INTRODUCTION
Approximately 1 million abdominal wall hernia repairs are performed in the United States each year [1]. Of these operations, roughly 69% are groin, 15% umbilical, 9% incisional, and 7% are miscellaneous, (eg, Spigelian, lumbar, traumatic). The term ventral is most commonly used to describe collectively those hernias that occur outside the groin and is the convention that is used in this article. It is estimated that about 5% of the population develop a ventral hernia at some time in their life. Most hernias are small and can be managed in general practice using standard techniques. However, there is a subset of these patients who develop enormous hernia sacs. These hernias are usually described as giant ventral hernias. Other synonyms used in the literature for giant ventral hernias are component separation, loss of domain, and incisional hernia.

Key points
• Repair of huge ventral hernias is technically challenging for the surgeon and a major operation for the patient and should be performed by experienced surgeons in centers that are used to caring for patients who are commonly massively obese, with significant comorbidities.
• Preoperative medical optimization of patients is an important part in the overall management of these large hernias.
• Conventional component separation with retromuscular mesh repair is the workhorse operation, which successfully deals with many giant ventral hernias, but multiple alternative strategies must be available to address situations in which myofascial elements are completely deficient or there is significant loss of domain.
• The complexity of this surgery is reflected by recurrence rates ranging from 10% to 30% and wound complication rates as high as 40% to 50% in experienced centers.
hernia are large, massive, huge, or complex ventral hernia. Sometimes, the whole visceral contents herniate into the hernia sac outside the abdominal cavity (Fig. 1). These hernias are usually associated with loss of domain, because the abdominal cavity proper shrinks with the visceral contents lying chronically outside it. If an attempt is made to force these extra-abdominal viscera into the native abdominal cavity, there can be severe respiratory and cardiovascular compromise, causing the abdominal compartment syndrome.

Repair of huge ventral hernias with significant loss of domain is technically challenging, with high mortality, morbidity, and recurrences rates [2,3]. Moreover, these hernias are often associated with overlying skin ulceration, persistent infection, enterocutaneous fistulas, diverting stomas, and morbid obesity. Many of these hernias have been repaired multiple times, and each repair has failed, distorting the anatomy of the abdominal wall further. This article focuses on the management of these complex huge ventral hernias and the newer advances in abdominal wall reconstructions.

**SURGICAL ANATOMY**

The root cause of a ventral hernia is failure of key anatomic elements to contain intra-abdominal viscera within the abdominal cavity. Repair of ventral hernias requires a thorough understanding of the anatomy of the abdominal wall [4]. The abdominal wall is made up of the centrally located rectus abdominis muscles and the 3 lateral muscles: the external oblique, internal oblique, and transversus abdominis. The linea alba is the midline confluence of the aponeuoses of these muscles.

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**Fig. 1.** (A, B) Computed tomography scan (axial and sagittal view) of a patient with giant ventral hernia. Note that a significant amount of the intra-abdominal viscera including the stomach is in the hernia.
The external abdominal oblique muscle (Fig. 2) is the most superficial of the 3 lateral abdominal muscles. The external abdominal oblique arises from the posterior aspects of the lower 8 ribs and interdigitates with both the serratus anterior and the latissimus dorsi at its origin. The direction of the muscle fibers varies from nearly horizontal in its upper portion to oblique in the middle and lower portions. The horizontal fibers, which originate posteriorly, insert onto the anterior portion of the iliac crest. The obliquely arranged anteroinferior fibers of insertion fold on to themselves to form the inguinal ligament. The remaining portion of the aponeurosis inserts into the linea alba after contributing to the anterior portion of the rectus abdominis sheath. The middle layer of the lateral abdominal group is the internal abdominal oblique muscle (see Fig. 2). This muscle primarily arises from the iliac fascia along the iliac crest and forms a band of iliac fascia fused with the inguinal ligament. The uppermost fibers course obliquely toward the distal ends of the lower 3 or 4 (floating) ribs. The muscle fibers of the internal oblique fan out, following the shape of the iliac crest, so that the lowermost fibers are directed inferiorly. The aponeurosis of the internal oblique (Fig. 3A) above the level of the umbilicus splits to envelop the rectus abdominis, then reforms in the midline to join and interweave with the fibers of the linea alba. Below the level of the umbilicus (see Fig. 3B), the aponeurosis does not split but rather runs anterior to the rectus muscle, continues medially as a single sheet, joins the anterior rectus sheath, and contributes to the linea alba. The aponeurotic portion of the internal oblique is widest at the level of the umbilicus. The transversus abdominis muscle arises from the fascia along the iliac crest and inguinal ligament and from the lower 6 costal cartilages and ribs, where it interdigitates with the lateral diaphragmatic fibers. The muscle bundles of the transversus abdominis for the most part run horizontally. However, the lower medial fibers may continue in a more inferomedial course toward the site of insertion on the crest and pecten of the pubis. The aponeurosis of the transversus abdominis joins the posterior lamina of the internal abdominal oblique, forming above the umbilicus a portion of the posterior rectus sheath. Below the umbilicus, the transversus abdominis aponeurosis is a component of the anterior rectus sheath. The gradual termination of aponeurotic tissue on the posterior aspect of the rectus abdominis forms the arcuate line of Douglas.

The rectus abdominis forms the central and anchoring muscle mass of the anterior abdomen. The rectus muscle arises from the fifth to the seventh costal cartilages and inserts on to the pubic symphysis and pubic crest. Each rectus muscle is segmented by tendinous intersections at the levels of the xiphoid process and the umbilicus and at a point midway between these 2. The lateral edge of the muscle is demarcated by a slight depression in the aponeurotic fibers, corresponding to the lateral edge of the rectus muscle; this depression is the semilunar line. It marks the site of initial lateral insertion of the aponeurotic tendons of the lateral abdominal muscles.

