Surgical anatomy, like other sciences applied to medicine, often passes through periods of neglect. The chief reason for this neglect is that there is confusion between contemporary advances in surgery and the knowledge required of each new generation of surgeons. Many surgical anatomic problems have been worked out long ago, but each surgeon must relearn for himself much of the information left behind by the most recent wave of surgical concern.

This does not mean that surgical anatomy is fossilized. It is not the mere recounting of layers and relationships. It must consider new interpretation learned by experience with old procedures, and at its best, should point out further refinements of existing methods.

In addition to preserving knowledge already gained and presenting it as usefully as possible, surgical anatomy must be prepared for the recent advances as well. Frequently, areas well known to the anatomist, but rarely explored by the surgeon, become important new approaches for surgical repair. Here surgical anatomy becomes the bridge between the textbook of anatomy and the operating table. To tell the modern surgeon that "it is all there on pages 1275 to 1293" is to ask him to spend valuable time at a task, perhaps not beyond his competence, but one well outside his primary concern. Surgical anatomy must take over here to organize the material in light of the surgeon's actual needs.

We wish also to emphasize the role of embryology. The student, the resident, and the surgeon will understand and appreciate surgical anatomy better if they understand surgical embryology. An obvious example is the dissection of a cyst or fistula of the second branchial cleft or pouch in the neck. Knowledge of surgical embryology and surgical anatomy of this area will ensure that the patient will have the ideal operation.

Surgical anatomy must continually re-interpret older surgical problems as well as be prepared to interpret the future problems of the practicing surgeon. For his part the surgeon must read today's surgical anatomy and embryology rather than rely on the half-remembered didactic lessons of his medical school days.
"Where should I cut the ring of the incarcerated or strangulated hernia?"

A strangulated hernia at any site is a surgical emergency. Delay in treatment will usually result in gangrene and eventual perforation of the incarcerated viscus.

The surgeon must know the precise anatomy of the constricting ring in order to incise it without injury to nerves and vessels or to the incarcerated viscus, as well as to provide conditions favorable for subsequent repair. In some cases the required incision is obvious; in others, it is easily performed only if the anatomy is well understood; in a few, considerable skill as well as understanding may be necessary to avoid a surgical catastrophe. In no case should the incision be longer than necessary to release the herniated viscus. To "nick the neck" will usually be sufficient.

We have made no effort to add to the number of excellent books on hernias which describe in detail the anatomy, etiology, diagnosis, and repair of hernias. Here we are concerned only with reduction of those hernias, already incarcerated or strangulated, that require surgical release before repair can be attempted. The illustrations are diagrammatic and have been made to emphasize the structures forming the ring and any special hazards involved in cutting it. We hope to answer only the first question presented by these incarcerated hernias: "Where should I cut the ring?"

Incarcerated Indirect Inguinal Hernia

Incarceration may occur at either the external or the internal ring. In children, constriction is usually at the external ring.

The ring is a triangular opening of the aponeurosis of the external oblique muscle, the base being part of the pubic crest with the margins formed by the two crura. The superior crus is formed by the aponeurosis itself, and the inferior crus is formed by the inguinal ligament.

Enlargement of the ring is accomplished by incising the aponeurosis of the external oblique muscle upward and parallel to the fibers. The incision should be only long enough to permit reduction of the hernia.

The internal inguinal ring, a normal defect in the transversalis fascia, is V-shaped. The arms of the V, the anterior and posterior crura, are formed by special thickenings of the transversalis fascia, forming a sling. In making the incision, retract the internal oblique muscle medially. Continue the anterior incision of the sac upward, far enough to release the incarcerated viscus. Keep in mind the possibility of a sliding hernia when you attempt to open the sac.
Incarcerated Interparietal Hernias

The hernial sac enters the internal inguinal ring as in indirect inguinal hernia. The sac passes between any two layers of the abdominal wall (properitoneal, interstitial, or superficial hernia). The sac may be multilocular, often having an indirect hernial component which emerges through the external ring.

