

INTRODUCTION

Reconstruction of abdominal wall defects is complex and the clinical considerations involved in determining the surgical approaches are challenging to general and plastic surgeons alike.

The anatomical factors, site of the wound and wound bed quality, infection, nutritional status of the patient, diabetes and smoking, also play a key role in how reconstruction plans develop (*Rohrich et al., 2000*).

Anterior abdominal wall serves several functions; it contains and protects the abdominal viscera. The muscles of the anterior abdominal wall also assist in pulling down on the ribs during forced expiration and coughing. These muscles help with defecation, micturation, child birth, fixation of the spine and assist in the rotation of the body (*Rohrich et al., 2000*).

There are different causes of abdominal wall defects either congenital or acquired as mentioned by (*Cohen, 2006*). The congenital causes can be divided into either herniation of abdominal contents at the umbilicus as Omphalocele (Exomphalos) with an incidence of 1:6,000 or full thickness abdominal defects situated always to the right of umbilicus like Gastrschisis with an incidence of 1: 20,000: 30,000.

Deficiency of the abdominal musculature which may be associated with urinary tract dilatation and cryptorchidism as

in Brune-Belly syndrome having an incidence of 1:50,000. (*Cohen, 2006*).

The acquired defects of the abdominal wall are caused primarily by trauma, infection, ablative resection of primary or recurrent tumors, complications of surgical procedures such as incisional hernia, radiation damage and burns. (*Cohen, 2006*).

These defects can be superficial, involving only some layers of the soft tissue of the anterior abdominal wall, or full thickness, extending into the abdominal cavity (*Cohen, 2006*).

The main goals of reconstruction of abdominal wall defects is the restoration of the structural and functional continuity of the musculofascial systems mentioned by *Dibello and Moore (1996)* with the preservation of the integrity of the abdominal wall and minimizing the complications such as infection, dehiscence and abdominal compartment syndrome as affirmed by *Dibello and Moore (1996)* as well as the achievement of stable local wound coverage.

The preoperative evaluation should include complete history, physical and general medical evaluation, basic laboratory work and other diagnostic and radiological studies (*Dibello and Moore, 1996*).

The evaluation of the extent of the defect and the associated pathology as presence of local inflammation or bowel adhesions so important in determining the timing and

the option used in reconstruction that is balanced with patient's general health, tissue requirements and wound bed.

Dibello and Moore (1996) stated that the ideal reconstruction should encompass four requirements; prevent visceral evisceration, incorporate the abdominal wall, provide a tension-less repair, and dynamic muscle support.

Reconstructive options for the abdominal wall repair are vast starting from skin and fascial grafting for covering extensive wounds after being healthy and granulating well. The staged tissue expansion as affirmed by *Wihelmi (1998)* through placement of the tissue expansion devices in between the different layers of the abdominal wall have role in the staged abdominal wall reconstruction.

Kilbride et al. (2006) outlined the Vacuum-assisted closure is considered a safe and effective alternative in treating the complicated cases, also the component separation technique which was considered as a preferred technique for the repair of large defects not amenable to primary repair (*Broud et al., 2007*).

The use of prosthetics (e.g. Prolene, Mersalin) and bioprosthesis (e.g. AlloDerm) have aided greatly in the management of complex abdominal defects as affirmed by (*Grevious, 2006*).

Flaps either local, pedicled, or free transferred plays an important role in the reconstruction of abdominal wall defects,

being necessary for the contaminated wound fields and coverage of applied mesh.

All these reconstructive techniques, and procedures will be described and improved treatment protocols will be presented. Thus management philosophies have changed completely, and the final outcomes improved significantly

AIM OF THE WORK

The aim of this work is to spot light on the recent and different techniques in surgical management of complex surgical abdominal wounds with emphasis on the techniques and outcome of each

STRUCTURAL & FUNCTIONAL ANATOMY OF THE ANTERIOR ABDOMINAL WALL

Knowledge of the anatomy of the anterior abdominal wall has enabled the reconstructive surgeons to achieve one of the goals in managing abdominal wall defects, which is restoration of the structural and functional continuity of the musculo-fascial system (*Grevious, 2006*).

