurism. Thomas Godart, London, 1880.

Gangrene of the hand from idiopathic arteritis. Thomas Godart, London, 1880.



Gangrenous toes from Raynaud's Disease, 5 year old boy with congenital syphilis. A.F. Stevens, 1891.

Photos at beginning not available.

Four months into effective care.

Most of the original wound is closed and healed. Open areas shown are part of the deliberate staging of this reconstruction.

— BUT ALSO —

Four weeks absent from hospital.

RA and inflammation have flared. Vascular stasis and signs of incipient tissue infarction give a sense of the original presentation.

Prompt correction after starting steroids and argatroban.

---

50 day interval from above image.

Small incidental wounds all healing.

Wound at confluence of flaps (low center) is expected.

Wound at base of spine (L2) is open by design to protect rest of the repair (to be repaired in a few weeks).

Stasis, cyanosis, ischemia, necrosis, infarction, & abnormal inflammation have all ceased with anticoagulants.

UNDERLYING PROBLEM

34 m :: Paraplegia and pressure ulcers.

Wound pathergy and progressive surgical wound infarcts resulting in translumbar amputation.

Recent onset severe Rheumatoid arthritis.

STATUS AT ACCEPTANCE

TLA infarcted, complex abdomino-pelvic wounds.

SUCCESS AFTER:

Proper wound care.

Coagulation w/u then Rx.

Heparin (–> HIT), then argatroban, then rivaroxaban.

Continuous argatroban during & after surgery.

Steroids & multimodal Rheumatoid Rx.

Proper staged surgery.

Positive Lab Studies

Species	*Value*	*Normal*
Fibrinogen	**632 H**	< 465
D-dimer	**892 H**	< 500
TAT cmplx	**4.5 H**	< 4.0
F.VIII	**231% H**	50-150
Protein C	**69 L**	70-140
RheumF	**108 H**	< 13
CCP IgG	**119 H**	< 16

Left top, wound & fistula.

Left bottom, start of the exposure.

Right top, completed surgery with stoma through hypogastric flap.

Right bottom, 8 days, no ischemia or necrosis except next to stoma, from perforating the flap, not coagulopathy.

UNDERLYING PROBLEM

52 m :: Diverticulitis & complications.

Wound pathergy and surgical infarcts resulting in abdominal wall loss and entero-cutaneous fistula.

Multiple infarcts & leaks: bowel, anastomoses, wall.

STATUS AT ACCEPTANCE

Complex abdominal wound, open bowel & fistula.

SUCCESS AFTER:

Proper wound care.

Coagulation w/u then Rx.

Heparin, then apixaban.

Continuous heparin during & after surgery.

Inflammatory bowel disease ruled out.

Proper staged surgery.

Positive Lab Studies

Species	*Value*	*Normal*
Fibrinogen	**1101 H**	< 465
MTHFR	**heterozyg**	neg
F.VIII	**271% H**	50-150
AT-3	**73 L**	83-128
Protein S	**53 L**	55-146
ANA	**pos H**	neg
Chrom.Ab	**pos H**	neg
dsDNA	**7.0 H**	< 4.0
Saccharo.A	**113 H**	neg
Saccharo.G	**76 H**	neg

A NOMENCLATURE OF THROMBO- & MICRO-OCCLUSIVE DISORDERS

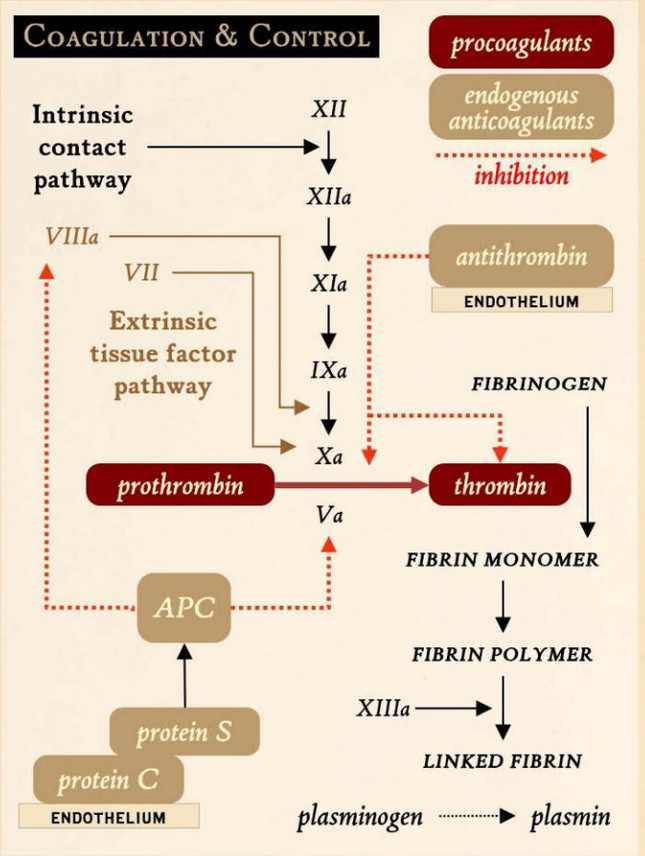
Hemodynamic Disorders	vessels, blood, & coagulation normal **Fluid Dynamics Abnormal**	Hemodynamics & macro-vasculopathies *Examples :* a-v malformations, atrial fibrillation vascular compression, low flow states
Endo-Vasculopathies	blood & coagulation normal **Vessels Abnormal**	Intrinsic disorders of blood vessels *Examples :* atherosclerosis thromboangiitis, alloplastic implants
Exo-Vasculopathies	blood & coagulation normal **Vessels Abnormal**	Extrinsic disorders of blood vessels *Examples :* vasculitis, hyperparathyroidism, immune cv-ct disorders
Non-Hypercoag Hemopathologies	vessels & coagulation normal **Blood Abnormal**	Altered blood elements, non-plasma *Examples :* formed element abnormalities, hemoglobinopathies, dys- & cryoproteinemias
Hypercoagulability	vessels & blood normal **Coagulation Abnormal**	Disorders of the plasma coagulation system Intrinsic: thrombophilic - prethrombotic disorders Extrinsic: immune-apl, estrogens, cancer

Micro-occlusive disorders are a major cause of chronic ulceration, impaired wound healing, and complications of trauma and surgery. Little appreciated by most physicians, this subject requires broader awareness. Here is a conspectus of the subject, and a nomenclature of disease, focused on hypercoagulopathies.