In some superficial hernias the sac may emerge from the external inguinal ring before entering the subdermal fascia between the external oblique muscle and the skin. The external inguinal ring may then be the site of constriction.

Incarcerated Femoral Hernia

The boundaries of the femoral ring are: anterior, the inguinal ligament; posterior, the pectinate ligament (of Cooper); medial, the lacunar ligament (of Gimbernat); lateral, the femoral vein.

The incision should divide the inguinal ligament anteriorly, or divide the lacunar ligament medially. The former incision is preferred since the lacunar ligament is only 0.5 to 1 cm in length and its division may not enlarge the ring sufficiently.

We prefer to divide the inguinal ligament because an aberrant obturator artery, arising from the inferior epigastric artery, may pass medial to the femoral ring in the edge of the lacunar ligament rather than lateral to the ring. Injury to this vessel will cause unnecessary complications.

If the approach is made from above the inguinal ligament, and thus through the floor of the inguinal canal, the aberrant obturator artery can be seen clearly so that the lacunar ligament can be cut with safety.

Incarcerated Obturator Hernia

The boundaries of the hernial ring are: superior and medial, the obturator groove of the superior ramus of the pubic bone; inferior, the pelvic fascia containing the edge of the obturator membrane, the internal and external obturator muscles; posterior and lateral, the obturator vessels and nerves. The vessels lie lateral to the sac in 50 per cent of patients and medial, anterior, or posterior in the remainder.

Since the relation of the sac to the vessels and nerves is variable, the incision should be made at the lower margin of the ring.

Incarcerated Sciatic Hernias

There are three sites through which sciatic hernia may occur: suprapyramidal, through the greater sciatic foramen; infrapyramidal, through the greater sciatic foramen; and subspinous, through the lesser sciatic foramen.
Incarcerated Suprapyramidal Hernia. The boundaries of the ring are: superior, the anterior sacroiliac ligament; inferior, the upper border of piriformis muscle; lateral, the ilium; medial, the upper part of sacrotuberous ligament and part of sacrum.

Transect the piriform muscle by a posterior and inferior incision, remembering that the following structures lie in, or pass through, the greater sciatic foramen which is the "Monemvasia," the only gate, of the gluteal area: piriformis muscle, superior gluteal nerve and vessels, inferior gluteal nerve and vessels, internal pudendal nerve and vessels, posterior femoral cutaneous nerve, nerves to internal obturator muscle, nerves to quadratus femoris muscle, and sciatic nerve.

Incarcerated Infrapyramidal Hernia. Its boundaries are: superior, the lower border of piriformis muscle; inferior, the sacrospinous ligament; posterior, the sacrotuberous ligament; anterior, the ilium. For the incision, transect the piriform muscle. Remember the proximity of the vessels and nerves passing through the greater sciatic foramen.

Incarcerated Subspinous Hernia. The ring is formed by the lesser sciatic foramen, the boundaries of which are: anterior, the ischial tuberosity; superior, the sacrospinous ligament and ischial spine; posterior, the sacrotuberous ligament. Decompression and reduction without incision is preferred. If reduction cannot be accomplished otherwise, incise the obturator internus muscle.

Incarcerated Supravesical Hernias

External Supravesical Hernia. Ring and incision as in inguinal hernia.

Internal Supravesical Hernia. There are several sites of herniation in the supravesical fossae. Through these, the hernial sac passes in front of, or to the side of the bladder. A single case of intravesical hernia is known. In retrovesical hernia, the sac passes behind the bladder.

Supravesical, prevesical, and paravesical hernial rings are similar. The boundaries may be described as follows: superior, the upward continuation of the vesical fascia and its fusion with the transversalis fascia and the peritoneum; inferior, fold of vesical fascia and peritoneum; lateral, the lateral umbilical ligament and peritoneum; medial, the medial umbilical ligament and peritoneum. Boundaries of the retrovesical hernia are: superior and anterior, the vesical fascia and peritoneum of the posterior bladder wall; inferior and posterior, the transverse vesical fold.

If enlargement of the ring is necessary, the posterior margins should be incised upward.