Embryology

The abdominal wall begins to develop from the lateral plate of intra-embryonic mesoderm. When differentiation proceeds, the intra-embryonic mesoderm becomes segmented into proliferating somites forming the abdominal wall. When the lateral plates grow and folds over four unique folds are formed.

In the third week of gestation, the embryo has the shape of an elongated disc and the disc has three germ layers in its cranial half: ectoderm, dorsally; mesoderm in the middle; and endoderm, ventrally. Also, at this stage the embryonic endoderm layer is continuous with the endoderm of the visceral yolk sac; while the embryonic ectodermis continuous with ectoderm over the amniotic cavity. Thus, the disc is positioned between the amniotic cavity dorsally and the yolk sac cavity ventrally (figs.1A&2A) (*Sadler, 2006*).

Neural tube development (neurulation) is initiated when the central(axial) portion of the ectoderm layer is induced to

form the neural plate whose lateral edges then elevate to form the neural folds (*Colas and Schoenwolf, 2001*).

These folds consist of neurectoderm and underlying mesenchyme as they continue to elevate and bend toward the midline where they will fuse dorsally at the end of the fourth week to form the neural tube (Fig. 2). The closure process begins in the cranial region and progresses caudally (*O’Rahilly and Muller, 2002*).

Initially, lateral plate mesoderm forms a solid block of tissue continuous with extra-embryonic mesoderm covering the amnion and yolk sac. During the third week, small cavities appear in this solid layer and these spaces coalesce to create two new layers bordering a new cavity. The new cavity is the intra-embryonic cavity that eventually will be separated into the pericardial, pleural, and peritoneal cavities (Fig. 2B–D) (*Sadler, 2006*).

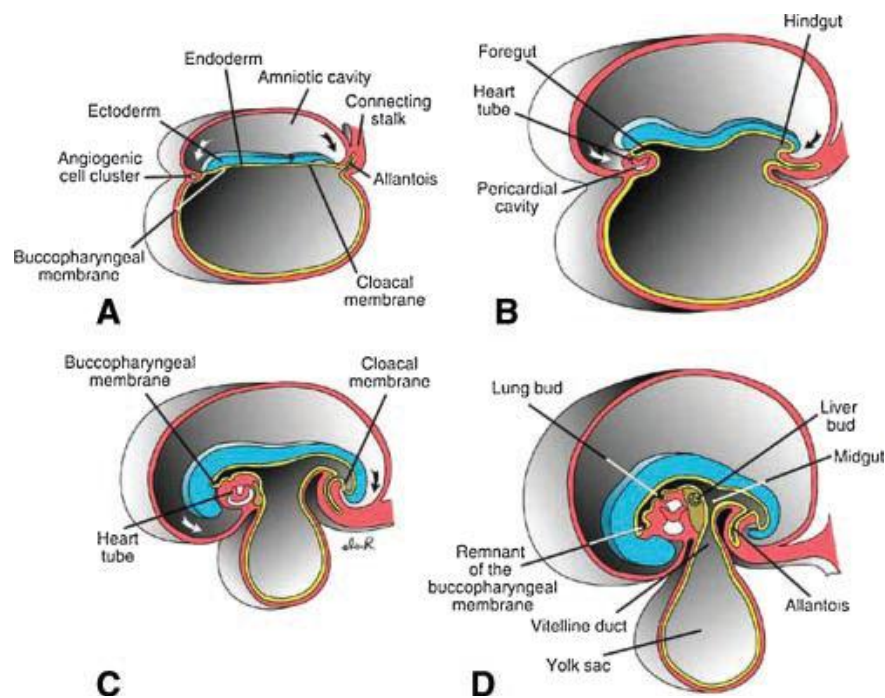


Figure (1): Sagittal sections through embryos (A) 17 days; (B) 22 days; (C) 24; (D) 28 days. Formation and progression of head and tail folds (arrows) cause the embryo to assume the fetal position. Together with the two lateral body wall folds, they also “pull” the amnion ventrally around the embryo to the region of the umbilicus, while concomitantly narrowing the gut tube’s connection to the yolk sac. By four weeks (D) this connection has narrowed considerably to form the vitelline (yolk sac) duct (*Sadler, 2006*).

