Theoretic and Evidence-Based Laparotomy Closure with Sutures and Meshes

Lauren M. Mioton, MD
Gregory A. Dumanian, MD, FACS

Chicago, Ill.

Summary: The ultimate tensile strength of newly apposed tissue is the sum of the strength of the physical construct holding the tissues and the strength of biologic healing. For successful incisional hernia repair, the total strength of the repair must remain greater than the forces applied. While much work has been done to understand the science of wound healing—the ability of an open defect to contract and close—far less has been done to understand the healing of 2 newly approximated previously nonwounded surfaces held together with sutures or other implants. In this article, the elements of ultimate tensile strength on laparotomy closure as well as their progression over time will be related to clinical studies of abdominal wall closure. (*Plast. Reconstr. Surg.* 142: 117S, 2018.)

THEORETICAL ABDOMINAL WALL CLOSURE

Sutures

Sutures as a medical device are taken for granted. Based on a 3,000 year-old design from ancient Egypt,[1] sutures are flexible linear elements that approximate tissues and maintain tension typically with the aid of a self-holding knot or hooks located along the device. It is the interaction of the suture with tissues at the suture-tissue interface (STI) that permits the magic of surgical interventions. There is a chemical reaction, due to the composition of the suture and its degradation products. There is foreign body reaction, where the body walls off the suture as foreign in a microencapsulation process. Finally, there are sizeable tensile and extensile forces that play a defining role for outcomes. The force experienced by each suture is the total force required to appose the 2 edges, plus an additional amount of force that actually compresses the 2 edges together,[2] divided by the number of grabbing points or number of sutures that cross the apposition line (Figs. 1, 2). At each STI, the local tension is the force divided by the surface area of contact—a function of the diameter of the suture. Fine sutures concentrate forces at the STI, while larger sutures have the potential to distribute forces and lower the forces at the point of concentration. (**See Figure, Supplemental Digital Content 1**, which shows a finite element analysis of the STI, demonstrating that a flatter larger suture will distribute forces better than a suture with a symmetric and smaller cross-sectional appearance. *White* represents the suture. *Red* and *orange* represent a high force on the tissue, *green* is intermediate, and *blue* is low tension with little to no force from the STI experienced on the tissues, ***http://links.lww.com/PRS/C940***.) When the forces at the STI are greater than the tissue's ability to withstand the force, "suture pull-through" or "cheesewiring" occurs (Figs. 3, 4).[3] This failure can occur abruptly, leading to a dehiscence, or slowly, as will be discussed, leading to an incisional hernia.

From the Division of Plastic Surgery, Northwestern Feinberg School of Medicine.
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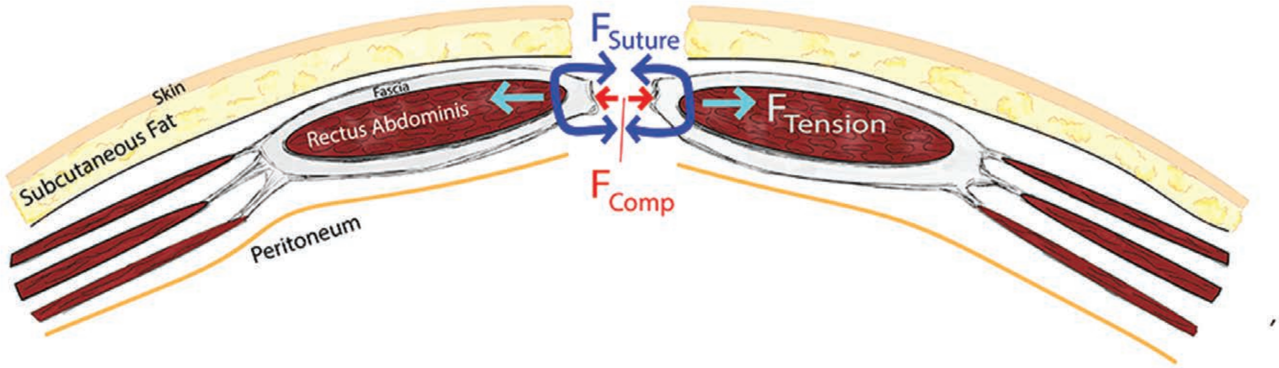

Fig. 1. Diagram of laparotomy closure and acting forces.

