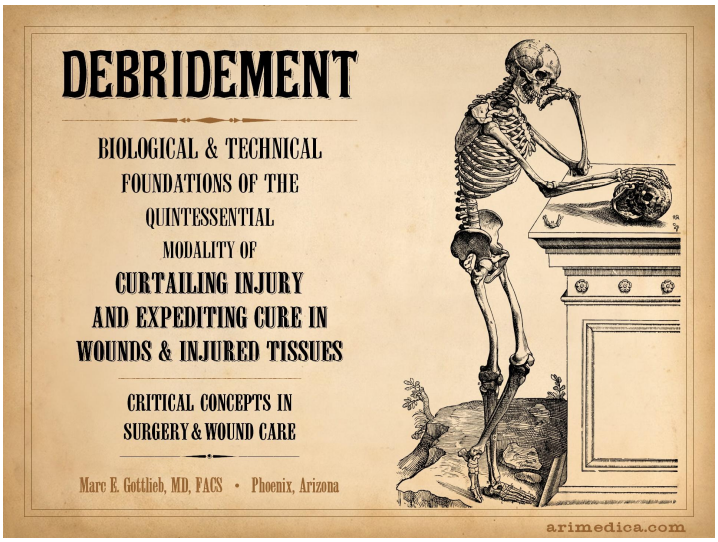
DEBRIDEMENT
Biological & Technical Foundations of the Quintessential Modality of Curtailing Injury and Expediting Cure in Wounds & Injured Tissues

Marc E. Gottlieb, MD, FACS

Phoenix, AZ

Revision 01-a, April 13, 2016, (original),



1

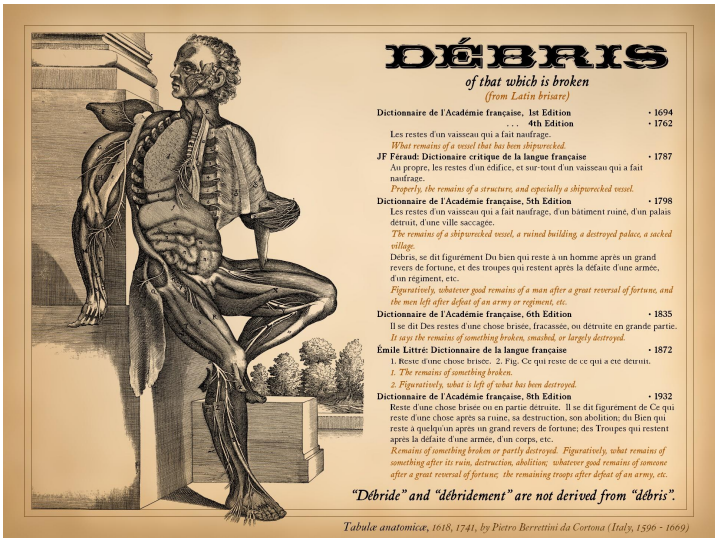
Debridement

Biological & Technical Foundations of the Quintessential Modality of Curtailing Injury and Expediting Cure in Wounds & Injured Tissues.
- Critical Concepts in Surgery & Wound Care. -

This presentation was given on April 13, 2016, in Atlanta Georgia, part of a debridement course sponsored by Integra Life Sciences. This specific presentation introduces the basic concepts of debridement, and it demonstrates the biological effects of removing injured, necrotic, and inflamed tissues from wounds.

This presentation and case studies by Marc E. Gottlieb, MD come from his practice of reconstructive plastic surgery in Phoenix and Scottsdale, Arizona. This presentation is available for viewing and download at Dr. Gottlieb's website *arimedica.com* which is used solely for the posting of presentations and other academic and instructional materials.

This illustration is taken from the works of Andreas Vesalius (1514-1564), native of Brussels, and eventually professor of anatomy at the prestigious medical school in Padua. His grand opus, De Humani Corporis Fabrica was first published in 1543. The book illustrates his anatomical dissections with many inspiring woodblock prints that are as exquisite in their artistic interpretation as they are in their scientific detail. The illustrations are exceedingly accurate, even by today's standards. This work established anatomy as a scientific discipline and ended a thousand years of stagnation in European medical knowledge. It is justly considered one of the great masterpieces of not just science and medicine, but also of the printer's craft (with all just accolades to printer Johannes Operinus) and even of human civilization itself. This image is especially important, because the skeleton is positioned such, with extremities flexed-extended and pronated-supinated, and with the accessory skull, hyoid, and ear ossicles displayed, that every single bone in the body can be seen in this one view. Nearly everyone who sees this image, contemplation of a skull upon a tomb, likens it to Hamlet's soliloquy upon Yorick, but Hamlet was not written until 1600. "Contemplation" is a worthy prelude to the study of debridement, pausing to understand the importance of this foundational concept, the central common procedural technique in the management of injuries, wounds, ulcers, and infarcts, that expedites resolution of injury and the onset and continuation of healing.



2

The etymology amd lexicology of the word *debridement* are not relevant to the act of debridement as it is being exercised upon a real patient or wound. However, the origin of the word is interesting, and it illuminates an historical perspective on the management of wounds and surgery, and how certain concepts that we now consider so centrally important are of relatively recent ascendancy. Nowadays, debridement is understood to mean a cleanup or elimination of morbid or non-viable tissues from a wound, that is, removal of debris from a wound. However, the terms *debris* and *debride* have altogether different origins and meanings. Confusion arises for English speakers in that the words, readily understood in their native French, are not just phonetically similar, but their inherent meanings seem to have a mutual implication.

Debris, or *débris*, refers to breakage, from *briser* (to break), *brisé* (broken), *bris* (breakage). Débris thus can be the prepositional object *de bris*, of that which is broken. Or, débris is an exaggerated form of briser, using the intensive prefix *de-*, thus Old French *debrisier*, or *débriser*, to break down or break emphatically to pieces. All are derived from Vulgar Latin *brisare* which referred to crushing grapes.

Usage in modern history is documented in standard dictionaries, beginning with the first edition of the *Dictionnaire de l'Académie française* (l'Académie being the official steward of the French language). The following and subsequent lists of dictionary excerpts were obtained from ARTFL (*The Project for American and French Research on the Treasury of the French Language. "Founded in 1982 as a result of a collaboration*

between the French government and the University of Chicago, the ARTFL Project is a consortium-based service that provides its members with access to North America's largest collection of digitized French resources.") at https://artfl-project.uchicago.edu/ .

DEBRIS
https://artfl-project.uchicago.edu/content/dictionnaires-dautrefois
http://artflsrv02.uchicago.edu/cgi-bin/dicos/pubdico1look.pl?strippedhw=d%C3%A9bris

Dictionnaire de l'Académie française, 1st Edition • 1694
. . . 4th Edition • 1762
Les restes d'un vaisseau qui a fait naufrage.
What remains of a vessel that has been shipwrecked.

JF Féraud: Dictionaire critique de la langue française • 1787
Au propre, les restes d'un édifice, et sur-tout d'un vaisseau qui a fait naufrage.
Properly, the remains of a structure, and especially a shipwrecked vessel.

Dictionnaire de l'Académie française, 5th Edition • 1798
Les restes d'un vaisseau qui a fait naufrage, d'un bâtiment ruiné, d'un palais détruit, d'une ville saccagée.
The remains of a shipwrecked vessel, a ruined building, a destroyed palace, a sacked village.
Débris, se dit figurément Du bien qui reste à un homme après un grand revers de fortune, et des troupes qui restent après la défaite d'une armée, d'un régiment, etc.
Figuratively, whatever good remains of a man after a great reversal of fortune, and the men left after defeat of an army or regiment, etc.

Dictionnaire de l'Académie française, 6th Edition • 1835
Il se dit Des restes d'une chose brisée, fracassée, ou détruite en grande partie.
It says the remains of something broken, smashed, or largely destroyed.

Émile Littré: Dictionnaire de la langue française • 1872
1. Reste d'une chose brisée. 2. Fig. Ce qui reste de ce qui a été détruit.
1. The remains of something broken.
2. Figuratively, what is left of what has been destroyed.

Dictionnaire de l'Académie française, 8th Edition • 1932
Reste d'une chose brisée ou en partie détruite. Il se dit figurément de Ce qui reste d'une chose après sa ruine, sa destruction, son abolition; du Bien qui reste à quelqu'un après un grand revers de fortune; des Troupes qui restent après la défaite d'une armée, d'un corps, etc.
Remains of something broken or partly destroyed. Figuratively, what remains of something after its ruin, destruction, abolition; whatever good remains of someone after a great reversal of fortune; the remaining troops after defeat of an army, etc.

The page at the above web link has more entries. The text on this slide is just an abstract of the whole page, but the linguistic evolution of the word *débris* can be appreciated. In 1694, the word referred to the remains of a shipwreck. A century later, the word referred to the remains of almost anything that had been ruined, including in a more figurative sense the remains of worth and reputation after great misfortune. (One can imagine that the French Revolution, 1789-1799, gave society ample opportunities to apply the word more broadly.) For the two centuries hence, the meaning of debris has remained static. It is easy to see however, that taken in its more literal sense, it could properly refer to that which is broken or the remains of tissue after injury and wounding. Nonetheless, “débride” and “débridement” are not derived from “débris”.

The illustration is by Pietro Berrettini (1596-1669). Known popularly by his city of birth, Pietro da Cortona was an artist and architect of the highest preeminence, epitomizing the High Baroque in Italy, and perhaps second only to his contemporary Gian Lorenzo Bernini in influence and cultural significance of that era. Along with his geometrically detailed baroque architecture projects and his extravagantly dramatic paintings and frescoes, he created one of the more interesting and beautiful bodies of work from that era, the Tabulæ Anatomicæ. The work is a mystery – no one knows why he did them, who commissioned them, under what circumstances he had the wherewithal to make them, nor even when exactly they were made, the best estimates being 1618, before he earned his fame and came to the attention of patrons and commissioners. They were published posthumously in 1741 , 72 years after his death, over a century after they were drawn. The circumstances of their rediscovery and impetus to publish them are not fully understood. In any work of this sort, the artist needs crucial collaborators, in this case the woodblock engraver being most likely Luca Ciamberlano, and his anatomist being probably surgeon Nicolas Larchée. Consistent with da Cortona’s style as seen in his paintings, all of the figures in the Tabulæ have drama, grandeur, and great artistry. Many of the skeletonized figures in the Tabulæ express grimness and pathos. In contrast, the engraving shown here, Pietro da Cortona’s Tabula VI, a view of the viscera in situ, has a certain sense of repose and contemplation. That repose stands in irony to his physical state of debris, his remains after some great assault by anatomical dissection, or perhaps he is glad that someone had the good sense to debride the damaged tissues no longer in view.

3

The terms *debride* and *debridement* come from the words *bride* and *débrider*. "Bride" is equivalent to Old Germanic *bridel* and English *bridle*. The bridle, *la bride*, is a horse's head harness, consisting of the head-stall, the bit, and the reins. Unlike *débris* in which *dé-* is used as an intensive prefix (smash with emphasis), in *débrider*, *de-* is used in its ordinary sense of reverse, remove, lessen, etc. Literally, débrider means to de-bride the horse, to remove the bridle. While it is easy to appreciate how *debris* could evolve from broken grapes to a shipwreck to the remains of anything broken, the evolution of *debride* to its current meaning is more abstract and figurative. Once again, it is easy to track this evolution by looking in dictionaries, as done below for the words *bride*, *débrider*, and *débridement*.

The illustration titled "The Old Trapper", 1888, is by renowned Western artist and illustrator Frederic Remington (1861-1909). It is included to depict the horse's bridle. This was the "golden age" of the artist-illustrator in the latter 19th century. It coincided with the romantic conquest of the Wild West in the United States, and Frederic Remington was the preeminent visual interpreter of both the subject and the style. This is also the time when the word "debridement" was coming into accepted use as a medical and surgical term. It is notable then that Mr. Remington died at the young age of 48 after a surgical debridement of chronic appendicitis.

BRIDE *French* • BRIDLE *English*
https://artfl-project.uchicago.edu/content/dictionnaires-dautrefois
http://artflsrv02.uchicago.edu/cgi-bin/dicos/pubdico1look.pl?strippedhw=bride

Dictionnaire de l'Académie française, 1st Edition • 1694
Frein, ce qui sert à gouverner un cheval. Le mors, les resnes & la testiere de la bride.
Brake, that which governs a horse - the bit, the reins, and the head-stall of the bridle.