Incarcerated Perineal Hernias

A primary perineal hernia may occur anterior or posterior to the superficial transverse perineal muscle.
Anterior Perineal Hernia. The defect is located anterior to the superficial transverse perineal muscle through the urogenital diaphragm into a triangle formed by the bulbocavernous muscle medially, the ischiocavernous muscle laterally, and the transverse perineal muscle posteriorly. The sac passes between the ischiopubic bone and the vagina, producing a swelling in the posterior half of the labium majus.

An abdominal approach is advised, with division of the deep transverse perineal muscle, including both layers of the urogenital diaphragm if necessary.

Posterior Perineal Hernia. The defect is located posterior to the superficial transverse perineal muscle, through the levator ani muscle, or between the levator ani and the coccygeus muscles. Koontz states: "It usually emerges halfway between the rectum and the tuberosity of ischium." If the levator ani muscle is not attached to the obturator fascia, a gap, the hiatus of Schwalbe, is formed: "It is important because a process of pelvic peritoneum may be pushed through the hiatus into the suprategmental space, and in this way occurs a hernia into the ischiorectal fossa." Division of the levator ani muscle is sufficient to release the incarcerated viscus.

Incarcerated Lumbar Hernias

Hernia Through The Inferior Lumbar Triangle (Petit's Hernia). The boundaries of the inferior lumbar triangle are: anterior (abdominal), the posterior border of the external oblique muscle; posterior (lumbar), the anterior border of the latissimus dorsi muscle; base, the iliac crest.

The floor of the triangle is formed by the lumbodorsal fascia and the internal oblique muscle, with some participation of the transversus abdominis muscle. If the hernia is small, the ring is formed by fibers of the lumbodorsal fascia and the internal oblique muscles. If the hernia is large, the ring is formed by the boundaries of the inferior lumbar triangle. The ring may be enlarged by a medial or lateral incision of the lumbodorsal fascia.

Hernia Through The Superior Lumbar Triangle. The boundaries of the superior lumbar triangle are: anterior (abdominal), the posterior border of the internal oblique muscle; posterior (lumbar), the anterior border of the sacrospinalis muscle; base (costal), the serratus posterior inferior muscle and the 12th rib.

The floor of the triangle is formed by the transversus abdominis aponeurosis through which herniation may occur. Thus, if the hernia is small, the ring is formed by the aponeurosis only; if the hernia is large, the ring is formed by the boundaries of the superior lumbar triangle. The ring may be enlarged by a medial or lateral incision (or both) midway between the 12th rib and the iliac crest.
Incarcerated Umbilical Hernia

Herniation at the umbilicus is of four types:

1. Hernia of the intestines into the cord during development of the fetus. This normal herniation spontaneously reduces before birth. If reduction fails to take place, an omphalocele results.

2. Intussusception of the intestines through a congenital, abnormally patent vitelline duct.

3. Infantile umbilical hernia. Bulging of the abdomen between the recti at the umbilicus is not uncommon in infants. Incarceration is rare and the defect usually repairs itself without requiring surgery.

4. Acquired umbilical hernia in adults. It is this type with which we are concerned.

The ring is formed by the fusion of all abdominal layers of the defect. Herniation is through the linea alba, usually at the upper margin of the ring so that the sac protrudes above or to one side of the umbilicus (paraumbilical).

The incision should be circular or elliptical, around, never over, the hernial sac. The ring should be incised laterally and the incision extended into the aponeurosis of the rectus abdominis with retraction of the muscle if necessary.

Incarcerated Epigastric Hernia

This is also known as hernia of the linea alba or ventral hernia. The ring is composed entirely of fibers of the linea alba. It may lie anywhere between the xiphoid process and the umbilicus. The ring can be enlarged by incising the linea alba upward or downward, sufficiently to release the incarcerated tissue or viscus.