The composition of the rectus sheath varies depending on axial level of the abdominal wall [5]. The anterior sheath superior to the umbilicus is composed
Fig. 2. Abdominal wall musculature (A) Superficial layers (B) Deep layers.
of the aponeurosis of the external abdominal oblique and the anterior lamina of the internal abdominal oblique. The transversalis aponeurosis does not participate in the formation of the anterior sheath at this level. The posterior sheath of the rectus muscle superior to the umbilicus is contributed to by the internal abdominal oblique and the transversus abdominis aponeurosis. The external abdominal oblique does not participate in the formation of the posterior portion of the rectus sheath. At a highly variable site inferior to the umbilicus, all the aponeurotic tendons pass anteriorly to form the anterior rectus sheath. The fibers of the posterior sheath are seen to attenuate gradually. This transfer of connective tissue away from the posterior rectus sheath causes the arcuate line of Douglas to form on the posterior surface of the muscle. The tissue covering the deep surface of the rectus muscle inferior to the arcuate line is primarily the transversalis fascia [6]. This anatomic layer is most significant to the surgeon performing a retrorectus hernia repair.

The innervation of the anterior abdominal wall muscles is multiple. The lower intercostal and upper lumbar nerves (T7–T12, L1, L2) contribute most of the innervation to the lateral muscles and the rectus abdominis. The nerves pass anteriorly in a plane between the internal oblique and the transversus abdominis, eventually piercing the lateral aspect of the rectus sheath to innervate the muscle. The external oblique muscle receives branches of the intercostal nerves, which penetrate the internal oblique. The blood supply of the lateral muscles of the anterior wall is primarily from the lower 3 or 4 intercostal arteries, the deep circumflex iliac artery, and the lumbar arteries. The rectus abdominis has a complicated blood supply derived from the superior epigastric artery (a terminal branch of the internal mammary artery), the inferior epigastric artery (a branch of the external iliac artery), and the lower intercostal arteries. The latter arteries enter the sides of the muscle after traveling between the oblique muscles. The superior and inferior epigastric arteries enter the rectus sheath and anastomose near the umbilicus.
DEFINITION OF GIANT VENTRAL HERNIA

There is no consistent or standardized definition of what represents a giant ventral hernia. In the past, ventral hernias have been categorized as large based on the transverse diameter of the defect. However, the critical importance of the definition of a giant ventral hernia is to determine inoperability, so that surgeons can avoid the potentially fatal scenario of being in the operating room with an open abdomen and no options to return the viscera to the abdominal cavity with skin coverage without causing intra-abdominal hypertension. The area of the hernial orifice in relationship to intra-abdominal and hernia sac volumes is more important in determining the severity and complexity of the hernia. Unique to the consideration of giant ventral hernia is the concept of loss of domain. These huge hernias with enormous hernia sacs tend to retain most of the abdominal viscera for prolonged periods. Lack of viscera in the abdominal cavity causes a decrease in abdominal wall muscle elasticity, abdominal wall muscular atrophy, and reduced volume because of disuse.

Decreased intra-abdominal pressure alters the ventilatory equilibrium in these patients, and diaphragmatic descent is often seen. Impaired venous and lymphatic return from long-standing prolapse of the intestines causes edematous thickened bowel, which is difficult to reduce back. In these conditions, forceful replacement of the viscera in the abdominal cavity may prove life-threatening because of development of abdominal compartment syndrome. To objectively quantitate the loss of domain, investigators have tried to calculate the volume of the giant hernia sacs and its ratio with the abdominal cavity volume using multidetector computed tomography scanning. However, these methods need to be validated before widespread application [7].

CAUSE

There are multiple factors that play a role in the development of incisional hernias. Technical flaws at the index operation, including slippage of knots, breakage of sutures, tearing of fascia by sutures, rough handling of tissues, closure of the abdomen under tension, and poor choice of suture material, result in incisional hernias [8]. In addition, several host factors play a role, listed in Box 1 [9,10]. An association between a patient’s comorbidities and the incidence of incisional hernia is well known. Genetic factors also play a role. An increased incidence of ventral hernia is noted in certain connective tissue disorders, for example, osteogenesis imperfecta, Marfan syndrome, and Ehlers-Danlos syndrome. Collagen imbalance (type 1–type 3), abnormal matrix metalloproteinase expression, and low growth factor levels are some molecular defects that may be linked to the cause of ventral hernia. A simple incisional hernia may continue to enlarge into a huge complex hernia, reaching a point at which it contains almost all of the abdominal visceral contents. The computed tomographic image shown in Fig. 1 highlights this. Previous unsuccessful attempts at repair of an incisional hernia weaken the abdominal musculature and result in a higher risk of recurrence [11]. Huge ventral hernias may develop after
extensive resection of the abdominal wall for tumors or massive trauma to the abdominal wall. Intra-abdominal catastrophe requiring serial abdominal washouts and closure achieved by secondary intention or with the aid of split-thickness skin grafts can also result in massive abdominal wall defects.

**CLASSIFICATION**

To make meaningful decisions about the appropriate management of an incisional hernia, the hernia has to be classified. A classification system developed by experts from the European Hernia Society is shown in Table 1 [6]. Important factors from which ventral hernias are classified are location of hernia, size of defect, reducibility, symptoms, presence or absence of obstruction, and recurrent nature of the hernia. Table 2 shows the Zollinger classification of ventral hernias, which is based on the cause of hernia. Ventral hernias have also been graded depending on the wound status and host factors [12]. Grade 1 (low risk) patients have no previous history of wound infection or comorbidities. Grade 2 patients have comorbidities that predispose to infection but no evidence of ongoing wound infection. These comorbidities include age, smoking history, steroid use, malnutrition, and diabetes mellitus. Grade 3 patients’ wounds are contaminated and grade patients 4 have active infection. We recommend customizing the hernia repair based on the grade of hernia.