Dictionnaire de l'Académie française, 4th Edition • 1762
La partie du harnois d'un cheval, qui sert à le conduire, & qui est composée de la têtière, des rênes & du mors.
The part of a horse harness which serves to lead, composed of head-stall, reins, & bit.
On dit figurément, Tenir quelqu'un en bride, pour dire, L'empêcher de faire ce qu'il veut.
Figuratively, hold someone to the bridle, meaning, prevent him from doing as he wishes.

Dictionnaire de l'Académie française, 8th Edition • 1932
Partie du harnais d'un cheval ou d'un mulet, etc., qui sert à le conduire, et qui est composée de la têtière, du mors, des rênes.
The part of a horse or mule harness which serves to lead, composed of head-stall, bit, & reins.
Fig. et fam., Tenir quelqu'un en bride, Le contenir, surveiller sa conduite.
Figuratively, hold someone to the bridle, contain him, surveil his conduct.

DÉBRIDE, DÉBRIDEMENT *French* • UNBRIDLE, UNBRIDLING *English*
https://artfl-project.uchicago.edu/content/dictionnaires-dautrefois
http://artflsrv02.uchicago.edu/cgi-bin/dicos/pubdico1look.pl?strippedhw=d%C3%A9brider
http://artflsrv02.uchicago.edu/cgi-bin/dicos/pubdico1look.pl?strippedhw=d%C3%A9bridement

Dictionnaire de l'Académie française, 4th Edition • 1762
• Ôter la bride à un cheval. *Remove the bridle from a horse.* [literal]
• Figur. dans le style familier, en parlant de certaines choses qu'on fait avec une extrême précipitation. *Figuratively, in familiar style, things done very precipitously or with urgency.* [fig: unleash, let go the brakes]

Jean-François Féraud: Dictionaire critique de la langue française • 1787
Ibid. [literal]; [fig: unleash]
• Sans débrider, adv. (Figuré.) Tout de suite et sans interruption.
Through to the end without interruption. [fig: without stopping]
• Débridement, pour exprimer l'action de débrider. Ce Lexicographe a beaucoup de ces substantifs, forgés d'après des verbes.
Debridement, to express action of debriding. This lexicographer has many nouns forged from verbs.

Dictionnaire de l'Académie française, 5th Edition • 1798
Ibid. [literal]; [fig: unleash]
• Il s'y joint alors une idée de repos, de cessation de mouvement.
From the literal joins an idea of repose, cessation of movement. [fig: cease, rest, halt]

Dictionnaire de l'Académie française, 6th Edition • 1835
Ibid. [literal]; [fig: unleash]; [fig: rest, halt]; [fig: without a stop]

Émile Littré: Dictionnaire de la langue française • 1872

Ibid. [literal]; [fig: unleash]; [fig: rest, halt]; [fig: without a stop]

• Terme de carrier. Détacher le câble de la pierre.

Quarry term, detach the cable from the stone. [by analogy]

• Terme de chirurgie. Pratiquer l'opération du débridement.

Surgery term, practice the operation of debridement. [by analogy]

• Débridement. Terme de chirurgie. Opération consistant à enlever les brides ou filaments dont la présence dans une plaie met obstacle à la libre sortie du pus.

Term of surgery, operation consisting of removing the adhesions or filaments whose presence in a wound obstructs the free exit of pus.

• [1877] Opération consistant à diviser un tissu membraneux ou aponévrotique qui comprime les plaies, ou à agrandir une solution de continuité pour donner issue à la suppuration.

Operation of dividing a tissue membrane or fascia that compresses wounds, or enlarge a break to give exit to pus.

Dictionnaire de l'Académie française, 8th Edition • 1932

Ibid. [literal]; [fig: unleash]; [fig: rest, halt]; [fig: without a stop]

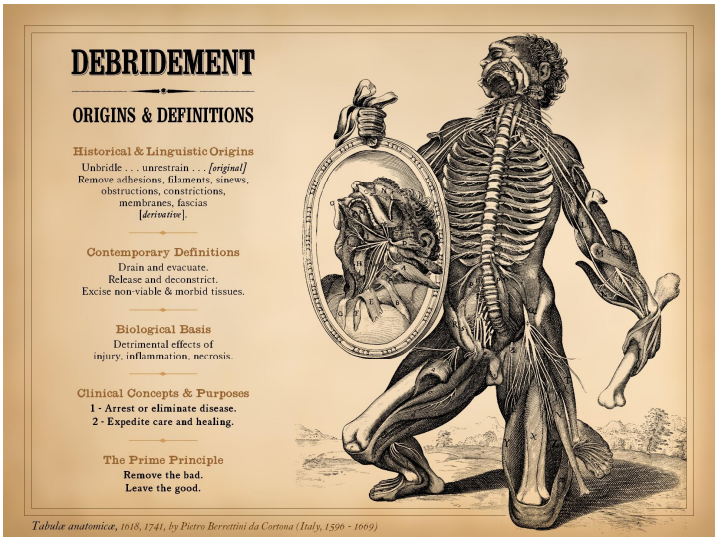
• Par analogie, il signifie Dégager ce qui est serré.

By analogy, it means free what is tight or constricting. [by analogy]

• Débridement. Terme d'Arts et de Chirurgie. Action de débrider. Le débridement d'une pierre. Le débridement d'une plaie.

Action of debriding, i.e. debridling, e.g. a stone or a wound.

As for *débris*, the pages at the above links have much more detail, and the text on this slide is an abstraction. In 1694, the word *bride* referred solely to a horse's bridle. A century later, and through until today, the word also took on a figurative sense of holding someone to the bridle, to prevent someone from doing as he wishes. The opposite was *débrider*, to debride, literally to remove the *bride*, the bridle, from the horse. By the 18th century, the word was beginning to take on figurative meanings and derivative forms. Extended meanings forked three ways: (1) the figurative sense of stopping, since removing the bridle was the symbolic end of a journey; (2) the figurative sense of unleashing or unbraking, allowing something to run "full throttle", since removing the bridle took away the rider's ability to restrain the horse; and (3) the analogous sense of removing the cables or harness on objects other than a horse, or (analogous and figurative) to remove whatever is tight or constricting. For example, *sans débrider*, "without debridling", meant to do something to the end without interruption, without stopping (e.g. "*faire dix lieuës sans débrider, make ten leagues without debridling, without stopping*", *and* "*elle a dormi huit heures sans débrider, she slept eight hours without interruption*"). The sense of precipitous or unrestrained hurry is in sentences such as "*voyez comme il débride, comme il mange*, *see how he rushes how he eats*". More sample sentences are at the links above. The analogous meanings of the word took the form of the original verb *débrider* but also the derivative noun *débridement*. Thus, in the quarryman's trade, *débrider* meant to detach cables from a stone. In surgery, *débrider* was to practice the operation of *débridement*. In dictionaries of the 1870's, the surgical operation of debridement was defined as *"term of surgery, operation consisting of removing the adhesions [les brides] or filaments whose presence in a wound obstructs the free exit of pus"*, and as *"operation of dividing a tissue membrane or fascia that compresses wounds, or enlarging a break to give exit to pus"*. The first use of these words related to surgery was circa 1842. Even in modern parlance, the word in French maintains its derivative meanings in ways that are intuitively correct to a French speaker, such as *débrider les yeux*. This is the term for *la blépharoplastie asiatique*, the oriental eyelid correction that is done as a cosmetic procedure to remove epicanthal folds and make eyes look less Asiatic. In that sense, it has the connotation of unclamping or deconstricting the eyes. To an English speaker though, the idea of "debriding the eyes" does not make sense (unless in a horror movie), since the way *debridement* has entered the English lexicon it refers to the concept of removing wound debris. Note how débris, derived from briser (to break), and débride, derived from bride (bridle) differ by the letter *s* versus d. Despite the visual, spelling, and phonological rough similarity of these words, they are different words with greatly different root meanings and figurative extensions.



4

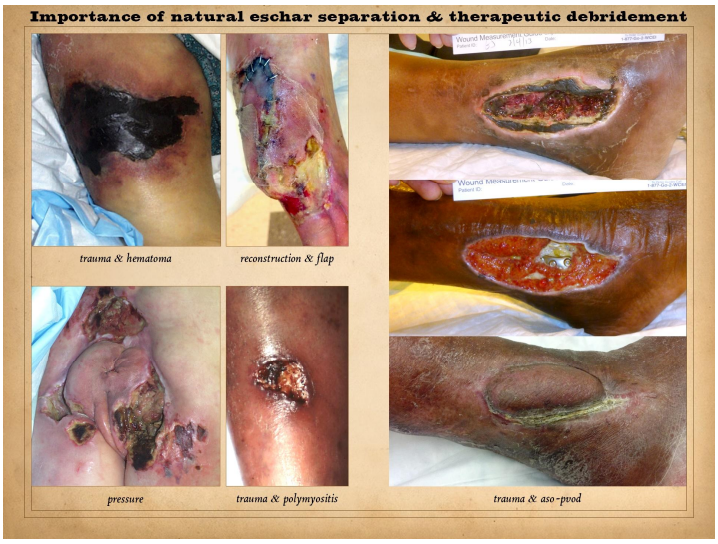
Debridement must be understood from several points of view. The historical and linguistic origins of the word were described on the preceding slides. Derived from its literal and figurative senses of unbridling and unrestraining something, its original surgical use was to remove or open tissue membranes to allow pus to exit freely. It was used then as we would now prefer the terms "drain" or "drainage". By extension, it took on the broader meaning of removing adhesions, filaments, sinews, obstructions, constrictions, membranes, and fascias, not just to allow pus to drain, but to remove those tissues when they themselves are injured, morbid, or devitalized and non-viable.

There are profound biological and therapeutic implications to debridement and the removal or morbid tissues. After injury, and especially in the presence of infarcted, infected, and necrobiotic tissue, there is intense inflammation and progressive tissue injury and lysis. Eschar and devitalized tissue will eventually separate due to natural processes, but the sooner that they and the resulting inflammation can be eliminated, then the sooner the wound can move on to its efferent phase of proliferative repair with less injury to residual viable tissues.

Debridement does two vital things for the wound and the patient. First, it arrests or eliminates disease, the focus of injury, inflammation, and

progressive damage or delayed healing. Second, by so doing, it expedites the care and healing of the wound and patient. Technically, debridement is simple. It has can have its caveats and special considerations depending on patient, pathology, and part of the body, but its quintessence is utter simplicity: remove the bad, leave the good. The following slides will explain the foundations for the biological effects and clinical necessity of debridement.

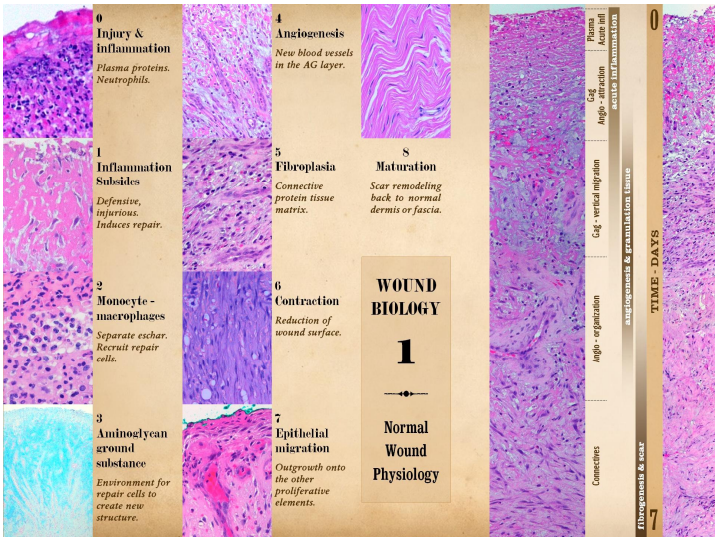
This illustration is also from Pietro da Cortona, Tabula XII, showing spinal nerves and some of the peripheral and cranial nerves. There is a certain pathos to this image, serving to remind that delays in debridement and control of disease risk progressive injury and loss of tissue and the need for more extensive eventual debridement. One would hope that the patient does not pay the wages of delay and thus end up looking like the poor model who posed for this drawing, and that unlike the many nerves shown, that a practitioner will not be nervous about doing a proper debridement.