Suture adds an element of injury or reactive inflammation during the approximation of otherwise nonwounded tissues. Supporting the concept that stitches are the main inducer of scar is the finding that 5-hydroxyproline (correlating with scar strength) is greater around the suture than in the interstitch zone. Animal-based evidence has shown there is an optimal amount of tissue injury required for the initiation of healing.[4] Minimally wounded tissue edges and low suture tension are associated with lower burst strength than the approximation of gently wounded tissue edges with appropriately tied sutures. Tissues approximated by tightly tied sutures also have measurably less perfusion[5] and decreased burst strength, implying that the suture tension curve is parabolic with an ideal approximation force. The optimal approximation force is difficult to obtain, as it is challenging to judge the tension applied on to a suture, even for experts.[6]

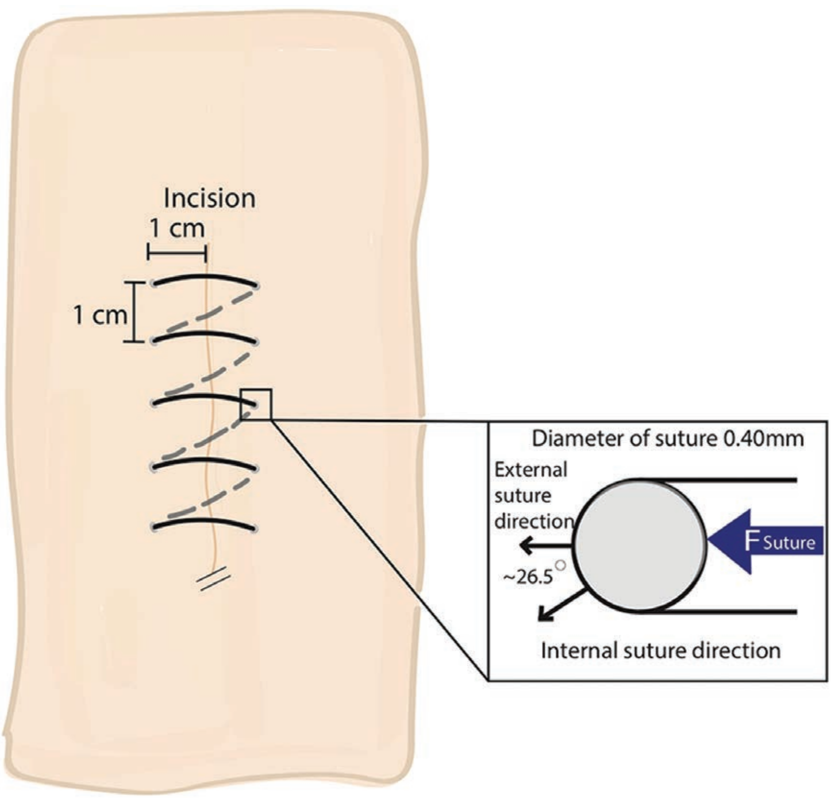

Fig. 2. Schematic of laparotomy closure. Total force of closure is divided by the number of gripping points by sutures to calculate the force experienced at each suture/tissue interface.

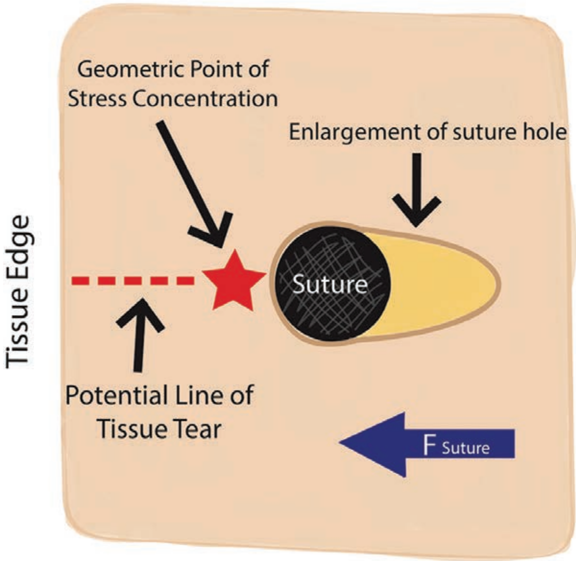

Fig. 3. Schematic of the forces causing suture pull-through.