Incarcerated Lateral Ventral Hernia (Spigelian Hernia)

Spigelian hernia occurs along the linea semilunaris of Spieghel. There has been confusion in the literature of the linea semilunaris and the linea semicircularis (semilunar fold of Douglas). Hollinshead writes: "Lateral ventral hernias are called Spigelian hernias because they are said to occur along Spieghel's (Spigelius) line, which is generally regarded as synonymous with the semilunar line." We agree with Hollinshead and believe that the confusion arose because most Spigelian hernias occur on the linea semilunaris where it intersects the linea semicircularis.

The linea semilunaris is usually taken as the lateral border of the rectus sheath, although Spieghel intended it to be the line of transition between the muscular fibers and the aponeurosis of the transversus abdominis muscle. These two lines define a "semilunar zone" from 0.3 to 3.7 cm wide, within which a Spigelian hernia may take place, anywhere from above the level of the umbilicus to the symphysis pubis.
If the hernia lies at the intersection of the linea semilunaris (Spieghel) and the linea semicircularis (Douglas), the ring is formed by the aponeurosis of the internal oblique muscle and the aponeurosis of the transversus abdominis muscle. If the hernia is above the linea semicircularis, but below the umbilicus, the ring is almost the same. If the hernia is above the level of the umbilicus, the defect is formed by a tear in the transversus abdominis muscle and a defect of the aponeurosis of the internal oblique muscle. The neck of the hernia may be tough and rigid; the sac may be covered by properitoneal fat.

A transverse incision should be made just above the swelling, keeping in mind that the sac is under the aponeurosis of the external oblique muscle which should be carefully incised. An oblique incision of the aponeurosis following the direction of the fibers will be anatomically most sound. The procedures for umbilical herniorrhaphy should be followed.

**Incarcerated Gastrochisis**

This is a congenital defect of the body wall always to the right or left of the midline, the ring being formed by all layers of the abdominal wall. The defect occasionally needs to be enlarged by incising the lower edge of the ring if the opening is too small to permit reduction of the herniated intestinal loops. The capacity of the abdominal cavity rather than the diameter of a constricting ring, is the usual problem.

**Incarcerated Paraduodenal Hernias**

**Incarcerated Right Paraduodenal Hernia.** The mouth of the sac lies behind the superior mesenteric artery or the ileocolic artery at the base of the mesentery of the small intestine (mesentericoparietal fossa of Waldeyer). The mouth opens to the left. The sac is directed to the right and usually lies in the retroperitoneal space behind the right mesocolon or transverse mesocolon. The boundaries are: superior, the duodenum; anterior, the superior mesenteric artery or ileocolic artery; posterior, the lumbar vertebrae.

To avoid vascular injury the incision must be in the lower part of the mouth. If vascular damage appears inevitable, the surgeon should open the mesentery and decompress the proximal intestinal loop before attempting reduction.

**Incarcerated Left Paraduodenal Hernia.** The mouth of the sac usually lies behind the inferior mesenteric vein and the left colic artery, at the left of the 4th part of the duodenum, at the duodenojejunal flexure. The mouth opens to the right. The sac is directed to the left and usually lies in the retroperitoneal space behind the left mesocolon. The boundaries are: superior, the duodenojejunal flexure or the beginning of the jejunum, pancreas and renal vessels; anterior, the inferior mesenteric vein and left colic artery; right, the aorta; left, the left kidney.

The incision should be made in the lower part of the mouth. McNair advises division of the inferior mesenteric vein. A downward incision of the mouth will avoid this sacrifice.
Incarcerated Transomental Hernia

The ring is formed entirely by omentum. A vessel will usually lie in one edge of the defect. Incise the omentum after clamping.

Incarcerated Transmesenteric Hernias

The mesenteric defect may be located in: (a) the mesentery of the small intestine, (b) the transverse mesocolon, (c) the sigmoid mesocolon. At least one free edge of the ring is usually formed by a branch of the superior mesenteric or inferior mesenteric artery. Since there is no sac and the obstructed loop is visible, decompress the dilated loop only and do not incise the neck. Vascular injury ad portas must be avoided.