These categories can each be subcategorized.

This presentation will focus solely on the further nomenclature of the hypercoagulable disorders.

COAGULATION PHYSIOLOGY & PATHOLOGY



1 - Normal coagulation

Thrombosis stops bleeding.
It is a complex control system tuned to not trigger if plasma stays within normal blood vessels.
Altered vessels or flow trigger the healthy process, rightly (trauma) or wrongly (e.g. vasculitis).

2 - Hypercoagulopathy

Normal blood does not clot in normal blood vessels, but System can become untuned:
- less prone to clot when it should (hemorrhagic hypo-coagulopathy), or
- overly prone to clot when it shouldn't (thrombotic hyper-coagulopathy).

Hypercoagulable blood clots spontaneously in vessels, or is more sensitive to ordinary triggers.

3 - Pathophysiology of errant clotting

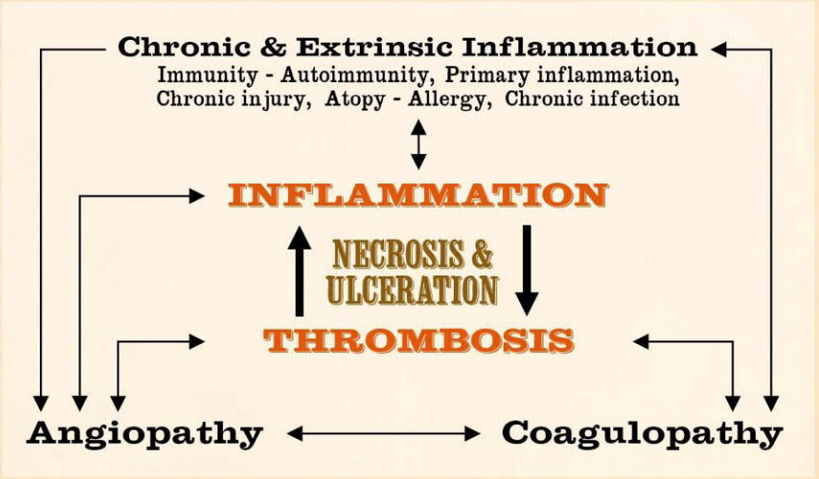
Thrombosis occurs in intact vessels, interrupting blood flow.
Large vessels and structures are at risk for major infarct or death.
Small vessel non-lethal events cause vascular stasis and micro-infarction (becoming ulcers).
Inflammation is triggered. Stasis & inflammation are in turn normal triggers for thrombosis.
Hypercoagulopathic events are thus auto-amplifying, and self-perpetuating.

4 - Biochemistry

The main sequence cascades and proteins are basic medical education.
Every step in the process has multiple promoters and inhibitors.
All can become unbalanced or dysfunctional to promote abnormal clotting.

5 - Dynamical disorder

This is a complex non-linear multi-control system. Dynamics are chaotic.
When healthy, it is self-stable.
When unstable or in a stable but unwanted state, unpredictable events can occur.
This means is that patients can be variably normal then abnormal.
Hypercoag. patients are not always hypercoagulable, even with hypercoag. genes.



A NOMENCLATURE OF THE HYPERCOAGULABLE DISORDERS

Primary Alteration	Effects	Example Species
Intrinsic disorders "Pre-thrombotic" or "Thrombophilic"	Defects, deficiencies, altered levels of primary clotting factors and para-thrombotic proteases. Includes gene mutations and acquired or episodic variances and imbalances of any of these factors.	proteins C & S, AT-3, f.VIII, fibrinogen, f.V Leiden (gene R506Q), prothrombin mut. (gene 20210G)
Extrinsic	Coagulation imbalances triggered by disease, injury, metabolism, drugs, hemodynamics, etc. Conditions causing inflammation, vascular stress or injury, stasis, platelet activation, plasma imbalance.	inflammation, platelets, hemodynamics, formed element hematopathologies, dys- & cryoproteinemias
Immune - inflammatory	Autoimmune procoagulants. Association with connective tissue disorders. Intimate association of clotting and inflammation - mutual triggers and breeders, dynamic amplification.	apl-abx (lupus anti-coagulant, anticardiolipin), anti: beta-2-glycopr, anca, mpo, pr-3, autoimmune disease, general inflammation
Metabolic	Extrinsic triggers from disorders of specific organs or pathologies, or dietary and acquired factors. (In distinction to the generalized extrinsic stresses of inflammation, injury response, and altered circulation.)	warfarin, homocysteine, gene MTHFR, estrogen, pregnancy, pnh, para-neoplastic

Trigger conditions	Coagulation balance	Implications & examples
Trauma	Normal trigger for thrombosis.	Local and remote, trauma and surgery.
Inflammation	Normal & errant trigger for thrombosis.	Acute & reactive, immune, circular amplification.
Hemodynamic	Errant trigger for thrombosis.	Macrovascular stasis & eddies, small vessel rheology.
Hematological	Elements that engage the plasma system.	Platelets, granulocytes, immune & lytic red cell events.
Metabolic & Pharma	Trigger offsets or hypersensitivity.	Whatever affects blood or coag, including Rx meds.
Disease Associations	Other serious dx.	Immune, cancer, infections, etc.
Dysdynamia	Chaotic behavior of integrated coag system.	Large effect of small perturbations, basins of stability.
Combinations	Effects & risks additive.	System more sensitive, closer to triggering.

CLINICAL PATHOLOGY OF THE HYPERCOAGULABLE DISORDERS

Macrothrombosis

Large vessel
Acute
Overt
Life and limb risk

"Old hat"
Often easily recognized
Defined clinical syndromes

large vessel arterial thrombosis
large vessel venous thrombosis
other peripheral thrombosis
various thrombophlebitis
pulmonary embolism

coronary artery thrombosis
intracardiac thrombosis
graft and valve thrombosis
cerebrovascular thrombosis

subclavian v. (paget-schroeder)
hepatic veins (budd-chiari)
pituitary apoplexy (sheehan)
retinal artery & vein occlusion
intracranial sinus thrombosis
spinal apoplexy
visceral apoplexy *(renal, adrenal, bowel)*

The underlying hypercoagulopathy might nonetheless be overlooked.