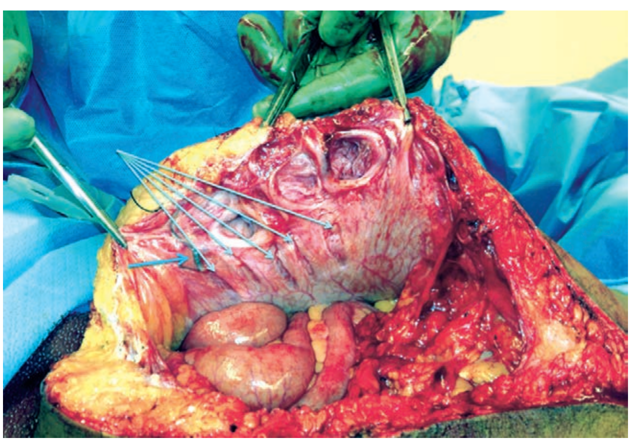

Fig. 4. Clinical example of laparotomy closure with running nylon suture. Green areas show multiple gashes of the abdominal wall. Single larger blue arrow shows the nylon suture.

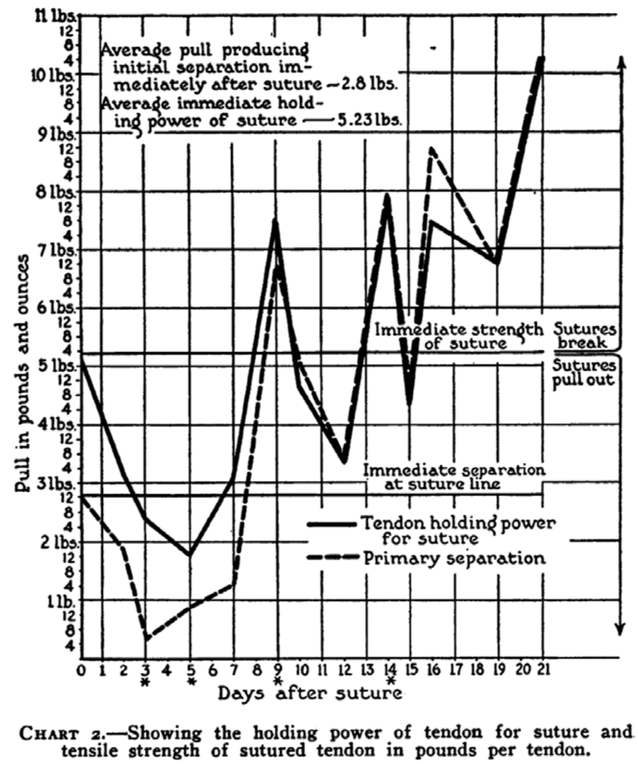

Fig. 5. UTS of newly repaired canine tendons. Reprinted from Mason ML, Allen HS. The rate of healing of tendons: An experimental study. *Ann Surg*. 1941;113:424–459.