Incarcerated Hernia Through The Epiploic Foramen Of Winslow

The boundaries of the foramen are: superior, the caudate process of the liver and the inferior layer of the coronary ligament; anterior, the hepatoduodenal ligament containing the portal vein, the hepatic artery, and the common bile duct; the cystic duct is also present in the free edge of the lesser omentum; posterior, the inferior vena cava; inferior, the first part of the duodenum and the transverse part of the hepatic artery.

For the incision open the hepatogastric omentum after clamping, and decompress the proximal intestinal loop. Do not incise the neck; the authors do not advise ligation of the hepatic artery. (Remember that there is no excuse for dividing the neck of the sac.)

Incarcerated Retroanastomotic Hernias

Markowitz is the authority on these iatrogenic hernias: "Construction of each gastrojejunostomy, either as a procedure by itself or as a step in the performance of a partial gastrectomy, results in the formation of an internal hernia ring which may be the cause of severe difficulties in the immediate or distant postoperative period."

Hernia Following Retrocolic Gastrojejunostomy. The boundaries of the ring are: anterior, the gastrojejunostomy and the efferent or afferent jejunal loop, depending on whether the afferent loop is attached to the lesser or the greater curvature of the stomach; posterior, the posterior parietal peritoneum; superior, the transverse mesocolon and posterior wall of the gastric remnant; inferior, the ligament of Treitz and the duodenojejunal peritoneal fold.

No incision should be made. An enterostomy should be performed to facilitate reduction of the loop. Recurrence is prevented by closure of the ring.

Hernia Following Antecolic Gastrojejunostomy. With the afferent loop attached to the greater curvature of the stomach, the boundaries of the ring are: anterior, the gastrojejunostomy and the afferent jejunal loop; posterior, the omentum and mesocolon; superior, the transverse colon and mesocolon; inferior, the ligament of Treitz and the duodenojejunal peritoneal fold.
With the afferent loop attached to the lesser curvature of the stomach, the boundaries of the ring are: *anterior*, the afferent jejunal loop with its mesentery; *posterior*, the omentum, transverse colon, and mesocolon; *superior*, the gastrojejunostomy, ligament of Treitz, and the duodenojejunal peritoneal fold; *inferior*, the jejunum with its mesentery. No incision of the ring is required.

**Incarcerated Diaphragmatic Hernias**

**Incarcerated Posterolateral Hernia (Hernia Through the Foramen of Bochdalek).** This hernia may be congenital or traumatic and is more common on the left side. The congenital defect, and hence the ring, is located in the posterior portion of the diaphragm, usually where it is attached to the 10th and 11th ribs. In 90 per cent of cases there is no hernial sac.

The choice of approach to the diaphragm in any particular case is beyond the scope of this chapter. The surgeon should always be prepared to perform a thoracoabdominal incision should an abdominal approach prove inadequate.

The diaphragm should be incised laterally from the margin of the ring toward the periphery. The surgeon should be familiar with Merendino's description of incisions that will avoid severing branches of the phrenic nerve. Bear in mind that if a sac is present, it should be excised.

**Incarcerated Anterior Hernia of the Diaphragm (Parasternal Hernia, Hernia Through the Foramen of Morgagni).** The diaphragm attaches to the xiphoid process by two narrow slips on either side of the midline and to the seventh costal cartilage just lateral to the xiphoid process. This attachment leaves three gaps, one medial and two lateral. The superior epigastric vessels pass through the lateral gaps, anterior to the diaphragm, together with some lymphatic channels. The gaps themselves are the sternocostal triangles, the foramina of Morgagni, or the spaces of Larrey. The hernial ring is thus formed anteriorly by the costal cartilage and the xiphoid process, and laterally, posteriorly, and perhaps medially by the diaphragm. There may or may not be a peritoneal sac. The right lateral foramen is the usual site of herniation.

According to Nakayama and Yanagisawa, the approach should be abdominal in the newborn and in children. A thoracoabdominal approach is preferred in adults. The diaphragm should be incised laterally along the costal margin only far enough to release the incarcerated loop.