**Box 1: Host factors associated with incisional hernia**

- Male sex
- Old age
- Morbid obesity
- Abdominal distension
- Cigarette smoking
- Pulmonary disease
- Mechanical ventilation
- Type 2 diabetes mellitus
- Oral anticoagulants
- Malnourishment
- Hypoalbuminemia
- Anemia/transfusion
- Malignancy
- Jaundice
- Corticosteroid therapy
- Chemotherapy
- Radiotherapy
- Renal failure
CLINICAL PRESENTATION

Most incisional hernias, particularly when reducible, are asymptomatic or cause minimal symptoms, with pain or discomfort when lifting heavy objects, coughing, or standing for long periods. On the other hand, patients with huge ventral hernias (as shown in a clinical image in Fig. 4) can be incapacitated by their hernia because of the presence of a huge pannus, which, in addition to being unsightly, is painful, restricts mobility, commonly causes back problems, and affects respiratory mechanics, with dramatic effects on the functional status and quality of life. These hernias are almost never reducible, not because of the size of the abdominal wall defect but because there has been loss of domain. Some patients may present with a bowel obstruction because the neck of the large hernia sac is very narrow or when there is a Swiss cheese fascial defect pattern. Unique to giant incisional hernias is the patient who has a bowel obstruction caused by factors exclusively within the sac, such as a volvulus or adhesions to the unprotected extra-abdominal bowel caused by inflammation. In this situation, patients can go onto abscess formation or even gangrene and not seem as toxic as patients with more classic intra-abdominal bowel obstructions, presumably because the process becomes relatively isolated from the abdominal cavity proper.

Table 1
Classification system for incisional hernias

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Categories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Vertical</td>
</tr>
<tr>
<td></td>
<td>Midline, above or below umbilicus</td>
</tr>
<tr>
<td></td>
<td>Midline, including umbilicus</td>
</tr>
<tr>
<td></td>
<td>Paramedian</td>
</tr>
<tr>
<td></td>
<td>Transverse</td>
</tr>
<tr>
<td></td>
<td>Above or below umbilicus</td>
</tr>
<tr>
<td></td>
<td>Crosses midline</td>
</tr>
<tr>
<td></td>
<td>Oblique</td>
</tr>
<tr>
<td></td>
<td>Above or below umbilicus</td>
</tr>
<tr>
<td></td>
<td>Combined</td>
</tr>
<tr>
<td>Size (cm)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>&lt;5</td>
</tr>
<tr>
<td></td>
<td>5–10</td>
</tr>
<tr>
<td></td>
<td>&gt;10</td>
</tr>
<tr>
<td>Recurrence</td>
<td>Primary</td>
</tr>
<tr>
<td></td>
<td>Multiply recurrent</td>
</tr>
<tr>
<td></td>
<td>Stratification for type of previous repair</td>
</tr>
<tr>
<td>Reducibility</td>
<td>Yes</td>
</tr>
<tr>
<td></td>
<td>Obstruction</td>
</tr>
<tr>
<td></td>
<td>No obstruction</td>
</tr>
<tr>
<td></td>
<td>No</td>
</tr>
<tr>
<td></td>
<td>Obstruction</td>
</tr>
<tr>
<td></td>
<td>No obstruction</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td></td>
<td>Symptomatic</td>
</tr>
</tbody>
</table>

<sup>a</sup>Difficult to measure consistently.
Table 2
Zollinger classification system for ventral abdominal wall hernias

<table>
<thead>
<tr>
<th>Type</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital</td>
<td>Omphalocele</td>
</tr>
<tr>
<td></td>
<td>Gastroschisis</td>
</tr>
<tr>
<td></td>
<td>Umbilical (infant)</td>
</tr>
<tr>
<td>Acquired</td>
<td>Midline</td>
</tr>
<tr>
<td></td>
<td>Diastasis recti</td>
</tr>
<tr>
<td></td>
<td>Epigastric</td>
</tr>
<tr>
<td></td>
<td>Umbilical (adult, acquired, paraumbilical)</td>
</tr>
<tr>
<td></td>
<td>Median</td>
</tr>
<tr>
<td></td>
<td>Supravesical (anterior, posterior, lateral)</td>
</tr>
<tr>
<td></td>
<td>Paramedian</td>
</tr>
<tr>
<td></td>
<td>Spigelian</td>
</tr>
<tr>
<td></td>
<td>Interparietal</td>
</tr>
<tr>
<td>Incisional</td>
<td>Midline</td>
</tr>
<tr>
<td></td>
<td>Paramedian</td>
</tr>
<tr>
<td></td>
<td>Transverse</td>
</tr>
<tr>
<td></td>
<td>Special operative sites</td>
</tr>
<tr>
<td>Traumatic</td>
<td>Penetrating, autopenetrating(^a)</td>
</tr>
<tr>
<td></td>
<td>Blunt</td>
</tr>
<tr>
<td></td>
<td>Focal, minimal injury</td>
</tr>
<tr>
<td></td>
<td>Moderate injury</td>
</tr>
<tr>
<td></td>
<td>Extensive force or shear</td>
</tr>
<tr>
<td></td>
<td>Destructive</td>
</tr>
</tbody>
</table>

\(^a\)Penetration from host tissue such as bone.

Fig. 4. (A, B) Clinical image of a patient with giant ventral hernia with significant loss of domain.
PREOPERATIVE WORKUP

Morbid obesity is a rule rather than exception in patients with giant incisional hernias. Therefore, a comprehensive abdominal wall reconstruction program should include a bariatric arm. A body mass index (BMI) of less than 30 is ideal before repairing the hernia. Incapacitating symptoms or signs of impending bowel obstruction commonly make a prolonged waiting period for weight loss impossible. Because compliance is so poor, bariatric surgery has to be a consideration, but most third-party payers require a minimum of 1 year of supervised weight loss before approving this. Nevertheless, patients need to be thoroughly counseled that the chance of a successful hernia repair is inversely proportional to their BMI and that weight reduction should be the first step.