The new mesodermal layer lying to the outside of this cavity and lining the inside of the body wall is the parietal (somatic) layer of lateral plate mesoderm; while the layer lining the inside of the cavity forms the visceral (splanchnic) layer of lateral plate mesoderm. The parietal layer is closely associated with ectoderm covering the body wall; whereas the visceral layer is intimately associated with the wall of the gut tube (*Sadler, 2006*).

While the neural tube is progressing toward closure dorsally, the sides of the embryo, which include the parietal layer of lateral plate mesoderm with overlying ectoderm, start to grow ventrally, (figs 2D&3A). These structures form the lateral body folds that begin their downward extension at the end of the third week. Head (cranial) and tail (caudal) folds also form and begin to “fold” the embryo into the “fetal” position (figs1) (*Sadler, 2006*).

The combined movement of all four body folds narrows the connection between the yolk sac and embryonic endoderm and assists in formation of the gut tube (figs1&3). As the visceral layer of lateral plate mesoderm and underlying endoderm merge in the midline the gut tube is formed (figs 2D&3). The tube extends from the pharynx to the anal canal and for most of its length it is suspended from the dorsal body wall by a dorsal mesentery derived from lateral plate mesoderm at the junction of the visceral and parietal layers (fig.3B) (*Sadler, 2006*).

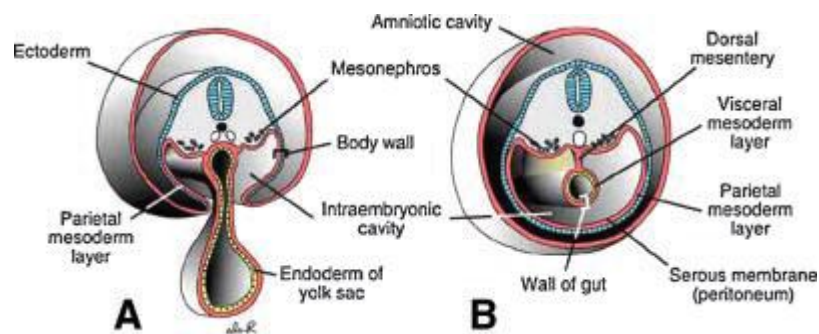


Figure (3): Cross sections through embryos and their membranes at: (A) 25 days; (B) 28 days. The sections show the approach and closure of the lateral body wall folds. As the folds approach the midline, the connection between the gut tube and yolk sac narrows (A) and is lost (B) except in the region of the umbilicus. Once the folds fuse, there is a considerable reorganization of cells in the amniotic membrane and embryonic ectoderm and parietal mesoderm to effect closure and produce the appropriate continuum of intact layers across the midline (*Sadler, 2006*).

Meanwhile, the lateral body folds continue to move ventrally and toward the midline where they fuse. As fusion occurs, the amniotic membrane, surface ectoderm, and parietal layer of the lateral plate mesoderm each becomes continuous with its counter part from the other side. The fusion process probably involves programmed cell death (apoptosis), formation of specialized cell-to-cell contacts, and cell migration which have been observed in other similarly fusing folds, including the neural tube (*Copp et al., 2000*).

The head fold appears to form by default as the cranial region of the neural tube overgrows the ventral part of the embryo., the tail fold forms by extension of the body axis with the dorsal region (neural tube) growing faster than the ventral region (*Sadler, 2006*). Undoubtedly, growth of these “folds”

assists in creating the “fetal” position of the embryo, but how much these folds contribute to closure of the ventral body wall is not clear.

The cephalic fold is the most anterior and contains the foregut, the stomach and the mediastinal / thoracic contents also it forms the epigastric abdominal wall. The caudal fold develops into the colon, rectum, bladder and the hypogastric abdominal wall (*Sabiston and Lyerly, 1994*).