5

This series of cases demonstrates the basic concept of natural eschar separation and therapeutic debridement. The left panel shows four cases. (Top left, thigh after vehicular trauma and shearing-avulsion injury with hematoma and overlying skin necrosis; top center, partial necrosis, tip of a foot flap after wound closure for sarcoma radiation and resection; bottom left, perineal and pelvic pressure ulceration and necrosis in a paraplegic patient; bottom center, leg ulcer and necrosis in a patient with polymyositis-dermatomyositis after a fall and impact injury to the leg.) These are very different patients and problems, but all have one thing in common. Disease or injury has resulted in zones of necrosis, ulceration, and inflammation. The body has a mechanism for responding to injury and injured tissues – the integrated process of inflammation and wound repair. Inflammation is the afferent arc that senses and responds to the injury, to protect and minimize risk to the host, then to clean up the detritus and debris in preparation for healing. Inflammation is obligatory after any injury, but lytic necrobiosis and tissues which have become infected provoke a greater inflammatory response. Inflammation, being inherently destructive, risks damage to the host if unregulated or over-stimulated. As part of inflammation's protective activities, it actively causes eschar separation, the natural separation of non-viable necrotic material from remaining living tissue. Once that happens, inflammation tends to subside because that which has provoked the inflammation is now gone. Until the eschar and inflammation are gone, the wound cannot fully enter the efferent proliferative healing phase. None of the 4 cases shown on the left have signs of a proliferative wound module or robust healing. That is pending once the eschar separates, but if the eschar can be removed more quickly than nature herself provides, then the efferent phase of healing begins more quickly as well.

The right panel shows the progression of an ankle wound after injury and surgery. The patient, who has diabetes and unreconstructable distal athero-occlusive disease, had trauma resulting in distal tibia-fibula fracture, treated by plate-and-screw fixation. The lateral fibular incision became necrotic. Top is the initial view at consultation. Necrosis of the skin edges has caused lack of adhesion then wide dehiscence of the incision. There are subtle signs of eschar beginning to separate, but arteriopathy and ischemia make even this process run slowly. Inflammation is under reasonable control due to active topical wound care, but allowing this process to run at its own impaired natural rate puts the fracture and ankle at risk. Middle, the wound has been surgically debrided, following which reparative processes proliferated quickly. The exposed plate and fracture dictate that surgery be done to cover them, and the improved wound status following debridement now permits that. Bottom, closure was done with a sural flap, with a long term healed wound and normal ambulatory function.



6

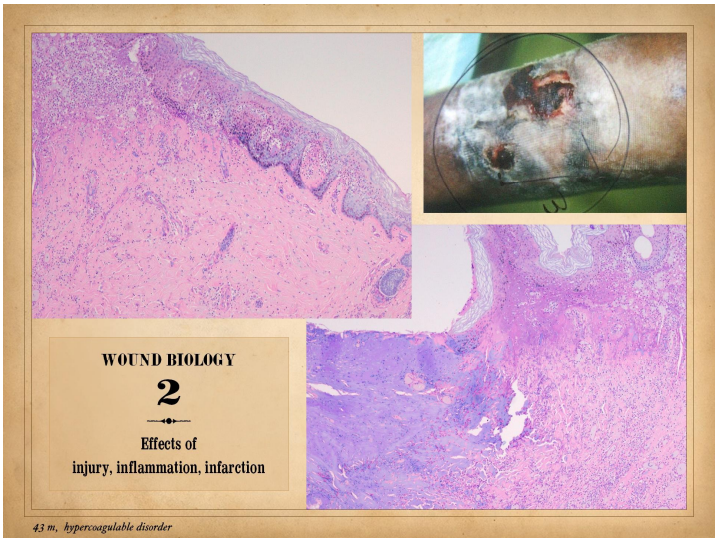
Normal Wound Healing

To understand the importance of eschar separation and debridement from a biological point of view, it first necessary to understand the general process of response to injury, inflammation, and wound healing. The anatomy of normal wound healing is summarized in the concept of the "wound module", the sequence of chemicals, cells, and events which occur and self-organize to repair the stroma after injury. Wound repair develops in time. In an open wound, new inflammation accumulates on the surface while repair events are occurring in older strata below. The deeper down you look from the surface, the older in time you are looking. When looking at wound histology, each specimen shows its own history. At the surface are events occurring now. As you go deeper, you are seeing, in sequence, events that happened yesterday, the day before, the day before that, and weeks before. The wound you see under the microscope did not happen all at once.

Right, the long vertical images show the full depth of a wound from the inflammatory layer at the surface to the organizing fibrous layer at the bottom. Scales are given to show the relative position of anatomical strata (plasma & acute inflammation, gag's & angio-attraction, gag's & vertical migration, angio-organization, connectives) and of temporal events (acute inflammation, angiogenesis granulation tissue, fibrogenesis & scar). The

small panes show individual events within these zones. Injury and inflammation must be controlled for repair to begin. After the wound is closed, i.e. fully re-epithelialized, the nominal clinical endpoint of complete repair, then the wound matures. In between injury-inflammation and maturation, there are 7 notable and clinically observable events: 1 - inflammation subsides; 2 - macrophages appear, separating eschar, and orchestrating local cells by cytokines; 3 - aminoglycan ground substance appears; 4 - angiogenesis occurs, visible as "granulation tissue"; 5 - histioblasts appear, leading to fibroblasts, which make connective proteins to hold the wound together; 6 - myofibroblasts are another histioblast derivative, which serve to contract the wound, responsible for much of the wound closure; 7 - epithelial growth continues until there is a complete epithelial (ectodermal or entodermal) interface between the environment and the mesenchyme.

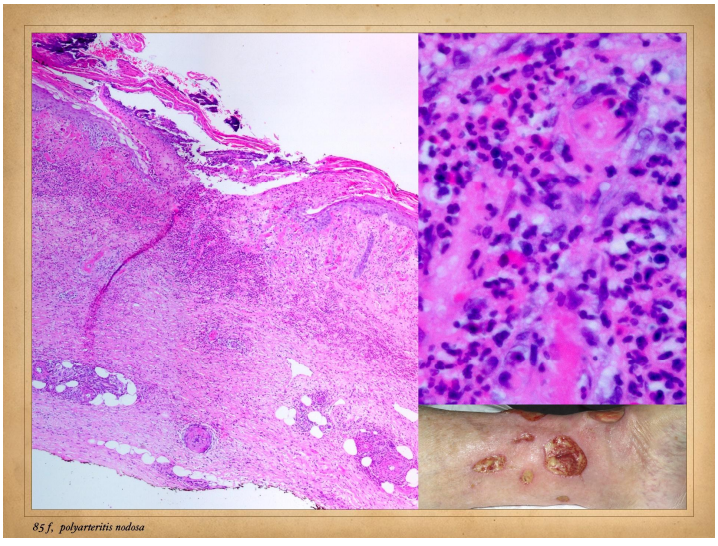
It is not the intent of this presentation to discuss the entire physiology of wound healing. Detailed discussion of this slide and other materials about the biology of wound healing can be read in other presentations at arimedica.com. The next few slides will focus on just the early phases of this process, injury and inflammation through eschar separation.



7

In the slide above, the time sequence of inflammation and repair starts with Event-0, the injury and resulting inflammation, and then Event-1, subsidence of acute inflammation. It is during the early inflammatory events that progressive injury and tissue damage occur. Regardless the cause of the injury, inflammation is destructive by nature, meant to defend and destroy exogenous agents, then dissolve host tissues so that the latter cleanup and repair processes can commence. This slide shows how acute inflammation and injury affect the tissues. Remember that "injury" is the inciting event. It can be any agent of damage, including mechanical injury, physical agents, immune and allergic events, thrombotic-occlusive-ischemic infarctions, etc.

Illustrated is a 43 year old man with leg ulcers due to a hypercoagulable disorder, deficiencies of protein C and protein S. Note the dry black eschar, and the otherwise indolent look of the tissues, free of edema and erythema, features typical of hypercoagulable ulcers where there is ischemic thrombo-infarction rather than an inflammatory-lytic process. The wounds all healed after treatment with anticoagulants and usual debridements and topical wound care. Two histological views are shown. Right lower is a broader view of a biopsy at the edge of an infarct and ulcer. Epithelium is missing from the ulcerated area to the left. The subjacent dermis is basophilic and amorphous or waxy looking, the histologic appearance of necrosis. The infarct is not invaded by inflammatory cells, nor is the pale pink immediate peri-necrotic zone heavily infiltrated, consistent with the hypercoagulable micro-occlusive origin of the problem. More peripherally, in the right upper quadrant of the image, the darker more purplish pink is infiltrated with acute inflammatory cells. Left upper image is a closer view of the peri-infarct inflammatory zone, seen in the left upper quadrant of the image. Ulcer and infarct are off-image to the left, and far from the ulcer, image right, the dermis and epidermis both look relatively normal and uninjured. The inflammatory zone between is recognized by cellular infiltration (leukocytes) and relative basophilia. Inflammatory infiltration is dissolving the tissue, recognized by the loss of pink staining collagen leaving a "see through" show of the background illumination. Epidermis to the right is intact, but in the peri-infarct zone, there is dissolution and loss of the basal layer and infiltration of the epidermis with inflammatory cells. This is the process of active ulceration. In the earlier phases of the whole process, inflammation is enlarging the ulcer area. In latter phases, this inflammatory dissolution eventuates in separation of the infarcted central zone, the eschar, and when that is gone, inflammation can begin to wind down. If the infarct and eschar are preemptively removed, debrided, then secondary inflammatory injury subsides earlier, and repair processes can likewise arise earlier.

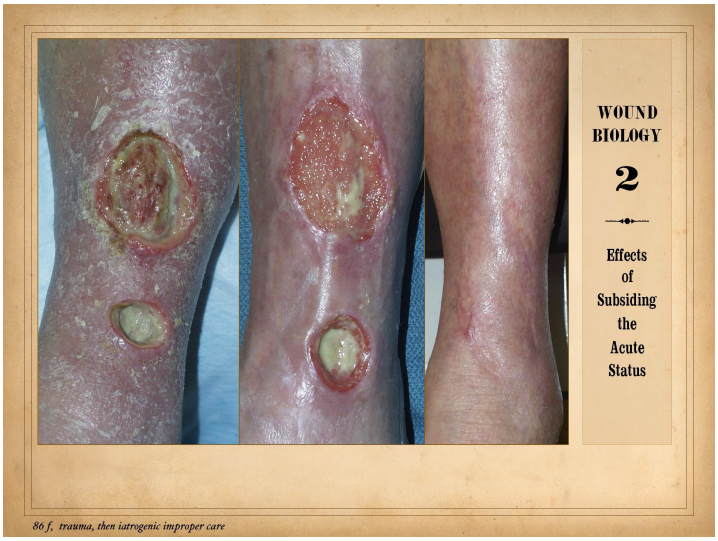


8

The example in the last slide was due to hypercoagulability, a set of disorders which cause microvascular infarction intrinsically free of inflammation. Inflammation that does occur is secondary, part of the proper reaction of the body to the injury. In contrast, the patient depicted here is an 85 year old woman with polyarteritis nodosa, an autoimmune and primary inflammatory disorder. Right lower, the depicted ulcers are already settled from some basic care, but they still have residual periwound erythema, edema, and some eczema. There are no dry infarcts, but there are residual unseparated elements of dermis and underlying fascias that were damaged by the inflammation.

Left, consistent with the differences in primary diagnoses, the broad histologic view shows a different mechanism of injury than the preceding example. Rather than a zone of indolent non-inflammatory infarction underlying the ulcer, the center zone of this specimen has active ulceration from intense inflammatory infiltration in the dermis. It is this primary inflammation which is lysing the skin and creating the ulcer, recognized by the destruction and separation of the overlying epidermis. Right upper, a closeup of the dense neutrophilic infiltrate in the tissue, along with amorphous pink eosinophilia consistent with destruction of the connective protein matrix, along with "poly dust" nuclear debris and other basophilia consistent with cellular lysis. In this case, ulceration is occurring due to a primary inflammatory disorder. Simple

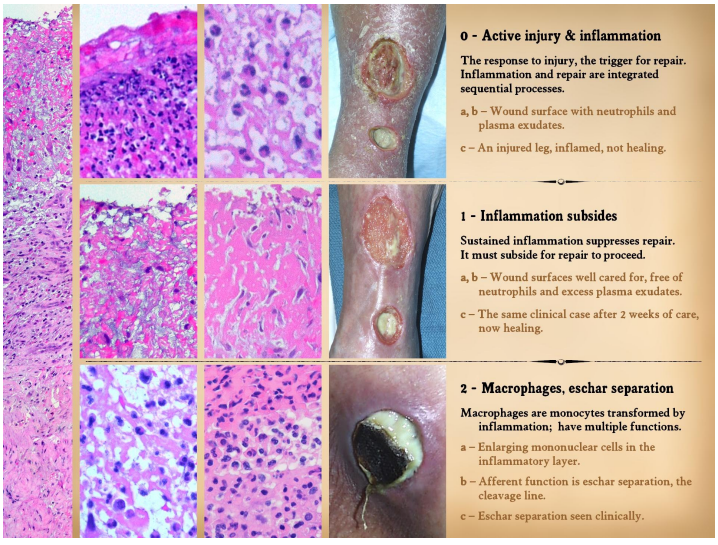
debridement or excision will not suppress or eliminate the primary disorder. Control of disease will depend on steroids or other pharmacological control. Nonetheless, regardless the cause of the ulcer, the process will incite even more inflammation as a proper response to injury. Debridement is required. Combined with pharmacological control of the primary disorder, physical elimination of the inflammatory cells and the damage they have created will expedite subsidence of the problem and then onset of wound repair.