Microthrombosis

Small vessel
Subacute, chronic, recurring
Occult, missed diagnosis
Tissue and wound risk

Under appreciated
Often non-obvious
Perplexing refractory problems

vascular occlusion not overt
often not life threatening
recognized by secondary events
young age
family history
associated diseases *(e.g. cvd-ctd)*
special tip-offs *(e.g. warfarin resistance)*
long history of failed care
long hx care for wrong diagnosis

complications of trauma & surgery
wound pathergy and infarction
non-anatomical flap necrosis
non-healing ulcers

miscarriage
complications of contraceptives

non-immune glomerulonephritis
primary pulmonary thrombosis
warfarin necrosis

Related Disorders

Other micro-occlusive classes
Hematological, vascular
Autoimmune cvd-ctd
Trigger diseases & conditions

Disease Associations

immune & chronic inflammatory
acute & chronic venous
estrogens, pregnancy
cancer (Trousseau)
parox. nocturnal hemoglobinuria

Others of Interest

primary pulmonary thrombosis
pulmonary hypertension
non-immune lupus nephritis, RPGN
digital ischemia of CTD / CVD
visceral infarcts & apoplexies
(e.g. pituitary, adrenal, bowel, spine)
an open field for inquiring minds

Hypercoag Syndrome

Tetrad — Pentad

Thrombotic or embolic event
Autoimmune cvd-ctd
Wound pathergy
Miscarriage
Family history of same

Core Pathophysiology

Normal blood is tuned to clot immediately on seeing non-endothelial matter, but never to clot when within normal blood vessels.

Hypercoagulable blood clots spontaneously within normal vessels.

Cf. Hypocoagulability

Consequences of hypocoagulability are often acute, overt, dramatic, immediately threatening, affect body and life as a whole, or else fit well defined dx (e.g. hemophilia).

The same is true for large vessel macro-vascular occlusive events.

In contrast, hypercoagulable states causing micro-thrombosis are often slow, subtle, insidious, chronic, occult, affect local or isolated tissues, and apt to be repeatedly missed, unrecognized, or misdiagnosed.

NECROSIS & ULCERATION – TWO GENERAL PATHOLOGIES & PATTERNS

THROMBO-INFARCTIVE

The pattern of ischemia and stromal deprivation.

Macro-occlusive

Micro-occlusive

Micro-angiopathies

Hemopathologies

Hypercoagulable / Coagulopathic

INFLAMMATORY-LYTIC

The pattern of inflammation and stromal predation.

Inflammatory

Autoimmune

Atopic, Suppurative

Connective Tissue Disorders

Lymphoreticular / Reticuloendothelial

CLINICAL PATHOLOGY OF HYPERCOAGULABLE WOUNDS & ULCERS

Onset of Illness

Acute micro-thrombosis & vascular stasis.
Severe local ischemia of skin and fascias.
Skin infarcts, progressing to ulceration.
Gross inflammation +/-, often absent.
Pseudo-inflammation from severe stasis.

Spontaneous -vs- triggered by an event.
Chronic or chronically recurring.

Common on lower extremities.
Can occur anywhere.

Link to Inflammation

Coagulation & inflammation are linked:
1° thrombosis triggers 2° inflammation.
1° inflammation triggers 2° thrombosis.

Some injuries purely one or the other domain.
Some wounds are inextricably mixed.

Strong association with CVD–CTD:
Ongoing trigger from chronic 1° inflamm.
Sustained 2° inflamm. induces autoimmunity.
Many patients have mixed lab profiles.

Findings

Ischemic infarction: skin, fascias, wounds.
Active ulceration, thrombo-infarctive.
Edema & gross inflammation often absent.

Periwound stasis, low TcpO2, pain.
Mixed wound module, non-healing.
No signs of other dx.
Good pulses.

Confirmatory blood tests & histology.

Acute necrosis.

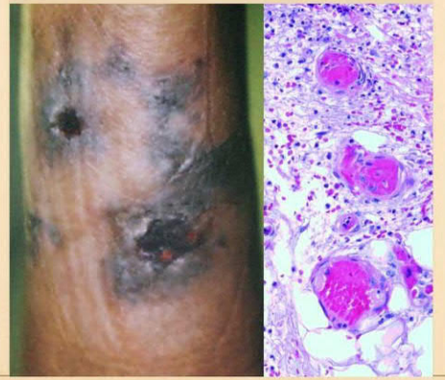
Skin infarcts are usually small, scattered, isolated, but sometimes large and life threatening.

35 yo woman, acute lupus.
Extensive skin infarcts (hips & thighs shown). Antiphospholipid antibodies. Low skin TcpO2s.

Stasis and infarcts.

Around the infarcts are zones of severe stasis which may die and ulcerate (or recover).

43 yo man, spontaneous leg ulcers.
Very low proteins C and S (leg & ankle shown). Small vessel thrombosis and organization, with adjacent stasis, congestion, and hemorrhage.



Chronic active ulceration.

Post-infarct eschar separates, leaving ulcers. The problem can be chronically active.

61 yo woman, protein S deficiency.
Long history DVT, PE, and leg ulcers. Perpetual stasis, inflammation, active infarction and ulceration. Old recanalizing thrombus shown.

Trauma pathergy, morbidity.

Trauma and injury can trigger microthrombosis, with unexpected wound infarcts, dehiscence, failed repair.

53 yo woman, rheumatoid arthritis.
Dogbite, forearm. Many complications of repeated surgery. Proteins C&S deficient. Confirmatory histology.