Before scheduling the patient for surgery, it is vital that any abdominal wall infection (ie, ulceration, open wounds, sinuses, enterocutaneous fistulas, and so forth) must be controlled. This control may entail performing multiple drainage procedures, removal of previous infected prostheses, serial debridements, appropriate wound care, and antibiotics. Because of the large pannus, intertriginous skin problems especially caused by yeast, Staphylococcus species and Corynebacterium are common and should be treated topically. The complication rate for ventral hernia repair in general is greatly increased in the presence of both clean-contaminated and contaminated cases [13].

All patients with huge ventral hernia must have a computed tomography scan of the abdomen and pelvis to assess the status of the abdominal wall musculature, the defect size, volume of the hernia sac, and also to exclude incidental unsuspected conditions. Some authorities prefer ultrasonography for estimation of the size of the defect [14].

Cardiovascular assessment

Repair of huge ventral hernias is an extensive operation, with a significant operative mortality because of cardiorespiratory complications and pulmonary embolism. The patient experiences a prolonged anesthetic and massive fluid shifts because of third spacing, which continues in the postoperative period. Surgeons are often faced with patients who, despite significant operative risk because of the magnitude of the hernia as well as associated comorbidities, are insistent on having the hernia repaired because their quality of life is so poor that they would rather die than have to continue to live the way they are. Patients should be informed about the potential need for postoperative ventilation, renal failure from intra-abdominal hypertension, and morbidity from wound-related complications. Because most of these operations are performed electively, the cardiopulmonary status of the patient can be thoroughly assessed and medically optimized. Almost all patients should at the minimum undergo pulmonary function studies and stress testing for dynamic cardiac function. Appropriate deep venous thrombosis (DVT) prophylaxis is critical and may even require the placement of a vena cava filter in certain high-risk patients.
A discussion about the repair of a giant incisional hernia is not complete without addressing prosthetic materials. This situation is because prosthesis of some type is always used, except in extenuating circumstances (eg, extensive infection or contamination, allergies, patient refuses foreign material). Use of prosthesis provides a scaffold for ingrowth of fibrous tissue and strengthens the weak abdominal wall. This situation significantly reduces the recurrence rate after incisional hernia repair [15]. Studies have shown that primary suture repair of ventral hernia is associated with a recurrence rate as high as 25% to 55% [3,14]. In a randomized, controlled study from the Netherlands, even small incisional hernias (defects less than 10 cm²) had a recurrence rate of 67% when primary suture repair was used [16]. In a systematic review published on incisional hernia repairs, recurrence rates ranged from 31% to 63% for direct suture repairs and from 0% to 32% (mostly less than 10%) for prosthetic repairs [17].

In recent years, numerous prosthetic materials have been introduced into the market, making it confusing to decide which should be used. To simplify this situation, the materials can be divided into several classes (ie, noncomposite heavyweight plastic, noncomposite heavyweight membrane, noncomposite lightweight plastic, composite prosthesis, coated prosthesis, absorbable, and biologic). Representative lists of products in each of these categories can be found in Boxes 2 and 3. So how does one decide? Most authorities agree that a synthetic material is preferred over a biologic in the absence of infection or contamination. The choices are between polypropylene (PPL), polyester (PE) or expanded polytetrafluoroethylene (ePTFE). PPL and PE have similar properties and can be used interchangeably. North American surgeons tend to prefer PPL, because they believe that it performs better when considering infection (ie, it less likely to get infected and less likely to have to be explanted when infected), whereas in Europe, PE is more popular, because surgeons there believe that PE has better handling characteristics. ePTFE is commonly referred to as mesh, but it acts more like a membrane. By definition, a mesh has grossly visible interstices (macropores), which allow ingrowth of scar tissue, whereas the pores in ePTFE cannot be seen except microscopically (micropores) and are so small that ingrowth is minimal; therefore, when implanted, the material is encapsulated rather than incorporated, as with a true mesh. The micropores in ePTFE are not so small that they can prevent bacterial penetration and this fact along with the lack of incorporation is believed to be the reason why it is more susceptible to infection and is almost never salvageable when it becomes infected. On the other hand, there is a good side to this lack of incorporation. When placed in contact with intra-abdominal viscera, it does not have a tendency to erode, and fistula formation is almost unheard of. ePTFE is therefore the gold standard material during abdominal wall reconstruction if the prosthesis cannot be isolated from the abdominal cavity and is in contact with the viscera. PPL and PE meshes are contraindicated in this circumstance, because of an unacceptably high incidence or erosion and fistula formation.
**Box 2: Commercially available synthetic prostheses for an abdominal wall hernia repair**

**Noncomposite Heavyweight Plastic Meshes**
- Prolene (Ethicon) PPL
- Marlex (Bard) PPL
- SurgiPro (Covidien) PPL
- Parietex (Covidien) PE
- Mersilene (Ethicon) PE

**Noncomposite Heavyweight Membranes**
- Gore-Tex (W L Gore) polyfluorotetraethylene (ePTFE)
- MotifMesh (Proxy Biomedical)
- Dual Mesh (W L Gore) ePTFE, one side roughened
- Dulex (Bard/Davol) ePTFE, one side roughened

**Noncomposite Lightweight Plastic Meshes**
- Ultrapro (Ethicon) PP + poliglecaprone-25
- Vipro II (Ethicon) PP + polyglactic acid
- ProLite (Atrium) PPL