9

This slide is a simple demonstration of the importance of subsiding inflammation if wound healing is to occur. This woman tripped and lacerated the front of her leg. It would have healed with simple basic care (topical wound hygiene and edema control). Instead, the patient was given improper care with injurious topical chemicals that irritated the skin provoking the diffuse contact and eczematous dermatitis shown. Progressive (and thoroughly unnecessary) ulceration eroded through subcutaneous fascias through the muscular fascias into the anterior compartment. Gravity dependent accumulation of inflammatory fluids settled at the retinacular ligaments causing the secondary distal ulcer. Every step of the way, the treating physician replaced ignorant care with more inept and injurious care, and the problem got worse. The patient has no prior medical problems, no risk factors for chronic ulceration. The patient was literally days away from scheduled leg amputation (her physicians working on the false principle that if you cannot fix it, throw it away). She wisely sought another opinion. The leg on presentation is shown left. Care consisted of superficial debridement (simple curettage), topicals (silver sulfadiazine for the wounds, desoximetasone for the skin), an anti-inflammatory contact layer (xeroform gauze, bismuth tribromophenate), and elastic compression wraps. The interval to the next picture middle is 2 weeks. Since the patient has no diseases or risk factors for wound healing problems, it is not surprising that alleviation of injury allowed wound healing to progress at a physiologically normal rate. Gross signs of inflammation are entirely gone, edema, and eczema are resolved, and the wound surfaces are properly proliferative and healing. The only caveat is that with the open tendons at the retinacular level, surgery is required to close that area. A small local flap was done to cover that area, and skin grafts were placed on the flap donor site and whatever else was not yet healed. The interval to the last picture right is just 8 weeks from the original image.

This patient got worse as inflammation was created by errant injurious care. The leg improved by simply eliminating injury and suppressing inflammation. This case demonstrates the quintessential central importance of controlling inflammation. In managing any wound, the initial phase of care is always about subsiding injury and controlling inflammation, thus inhibiting secondary damage and allowing efferent reparative processes to appear. Controlling primary disease is a variable process dictated by the nature of the primary disorder. Controlling inflammation (and related issues of edema and thrombosis) may require the help of steroids or other pharmaceuticals, but the main modalities of general control are topical and non-specific – good wound hygiene by debridement, bathing, and topicals, and edema control by elevation and compression wrapping. The more expeditiously that damaged tissues are removed – debrided – the more quickly inflammation subsides and wound repair begins.



10

The above case is repeated here where control of inflammation and afferent wound processes re correlated with wound anatomy. The basis of this slide are the sequential images of wound histology from Slide 6. Detailed discussion of each image and each phase of wound healing can be found on the arimedica.com website. This panel will focus only on events from injury to eschar separation. On the left edge is a vertical wound image. It seems to be split into almost exact thirds, each area with its own distinctive architecture. The “exact thirds” split is just an artifact of how the photograph was cropped, but it does clearly illustrate the progressive development of wound anatomy as the physiological events evolve. The upper third has zones 1, 2, & 3 – inflammation, angio-attraction, and angio-organization – zones made of plasma then aminoglycans without connective matrix, The middle third is the area of young fibroplasia, zones 4 & 5, the histio-attraction and histio-organization layers where fibrous matrix is being made. The bottom third is zone 6, the fibrous collagen layer, the formation of a scar and the conclusion of the mesenchymal wound events. These strata also reflect the timewise events in the wound.

0 - Active injury & inflammation. Inflammation is the initial response to injury, to contain damage, clear debris, and prepare for repair. It is also the trigger for repair. Inflammation and repair are integrated sequential processes. Left: a normal wound surface. Proteinaceous plasma exudates are the medium, the only environment that exists at this level. What can live and function there are those cells which normally live in plasma – leukocytes. Neutrophils are there in great numbers because they are chemotactically attracted by inflammatory signals. Other leukocytes arrive in the wound more or less in proportion to their concentrations in the blood, monocytes being especially important as the keystone or bridge between afferent (inflammation) and efferent (repair) wound events. Center: a close up view of normal wound neutrophils in the upper plasma protein layer of the wound. Their presence indicates active inflammation, disease, or injury of one sort or another. The greater the activity of disease or injury,

then the greater the neutrophils at this level and the greater the degree of acute inflammation, and the less likely that the wound can transition into the repair phase. Right: an injured leg, inflamed and not healing. This was the result of a superficial laceration in a healthy person. Progressive dermatitis, panniculitis, and ulceration were a consequence of inept care with injurious topical chemicals. Even when sustained injury does not result in progressive ulceration, it will keep repair suppressed.

1 - Inflammation subsides. Because sustained acute inflammation suppresses repair, it must subside for repair to proceed. Left & center: healthy wound surfaces well cared for. These show the top stratum, the plasma protein layer. Under the influence of basic hygienic wound care (regular bathing, silver based dressings, edema control), both of these specimens are nearly devoid of neutrophils, stippled basophilia, nor any other evidence of leukocyte activity and acute inflammation. The cells that are present in the upper plasma layer are all large and migratory – monocytes, macrophages, and some arriving angioid cells. The subsidence of inflammation means release from inhibitors that suppress reparative events. Assuming these wounds are otherwise healthy, they can now start healing. Right: the same clinical case after 2 weeks of care. Acute injury and inflammation are gone, and the wound is now healing.

2 - Macrophages, eschar separation. Macrophages are monocytes transformed by inflammation. They have multiple functions in the wound. Their afferent function is as phagocytes that clean up the debris and damage of the acute injury and subsequent acute inflammation. Left: mononuclear cells are distinctive in the topmost plasma protein inflammatory layer, appearing as typical "compact" (blood borne morphology) monocytes, or in transition as they accumulate cytoplasm and nucleoplasm, or as fully matured macrophages. Center: this image shows the cleavage plane between necrotic eschar (above) and viable tissue (below). The cleavage plane represents tissue lysis and processing by neutrophils and macrophages. Right: eschar separation seen clinically. This is a pelvic pressure ulcer several weeks after the pressure exposure and necrosis. The separation will continue until complete, all necrosis eliminated, leaving behind healing wound surfaces.

11

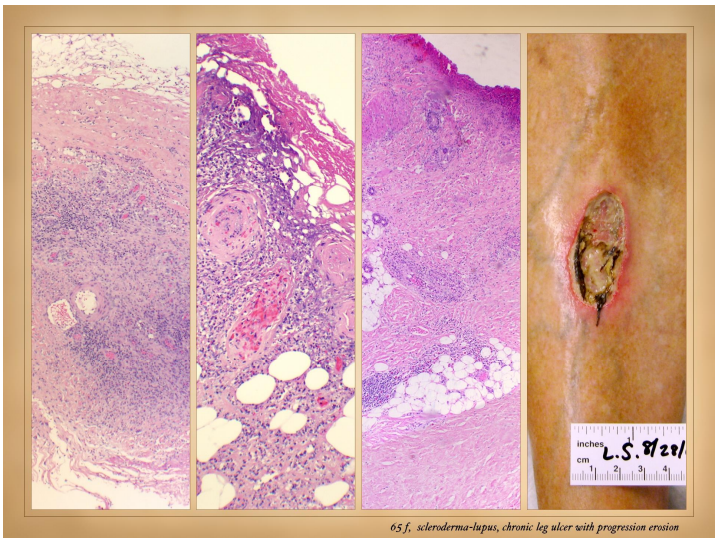
Eschar separation is a natural part of the process of inflammation and wound healing. It is nature's method of debridement. It is not only an obligatory free-running self-organizing process within a wound, but it is necessary and essential for the wound to transition from afferent inflammatory response to injury into efferent phases of wound repair. Eschar separation is simply the cleavage and separation between that which is dead or destined to die in the wound, and that which is viable and destined to survive. It is a process that operates tangential to the surface. It is easy to understand the passive self-organizing nature of the process. In a wound with damaged tissue, bulk tissues in the center lyse or infarct from inflammation throughout the injured zone or from the loss of circulation that ensues as the tissue degrades. Without circulation, inflammatory cells cannot arrive, so rather than lysing, residual tissues desiccate and are sequestered from the surrounding areas. At the periphery of the injured zone, there is no stimulus to inflammation, and neutrophils and macrophages that are delivered with inflammation lose any stimulus to arrive or remain. At the interface of the two zones, conditions remain viable and vascularized, but also persistently injured from obligatory wound inflammation, long after original injury and inciting events have subsided. Thus, residual inflammation becomes focused and concentrated in that interface zone, continuing to lyse tissues in that thin zone until the central bulk infarct no longer has any physical connection to the body. As the eschar falls off, what remains is simply the surface of the viable wound, with reparative proliferative processes already underway.

Left, a panel of four images illustrating the timewise sequence of eschar separation. Left top, pressure injury and skin necrosis due to errant placement of an ankle splint causing pressure on medial side of the first toe MP joint. Naive observers tend to misrecognize this as "bruise" or "ecchymosis", but it is dead escharotic skin and fascias. However, the boundary between living and dead skin is still physically intact, no separation of eschar yet, so it represents no risk to the patient for the moment. There are no gross signs of inflammation as erythema or edema, but microscopically, it is occurring as a necessary, obligatory, integrated, programmed part of response to injury. Left 2nd, a buttock ulcer representing the next phase of the eschar. Eschar is starting to separate, manifest outwardly as a thin break or separation between viable and non-viable tissues. With this break comes exposure of the mesenchyme and wet tissues underneath. Exposure of subepithelial tissues to environment and microbiota now induces reactive inflammation to a degree meant to defend the body, thus a denser infiltration of leukocytes and the appearance of overtly recognizable signs of inflammation at periphery of the wound. The process seen at the skin level is occurring along the entire interface of viable versus non-viable tissues regardless how deep or broad that process may be. Left 3rd, another buttock pressure ulcer. Progressive eschar separation is occurring. As the process more thoroughly cleaves the boundary of viable and non-viable, physical separation of the necrotic body becomes more apparent to the outward observer. Skin has turned black and leathery. Subjacent adipose has become lytic and necrobiotic. Note that the periwound is free of gross inflammatory signs. This is because (1) separation of eschar now removes some of the stimulus to inflammation (teleologically, that is why eschar separation occurs, so that "the bad" is eliminated and the body no longer needs to react or respond to threats and injury), and (2), the patient presents with some degree of topical wound care already started which is keeping the remaining surfaces hygienic and clean. At this stage of separation, the appearance is "ugly and gross" and inflammatory fluids are draining, and the wound has all of the pungent odors of necrobiosis that occur regardless the presence or not of any bacteria. Thus, this is typically the time, perhaps 4-12 weeks after initial injury, that patient, family, and naive caretakers begin to appreciate the true nature of the "bruise" they saw in the first image and then start a panic. This image represents the body's own efforts to protect and repair itself, and absent any signs of gross inflammation or secondary complication, this stage of the process is safe and welcome. Deliberate debridement will of course eliminate the "freak out" factor that affects so many naive observers, making the situation more pleasant to eye and nose, and legitimately expediting resolution and progressive care of the problem. Left bottom, an ischial pressure ulcer, necrotic eschar now almost fully separated. Just a few shreds remain of denser tendon and skeletal fascias.

Periwound is free of inflammation. Remaining wound surfaces are clean, healthy, and proliferative. The wound is now finally able to contract and close itself or else is eligible for operative closure.

Bottom, a histological view of eschar separation. The upper stratum is necrotic tissue that must be eliminated. The lower stratum is viable reactive wound and mesenchymal stroma reacting to injury. The pale lucent zone across the center is the cleavage plane between viable and non-viable tissues. Leukocytes (neutrophils and macrophages) are lysing the interzone, and soon enough the necrotic material above will peel away from body.