The two internal folds develop into midgut and the lateral segment of the abdominal wall. All of these segments coalesce in the midline at the umbilicus (*Sabiston and Lyerly, 1994*).

Because the alimentary tract grows rapidly, at 6 to 8 weeks of gestation, all fetuses demonstrate a physiological herniation of the midgut by the 11th week, the midgut rotates and return back into abdominal wall cavity with the alimentary tract in continuity (*Sabiston and Lyerly, 1994*).

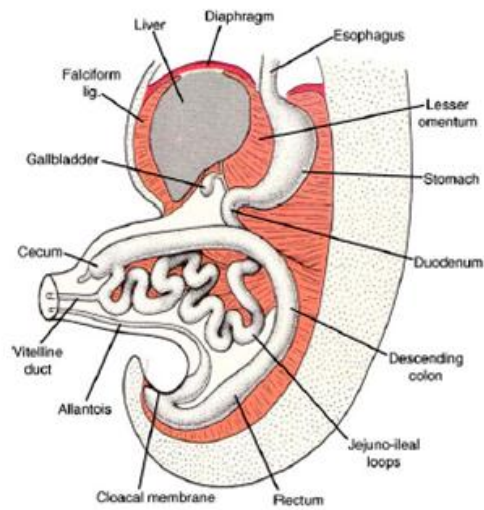


Figure (4): Embryo at 12 weeks at time of abdominal wall formation (*Sadler, 1990*).

At an early stage (5th week) the abdominal wall is composed of only a membrane of connective tissue that is soon replaced by muscular buds from the dorsal myotomes. These muscular buds are segmentally connected to their corresponding neurovascular bundles. These buds fuse to form the definitive muscles (**Sadler, 1990**).

Gross Anatomy

The anatomical layers of the abdominal wall include six layers which are skin, subcutaneous tissue, superficial fascia, deep fascia, muscles, extra-peritoneal fascia and peritoneum. This anatomy may vary with respect to different topographic regions of the abdomen. The major source of the structural integrity and strength of the abdominal wall is provided by the musculofascial layer (*Grevious, 2006*).

The skin

Skin was described by ***Mahadevan (2003)*** as being thin compared with that of the back and relatively mobile over the underlying layers except at the umbilical region, where it is fixed.

Natural elastic traction lines of the skin (Kraissl's Lines) of anterior abdominal wall are disposed transversely, above the level of the umbilicus these lines run almost horizontally while it runs with a slight inferio-medial obliquity below the umbilical level.

Incisions made along, or parallel to these lines tend to heal without much scarring, where as incisions that cut across these lines tend to result in a wide or heaped-up scars (***Mahadevan, 2003***).



Figure (5): kraisissl's lines (***Jon Armitage and lockwood, 2011***).

The superficial fascia

The primary function of the superficial fascial system (SFT) as stated by **Lockwood (1991)** is to encase, support, and shape the fat of the trunk and extremities and hold the skin to the underlying tissues.

The skin and fat together with the superficial fascial system provides a protective cushion over the musculoskeletal framework and supports the position and weight of fat deposits. They also help in preventing shifting of excessively obese or aged soft tissues onto another anatomic region.

The superficial fascia was described by **Lockwood (1991)** as a connective tissue network that extends from the sub dermal plane to the underlying muscle fascia. It consists primarily of one to several thin, horizontal membranous sheets separated by varying amounts of fat with interconnecting vertical or oblique fibrous septae.

There are two distinct layers of superficial fascia as stated by **Mahadevan (2003)**, An outer adipose layer immediately subjected to the dermis. And inner fibro-elastic layer termed Scarpa's fascia which is more prominent and better defined in the lower half of the anterior abdominal wall.

Superiorly, Scarpa's fascia crosses superficial to the costal margin and becomes continuous with the retromammary fascia. Laterally, it fades out at the mid-axillary line. Inferiorly, it crosses superficial to the inguinal ligament. Below the level