**Composite Prosthesis (ePTFE + Plastic Mesh)**
- ePTFE + heavyweight plastic mesh
  - Composix EX (Bard/Davol) ePTFE + heavyweight PPL
  - Bard Kugel Hernia Patch (Bard/Davol)
  - Ventralex (Bard/Davol)
- ePTFE + lightweight plastic mesh
  - Composix LP (Bard/Davol) ePTFE + lightweight PPL

**Coated Prosthesis**
- PPL mesh + coating
  - Glucamesh (Brennen) complex carbohydrate, oat β glucan (50 g/m²)
  - Sepramesh (Genzyme), carboxymethylcellulose-sodium hyaluronate-polyethylene glycol (102 g/m²)
  - Proceed (Ethicon) polydioxanone-oxidized regenerated cellulose (45 g/m²)
  - C-Qur (Atrium) omega-3 fatty acid (50 g/m²)
  - TiMesh (GfE) PP + titanium coat
- PE mesh + coating
  - Parietex composite (Covidien/Sofradim) collagen-polyethylene glycol-glycerol (75 g/m²)

**Absorbable Synthetic**
- Polyglactin mesh (Vicryl)
- Polyglycolic acid (Dexon)
The coated meshes were developed as an alternative to ePTFE when contact with intra-abdominal viscera cannot be avoided. Representative examples along with their specific coating are listed in Box 2. The coating is designed to prevent erosion into viscera. The coating is usually applied only to 1 side of PPL or Polypropylene (PL), with that side facing the viscera and the uncoated side facing the abdominal wall, where ingrowth is desirable. Neither ePTFE nor the coated meshes completely prevent adhesions, as shown by the findings when patients who have had them implanted are reexplored for some reason. It remains to be seen if the coated prosthesis affords the same protection on long-term follow-up as ePTFE, with its long track record of safety.

Lightweight, large-pore prosthetics have become popular in recent years, because they seem to be associated with less chronic discomfort and better abdominal wall mechanics. It is not clear whether the lightweight (eg, less than 35 g/m²) or the larger pores or both are responsible for this popularity. Regardless, there have been reports of burst mesh with the lightweight material, and therefore, they should be used with caution when considering giant hernias [18].
Another new class of prostheses is the biologics. They are called biologics because they are derived from living tissue (ie, human, porcine, or fetal bovine skin, porcine small intestine submucosa, and bovine pericardium), as shown in Box 3. These donor tissues are processed to remove hair, cells, and cell components as well as other antigens present in the matrix, leaving only the highly organized collagen architecture and surrounding extracellular matrix. In general, these tissues possess the physical and mechanical characteristics of a clinically acceptable synthetic prosthesis, in that they initially have sufficient mechanical strength to withstand the physiologic and anatomic stresses of the abdominal wall and present a biological scaffold to support tissue regeneration. It has been theorized that they might be more physiologic and therefore result in less chronic pain and better recovery than the synthetics, although this theory remains unproved. It seems that they can be safely placed in contact with intra-abdominal viscera and are believed by many authorities to perform better than a synthetic mesh in a contaminated wound. Their major drawback is expense when compared with a synthetic.

**OPERATIVE APPROACHES**

The basic principles that guide the repair of a giant ventral hernia are modified from those used for simple incisional hernias. However, they almost always include the use of some type of prosthesis, if any chance of success is to be achieved. Rarely, a pure tissue repair can be accomplished using component separation techniques (as discussed later), which are reserved for specific contraindications to a prosthesis such as allergy or the patient’s personal preference. Most giant ventral hernias are located in the midline and therefore, for simplicity, the descriptions given later assume this location. The basic principles discussed must be adapted for off-midline or lateral abdominal wall hernias.

Prosthetic material can be positioned in 3 different anatomic planes, as depicted in Fig. 5. An overlay may be placed on top of a simple suture repair, but most authorities believe that it offers minimal advantage over the primary repair and has the same excessive recurrence rates as without a prosthesis [6]. Proponents of the overlay technique mitigate the tension associated with the primary closure by incising the anterior fascia several centimeters lateral to the defect of the hernia, rotating it medially in continuity with the posterior fascia, in effect lengthening the posterior fascia enough to allow for primary closure, isolating the intra-abdominal viscera from the fascia. In the classic procedure described by Chervel, a large synthetic prosthesis widely overlapping the unclosed anterior fascial defect is then placed (Fig. 6) [19].