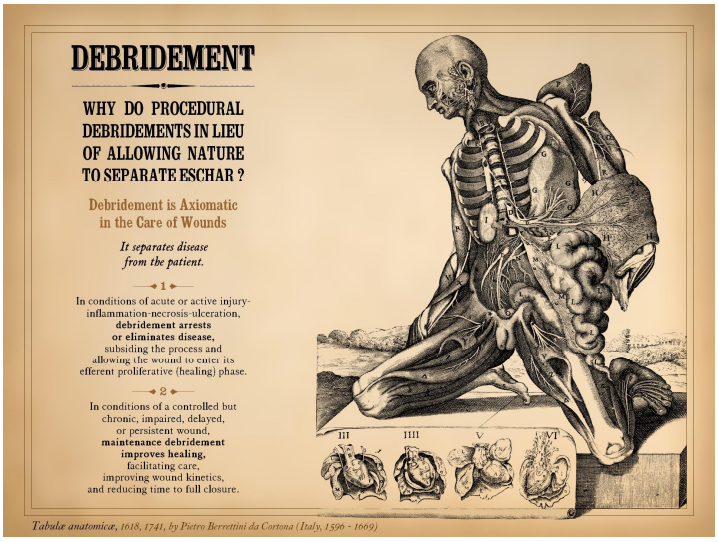
Upper tier, the other clinical images show eschar separation under various circumstances. Upper left, a finger tip injury has undergone necrosis and eschar separation. Subjacent viable skin has contracted. There is no gross inflammation. This finger is fully healed except that separated eschar remains attached by the bony tuft of the distal phalanx. Bone is subject to the same process of eschar separation that affects soft tissues, but it takes much longer. In this case, simple transection of the residual bone resolved the problem. However, even left to itself, the eschar would have eventually separated. Bone eschar is also demonstrated upper right, a sacral wound in which all soft tissue has separated, but there is necrotic bone that, left to itself, would take weeks or months more to separate. Good topical care has kept the wound from being grossly inflamed or threatening, but operative debridement will be required if compete resolution and then closure are to be accomplished anytime soon. Right lower is another sacral ulcer. Eschar is mostly separated, with a few shreds remaining of fat and fascias that will separate soon enough if not actively debrided. The periwound is not inflamed, and reparative processes are active. In pressure injury, everything from skin to surface of the underlying bone is necrotic, so when the eschar separates, the resulting void has a large cavitary three dimensional aspect to it. In contrast, upper center is a wound resulting from pyoderma gangrenosum, an inflammatory disorder that affects only the subdermal plane, sparing the subcutaneous adipose fascias. The skin eschar is now peeling off, and the base of the wound is flat and superficial, as expected (and confirmatory of a superficial process such as pyoderma gangrenosum). Proliferative wound healing is robust on the separated surface, and this wound will contract and epithelialize quickly.



65 f, scleroderma-lupus, chronic leg ulcer with progression erosion

12
This slide demonstrates a wound with several zones representing different times and events correlated with the histology of the process, the focus here being on eschar separation, nature's own process of debridement. The patient is a 65 year old woman with 10 year history of scleroderma-crest with other lupus or mixed connective tissue symptoms, presenting with a chronic ulcer at mid leg over the tibia. The patient has a history of thrombophlebitis and miscarriage, and she is Factor V Leiden heterozygous. The gross findings show infarction as well as lysis, and histology of the excised wound shows acute and chronic thrombi and peri-thrombotic infarcts.

Right, the gross appearance of the ulcer at the time of consultation. The erythema and dermal lysis at the 6 through 9 o'clock position is a typical inflammatory-lytic pattern of ulceration, whereas the yellow fat necrosis at the base and the black shreds of dermis are characteristic of thrombo-infarction. Left, is from an area that is at the front end of the active process and is most representative of the primary inflammatory pathology. The tissues here are all viable, no necrosis. Scar or dermis (upper pink area) is relatively bland, with little neutrophilic infiltration, neither diffuse nor concentrated. The basophilic nodule in the center is due to dense cellularity. This is a vascular locus with typical hypertrophic and migratory angiocytes, dense vessels, and also some fibroblasts and young matrix, i.e. relatively normal "granulation tissue". However, it is heavily surrounded and infiltrated with lymphoid cells, mostly lymphocytes with some plasma cells. While neutrophils are not prominent in the pink matrix areas, they are present within the lymphocyte-infiltrated central area. This is typically how autoimmune ulceration commences and progresses, activation of acute neutrophilic inflammation by chronic perivascular lymphoid cells. If this becomes overly incited, then inflammatory lysis and necrosis will occur. This corresponds to the tissues seen at the margin of the gross ulcer at the 11-12 o'clock position. Center left, is from an area of necrosis. This is representative of the thrombotic and reactive components of the process. There is thrombus and organization in vessels. Lymphoid cells are present, but they are relatively few compared to the dense neutrophilic infiltration characteristic of acute inflammation. The darker granular basophilic material toward the top is active infarction and necrobiosis of cells and tissue, and the pink area atop that is a ghost zone of established necrosis, all having predictably occurred around the thrombosed vessels. This corresponds to the active infarcts and ulceration seen at the margins of the ulcer in its lower pole. Center right, a view at the ulcerated surface. The deepest stratum below is dermis which is quite normal without inflammation. The upper pink areas are viable wound undergoing normal reaction and proliferation, i.e. "granulation tissue". Inflammatory cells are present, but their density is low and non-suppurative, consistent with good topical care (wound hygiene and silver sulfadiazine). In between is a zone where intradermal vessels are surrounded by lymphoid infiltrates. These are the lymphoid tertiary organs that are the mediators of immune events in the wound. As the acute injury and initial inflammatory process subside, acute inflammation is no longer seen around them, and the top of the wound looks like any ordinary wound now healing with granulation tissue at the surface. This is the tail end of the process. It corresponds to the granulation tissue seen at base of the wound in its upper half. Whatever necrosis was present here in days prior, such as that still seen in the lower half of the wound, that has now separated. As can be appreciated, eschar separation directly correlates with maturity as the wound settles down and moves from afferent inflammatory phase to the efferent proliferative wound healing phase.



13

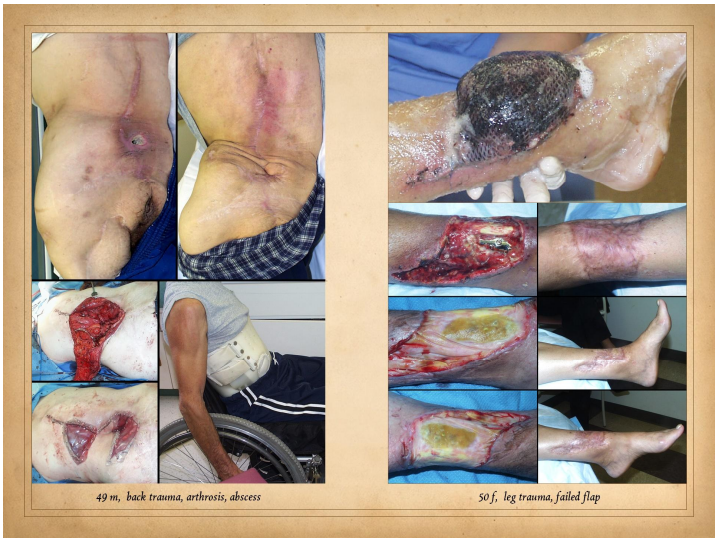
Debridement

Why do procedural debridements in lieu of allowing nature to separate eschar?

Why bother to do procedural debridement, cutting, scraping, shaving, incising, or otherwise peeling away wound detritus from the body? Why not let nature use its own process of eschar separation to accomplish that? As we have seen, nature does a good job of that process. However, we have also now seen that there are significant benefits that follow once eschar and inflammation are gone. Removal of injured, inflamed, and non-viable tissues permits the wound to move into an efferent phase of proliferation and wound healing. Expediting the removal of the injured material expedites the onset of the favorable back end of that integrated process.

Debridement is axiomatic in the care of wounds. It separates disease from the patient. Debridement is relevant in two general sets of conditions. (1) In conditions of acute or active injury, inflammation, necrosis, and ulceration, debridement arrests or eliminates disease, thereby subsiding the process and allowing the wound to enter its efferent proliferative healing phase. In these scenarios, *active* or *acute* or *primary debridement* generally refers to the elimination of native tissues that have been injured or devitalized by the trauma or disease. (2) In conditions of a controlled but chronic, impaired, delayed, or persistent wound, *maintenance debridement* is more relevant. That is the periodic removal of any new necrosis, but also curettage, planing, or paring away the upper strata of the wound where inflammatory cells reside. In these circumstances, periodic debridement improves healing by facilitating care, improving wound kinetics, and reducing time to full closure.

Another illustration from Pietro da Cortona, Tabula IX, revealing the internal viscera. Like all of the Tabulæ Anatomicæ, it has a sense of artistic pose and drama rarely matched in other anatomical studies. The art is eminently Baroque and eminently da Cortona. It is included here to remind that the body is highly structured, and disease and injury can affect all structures and tissues, not just skin. The physiology of responding to damage, via inflammation then wound repair, applies to any injury of any organ. The principles of debridement and other care apply equally to all injured tissues, even if some technical adjustments are required here and there.



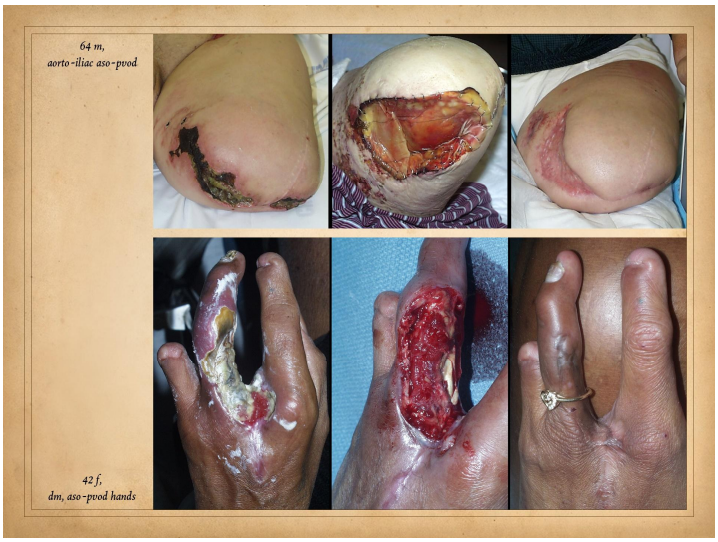
14

Wounds cannot heal until injury and inflammation subside. Open wounds cannot be closed surgically if there remains a focus or nidus of disease, debris, or inflammation that will cause ongoing drainage and wound failure. The one and only central principle in the preparation for wound closure is that all non-viable, inflamed, infected, or pathological tissue be removed before closure. Despite the central importance of this principle, there are far too many examples of surgeons who have eschewed this principle. You, the reader, may care or not to speculate how it is that this lapse in knowledge, technique, and practice of the art can be so commonly ignored or trivialized by those who should know better. Nonetheless, for reconstructive and wound surgeons who make a career of this business, the patients and stories are all too common of seeing patients who have suffered due to violation of this principle from surgeons primary in other specialties. Cheating this principle cannot win, and it is the wound and patient who pay the price, at minimum by a failed and persistent wound, or at worse by progressive morbidity, illness, and disability. Whatever else you might choose to call the preparation of a wound for closure, regardless whether that closure is by surgery or by natural contraction, it is debridement.

This and the next slide demonstrate examples of patients where improper preparation of a wound and premature closure resulted in serious progressive problems or failed results requiring do-overs. Left, a 49 year old man had trauma and spine injury with paraplegia. Hardware fixation of the back was complicated by abscess. Multiple attempts were made to "debride" then close the wound without actually removing pathological tissue and materials, and without going through a period of intermediate care to get the wound clean and settled after debridement. Over a period of years, soft tissues became progressively pathological and scarred, and abscess and suppurative osteitis progressed through the zone of the original trauma and fixation. Left upper, before and after pictures are shown. *Left* is the back wound as seen on consultation when just a small aperture in the skin provides the only drainage for the wound. *Right* is the result after proper care. That included complete vertebral ostectomy L1-L5, then closing the defect with flaps and regenerative matrices (Integra dermal regeneration template). The stages of surgery are shown in the *bottom* panel. Even with the lumbar spine missing, the patient is healed, healthy, and fit, seen here in his wheelchair supported by a lumbar orthotic.