Physical Construct Strength

The clinical surgeon can see changes to the tissues held under tension within an approximating loop of suture. A porcine laparotomy model revealed a 25% decrease in suture tension only 30 minutes after fascial closure, which progressed to a 50% decrease by 23 hours.[7] This is separate from the ubiquitous phenomenon of tissue creep and stress relaxation, whereby tissues placed under tension elongate over time (and serve to lower $F_{tension}$). Inflammation of tissue within the suture loop weakens the tissue either by the release of collagenases or by neutrophil-mediated mechanisms,[8] further facilitating the failure of the physical construct at the STI.[9] This initial weakening of the physical construct can be visualized and was first demonstrated in the initial downward strength curve of a repaired dog tendon published in 1941 by hand surgeons at Northwestern (Fig. 5).[10]

The total strength of the physical construct is a complex interplay of the ultimate tensile strength (UTS) of the suture, the number of "grabbing points" that the suture makes with the tissue, the ischemia caused by these sutures, the strength of the tissue, the ability of the collagen fibers to resist pull-through, and finally, the strength at the knot. Additional sutures placed along a suture line will increase construct strength, but at the expense of additional ischemia at the wound edge. Sutures tend to break at the knot rather than along the length, leading many surgeons to prefer running over interrupted closures. The US Pharmacopeia requires a single knot to be placed along the suture during the determination of ex vivo suture breaking strength. Suture size is based on a minimum standard by the US Pharmacopeia (rather than size or diameter), with a 2-0 suture requiring a UTS of 26.3 N, and an 0-suture having a UTS of 38.2 N.[11]

When the strength of the physical construct added to the strength of biologic healing remains greater than the forces applied along the length of the closure at all times, the repair will remain intact. Forces greater than the strength of the repair will cause a dehiscence. A middle ground exists for episodic forces applied onto the abdominal wall that cause tearing and weakening, but without frank rupture. Termed "gap formation" in orthopedics, the separation of a repair due to tension foretells a poor outcome. In hand surgery, gap formation of 2 mm is associated with tendon ruptures and scarring.[12] Similarly, 12 mm of gapping of a laparotomy closure over the first 30 days after repair is associated with a 94% chance of incisional hernia formation.[13] By 4 months,[14] the physical construct strength will reach a relatively steady-state characterized by scar formation and collagen remodeling. As such, many surgeons feel that a slowly absorbing suture that retains its tensile strength until this time point is optimal. Permanent sutures will be encapsulated in the foreign body reaction, leaving a certain amount of local scar at the zone of implantation.[15] In contradistinction, absorbable sutures initially create a foreign body reaction–induced scar, but this scar is thought to resorb when the foreign body dissolves.[16] As each absorbable suture has a different degradation profile, suture selection depends on the knowledge of how long the newly closed wound will require support by the physical construct.[17] The long-term strength of a laparotomy closure using absorbable sutures is approximately 70% of the unscarred linea alba.[18] This dovetails with many studies of scar strength.[19] Although this may suffice in the majority of individuals, the scar can remodel and weaken due to the underlying forces applied, causing an incisional hernia to form. Unlike normal unscarred tissues that follow Davis's law by tending to enlarge or hypertrophy with exercise and added use,[20] scar does not have pulsatile blood flow, and it deforms rather than gains strength. The weakening of the construct strength due to either suture pull-through or the suture absorbing perhaps explains the nearly identical incisional hernia rate when absorbable and nonabsorbable sutures are compared.[21]

Meshes

Meshes have clearly been shown to improve outcomes after incisional hernia repair,[22] though the mechanisms by which this is achieved are less well conceptualized. In a so-called "tension-free" repair, the hernia defect is left as an empty space or hole, and the mesh is sewn edge-to-edge to the abdominal wall. The quality of the repair is a complex interchange involving the strength of the adherence of the mesh to the abdominal wall edges (physical construct), the ability of the mesh to resist bursting, and the capacity of the body to envelop the mesh fibers in the foreign body response. The tension-free hernia repair is truly a misnomer, as there is always tension on the mesh with coughing, the Valsalva maneuver, and even respiration. However, the increased sarcomere overlap from the abdominal muscles not being brought back to their original resting length causes a decreased ability to create a strong contracture. Tension-free placements of mesh commit the patient to being on the lower end of the Starling muscle/force curve. Perhaps it is the decreased forces applied onto the mesh that allows these mesh constructs to remain intact over time.