CLINICAL PATHOLOGY OF HYPERCOAGULABLE WOUNDS & ULCERS

Dynamical Behavior

refractory impaired wound behavior
characteristic of severe ischemia

recalcitrant and continuously pathological
persistent active necrosis and ulceration
can be self-perpetuating and amplifying

chaotic dynamics
net misbehavior over time
rapid evolution, but (very) slow resolution
variable state with each observation

Complications

necrosis, dehisce, ulcerate after biopsy
necrosis, dehisce, ulcerate after debride
necrosis, dehisce after trauma and surgery
necrosis, dehisce, failed repair or closure
graft loss, flap necrosis
potentially lethal severity and extent
intercurrent thrombotic events

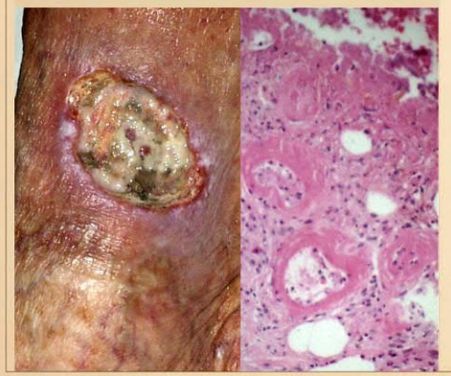
Treatments & Outcomes

chronic, persistent, recurring
consistent failures of general wound care
multiple failed procedures
patient and provider frustration
chaotic dynamics of therapy
warfarin hard to regulate

Surgical complications.

For surgery & controlled injury, risks are the same. Patients need perioperative anticoagulation.

69 yo woman. Wound dehiscence.
Complication of active ulceration after biopsy for minor skin lesion. Protein S deficient, and cryoglobulins. Histology shows thrombi, vessel and tissue necrosis.



Failed therapy.

Ischemia and necrosis impair healing and impede success, often repeatedly, for even mundane benign events.

72 yo woman, high anticardiolipins, ANA.
Ulceration and impaired healing of primary leg wound; then same for skin graft donor site, stasis & infarcts shown.

Unexpected profiles.

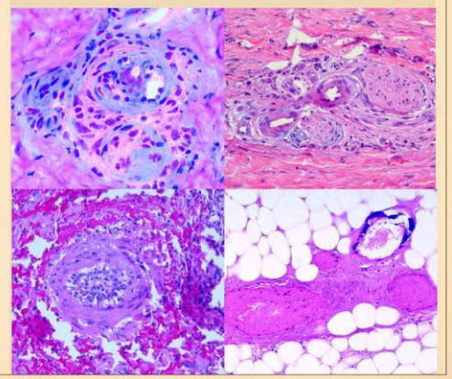
Think of hypercoagulopathies for young patients with peculiar ulcer histories and features.

39 yo man. Refractory leg ulcers.
Chronic since femur fracture & DVT at age 14. F.V Leiden (young men with venous ulcers have this mutant gene). Healed with 2 months of warfarin.

Histology.

Beyond general wound histology, the microscope reveals: thrombi in various stages, stasis, vessel and tissue necrosis, overlying ulceration, micro-angiopathy, 1° and chronic 2° vasculitis, vessel fibrosis and stenosis.

4 patients with various diagnoses.



DIAGNOSIS & APPROACH TO THE HYPERCOAGULABLE DISORDERS

1-A · Personal History

Any recurrent, unexpected, or inexplicable thromboembolism

arterial
deep venous
pulmonary
common types *(mi, cva)*
peculiar or rare events
(e.g. Budd-Chiari, Padgett-Schroeder)
events triggered by illness, injury
events in healthy young people
events in spite of treatment

peculiar profiles
(e.g. tardive paraplegia after non-cord spine injury, retinal artery occlusion in young person)
absence of common risks

Related diseases and events

miscarriages
venous disease
autoimmune, cvd-ctd
visceral autoimmune disease
angiopathies, blood disorders
cancer *(Trousseau)*, PNH
estrogens, warfarin resistance
absence of these or other risks

Hypercoagulable ulcers are NOT diagnoses of exclusion.

These diagnoses can be made on specific criteria.

1-B · Family History

Equally important as personal hx, diagnostic when personal history is weak or lab tests are negative.

miscarriages
thrombosis & embolism
autoimmune disease

1-C · Wound & Tissue History

Wounds and ulcers

continuous pathological behavior
absence of identifiable injury
long history failed rx
pain

Other events

trauma-induced pathergy
(tissue infarction, dehiscence, etc.)
complicated or failed operations
identified event *(e.g. warfarin, oral contr.)*
multiple such events
things that just don't add up
or defy the logic of common ailments

2-A · Physical Exam - Wound

Distinctive or consistent findings

sick / active wound
impaired / non-healing wound
thrombo-infarctive pattern
necrosis & infarcts *(as opposed to lysis)*
absence of inflammation *(or presence)*
progressive ulceration
persistent pathological behavior
pathergy/necrosis after debridement
signs of severe ischemia
vascular stasis, periwound cyanosis

Discrimination from other diagnoses

infarction vs. lysis
inflammation, or not
venous changes, or not
pulses / macrovessels normal
peculiar or non-specific locations
not in pressure / mechanical areas
not confined to tendons, synovium

DIAGNOSIS & APPROACH TO THE HYPERCOAGULABLE DISORDERS

2-B · Physical Exam - General

age *(any age, including young)*
vascular & skin exam
signs of previous ulcers or infarcts
rheumatoid & immunopathic signs

2-C · Exam & F/u – Response to Rx

Failures of general care

behaviors of severe ischemia
resistance to ordinary treatment
failed response to customary care
progressive infarction in spite of rx
failed therapy for other diagnoses
failed rx: steroids, anti-immune

Complications of specific care

pathergy / necrosis after debride
necrosis, dehiscence after surgery
failure, complications of surgery

Aberrant Response to Care

warfarin necrosis
warfarin resistance
difficulty regulating PT-INR

"things that just don't add up"

Hypercoagulable ulcers are NOT diagnoses of exclusion.

These diagnoses can be made on specific criteria.