Right, an ankle defect following trauma, tibia fracture, orif, and then skin necrosis and ulceration. Closure was attempted with local then several free flaps (the conventional solution for this situation) all of which died. When seen in consultation, more flaps were obviously ineligible. Instead, Integra collagen-gag matrix was used to provide essential coverage over the hardware, fracture and tendons. While serving as an effective skin substitute in the short term, it also became the agent of skin regeneration. It conducted new regenerative tissues tangentially through the matrix,

resulting in a healed wound over hardware that required no further surgery and which preserved normal motion of ankle and tendons. That good result did not happen just because of the choice of surgical technique. It required a period of topical wound hygiene and preparation in order to get a healthy healed result. The problem was that the several free flaps and other care done by the original surgeon had no interval periods of debridement and wound care before or between procedures. Dead flaps covering pus, as seen in the *top* image, were excised and immediately replaced with other flaps covering the same debris and pus. Proper acute debridement to remove the necrotic flaps and the a period of topical care and maintenance debridement converted the wound into a healthy entity that could then be covered by the semi-biological matrix.

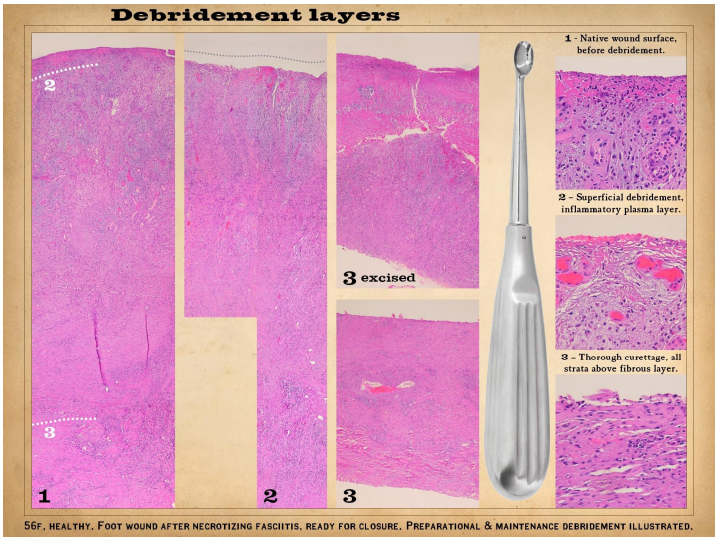


15

Upper panel, a 64 year old man with aorto-iliac occlusive disease and Leriche syndrome. A toe injury resulted in progressive levels of necrosis and amputation. With each failed amputation performed by the original surgeon, there were no interval periods of simple debridement, topical care, or any other supportive care. Unfortunately, by the time the amputation progressed to thigh level, there was insufficient femoral artery runoff to maintain large vessel patency, preventing efforts to directly revascularize that lower extremity. Left, when seen in consultation, no more acute surgery was done. Simple debridement and topical wound care were supplemented by hyperbaric oxygen therapies. Center, when acute phase inflammation and infarction subsided, the skin was reconstructed with a dermal regeneration template. Left, the wound then healed without other complications or loss of tissue. Exercise of basic wound care and vascular surgery principles when the problem was confined to the foot, or anywhere along the way, would have averted the original or progressive amputations.

Lower, a 42 year old woman with diabetes and upper extremity atherosclerosis. Comparable to the story above, a small fingertip injury led to progressive segmental amputations and wound closures without any "backing off" of surgery and applying proper principles of wound care. Progressive abscess, necrosis, and incremental amputations eventuated in ray amputation. Before amputating the whole hand, the patient was sent for consultation. Left, the long finger is already missing, and ring finger is now undergoing infarction and pending loss. Proper topical care has been initiated. Center, after topical care and debridement, the wound is stable and ready for closure. Closure was done with a regenerative dermal template. Right, the hand is healed without further problems.

The two cases on the preceding slide were healthy patients with no wound healing risks. Necrosis, wound failure, and complications were attributable solely to overly ambitious surgery and improper attention to the biology of wounds and wound care. There is no justification for that lapse of care or abrogation of basic surgical principles. For both of them though, despite the nuisance of all they went through and the ultimate long term risks, those wounds presented little immediate risk to their general health, allowing ample opportunity to make more such mistakes and ineffective care. In contrast, the two patients on this slide have wounds subject to *wound pathergy*. Their severe atherosclerosis and vascular insufficiency put the wounds at extreme risk of progressive infarction with any subsequent injury or inflammation. For them, life as well as limb were in legitimate jeopardy, and the details of care mattered with regard to controlling progression of disease and then getting the limbs healed without further risk or loss. The greater the risks of wound pathology and pathergy, the more sensitive the wound is to each detail of disease or care, then the more essential it is to induce a state of relief from injury, inflammation, and thrombosis. Proper topical care and meaningful debridement are necessary if wounds such as seen here are to have a chance to subside and then be eligible for closure.



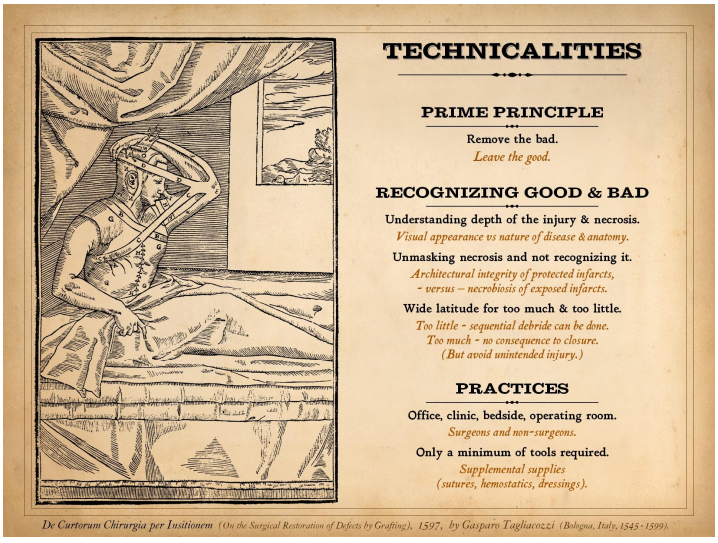
16

When we do debridement, what are we really removing? For an acute or primary debridement, the object of the activity is self evident – whatever tissues or structures have been damaged or devitalized by the initial trauma or disease. For maintenance or incremental debridements that are most apropos for the management of chronic wounds, the intent is to remove the upper strata of the wound where inflammatory cells and their debris reside. Because the afferent and inflammatory events in a wound are inhibitory or even destructive to efferent and reparative processes, removal of these strata has the opposite effect. Their elimination removes further triggers for afferent inflammatory behavior, and it disinhibits the repair phases. The upper strata are of course obligatory components of a wound, so they are self regenerating, and thus periodic debridement and re-removal is deemed valuable,

Wound anatomy and strata are presented most minimally on Slide 6. Detailed discussion of this subject can be found on the arimedica.com website. For the sake of this slide, it is important to remember the following. The topmost stratum in the wound is the *plasma protein inflammatory layer*. Regenerating blood vessels below are leaky, allowing plasma exudates to reside on the wound surface. Leukocytes live in blood, i.e. plasma, so they are at home in that top layer. That layer therefore serves as the protective interface to the ambient world. Depending on quality of topical wound care, the plasma inflammatory layer will have greater or lesser amounts of neutrophils depending on how clean the surface has been kept. A well cared for wound can have virtually zero

leukocytes, but the plasma layer is there at all times. Below that is the *aminoglycan* and *angio-migration* and *angio-organization strata*. In normal post-inflammatory wound healing, histogenesis starts with the arrival of angiocytes and their reassembly into blood conducting vessels (their leakiness when not yet reassembled is the origin of the plasma layer above). The angiocytes need a medium in which to do their business, and that is the glycosaminoglycan ground substance that is generated by nearly all cells including the leukocytes and angiocytes. Once vessels are established, fibroblasts deep to them have an environment in which they can make connective proteins. Since these histogenetic events are suppressed by inflammation, they cannot function unless inflammation is gone. Thus, the deeper histogenerative strata of the wound never have inflammatory leukocytes (unless some new insult has flared up injury). In the intermediate aminoglycan angio-organization strata, leukocytes are often abundant, but they are migrating from the new vessels to the plasma layer above, attracted by the chemotaxis of the events at the surface. The aminoglycan layer is not the target of the leukocytes, but their migratory tropism confirms that they have been activated. Thus, the leukocytes and their debris and effects, those agents that can cause more inadvertent injury or else inhibit wound healing, they exist in those upper strata only. Fully activated leukocytes are in the topmost plasma layer, and migratory leukocytes are in the subjacent aminoglycan layer. Since neither of these zones has connective proteins, they can readily be removed. Since the even deeper fibroplastic strata never have leukocytes, and since they cannot easily be removed, it is thus easy during debridement to find the effective plane. That which curettes off easily removes leukocytes. That which cannot be readily removed is benign.

The histology shown comes from a single wound at the time of operative closure. The patient is a 56 year old woman who had streptococcal necrotizing fasciitis. After acute phases of care, the wounds were clean and ready for closure. She is otherwise healthy with no disorders or risks of wound healing. These images show a healthy wound with no aberrations from normal wound healing physiology. Illustrated is a curette used to do the debridement. The three small images on the right are closeup excerpts of the three long images on the left. Image #1 is a biopsy of the native prepared wound prior to any instrumentation. This is a prototypical healthy wound with all of the expected strata. The thin pink rim at top of the wound is the plasma protein layer. As seen in the zoom-in on the right, leukocytes are sparse since the preparatory care has been good, but they are there. The more purplish zone in the upper third of the specimen is the angio-organization layer, looking purple from the basophilia of cells, and absence of any pink staining eosinophilic connective proteins (special stains are needed to see the aminoglycans in that layer). The zoom in view confirms the presence of leukocytes in the reassembling blood vessels. Dotted line “2” shows the rough boundary between plasma and aminoglycan layers, the zone that can be easily removed by light curettage. Dotted line #3 shows the rough boundary between the aminoglycan-vascular zone and the connective protein layer below, a boundary that can be removed with a bit more pressure and force on the curette. As can be appreciated during actual debridements, curettage of the sticky upper plasma layer is associated with minimum if any bleeding, whereas deeper curettage down to fibrous layers always causes some bleeding. Image #2 (aligned vertically to the first) is a biopsy of the same wound after light curettage has removed the topmost plasma protein layer. The grey dotted line shows where the surface would have been. The zoom-in on the right confirms that the plasma layer is gone, new vessels and non-collagenous stroma are present, and that numerous leukocytes are still present. Image #3 is a third biopsy after more vigorous curettage removed granulation tissue down to the white fibrous layer underneath. Histologically, only connective tissue and scar remain. The zoom-in confirms fibrous tissue without leukocytes or other inflammatory cells. Image #3-excised looks at what was curetted away for specimen #3. It confirms that the debrided material includes the leukocyte harboring plasma and aminoglycan layers. Had this wound not been closed, then normal wound healing biology would have restored the upper strata within the next 4-7 days, and the wound would then be eligible for repeat curettage debridement if that is deemed necessary to maintain health of the wound.



De Curtorum Chirurgia per Insitionem (On the Surgical Restoration of Defects by Grafting), 1597, by Gaspare Tagliacozzi (Bologna, Italy, 1545-1599).

17

This presentation is not about the techniques of debridement, but it is worth mentioning a few technicalities that are often not mentioned, if for no other reason than to help build the confidence of those just learning how to do this most important of modalities.

Conceptually, debridement is simple. Adhere to the prime principle of removing the bad and leaving the good, and you are unlikely to do wrong. Even if something is removed that might seem important, if it is already dead, then sooner or later the body will get rid of it anyway. The sooner you get rid of it, the safer you keep the person and the more quickly you can close or reconstruct the defect. The art is not in how to debride, but in how to recognize the good and the bad.

Recognizing the good and the bad begins by understanding (1) the disease or trauma that caused the injury (and thus what to anticipate is the extent of damage), (2) the underlying anatomy (to be prepared for underlying structures that might appear during the debridement and the likelihood that they are healthy and to be preserved versus injured and in need of repair or in need of debridement), and (3) the inherent nature of injury and necrosis and how they should appear. A good example is bleeding. Bleeding is generally a good indicator of viability and the limits of where a debridement needs to be taken. However, certain tissues are sparsely vascularized. For instance, tendons and ligaments are dense with collagen but sparse with cells. Vascular density in tissues is governed by cell density and metabolic rate rather than bulk tissue mass or volume. Thus, a normal tendon looks white and avascular even when perfectly healthy, and expecting to see bleeding is misleading. Understanding what a healthy versus necrotic tendon should look like is matter of experience.