In the inlay procedure, the prosthesis is circumferentially sutured to the edges of the fascial defect; this was the standard of care for most of the latter half of the twentieth century. It was attractive because it was tension free and easy to perform. Long-term follow-up studies have shown this operation not to be durable, with frequent recurrent herniation at the mesh-tissue interface. It is now considered unacceptable in most instances. However, there
are situations in which the hernia defect is so large that abdominal wall closure cannot be obtained any other way, even with sophisticated adjuvant strategies such as component separation. It therefore needs to be a part of the armamentarium of the complex abdominal wall reconstruction surgeon. When a hernia defect is bridged, the abdominal viscera must be protected to prevent erosion and subsequent fistula formation. A peritoneal flap constructed from the hernia sac or omentum is sometimes technically possible to isolate the viscera from the abdominal wall. When contact with intra-abdominal organs cannot be avoided, ePTFE or a composite prosthesis is indicated. The coated prosthesis and the biologics also are applicable in this situation but do not have a track record to ensure that erosion into the abdominal viscera does not occur [20].
In addition to the high recurrence rate associated with the inlay mesh repair, even when there is no true recurrence of hernia, many patients perceive that the hernia bulge has come back because of lack of muscular covering overlying the prosthesis, which stretches over time. Therefore, the current gold standard open repair for a midline incisional hernia is primary repair of the posterior rectus sheath followed by reapproximation of the rectus muscles after a prosthesis has been placed behind them. This operation was initially described for treatment of large and multiply recurrent hernias requiring abdominal wall reconstruction but is now being used even for smaller hernias [21]. Minor modifications of the basic procedure have been published by Rives, Stoppa, Velamenta, and others [22,23]. The term Rives-Stoppa technique is commonly used to refer to the procedure in the literature, but retromuscular prosthetic repair is more descriptive and is preferred [24,25]. The operation is characterized by placement of a large prosthesis in the space between the rectus muscle and the posterior fascia or the transversalis fascia, depending on which part of the abdomen is being repaired (ie, there is no posterior fascia below the arcuate line). It involves creating extensive flaps by dissecting the skin and subcutaneous tissue off the external fascia, well lateral to the hernia defect on either side. This strategy often allows the musculoaponeurotic components of the abdominal wall to be advanced to the point at which the posterior and anterior fascial layers can be closed primarily. Once the flaps have been created, the fascia is opened at the edges of the defect, thereby affording entry into the plane between the posterior surface of the deepest muscle and the underlying peritoneum and posterior fascia. A combination of blunt and electrocautery dissection works best for creating a large space to accommodate a sizable prosthesis extending at least 5 cm beyond the defect [26]. The posterior rectus sheaths are approximated to each other primarily if possible. If the posterior sheath cannot be approximated because of tension, then the use of ePTFE or dual-layer prosthesis should be considered instead of the standard mesh. The prosthesis is then placed in the space deep to the muscle and secured in this position with sutures placed via a suture passer at the periphery of the retromuscular pocket (Fig. 7). The sutures pull the prosthesis laterally and firmly affix it to the abdominal wall; they are then tied in the subcutaneous tissue above the fascia. Some surgeons prefer to avoid using these full-thickness sutures because of concern over the possibility of wound pain resulting from neuromuscular entrapment and use sutures or staples to secure the mesh to posterior fascia as far laterally as possible. The retromuscular mesh repair is the most common approach for repair of large incisional hernias.

A laparoscopic repair has now become an accepted technique for ventral hernias, with acceptable short-term and long-term outcomes. However, it has found little place in the repair of huge ventral hernias, because of the wide separation of muscular elements of the abdominal wall. Prosthetic material that is used to bridge a huge gap even when wide overlap can be obtained invariably balloons through the original defect with time, making the patient feel that the hernia has returned. Further contraindicating laparoscopy is the commonly associated
extensive intra-abdominal adhesions caused by multiple previous operations. Laparoscopy can occasionally be helpful as an adjuvant to aid in initial adhesiolysis and to confirm complete fascial closure without incorporation of intra-abdominal viscera at the conclusion of an open procedure. The concepts of minimal invasion for giant ventral hernias have also been applied in the form of endoscopic component separation, which is described in a separate section later.

COMPONENT SEPARATION TECHNIQUE
Closure of rectus sheath commonly fails with giant hernias, with significant loss of domain, because the fascial edges are widely separated from each other. Under such circumstances, there is a role for component separation of the

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**Fig. 7.** The technique for placing a prosthesis behind the rectus muscle as described by Flament. (A) Creation of the retromuscular space. (B) Preparing the prosthesis with sutures with long tails circumferentially. (C) A suture passer being used to pass the suture at the periphery of the retromuscular pocket into the subcutaneous plane. (D) Sutures tied down in the subcutaneous space, securing the prosthesis to the lateral side of the rectus envelope. (E) Final appearance of mesh over the posterior rectus sheath but behind the rectus muscle.
abdominal wall, which allows medialization of the rectus muscles to achieve approximation of the abdominal musculature in the midline.

Component separation was first popularized by Ramirez and colleagues in the 1990s [27]. Several modifications of the operation have been published since then [28–30]. Originally, it was proposed as a tissue repair for incisional hernias with contaminated wounds to avoid the use of a prosthesis. However, this technique is being commonly used synchronously with a sublay prosthesis for repair of huge defects of the abdominal wall [24].

The technique involves making a long midline incision through the scar to expose the hernia. The hernial sac is dissected to the fascial edge. Flaps are fashioned by dissecting the skin and subcutaneous fat off the underlying anterior sheath of the rectus abdominis and the aponeurosis of the external oblique muscle. The aponeurosis of the external oblique muscle is then transected longitudinally, just lateral to the lateral edge of the rectus sheath (Figs. 8 and 9). It is important to extend this incision in the external oblique aponeurosis onto the chest wall at least 5 to 7 cm cranial to the costal margin. The external oblique muscle is separated from the internal oblique muscle as far laterally as possible. This step is safe because the neurovascular bundle (comprising the intercostal nerves and vessels) lies deep to the internal oblique muscle. The result is that the internal oblique muscle and the rectus abdominis muscle slide medially for 7 to 10 cm, so that the edges of the rectus sheath defect can be brought together without undue tension and sutured primarily in the midline [28]. If primary closure is still not possible without undue tension, an additional 2 to 4 cm can be gained by separating the posterior rectus sheath from the rectus abdominis. For very large hernias, transection of the transverse abdominis muscle may give extra gain [31]. Care must be taken not to damage the neurovascular bundle, which runs between the internal oblique muscle and the transversus abdominis to enter the rectus sheath posterolaterally.

**Fig. 8.** Conventional component separation technique. Identification of the components (A) and their separation (B) during the component separation technique.
Several investigators have published good outcomes of component separation technique for huge ventral hernias [28,29,32–37]. The rate of recurrence after component separation has been reported to range from 10% to 30% after a 2-year follow-up [38]. Most recurrent hernias after this technique are small and require no further treatment. We believe that to prevent long-term recurrence of hernia, there is a complementary role of mesh alongside the component separation technique, as has been shown by other investigators as well [2,30,39]. A study comparing only component separation versus component separation with overlay of biologics reported a significantly lower recurrence rate in the latter (13% vs 0%) [39]. de Vries Reilingh and colleagues [2] compared component separation technique with prosthetic repair in a randomized controlled trial for patients with giant midline abdominal wall hernias. Recurrence rates were 60% after prosthetic repair versus 53% after component separation during a follow-up of 36 months (statistically no difference). The component separation technique is frequently complicated by wound healing disturbances. Certain modifications of the classic Ramirez technique for component separation have been described to save the blood supply via the periumbilical epigastric perforators to prevent wound breakdown [28,40].