Far better than the bridged "tension-free" repair is the "direct supported" repair, where the mesh is used to augment the strength of the approximated abdominal wall. Clinically, bridged repairs have far higher hernia recurrence rates than direct supported repairs due to the latter having a higher initial construct strength and a better distribution of forces to limit suture pull-through.[23] Analogous to the strength of sutured tissue described above, the UTS will be the strength of the physical construct added to the strength of biologic healing. The strength of the construct is based on the quality of mesh fixation to the tissues. Here, there is great variability in published techniques in terms of mesh type (biologic versus synthetic), mesh size, mesh location, and mesh fixation. Retrorectus placed narrow meshes can be held with transfascial sutures. The physical construct is quite effective at medializing the borders of the rectus muscle, so that additional sutures used to approximate the midline are under less tension, avoiding suture pull-through and failure.[24] An alternative strategy is to use large unfixed meshes from mid-axillary line to mid-axillary line in the plane behind the rectus muscle and the tranversus abdominis after division of the anterior-most fibers of the latter muscle (the transversus abdominis release).[25] Few if any sutures are used to hold the mesh in place, so the physical construct relies on friction between the mesh and the tissues initially. Quickly, the mesh is incorporated to provide an increased UTS for the closure. A third alternative between numerous transfascial sutures and large unfixed meshes is self-gripping mesh that increases the friction between the material and tissues with micro-hooks.[26] Meshes placed on the anterior surface of the abdominal wall must be secured either with sutures to the anterior fascia or with surgical glue to distribute forces for increased UTS, to avoid postoperative pain potentially associated with transfascial suture fixation, and to facilitate rapid incorporation.[27]

Biologic Healing Strength

Factors that aid in the rise in biologic strength have been intensively researched.[28,29] A more rapid trajectory in healing would serve to keep the UTS of the abdominal wall higher than the forces applied and to prevent gapping more than 12 mm at the midline laparotomy closure. Factors associated with poor wound healing shift the biologic healing curve rightward and include any interruptions of the normal wound-healing cascade of inflammation and fibroblast deposition of collagen (Fig. 6). A current focus in hernia repair is patient optimization to maximize the trajectory of biologic healing strength.[29,30] This includes smoking cessation, tight glucose control, nutritional stability with an albumin over 3.0 mg/dl or prealbumin over 15 mg/dl, proper vitamin repletion, and the avoidance of surgery on elderly patients or patients taking steroids or immunosuppressants such as sirolimus. Surgery sites should be without infection, tissue necrosis, ischemia, foreign bodies, neoplasia, and radiation. Core body temperature should be maintained for optimal initiation of healing.[31]

While much has been attributed to slowing the biologic healing trajectory, less has been described on the augmentation of biologic healing. Provision of exogenous cytokines (platelet-derived growth factor, basic fibroblast growth factor, and transforming growth factor beta) and stem cells to the healing site is not practical, but is theoretically possible. Prewounding tissue akin to a surgical delay does upregulate these factors and essentially ignites the inflammatory cascade in advance of the laparotomy closure. A new strategy that has been shown to improve early wound strength is a novel mesh suture design.[32] The mesh suture is made of multiple small filaments braided and bonded to each other to equal the strength of a larger monofilament suture. The surface area of the 0 mesh suture is almost 8 times that of the surface area of standard 0 monofilament suture, perhaps augmenting the foreign body reaction or providing a scaffold for tissue integration.[16] As these sutures are 10 times larger in diameter than standard sutures, there would also be a physical resistance to suture pull-through. Mesh sutures are mostly air, and so the filaments collapse at the knot to become far lower profile than a similarly sized solid suture. There may be an improved biologic tissue response of small diameter filaments as compared with the use of larger filaments for laparotomy closure.[33] Similar strategies that attempt to augment the natural tendency of humans to form scar and enhance biologic healing include the application of nanofibers made from polyesters[34] and microparticles of silver.[35]