A caveat of debridement is that necrosis can be unmasked but not recognized. This is because necrotic tissue can look entirely normal to the inexperienced observer (and even sometimes the experienced observer). The issue is that when tissues are necrotic and exposed to the ambient world or to inflammatory biology, they degrade quickly. The liquefactive or desiccated escharotic appearance of necrobiotic tissue is familiar to all. However, necrotic tissue that remains sequestered and protected from the ambient world or degradative processes can maintain a nearly normal architecture and appearance for a long time. Consider for instance what the infarcted myocardium looks like after an MI. Unless you are a

pathologist or a heart surgeon, you likely have not seen it. However, if the patient recovered, that heart muscle must have maintained its structural integrity long enough to fibrose, otherwise ventricular rupture would have occurred, which does happen, but infrequently. Imagine how that juicy delicious steak looks in your refrigerator waiting to be grilled, or how a side of beef or a cured ham looks while hanging for days, weeks, or months before being ready to serve. If infarcted tissue is sequestered by vascular exclusion, such as an MI, or a hypercoagulable or pressure ulcer, or a compartmental muscle infarct that leads to Volkmann's contracture, then leukocytes and inflammatory cells cannot arrive in that devascularized zone. If inflammatory cells cannot invade the infarct, then the tissue cannot lyse. Under those circumstances, newly dead tissue runs down its metabolic energy to equilibrium after which chemistry in the cells and tissues ceases. For infarcts that eventually get exposed, exogenous elements or energies will then alter or degrade them, but until such time, the dead tissue remains architecturally intact. The practical side of this with regard to debridement is that you can excise dead skin that looks escharotic and believe that fascias underneath look healthy because their architecture seems intact. However, having exposed those deeper layers, necrobiosis will ensue, and within hours to days, the nature of the infarct becomes apparent. If you understand in advance where the depth of injury should be based on the nature of the injury, then you can be more certain of a thorough debridement the first time.

The good news for practitioners is that debridement is a forgiving activity. There is wide latitude to safely remove too much or too little. If you debride too little, you have done no damage, and you have still accomplished something useful even if incomplete. Within so many days, the nature of the remaining eschar will become evident, and repeat debridement can then remove what remains. If you remove too much, well, perhaps not really. Novice practitioners tend to be unnecessarily shy about adequate debridement. If the debridement does carve out some normal tissue, so what? A few millimeters, a few centimeters – depending on the size and location of the wound, that is trivial. To remove extra skin, fat, or muscle is inconsequential in most areas, and the more thorough the debridement, the more effective it is. Removing living tissue that is scarred and dystrophic can even be of great benefit in promoting normal wound mechanics and closure, so excision of living tissue can be a vital part of a good debridement in certain circumstances. The caveat is to not indiscriminately remove tissue until important living structures underneath are damaged. A chronic abdominal wound sitting on open bowel risks perforation and fistula if the debridement gets too aggressive or uncontrolled. If you are not a surgeon or this subject is new to you, odds are you will err by not doing enough, which is safe. In circumstances where accidental injury is possible or to be avoided, then being circumspect by removing too little and then doing more later as needed is the prudent approach.

Debridement can be "big" or "small" depending on the nature of the patient and problem. It can be done in whatever venue is appropriate for safety or efficiency, be it the office, clinic, bedside, or operating room. Large trauma cases may be best served in the operating room, but smaller injuries and nearly all chronic wounds can and should be debrided in office or clinic. Regardless the venue, only a few tools are needed – scissors, forceps, scalpel, curettes, rongeurs. Supplemental supplies include sutures, hemostatic agents, dressings and bandages, and local anesthetics. Local anesthesia is rarely needed for maintenance debridements or to trim eschar that is already nearly separated, but primary debridements of recently injured tissues may need it. Practitioners ought to make a habit of having anesthetics and hemostatics handily available even if used on only a small percentage of cases.

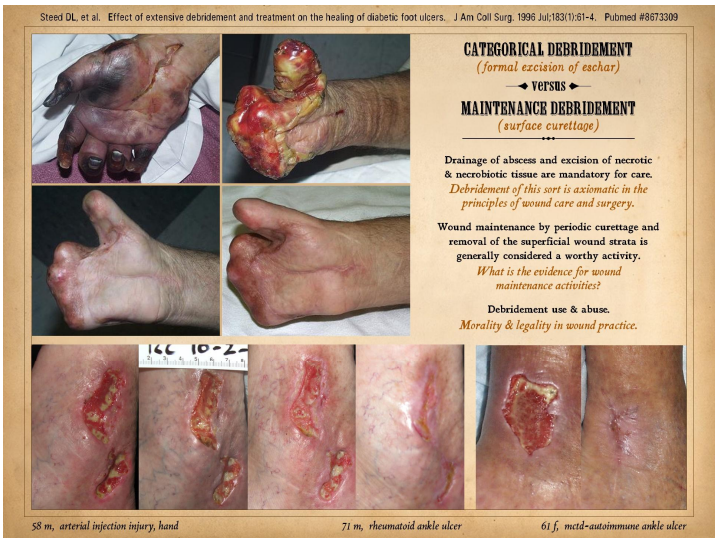
This illustration is from De Curtorum Chirurgia per Insitionem, "On the Surgical Restoration of Defects by Grafting" (or alternately, "On the Surgery of Mutilation by Grafting"), 1597, by Gasparo Tagliacozzi (1545-1599). Tagliacozzi was an Italian surgeon and anatomist, professor at the University of Bologna. In Renaissance and Baroque Italy, rapier dueling was popular, resulting in many nasal amputations. Nasal reconstruction by flaps has a long tradition in the history of surgery, recorded as far back as in the ancient Indian Ayurvedic text the Sushruta Samhita (circa 1000-600 BCE). This knowledge made its way to Italy via Arabic translations, and in the early 15th century, pioneering Italian surgeons were trying variations of the method to reconstruct a missing nose. Tagliacozzi built upon the work of prior Italian surgeons, notably Antonio Branca, to refine the concept of the arm flap. His book, the De Curtorum, illustrates the various steps in the procedure, but the woodcut shown, with the transferred flap and arm supported by an elaborate harness, has become an iconic symbol of the history of plastic surgery. The procedure however is not a very good one, and it rapidly dwindled out of interest until a brief revival around the turn of the 20th century. This image is included here to remind that proper debridement and preparation of a wound are mandatory before attempting wound closure or reconstruction, and that technique is important. Recall too the literal and derivative meanings of "débridement" as they come to us from the French. By analogy, removal of that harness would have been a debridement, a debridling, but one would hope that the flap was healthy and did not need surgical debridement for flap necrosis.

18

This presentation has referred to the distinction between primary debridement and secondary maintenance debridement. These final few panels will address the importance of maintenance debridement in the management of chronic and pathological wounds. This slide illustrates three circumstances of debridement. They are all debridement, but they serve different purposes for different types of wounds, patients, and pathways of care.

Left upper, an 82 year old woman who fell resulting in a shearing-avulsion injury with hematoma of the leg. The avulsed skin over the hematoma is necrotic and requires excision so that the eschar is eliminated and other care can begin. This is an example of primary debridement of initially injured tissues. Left lower, a 67 year old woman with active rheumatoid arthritis whose laparotomy incision dehisced. It is being managed as a chronic wound, using modalities meant to coax the wound to contract and epithelialize without doing surgery. Periodic curettage is being done as a means of maintenance debridement. Right, a 73 year old man with sternal dehiscence after coronary bypass. Right upper is the wound not long after initial primary debridement and start of topical wound care, showing that acute injury and inflammation are under control. Right middle is the wound after a week or two of topical care.

It is proliferative and ready for closure. Had this been an impaired or pathological wound, then intermediate maintenance curettage would have been done during followup office visits, but as a strictly healthy wound, maintenance curettage was not mandatory. Right lower, the wound in surgery, the first step before sternal ORIF being to fully excise or curette away the existing surface and expose the normal tissues underneath.



19

Top, a panel showing the reconstruction of a hand in a 58 year old man after arterial injection injury. After treatments to maximize the zone of survival, necrotic tissues were removed and the hand was reconstructed. Rather than recess viable bone in order to get skin coverage, all viable tissues were preserved, and missing skin was restored with a regenerative matrix. Miscellaneous activities such as deepening the web space, tendon reinsertions, and metacarpal osteotomies and prosthetic joints restored a reasonable degree of function. In this case, debridement is primary, an activity of care categorically required to initiate the reconstructive process.

Although this was a case of dry sterile eschar, tissue injury in any form must be eliminated before healing or wound closure. Whether drainage of abscess, excision of necrotic and necrobiotic tissues, or other methods to eliminate injured and inflamed tissues, these activities are mandatory for care. Primary debridement of this sort is axiomatic, a foundational principle in the care and surgery of wounds.

Bottom left, an ankle wound around the lateral malleolus, one of several in a 71 year old man with severe active rheumatoid arthritis and rheumatoid ulceration. All healed with wound care modalities and treatment of his rheumatoid arthritis without the need for wound closure surgery. Care included systemic steroids (prednisone), good wound hygiene, topicals, intra-lesional steroids (triamcinolone), and perfunctory wound curettage on every clinic visit. This wound would not have healed by basic wound modalities alone, including the maintenance debridements. Management of the primary autoimmune state and inflammation (manifest by complete control of arthritis and synovitis) were the key treatments. However, good wound care per se can never be ignored or trivialized, and the steroids would not have succeeded if they were the only thing prescribed. Bottom right, another ankle ulcer in a 61 year old woman with lupus or mixed connective tissue disorder, disease currently controlled and quiet. This ulcer also healed, by the simplest of approaches, using only good wound hygiene and topicals, edema control, and periodic maintenance debridement, i.e. curettage during scheduled office visits.

The question for the sake of this presentation about debridement is how effective or necessary is repetitive curettage and maintenance debridement? This activity to remove the superficial wound strata is generally considered a worthy activity. Is there evidence for wound maintenance activities? Is there benefit to doing maintenance debridements to remove the superficial wound strata, or is there detriment to repetitive mechanical re-injury of the wound? Does this practice promote healing by removing inhibitory elements in the wound, or does it delay healing by removing wound module elements already in place? These questions were common and appropriate for a long time, but since circa 2000, there has been a general acknowledgement that regular or periodic maintenance debridement is beneficial for chronic wounds when included as a part of comprehensive care that strives to get the wound closed.

Evidence for the benefit of regular debridement is both clinical and physiological, but none of it admittedly is robust or investigated from explicitly that point of view. Wound research of the decade circa 1995-2005 emphasized the role of inflammatory wound chemistry such as MMP's (matrix metallo-proteases), and they demonstrated to variable degrees that their elimination by one means or another was stimulatory or dis-inhibitory to wound healing. These were indirect implications of the validity of debridement to eliminate inflammation. The oft cited clinical study that demonstrated the effect of debridement is "the Steed paper". Steed et al published their results of a clinical trial for becaplermin (recombinant PDGF-BB, Regranex®) as a topical wound healing agent for diabetic foot ulcers. The original paper was *Steed DL, et al. Clinical evaluation of recombinant human platelet-derived growth factor for the treatment of lower extremity diabetic ulcers. J Vasc Surg. 1995;21:71-81.* Two groups of patients were studied, assigned to test drug versus placebo. In the analysis, the drug made more ulcers heal and heal faster than placebo. Primary and followup debridements were done as discretionary activities, so their effect was addressed in the analysis. The paper quoted:

> *There was no statistical difference in the number of debridements carried out in the two treatment groups. On average a patient underwent debridement on 47.4% of the study visits in the placebo group and 43.3% of the study visits in the rhPDGF-BB group. There was a possibility that there may have been a center effect on the number of debridements carried out and this may have affected the rate of ulcer healing. However, neither the treatment nor treatment-by-center interaction showed any statistically significant differences in the number or percentage of visits during which debridement occurred ($p > 0.2$).*

The implication was that the two groups were well matched and that debridement rates did not taint the data or statistical conclusion that the test drug performed better than placebo. However, a year later that group published a reanalysis of the study, *Steed DL, et al. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. J Am Coll Surg. 1996:183:61-64. Pubmed #8673309.* They described the study conduct and then results and conclusions as follows:

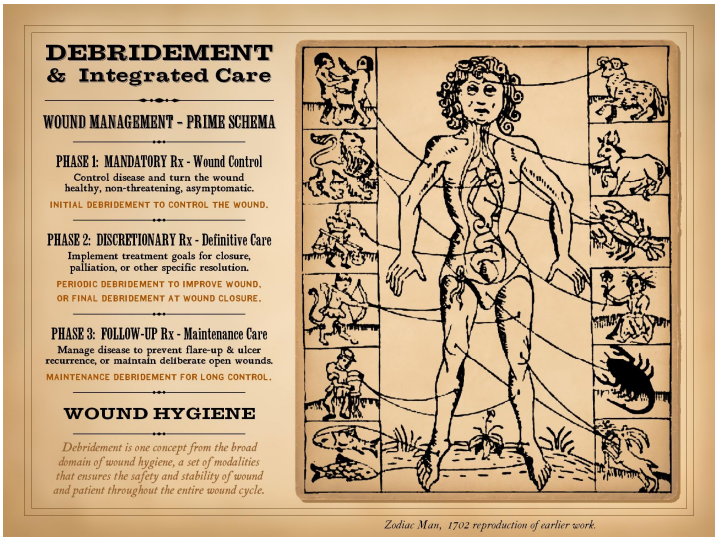
> *All patients had aggressive sharp debridement of their ulcers before randomization and repeat debridement of callus and necrotic tissue as needed. The influence of debridement was evaluated by reviewing the records of the office visits where debridement was performed. . . . Results: Forty-eight percent of patients treated with rhPDGF healed compared with 25 percent of patients who received placebo ($p = 0.01$). The mean percentage of office visits where debridement was performed was comparable for the two treatment groups: 46.8 percent (rhPDGF) and 48.0 percent (placebo). In general, a lower rate of healing*

was observed in those centers that performed less frequent debridement. The improved response rate observed with more frequent debridement was independent of the treatment group. However, for any given center, the percentage of patients who healed was greater with rhPDGF than placebo. . . . Conclusions: Wound debridement is a vital adjunct in the care of patients with chronic diabetic foot ulcers.