**ENDOSCOPIC COMPONENT SEPARATION**

Endoscopic component separation was developed to address the well-known wound-related complications of open component separation caused by the large undermining of skin flaps with disruption of the blood supply to the abdominal wall such as seromas, abscesses, or flap necrosis. The technique involves making a 2-cm to 4-cm incision just below the costal margin in the anterior axillary line [41]. This incision must be lateral to the attachment of external oblique aponeurosis to the rectus sheath. Some investigators make this skin incision from within the peritoneal cavity after palpating the lateral border of the rectus sheath [42]. In

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**Fig. 9.** Clinical picture of a patient who developed ischemia and skin necrosis of the right skin flap after component separation for a giant ventral hernia (note that the left skin flap is viable).
either case, the space between the external and internal oblique muscle is bluntly dissected out to make space for the dissector balloon (Covidien, Mansfield, MA). Under camera guidance, the dissector balloon is inflated. The balloon bluntly dissects the space between the external oblique and internal oblique muscles lateral to the linea semilunaris and is aided further by carbon dioxide insufflation. The external aponeurosis, which is seen separate from the deeper internal oblique, is then divided from the costal margin to the pubic symphysis using Endo Shears (Covidien, Mansfield, MA).

OTHER SURGICAL OPTIONS
Wide separation of the medial edge of rectus muscle along with an atrophic abdominal wall musculature with huge ventral hernias is sometimes a recipe for failure of abdominal wall reconstruction, even with the combined application of bilateral component separation and the retromuscular prosthesis. Often, the volume of hernia sac exceeds the volume in the abdominal cavity. Closure of the abdominal wall under such circumstances has a high risk of intra-abdominal hypertension and abdominal compartment syndrome [43]. For such complex cases; it is always wise to plan backup options.

MEDIALIZATION
A recent study of a small series of patients (n = 8) proposed a medialization procedure that consists of temporary closure of the abdominal wall with ePTFE followed by scheduled serial visits to the operating room to tighten the prosthesis by excising a strip in the middle and then suturing the edges together [44]. Definitive closure of the abdominal wall was achieved using component separation after a mean 6 serial visits to the operating room in this study.

ROLE OF PROGRESSIVE PNEUMOPERITONEUM PRECEDING VENTRAL HERNIA REPAIR
The use of preoperative progressive pneumoperitoneum (PPP) for repair of huge incisional hernias was first described by Goni Moreno in 1940 [45]. PPP stretches the abdominal cavity by increasing the length of the abdominal muscles and the diaphragm [46–48]. Room air (or CO\textsubscript{2}) is injected intraperitoneally daily via a needle or an implantable catheter with or without a subcutaneous port, to tolerance (ie, development of shoulder pain, difficulty breathing, or development of subcutaneous emphysema). Patients or family members are taught to administer this injection so that the procedure can be performed as an outpatient. Some surgeons prefer to use pressure monitoring, keeping less than 15 mm Hg, but this complicates the technique because of the need for in-hospital monitoring. In addition to increasing the capacity of the abdominal cavity, pneumoperitoneum may also cause adhesiolysis and improve diaphragmatic function.

PPP has been more commonly applied in Europe with good results for large ventral hernias [49,50]. In a recent study, safety and feasibility of this approach
were shown in 16 patients [51]. Nonetheless, 7 of these 16 patients still required ileocectomy or lateral relaxing incision on the rectus sheath to achieve abdominal wall closure. In another small series of 9 patients, pneumoperitoneum was sufficient to reduce the visceral contents, permitting a retrorectus repair in 80% patients [52]. Of 9 patients, 1 patient with diabetes mellitus developed an abdominal wall abscess requiring drainage in the postoperative period. Many surgeons in North America are skeptical about the approach, questioning how one ensures that the air injected stays in the peritoneal cavity and does not leak into the hernia sac, enlarging it further. Also of concern is the possibility of infection and the cost, especially if in-hospital monitoring is needed.

**ROLE OF FLAPS AND GRAFTS**

The use of flaps and dermal grafts for abdominal wall reconstruction is usually reserved for patients in whom the conventional component separation repair with a retromuscular prosthesis is not feasible. Most experience has been with fascia lata, which has been shown to retain its shape and tensile strength for an extended period [53]. Harvesting is easy and donor-site morbidity is low (13%–17%). A longitudinal incision on the lateral thigh is used to dissect the fascia from the fascia lata muscle. Girotto and colleagues [34] used free fascia lata grafts along with component separation to repair very large, contaminated abdominal wall defects in 78 patients. Wound infection occurred in 32 patients, with recurrence in 29% of the patients after a follow-up of 6 months. Full-thickness skin has been used to bridge fascial defects for incisional hernia repair. Autodermal grafts are easily available because of the redundant skin overlying the hernia, and its harvesting does not increase morbidity. After the harvest, the subcutaneous layer is removed and the graft is placed with the dermis toward the peritoneal contents. A wound complication rate of 20% has been reported without any complications from overgrowth of adnexal skin structures [14].