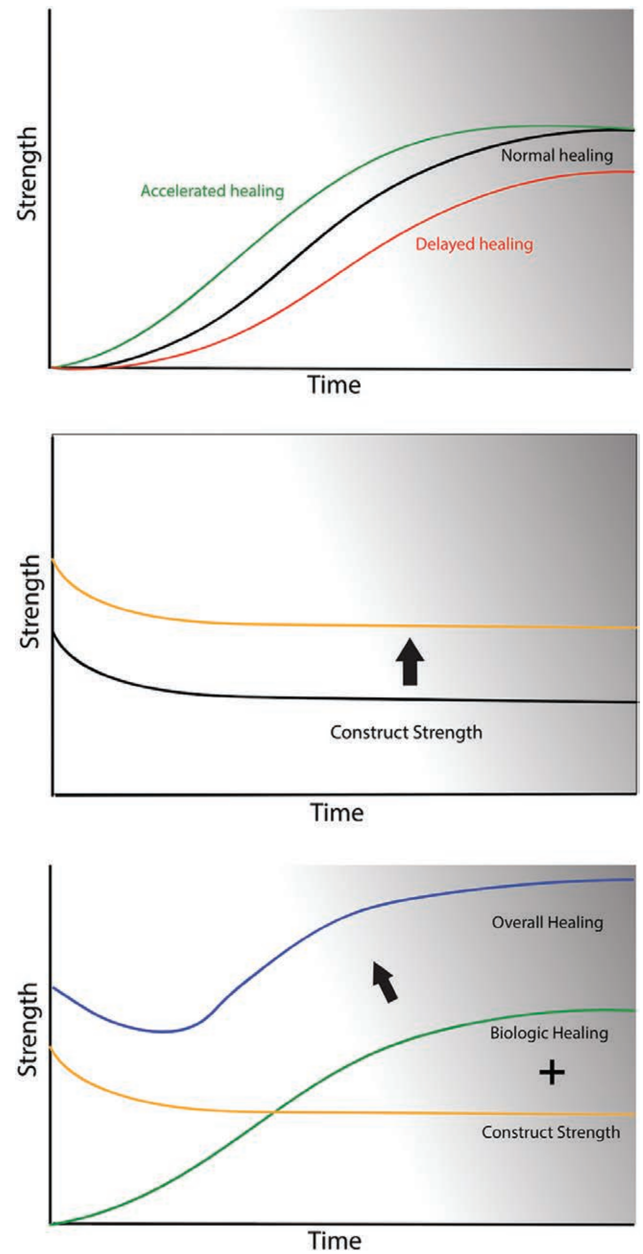

Fig. 6. Graphical representation of expedited biologic healing denoted in *green* and delayed healing denoted in *red* (*above*), improved physical construct strength (*center*), and components of overall strength of repair over time (*below*).

CLINICAL ABDOMINAL WALL CLOSURE

Translating theoretical concepts on abdominal wall closure into clinical practice has led to further nuances in surgical approach and materials. However, all these clinical trials referenced below begin with good surgical technique. As written in a recent editorial,

> "Preserving the integrity of fascial edges along with sparing of the rectus muscle is a key element of successful midline closure, whether performed with running sutures, slings, meshes or, perhaps in future, mesh sutures. To preserve fascial edges it is essential to identify and create a clean margin zone of some 2–4 cm along the linea alba. Blind stitches through subcutaneous fat or attempts to grasp recessed fascial edges with the needle inevitably lead to weak spots along the suture line. Preparation of the fascia is not time-consuming and should be seen as an essential step in all closure techniques."[36]