Platelet releasates and then becaplermin were the first generation of biologics for wound healing, introduced in the 1990's. Debridement was always emphasized as a necessary activity during application of the topical treatment agents, because a signaling peptide was being applied. Failure to clean the wound prior to application meant that the chemical would be buffered or degraded by "wound junk" such as proteases or non-specific opsonins before it had a chance to bind to its receptors on the target cells (angiocytes and fibroblasts). However, debridement and wound preparation rates among physicians using these materials were always variable and not strictly enforced, even in a controlled study such as the becaplermin trials. The quote from the first Steed study hints at the idea that debridement might have made a difference in healing rates. In re-analyzing their data to address that question, the results showed that regardless of which agent was applied to the wound, healing rates improved with more frequent debridement. If only the treatment group had been looked at, one could question whether debridement had a genuine direct effect on the biology of healing versus just facilitation of the potency of the drug for the peptide binding reason just described. That the improved healing rate was also observed in the placebo group might therefore imply a favorable direct effect of debridement on wound biology. However, this conclusion also has a caveat, revealed in the statement "a lower rate of healing was observed in those centers that performed less frequent debridement". Notice how the study was not designed to measure the effect of debridement, nor could the analysis directly extract that conclusion. All the analysis could state was that better healing rates occurred in certain centers. Association does not imply causation, and the better rates in the debriding centers might reflect not the debridements per se but rather a generally more detailed and punctilious approach to overall care in those centers. So, readers and practitioners must draw their own conclusions, but ever since the "Steed study", the concept of frequent maintenance debridement of wounds has been accepted as preferred practice.

While stringent studies might not exist to validate the role of maintenance debridement, certain analytical, inferential, experiential, and biological insights about wounds can be accepted as axioms to govern practical daily wound care. Upper wound strata harbor the microbiota that can destroy a graft or topical agent or provoke destructive inflammation (proportional to the degree or lack of good ongoing topical wound care). The upper wound strata are inflammatory, and keeping inflammation minimized is beneficial to wound dynamics and the promotion of efferent proliferative processes. Upper wound strata are inflammatory and can degrade the therapeutic agents (pharmaceuticals and biologics) applied as a wound stimulant. Anything that is done to minimize the load or effects of these detrimental factors must be considered favorable and a necessary component of practice.

A final word about the practicalities and practices of debridement, concerning use, abuse, and practitioner ethics. Debridement is necessary axiomatic, and unequivocally legitimate as a medical practice, and as just discussed, more is better. Debridement is also unequivocally accepted as legitimate by third party payors, those government and private insurers who pay the patients' bills – but within reason and morality. A new injury with eschar or a hot abscess needs primary debridement. The payors will not argue against that, and if prompt debridement prevents a more serious more expensive illness, then so much the better. Depending on the nature or circumstances of the wound and patient, a genuine primary active excisional debridement of real eschar or necrosis will be required none or once or perhaps twice. One good get-the–job-done debridement is the benchmark of good care. The problem is that beginning in the early 2000's, legitimate surgeons and practitioners started to get harangued by the insurers for documentation of the legitimacy of a debridement. How could there be any suggestion of impropriety, because who other than qualified surgeons would be doing debridements for messy unsavory problems? Who had raised the red flag of suspicion by doing too many unqualified and unnecessary debridements? Well, it turns out that the new cadre of wound centers and physicians spawned by the new wound products and biologics of the 1990's were submitting bills for every perfunctory wound maintenance activity that was done. Each scrape of the curette was charged out à la carte, not part of the "cover charge" of just showing up for the office visit. A visit with the doctor implies certain activities related to patient interview, hands on examination, diagnosis, treatment, and the physical activities that support the process. You can charge to give a vaccine, but you cannot charge extra to swipe an alcohol pad over the injection site. You can charge a fee to evaluate heart disease, but you cannot limit that to taking the pulse and then charge extra to listen with the stethoscope. Maintenance care of the wound, including curettage and minor debridement, is not a separately billable service in the eyes of the payors, and abuse of the process risks unwanted scrutiny and possible unpleasant legal consequences. One would hope that if you care to do well, to do well for the number of "wins" that you can tally on you healed wound score card, to do well by the patients who put their trust and hopes in you, to do well by a society that spends and wastes far too much money on ineffective and unnecessary care, that you would care to do it righteously and morally. Don't be the knucklehead who spoils it for the rest of us.



Zodiac Man, 1702 reproduction of earlier work.

Where does debridement fit into the overall structure of wound medicine and wound care, into the treatment plan and workflow for an individual patient or wound? Regardless the disease being treated, management of medical problems follows a schema that generally has three phases. This is especially apparent when treating the acute flareups or complications of a chronic problem, including most problem wounds which are simply the secondary manifestation of some underlying primary disorder.

Phase 1: Mandatory Treatment. This is the phase in which the acute process is acting up or out of control, and it must be controlled or subsided to protect the patient, preserve health, and prevent progressive injury or morbidity. Consider for instance the patient who comes to the emergency room in diabetic ketoacidosis or the patient with Crohn's disease who presents with an intestinal perforation and peritonitis. For the diabetic patient, acute mandatory care means getting sugar into cells, correcting acidosis and electrolyte alterations, and correcting the hyperosmolar state. For the Crohn's patient, acute mandatory care means exploring and draining the abdomen, diverting the stools, and giving antibiotics and general fluid and metabolic support until stable. For wounds that are actively inflamed or progressively ulcerating, acute phase mandatory care means controlling the wound and the underlying causative disease, turning the wound into a stable, non-threatening, asymptomatic condition that the patient can safely coexist with. This includes drainage, debridement, initiation of topical hygiene and edema control, and alleviation of the causative agent or control of the underlying disease, e.g. eliminating pressure from a pressure ulcer, or controlling autoimmune inflammation in a rheumatoid patient, or revascularizing an extremity in an arteriopath. It is in this phase that primary debridement to remove the detritus of the acute event is most relevant.

Phase 2: Discretionary Treatment and Definitive Care. This is the phase after the acute threatening state has been controlled. You now have the time to think through the definitive resolution of the underlying disease or causative problem. For the diabetic in DKA who is now stable, this means initiating a regulated diet and getting insulin or other medications started and optimized. For the perforated Crohn's, this phase means initiation of relevant pharmaceuticals and learning how to manage diet and the colostomy or ileostomy until the bowel can be anastomosed at a latter procedure (if that is a possibility). For wounds, it means implementing the treatment goals for closure, palliation, or other specific resolution. This could include preparation for closure then the surgery itself. It could mean a program of topical agents or biologics to promote closure by non-operative biology. It is in this phase that periodic debridement is done to expedite the time to closure, and that final debridement is done at the time of operative wound closure.

Phase 3: Follow-up Treatment and Maintenance Care. This is the long term phase, after the acute event has been fully resolved and the patient is getting back to a normal or at least a stable life, when acute care treatments are no longer required. For the diabetic, it means regular maintenance of diet and medications, and long term surveillance for the secondary sequelae of the disease such as nephropathy or retinopathy. For the Crohn's patient, it means maintenance of anti-immune medications, bowel care, and diet. For the wound patient, it means keeping the primary underlying disease well controlled, such as diabetes or rheumatoid, maintaining good edema control, preventing accidental injury, maintaining orthotics, etc. If the wounds are healed, then the focus is on preventing new ulcers. If the wounds are open and being managed as long term chronic wounds, then perfunctory maintenance debridement serves no purpose, although debridement as required is appropriate if the wound shows hints of starting to worsen.

Wound Hygiene. Although "debridement" tends to get attention as a procedure and wound care modality (and perhaps because it is a fancy sounding doctor word), there is a concept and mode of care that transcends debridement, the superset of all basic wound care modalities of which debridement is just one element. That concept is *wound hygiene*, the comprehensive set of modalities that ensures the safety and stability of wound and patient throughout the entire wound cycle. From simple bathing and dressing changes to maintenance curettage or active debridement when needed, these are the activities that ensure that the wound stays clean and free of inflammatory inciters and inflammatory debris. Basic wound hygiene requires little more than soap and water. When fancy doctor tools are unavailable for formal debridement, tongue blades serve well as curettes. The clever physician and patient can find many convenient and inexpensive ways to maintain good wound hygiene and good health.

Illustrated is a "Zodiac Man", a normative figure in the illustration of mediaeval and Renaissance medical books. The woodcut shown is from a 1702 work, and it is an obvious reproduction or reprint of an earlier source, best guess probably latter 15th century Germany. The zodiac man concept is rooted in astrological beliefs that equate diseases and body parts to celestial signs and the times of the calendar – which constellation ruled which organ. It is included to remind that good wound care and good results are not a matter of medieval faith or mystical beliefs. We know much about what makes a wound heal or not heal, and debridement is one of the simplest, cheapest, and most effective means of regulating the factors that promote wound healing and eliminating those factors that would inhibit it.

21
Summary

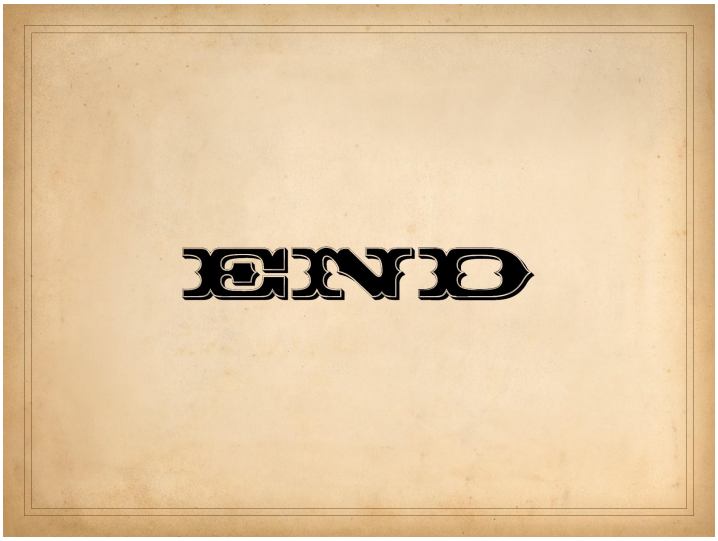
Debridement is an axiomatic and quintessential modality of wound care. It is necessary for two reasons. First is for patient safety. Primary debridement is done to eliminate infarcts, necrosis, abscess, debris, and active inflammation in a wound, factors which risk progressive injury and morbidity. Second is for effective results. The more thoroughly a wound is debrided, the more quickly it moves into the efferent reparative phase of wound biology, and the more certain it is to respond to the discretionary options chosen make the wound heal, be they pharmaceuticals, biologics, or surgery.

Debridement is based on one central principle - remove the bad and leave the good. For novices just learning to do this, time and experience will build your ability to recognize the difference, but that is truly all there is to the concept of debridement. By draining and debriding, you arrest or remove inflammation, necrosis, and active local disease, all of which promote that transition to the prolifertive phase.

This is another illustration from Pietro da Cortona, Tabula XVII, a view of the back and spinal nerves. This specimen stands proudly, showing off the healthy anatomy that survives under the layers that have been removed. Healthy anatomy and healthy wounds are the reward for those who have successfully and thoroughly debrided the debris, thereby restoring healthy wound biology.

This presentation and annotations are available for reading and perusal at arimedica.com.

Marc E. Gottlieb, MD, Phoenix, Arizona
Version 1, April 13, 2016.



22
END