3-A · Lab - Clinical

General studies

CBC, platelet, CMP, U/A

Thrombotic species

gene: factor-V.Leiden (R506Q)
gene: prothrombin mut. (20210G)
antithrombin III, protein C, protein S
factor-VIII, thrombin generation
fsp, d-dimer, TAT, plasminogen
fibrinogen (common pathway)
gene: MTHFR, homocysteine

Immune procoagulants

apl: anticardiolipin
apl: lupus anticoagulant
anti: beta-2-glypr, anca, mpo, pr-3

Autoimmune

Screen CVD-CTD, vasculitis:
sed rate, crp, ldh
ANA w/reflex, & specific abx
complement

Other micro-occlusive

SPEP / SIFE
PF4, Hgb, cryoglobulins, cryofibrinogen
new and future tests

3-B · Lab - Special

Vascular

TcPO2, laser doppler
imaging
periwound hypoxemia
(not useful: abi, pvr, ppg, doppler)

Histology

microthrombi, aggregates
platelet thrombi, fibrin thrombi
reorganization, recanalization
tissue infarction, vessel infarction
minimum inflammation
microangiopathies
vascular fibrosis, stenosis
vasculitis, acute (neutrophilic)
vasculitis, chronic (lymphoid)

3-C · Differential Dx & R/O

pyoderma, immune dermatoses
immunopathies, CVD-CTD
vasculitis, angiopathies
hematological, other micro-occlusive

DIAGNOSIS & APPROACH TO THE HYPERCOAGULABLE DISORDERS

Interpretation of Common Hypercoagulable Tests

fibrinogen	Typically high, common final pathway.
d-dimer	Often high, reflecting persistent microthrombosis.
protein C **protein S** **AT-3**	If low, these are hypercoagulable entities. If high, they are upregulation of endogenous anticoagulants . . . reflecting chronic active microthrombosis.
f.VIII	If high, microthrombosis is occurring.
lupus anti-coag **anti-cardiolipin**	Imply an associated autoimmune disorder. Expect high ana, and possibly rf, ccp, ds-dna, or others.
f.V Leiden **prothrom. 20210G**	Genes, thus system roots, immutable evidence of pathology. These confirm a breeder disorder for cvd-ctd.

78F Sjögren's

fibrinogen	565	++
protein C	60	–

67F Rheumatoid Arthritis

F.V Leiden	heterozyg	+
fibrinogen	640	++
plasminogen	135	+
protein C	136	+

57M Cirrhosis

bili	2.1	+
alk phos	160	+
RF	44	+
ANA	1:80	+
AT-III	47	–
protein C	35	–
protein S	55	–

Hypercoagulable disorders & ulcers are NOT diagnoses of exclusion.

They can be made on specific criteria.

Diagnosis is often made by just:
patient history
family history
physical exam

If history and physical seem certain,
positive blood tests are confirmatory.

If history and physical are equivocal,
positive blood tests are confirmatory.
(Remember, tests were ordered for suspicion.)

If history & physical are certain,
then even if labs are negative,
the diagnosis is made.

The Hypercoagulable Syndrome

Tetrad – Pentad

Thrombotic or embolic event
Autoimmune cvd-ctd
Wound pathergy
Miscarriage
Family history of same

Highly correlated with lab findings and response to rx, the basis for ordering confirmatory lab tests.

54M No prior diagnosis

F.V Leiden	heterozyg	+
ANA	1:80-spkl	+
lupus anticoag	pos	+
cardiolipin IgA	15	+
cardiolipin IgG	>150	+++
cardiolipin IgM	20	+
protein C	60	-
protein S	56	-
homocysteine	14.6	+

66F Scleroderma / MCTD

rheumatoid factor	35	+
ANA	1:1280-centro	++
protein S	62	–
fibrinogen	499	+

81F Leg ulcer

ANA	1:1280-homo	++
rheumatoid factor	27	+
lupus anticoag	pos	+
cardiolipin IgM	51	+
fibrinogen	429	+
homocysteine	19.3	+
protein C	142	+

76F Scleroderma

sed rate	56	+
C-reactive protein	7.4	+
ANA	1:1280	++
cardiolipin IgM	134	++
fibrinogen	477	+
protein S	58	-
plasminogen	>150	+

69F Rheumatoid

F.V Leiden	heterozyg	+
protein C	51	–
protein S	52	–

72F Polycythemia Vera

ANA	1:160	+
cardiolipin IgM	80	++
protein S	53	–

75M Anemia / Cythemia

rheumatoid factor	2780	++
cardiolipin IgM	70	+
protein C	65	-
cryoglobulin	pos	+

80F Leg ulcers, brain infarct

fibrinogen	386	+
protein C	12	- - -
protein S	43	-

TREATMENT & MANAGEMENT OF THE HYPERCOAGULABLE DISORDERS

4-A · Management - General

Major thrombotic events
urgent management as required
thrombolysis, target specific
thrombolysis, optional general

Associated risks and diseases
treat each accordingly
workup & treat immunopathies

After w/u and confirmed diagnosis
start anticoagulation
option heparins / inhibitors short term
warfarin, heparins, inhibitors long term
optional steroids for inflammation
regulate and monitor warfarin

of uncertain relevance:
anti-platelet drugs
rheologicals

Without a correct diagnosis or treatment, hypercoagulable ulcers are prolonged, persistent, frustrating, refractory, and resistant to care.

4-B · Management - Wounds & Tissues

Basic wound care and control
wound hygiene
debridement *(manage to avoid pathergy)*
topicals *(those for acute control)*
edema control

Problem specific management
for associated or derivative disorders:
other hematological
arterial, venous
immunopathic

Management for closure
basics *(topical care, natural contraction)*
repair, grafts, flaps as required
regenerative biomatrices
hyperbaric oxygen *(selective)*

Once a correct diagnosis is made and anti-coagulants are started, the wounds are usually easy to resolve, at times by anticoagulation alone, or with other necessary treatment.

4-C · Management - Long Term

General
manage underlying diagnoses
control associated risks & triggers

Wound support and prevention
compression and edema control
general skin care
topical steroids for dermatoses

Anticoagulation
until healed, plus 3-6 months
limited use for antiplatelet drugs
long term or lifetime anticoagulation,
(depending on diagnosis and risks)

PROPHYLAXIS FOR PROCEDURES

Principles of Anticoagulation
Restoration of Normal Profile

Different than ordinary anticoagulation.
You are not "thinning" normal blood.
You are restoring "sticky" blood to normal.
For warfarin, high INR required, 3.0 - 3.5 (or higher)

After adequate anticoagulation, necrosis stops, tissues start to revascularize, and wound healing resumes.