Pedicled or free vascularized flaps are used as the last resort, because these are complex procedures and leave large donor-site defects, causing additional morbidity. The tensor fasciae latae (TFL) pedicle flap is most commonly used for lower abdominal wall hernias, because it combines well-vascularized tissue with a strong fascia that can resist intra-abdominal pressure. Williams and colleagues [54] reconstructed abdominal wall defects in 9 patients with a pedicled TFL flap; in 5 patients, there was a contaminated or infected environment. Three flaps needed revision because of flap necrosis. Donor-site morbidity was 50%. There were no recurrences after a mean follow-up of 21 months. Harpf and colleagues [55] used this flap in 4 patients. After a follow-up of more than 11 months, none of the patients had a reherniation. Other flap options for the lower abdomen include a rectus femoris flap, a gracilis, or a vastus lateralis flap. The rectus femoris myocutaneous flap is a long but narrow flap that can be used to repair defects in the lower abdomen. It is pedicled on the lateral circumflex femoral artery. Caulfield and colleagues [56] reconstructed 13 abdominal hernias with this flap. Only 2 had major
complications: hematoma and loss of a split skin graft at the donor site. There were no recurrences after a follow-up of 3 to 18 months. A significant loss of muscle strength during knee flexion was noted after flap harvest. For the upper abdomen, the workhorse is the latissimus dorsi flap.

Free flaps represent the next level in extraordinary strategies for abdominal wall reconstruction. Upper abdominal defects may be repaired using a free vascularized TFL flap. The results using this free flap have been described in 4 retrospective case series containing a total of 20 patients [38]. Partial flap necrosis was seen in 5 of 20 patients. A free latissimus dorsi flap for abdominal wall defects has also been used [57]. Anterolateral thigh and rectus femoris have all been described in the literature. Abdominal wall transplantation represents the extreme when considering free flaps and has been reported [58].

TISSUE EXPANSION
Tissue expanders can be used in cases in which either skin or the myofascial elements of the abdominal wall are deficient [59]. They are implanted subcutaneously or subfascially, depending on the tissue needed. The expanders are then gradually inflated until the desired amount of extra tissue is obtained. The limitation of the technique is the delay in definitive repair while the expansion is ongoing and the considerable amount of pain that patients complain of. The intra-abdominal placement of tissue expanders has also been described to address the issue of loss of domain, but this is primarily confined to the pediatric literature [60].

RESECTION OF INTRA-ABDOMINAL VISCERA
There are situations when, despite applying all these techniques, even the most experienced surgeon is not able to replace the abdominal contents and close the fascia without causing significant increase of intra-abdominal pressure. In these circumstances, the last resort of the surgeon is to resect intra-abdominal viscera to reduce the volume needing to be reduced. The options include performing total omentectomy, subtotal colectomy, and removal of retroperitoneal fat. The downside of opening the gastrointestinal tract is the lack of freedom in using all possible prosthesis in such a wound, especially if there is any contamination.

COMPLICATIONS OF GIANT VENTRAL HERNIA REPAIR
In a meta-analysis, the overall complication rate after open ventral hernia repair was 27% (Box 4) [61]. Prosthesis-related infection remains a major problem repair and is one of the most important risk factors for recurrence. The chance of prosthesis infection is increased with preexisting infection, skin ulceration, obesity, incarceration, and enterotomy during repair. Seromas, which can lead to infection, are a common occurrence, as a result of extensive flap dissection and the large prosthesis required for giant incisional hernias. Suction drains are useful but are likely to result in prosthesis infection if left in place too long. Topical instillation of talc, tetracycline, and numerous other agents
has been tried to prevent or decrease seroma formation, but none has been shown to be consistently effective.

Lowe and colleagues [35] retrospectively looked at the complications after reconstruction of abdominal wall defects with component separation. The commonest complications seen were postoperative ileus (27%), cardiorespiratory complications (46% cumulative), wound ischemia (20%), wound infection (40%), and enterocutaneous fistula (43%). Other investigators have also reported wound complication rate as high as 30% to 50% [2,28,35,62]. In a systematic review of studies looking at the outcomes of component separation, wound complications were seen in 85 of the cumulative 354 patients (23.8%, confidence intervals 18.3 to 29.8%) [38]. Wound infection was the most common complication found in 18.9%, seroma in 2.4% hematoma in 2.4%, and skin necrosis in 1.5%. High rates of wound-related complications after component separation are caused by extremely large wound surfaces,

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**Box 4: Complications of giant ventral hernia repair**

**Postoperative Complications**
- Bleeding
- Postoperative ileus
- Myocardial infarction
- Deep venous thrombosis
- Pulmonary thromboembolism
- Enterocutaneous fistula

**Complications Caused by Loss of Domain**
- Abdominal compartment syndrome
- Renal failure
- Need for prolonged ventilation

**Wound-Related Complications**
- Seroma
- Ischemia of the skin flaps
- Skin necrosis
- Skin ulceration
- Wound infection
- Prosthesis infection
- Dehiscence (superficial or deep)

**Long-Term Complications**
- Recurrence
which are formed by mobilization of the skin and subcutaneous fascia from abdominal wall muscles. The other explanation is division of perforator vessels while raising these flaps, which causes ischemia, skin necrosis, and infection. Perforators are vital for the abdominal wall, because blood supply by the intercostals, superficial circumflex iliac and external pudendal vessels is often already compromised from previous incisions.

**SUMMARY**

Repair of huge ventral hernias is technically challenging for the surgeon and a major operation for the patient and should be performed by experienced surgeons in centers that are used to caring for patients who are commonly massively obese with significant comorbidities. Preoperative medical optimization of patients is an important part in the overall management of these large hernias. Conventional component separation with retromuscular mesh repair is the workhorse operation, which successfully deals with many giant ventral hernias, but multiple alternative strategies must be available to address situations in which myofascial elements are completely deficient or there is significant loss of domain. The complexity of this surgery is reflected by recurrence rates ranging from 10% to 30% and wound complication rates as high as 40% to 50% in experienced centers.

**References**


GIANT VENTRAL HERNIA