**Incarcerated Hiatal Hernia.** In about 50 per cent of patients, the esophageal hiatus is formed by the right and left limbs of the *right crus* of the diaphragm. In the remainder of patients, both the *right* and *left crura* participate in the formation of the hiatus.

In the patient with hiatal hernia, the fibrous attachments of the esophagus to the diaphragmatic ring are altered, and the relations in the newborn and in the adult differ. The details of these differences are highly controversial and are beyond the scope of this paper.
The ring must be considered to be muscular, although two inconstant ligaments should be mentioned: *anterior*, the transverse ligament of the central tendon, which is the point of union of the two limbs of the right crus; *posterior*, the median arcuate ligament joining the crura just above the celiac axis.

The left lateral incision must be toward the central tendon to avoid injury to the phrenic nerve. Remember that the left phrenic nerve supplies the left half of the right crus as well as the left leaf and left crus of the diaphragm, and that a hernial sac is always present.

**Incarcerated Aortohiatal Hernia.** In many patients the median arcuate ligament which forms the posterior boundary of the hiatal hernia ring is absent. As the transverse ligament of the central tendon of the diaphragm is strong, enlargement of the hiatus can take place only posteriorly, occasionally reaching the aortic hiatus. In such cases the aorta becomes the posterior boundary of the ring.

Incision should be made as in hiatal hernia.

**Summary**

An incarcerated hernia must be reduced before any attempt at repair can be made. We have described the surgical anatomy of the ring in most of the hernias of the abdomen, both internal and external. In addition we have described the incision or other procedure required for safe reduction of the hernia. We believe that this information about "where to cut," none of it new, has not been previously assembled.
Anatomic Basis of the Surgical Treatment of Portal Hypertension

Atef A. Salam, W. Dean Warren


Since the demonstration by Whipple and his associates, in 1945, that a portacaval shunt can be performed successfully in man, anatomy of the portal venous system has been a subject of considerable interest to the surgeon. This interest has been further stimulated by the more recent advances in portal angiography and by the introduction of newer techniques for the construction of portasystemic shunts. The discussion which follows deals with vascular anatomy as it relates to the modern concepts in pathophysiology and therapy of portal hypertension.

Intrahepatic Circulation

Kiernan, in 1833, introduced the concept of the hepatic lobule as the unit of liver architecture. He described circumscribed pyramidal lobes consisting of a central tributary of the hepatic vein and, at the periphery, a portal tract containing bile duct, portal vein radicle, and hepatic artery branch. Columns of liver cells and blood containing sinusoids extended between these systems. The existence of the hexagonal lobule has been recently questioned. Rappaport described a microcirculatory unit which consists of a terminal portal vein and a glomus of sinusoids branching off from it. The associated hepatic arteriole forms a plexus from which efferent arterial capillaries join the sinusoids close to the terminal portal venule. This microcirculatory unit provides the vascular framework for the structural and functional hepatic unit, the liver acinus. According to this concept, a simple liver lies between one or more terminal hepatic venules.

Intrahepatic obstruction to portal blood flow may result from inflammatory obliteration or from distortion by regenerating nodules at any part of the microcirculatory unit. Portal hypertension is conveniently divided into two major categories on the basis of whether the obstruction to flow occurs before (presinusoidal) or in and beyond (sinusoidal and postsinusoidal) the hepatic sinusoid. In hepatic schistosomiasis, fibrotic obliteration of the terminal venules secondary to inflammatory reaction around the ova leads to presinusoidal portal hypertension. Conversely, in alcoholic or postnecrotic cirrhosis, the block to portal blood flow occurs at or beyond the hepatic sinusoid. A possible explanation for the location of the circulatory block in these two types of cirrhosis is offered by Rappaport's thesis, which indicates that the cells of the hepatic acinus lying between the terminal part of the sinusoids are more vulnerable to hepatotoxins because of a lower PO₂ in the periphery of the microcirculatory unit.