BEFORE WARFARIN

RIGHT LEFT

AFTER WARFARIN

29 M

Multiple leg ulcers, many years.
Suspicious history.

Otherwise healthy.
No other illness or explanation.

Family history multiple miscarriages.
Family history.

Lab: high anticardiolipins.
Confirmatory blood tests.

Healed, 14 weeks after warfarin start,
8 weeks after PT-INR stable 2.5 – 3.5.
Healed with anticoagulation only.

Dx: Antiphospholipid antibodies

43 F **Dx: Proteins C & S deficiency**

Refractory leg ulcers, many years.
Multiple DVT & PE. (Otherwise healthy.)
No venous reflux or hypertension.
Suspicious history. No other illness or explanation.

Lab: low Protein C & S, low skin TcpO2.
Confirmatory lab tests.

Healed after high-INR warfarin (*& regenerative biomatrix*).
Recurrence after lapsed warfarin.
Characteristic response to anticoagulation.

61 F **Dx: Protein S deficiency**

Refractory leg ulcer, many years.
Multiple DVT & PE. (Otherwise healthy.)
No venous reflux or hypertension.
Suspicious history. No other illness or explanation.

Lab: low Protein S.
Histology shows old and recanalizing thrombi.
Confirmatory lab tests.

Healed after high-INR warfarin (*& regenerative biomatrix*).
Recurrence after INR drop to 2.5 – 3.0.
Rehealed after INR restored to 3.5 – 4.0.
Characteristic response to anticoagulation.

67 F

Acute skin necrosis.
Suspicious history.

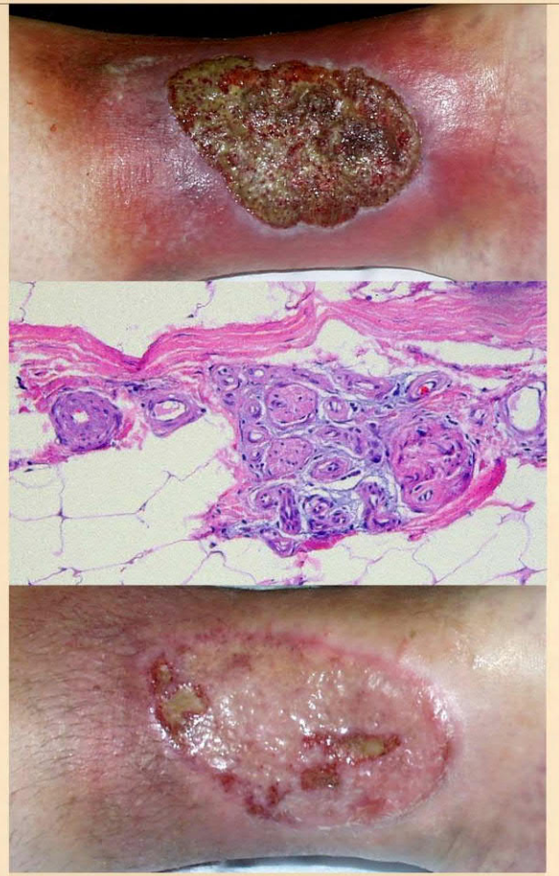
No prior risks or history.
Good pulses in feet.
No other illness or explanation.

Pure thrombo-infarctive pattern.
Confirmatory exam.

Lab: anti-thrombin-3 deficiency.
Confirmatory blood tests.

Healed: warfarin, hbo, regenerative biomatrix.
No recurrence on anticoagulation.

Dx: Anti-thrombin-3 deficiency



30 F **Dx: Mixed coagulopathy**

Refractory active ulcers. Severe ischemic pain.
History miscarriage.
Suspicious history.

Wound surface "granulation tissue" infarcts.
Confirmatory exam.

Lab: protein C deficiency, lupus anticoagulant.
Low skin TcpO2, with normal pulses.
Histology shows old and recanalizing thrombi.
Confirmatory tests.

Healed: warfarin (hard to regulate), hbo, matrix.
No recurrence on anticoagulation.

46 F

Refractory active ulcers.
Multiple miscarriages.
Suspicious history.

Mixed but mainly thrombo-infarctive pattern.
Good pulses in feet.
Confirmatory exam.

Lab: prothrombin 20210G mutation.
homocysteine very high.
p-anca & anti-mpo high.
Confirmatory blood tests.

Healed: warfarin, etc.
Healing on anticoagulation.

Dx: Primary hypercoagulopathy
Secondary immunopathy

42 M

Refractory leg ulcer, many years.
Multiple DVT.
Mother has same hx.
Suspicious history.
Wound edge infarcts.
Confirmatory exam.
Healed: warfarin, compression.
Healed with warfarin.

Lab:
protein C & AT-3 low.
anticardiolipins high.
homocysteine high.
f.V Leiden heterozygous.
Confirmatory blood tests.

Dx: Multi-factorial hypercoagulopathy

38 M **mixed hypercoag**

Refractory leg ulcer, many years.
DVT.
Suspicious history.
Lab: fibrinogen high.
protein C & AT-3 low.
f.V Leiden heterozygous.
Confirmatory blood tests.

33 M **venous disease**

Refractory leg ulcer, many years.
DVT after femur fx, age 9.
Suspicious history.
Lab: f.V Leiden heterozygous.
Confirmatory blood tests.

Young men with chronic Venous Disease.

Venous hypertension, panniculitis & dermatitis, ulceration.

Classical clinical syndromes yield to new understanding.
"Venous disease", when presenting with complications or refractory sequelae, has greater cause and implications than simple "post-phlebitis", both for origin of the illness, and effectiveness of care.