Very good evidence exists from the STITCH trial that, for thin patients [average body mass index (BMI), 24] undergoing midline laparotomy, small bites 5 mm from the edge of the incised linea alba and spaced 5 mm from each other using a relatively small 2-0 polydioxanone suture resulted in fewer incisional hernias than the traditional 1 cm bites spaced 1 cm from each other with a number 1 polydioxanone suture.[37] A relatively high rate of incisional hernias was found in both arms of the study (small bites 13%, large bites 24%), though this was attributed to radiologic identification of small abdominal defects with routine ultrasound. A 2-0 polydioxanone suture sounds too small for closure of patients with higher body mass indices, yet this particular suture has a high modulus of elasticity and a higher breaking strength than an 0-polypropylene suture (internal data, not published). This study builds on prior clinical work that the working length of a running suture used to close the abdominal wall should be over 4 times the length of the incision it closes for the lowest hernia rate.[38] It should be noted that the small bite closures used more suture than the large bite closures—serving to better distribute forces and to increase the area of the suture/tissue interface. The small bite closures had double the gripping points to hold the tissue in apposition in comparison to large bites, causing the force at the STI to be halved. However, the 2-0 suture has a smaller diameter than a number 1 (0.35 mm versus 0.5 mm), that would serve to increase the force locally and increase the tendency to cheesewire, as shown by finite element analysis (**see Figure, Supplemental Digital Content 1, *http://links.lww.com/PRS/C940***). Overall, the increase in the construct strength achieved by the 2-0 small bite closure clinically was an improvement over the large bite number 1 closure. Suture tension was not measured or controlled in this study—rather, surgeon technique would be balanced by the randomized nature of the study protocol. This study, though expertly performed, has not gained wide adoption due to the low BMI of the study patients and the concern that greater forces at the STI from obesity may alter outcomes.

Other than alteration of sewing technique with small bites, a recent well-done meta-analysis shows no differences between laparotomy closures with interrupted versus running sutures, and no differences of outcomes with suture composition (slow absorbable versus fast absorbable versus permanent). The European Hernia Society has established guidelines for running laparotomy closures to save time, and slow absorbable sutures to avoid delayed stitch abscesses.[39] Perhaps reliance on the simple linearly designed suture for wound closure is what is truly limiting. To that end, a completely new tissue approximation technique has been developed and used clinically over the last several years. Termed a "mesh sutured closure," strips of macroporous mesh 2-cm wide are passed on either side of the abdominal wall and tied like a suture.[40] The large size of the mesh strips resists pull-through, while the heightened total surface area of the device filaments encourages a local deposition of collagenous scar to achieve a durable closure. Clinically, despite using a permanent material, these mesh sutures resist bacterial colonization and have been used repeatedly to approximate large contaminated hernias with a 1-year hernia recurrence rate under 10%.[33] Tissue relaxation techniques including open anterior components release are helpful to reduce $F_{tension}$ and cheesewiring.

Recent studies including the PRIMA trial have moved toward using both sutures and mesh to achieve a lasting abdominal wall closure in higher risk patients—defined as those with a BMI over 27, or undergoing open abdominal aortic aneurysm repair.[41,42] Termed primary mesh augmentation (PMA), the mesh is placed prophylactically to distribute forces and to limit tearing at the STI. The mesh serves to add additional foreign body reaction scar around the filaments of the mesh. Risk of seroma formation is greater in PMA compared with primary suture repair, in particular with onlay placement (18.1%) compared with primary suture

(4.7%) and sublay mesh placement (7%). It makes sense that there is a cost for achieving a stronger physical construct in terms of the soft-tissue elevation required to place the mesh. Importantly, the higher seroma rates did not translate into higher rates of surgical-site infection, dehiscence, or reoperation. Another criticism is that mesh fixation with tissue adhesives would yield a smaller increase in physical construct strength relative to transfascial suturing. Glues require tissue incorporation and the foreign body response, which take time to occur. Fully 5% of the meshes were removed during the clinical care of these patients, and this must be balanced against the reduction of incisional hernias from 30% (sutures only) to 13–18% (PMA groups). Similarly, the AIDA trial using a prophylactic onlay mesh at the time of abdominal aortic aneurysm repair decreased the 2-year incisional hernia rate from 23% to 4%, at the cost of early problematic seromas.[43]

CONCLUSIONS

The midline laparotomy incision is a model for all types of internal high-tension closures. Advances in understanding will lead to improvements for all fields of surgery that still rely on the simple suture to approximate tissues.

Gregory A. Dumanian, MD, FACS
Northwestern Feinberg School of Medicine
Division of Plastic Surgery
675 N. St. Clair, Suite 250
Chicago, IL 60611-4807
gdumania@nm.org

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