Hepatic vein catheterization is used to study the intrahepatic hemodynamic changes in patients with portal hypertension. An endhole catheter, introduced via the brachial or femoral vein, is advanced until its tip is wedged into a peripheral hepatic venule. Wedged hepatic pressure as determined by this technique is an indirect measure of the sinusoidal pressure and is therefore elevated in patients with postsinusoidal portal hypertension. The combination of a normal wedged hepatic pressure and an elevated intrasplenic pressure is characteristic of presinusoidal portal hypertension. Hepatic catheterization is also used to study
the changes in hepatic blood flow. Under normal conditions, when contrast medium is injected into a hepatic lobule, the sinusoidal bed is opacified and the contrast medium fills the corresponding hepatic venule. With normal or near normal portal venous perfusion, none of the radicles of the portal vein are seen. In cirrhotic patients with severe restriction of portal blood flow, a wedged hepatic venogram may show retrograde filling of the intrahepatic branches of the portal vein. The extent of this filling is proportionate to the extent of reduction in portal blood flow, and in extreme cases where the direction of portal blood flow is completely reversed, the main trunk of the portal vein may be visualized. In the presence of reversal of flow, splenoportography frequently fails to visualize the portal vein, and in such cases hepatic wedged venography is most valuable in establishing the patency of that vein.

Collateral Circulation

General Considerations

The portal circulatory bed is unique in being a conduit from one set of capillaries to another. When the portal circulation is obstructed, whether it be within or outside the liver, collateral circulation develops between the portal and systemic veins. The most important site is the lower end of the esophagus, where opening of collaterals between the coronary vein of the portal system and the azygous veins of the caval system leads to the formation of esophageal varices. Bleeding from these varices accounts for the death of approximately one-third of patients with liver cirrhosis. The opening of collateral circulation between the superior and inferior hemorrhoidal veins explains the high incidence of hemorrhoids in patients with portal hypertension. Similarly, collaterals form in the retroperitoneal tissue and across parietal adhesions caused by previous abdominal operations. Because of this increased vascularity, unnecessary retroperitoneal dissection or lysing of adhesions should be avoided in patients with portal hypertension.

The umbilical vein, which is the relic of the umbilical circulation in the fetus, is often dilated and tortuous in patients with portal hypertension. Transumbilical catheterization of the portal vein has been advocated as an alternative approach to evaluate the hemodynamic and angiographic changes in the portal venous system in cirrhotic patients. This technique requires only local anesthesia, and it is free from the hemorrhagic risks of percutaneous splenic puncture. It is particularly useful in splenectomized patients presenting with recurrent variceal bleeding.

Collateral Circulation and Hepatic Blood Flow

Studies in cirrhotic patients have shown that the effect of spontaneous diversion of portal blood by collateral channels on total hepatic blood flow varies considerably among patients with an apparently similar clinical condition. At one extreme is the patient in whom there is normal or near normal total hepatic blood flow, with the great majority of portal venous flow shown by angiography to perfuse the liver. At the other extreme is the patient in whom there is moderate depression of hepatic blood flow, with complete cessation of portal venous perfusion of the liver and spontaneous reversal of flow in the portal vein. Following a conventional portacaval shunt, the portal blood is completely diverted away from the liver into the inferior vena cava. This total loss of portal blood flow to the liver leads to a more substantial drop in total blood supply of the liver in patients with high hepatic portal blood
flow, as compared to those in whom most of the portal blood has already been spontaneously diverted to the systemic circulation. Several studies have shown that the patients who suffer the most severe reduction in liver blood are the ones who tolerate the portacaval shunt poorly. This observation supports the thesis that total loss of portal venous perfusion to the liver is related to the increased incidence of encephalopathy and accelerated rate of liver failure in the postshunt patients.