42 M **venous disease**

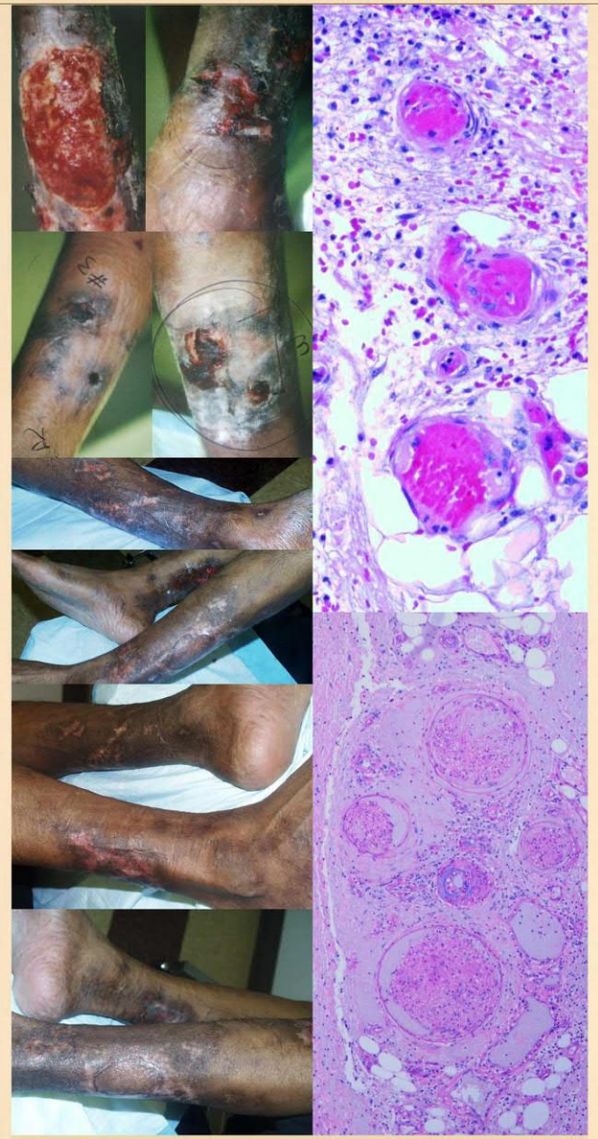
Chronic venous hypertension,
recurrent panniculitis & dermatitis.
Suspicious history.
Lab: f.V Leiden heterozygous.
Confirmatory blood tests.

(Top, active before rx; bottom, 1 year later.)

57 F **Factor V Leiden Hetero**

Acute leg & ankle skin infarcts & ulcers.

Healed by warfarin only (& basic topicals).



43 M **Very low Proteins C & S**

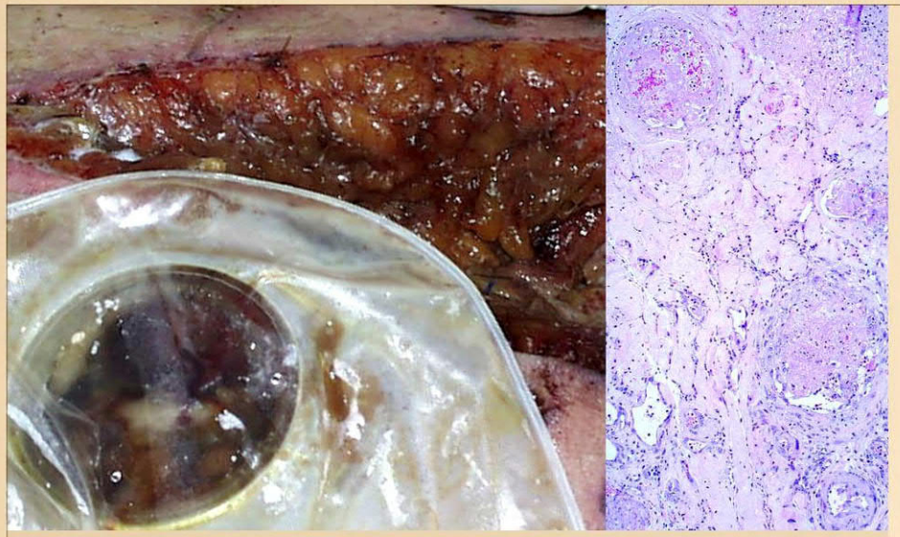
Acute spontaneous vascular stasis and skin infarcts.
Diffuse micro-thrombosis.
Healed by warfarin only (& basic topicals).

38 F **Lupus, APL syndrome**

Acute skin infarcts of leg.
Ischemia and ulcers, hands.
Protein S low, Anti-cardiolipins high
Healed by warfarin only (& basic topicals).

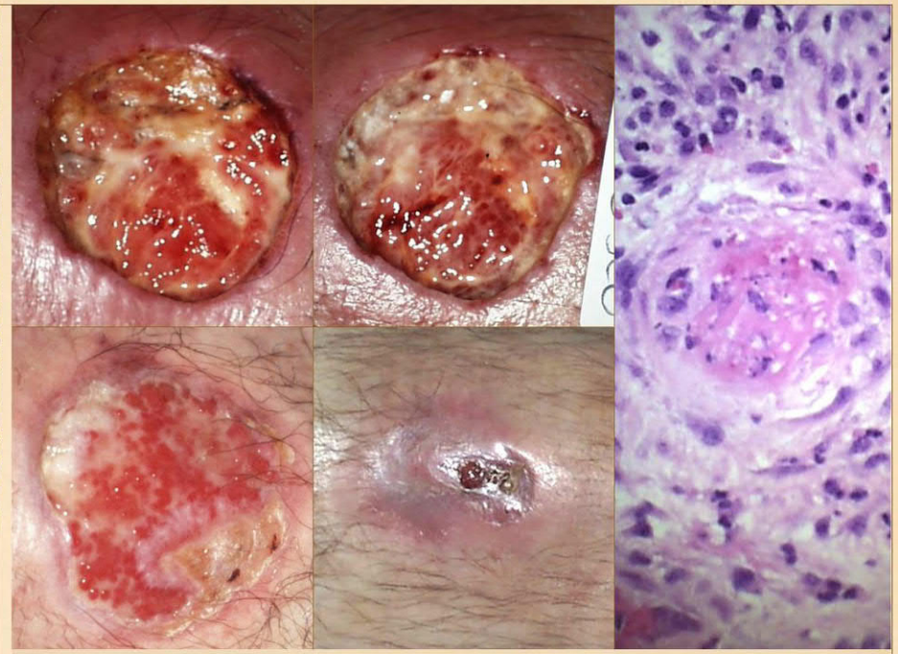
Note the potency of anticoagulants alone to restore normal wound healing, allowing eligible wounds to heal by natural contraction.

Hypercoagulable Disorders – Implications for Wound Pathergy, Acute Wounds, Chronic Ulcers, Trauma, and Surgery.



62 M **Dx: APC resistance, probable factor V Leiden**

Diverticular colo-vesical fistula.
Wound pathergy, then multiple infarcts and complications after surgery.
Bowel necrosis, abdominal wall necrosis.
Hx DVT - PE.
Hx finger necrosis after minor trauma.
Suspicious history.
Lab: APC resistance high.
Confirmatory tests.
Died before anticoagulation and diagnosis-specific Rx.



34 M **Dx: Lupus, Antiphospholipid antibody syndrome**

Wound pathergy, then multiple failed surgery after trivial hand trauma.
Multiple subsequent wounds from failed grafts and flaps.
Second set of wounds after elective hip replacement (for lupus arthritis).
Suspicious history.
Lab: anticardiolipins high.
Confirmatory tests.
Healed by warfarin only (& basic topicals).
Proper wound behavior only after warfarin.
Later: mva, abdominal trauma, abdominal wall infarct with wounds & colon fistula.
Later: home fall, minor non-skeletal back injury, tardive 2° cord infarct, paraplegia.

39 M **Dx: Factor V Leiden**

Tardive paraplegia from non-skeletal non-cord back injury

Minor fall without spine injury or neuro deficit, then paraplegia in coming days.
Recurrent infarctive wounds of feet from minor wheelchair trauma or pressure.
Lab: factor V Leiden, histology - diffuse thrombosis.

When trauma and surgery interact with the hypercoagulable disorders, the results can be extremely morbid, often fatal.

Note the importance of the "hypercoagulable therapeutic triad" – anticoagulants, hyperbaric oxygen, regenerative biomatrices.

44 F **Dx: APL syndrome**

Achilles rupture, multiple failed surgery.
Blind from retinal artery occlusion.
Suspicious history.

Otherwise healthy.
No other illness or explanation.

Lab: anticardiolipins high.
fibrinogen high.
periwound TcpO2 low.
Confirmatory tests.

Healed: warfarin, hbo, biomatrix.
Proper wound behavior only after warfarin.

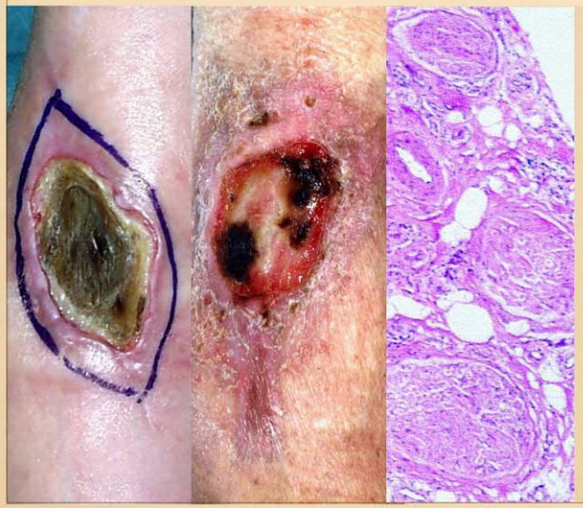
67 F **Dx: Factor V Leiden**

Back wound necrosis after spine surgery.
Family hx strong for DVT & leg ulcers.
Suspicious history.

Thrombo-infarctive, vascular stasis.
Confirmatory exam.

Lab: Factor V Leiden.
fibrinogen high.
plasminogen & protein C high.

Healed: warfarin, then surgery.

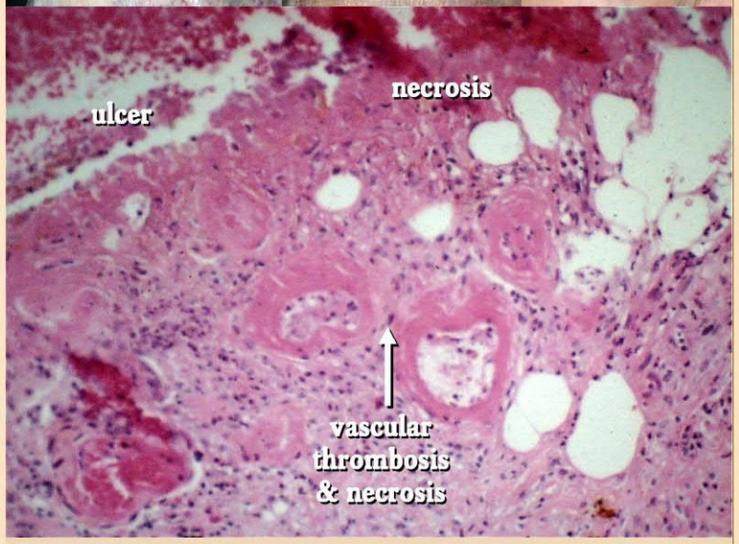


53 F **Dx: protein C & S deficiencies**

Wound infarct after dogbite injury, u.e.
Multiple failed surgery.
Rheumatoid arthritis.
Suspicious history.

Lab: protein C low.
protein S low.
histology - thrombus, organization.
Confirmatory tests.

Healed: warfarin, biomatrix.
Late re-ulceration after warfarin stop.
Proper wound behavior only after warfarin.



69 F **Dx: mixed coag**

Spontaneous skin ulcer.
Venous perforator thrombosis.
Wound pathergy after biopsy.
Suspicious history and exam.
No prior illness or explanation.

Lab: protein C low.
cryoglobulins present.
Confirmatory tests.

Healed: warfarin, biomatrix.
Proper behavior only after warfarin.

Each has its role to (1) arrest pathology, (2) restore physiological deficits, and (3) allow healing without risk of wound pathergy.