Collateral Circulation in Extrahepatic Portal Hypertension

Portal Vein Occlusion

Portal vein occlusion may occur as a complication of cirrhosis, infection, tumors, or pancreatitis. Most commonly, however, the history and physical examination do not suggest any specific etiologic factor. Developmental aberrations of the portal vein, extension of the normal obliterative process from the umbilical vein and ductus venosus, and unrecognized postpartum thrombosis secondary to omphalitis or generalized sepsis have all been proposed as possible mechanisms of portal vein occlusion in these cases. In patients with extrahepatic portal venous obstruction, collateral circulation develops not only with the systemic veins, but also between the tributaries of the portal vein and the intrahepatic vascular bed past the obstruction. The distribution of these portaportal collaterals varies according to the extent of the obstructing process. Angiographically, three types of portal venous obstruction can be recognized. Type I, where occlusion is limited to the hilum of the liver, is the least common form and is characterized by the formation of hilar caput medusae and marked enlargement of the cystic vein. In Type II the entire trunk of the portal vein is occluded and replaced by multiple racemose channels, the so-called cavernous malformations of the portal vein. It is not known whether these channels represent enlargement of the perivascular vessels (vena comitantes) into collateral channels or recanalization of thrombosed segments of the portal vein. In Type III, the portal, superior mesenteric, and splenic veins are occluded. The extent of the occlusive process determines the choice of operative procedure for the treatment of these patients. A portacaval shunt is only feasible in Type I, but in Type II patients, where the mesosplenic junction is not involved, both compartments of the portal bed can be effectively decompressed by a mesocaval or standard splenorenal shunt. Type III patients are not usually suitable for any form of conventional portasystemic shunt, but occasionally a makeshift shunt can be made between a large collateral vein and the inferior vena cava or the renal vein. The main disadvantage of this type of shunt is the high incidence of shunt thrombosis because of the limited blood flow in such collateral veins.

Splenic Vein Occlusion

Normally, no cross circulation can be demonstrated angiographically between the splenic and the superior mesenteric compartments of the portal circulatory bed. In patients with splenic vein thrombosis, a compartmental form of portal hypertension develops and a pressure gradient is created between the splenic and the superior mesenteric venous beds. Collateral channels thus form not only with the systemic veins of the esophagus and retroperitoneal tissue, but also with the tributaries of the superior mesenteric and portal veins. Normally, the fundus and part of the body of the stomach are drained by the short gastric and left gastroepiploic branches of the splenic vein. An imaginary line between the esophagus and the midpoint of the greater curvature marks the boundaries between this zone and that portion
of the stomach which is drained by branches of the portal vein. In splenic vein thrombosis, a pressure gradient develops across this line, and splenoportal collaterals form both within and outside the stomach wall. Intrinsic gastric collaterals lead to the formation of gastric and duodenal varices, which are frequently the source of bleeding in this syndrome. Unless their presence is suspected, the endoscopist may mistake these varices for hemorrhagic gastritis, particularly if the esophagus is not involved. The extragastric component of splenoportal collateral circulation is mainly represented by the left and right gastroepiploic veins, which together form one venous arch connecting the splenic to the portal vein. In our experience, dilation of the gastroepiploic veins is constantly seen in patients with splenic vein thrombosis, and it is the most characteristic single feature of this syndrome. Angiographically, these changes in vascular anatomy are shown as nonvisualization of the splenic vein and marked dilation of the gastroepiploic veins. In contrast to generalized portal hypertension, the varices are seen early in the study, while the coronary and portal veins are seen only in the late films. Since the direction of blood flow in these cases is from the varices to the portal vein, neither the coronary vein nor the varices can be seen on a portal venogram obtained either indirectly by selective injection of the superior mesenteric artery, or directly by operative cannulation and injection of a branch of the superior mesenteric vein.

Splenic vein thrombosis is a rare form of portal hypertension which is cured by splenectomy. Since it is most commonly seen as a complication of alcoholic pancreatitis, a history of heavy drinking is frequently obtained, and variceal bleeding in these patients may be erroneously attributed to alcoholic cirrhosis. An ill-advised portacaval shunt based on such a misdiagnosis would unnecessarily deprive the liver of its portal blood supply. During an emergency operation for gastrointestinal bleeding, the finding of a normal liver, an enlarged spleen, and a markedly dilated gastroepiploic vein should alert suspicion of this condition. The diagnosis is confirmed if, upon catheterization of the portal vein, both the pressure and the venographic appearance of the vessel are shown to be normal.

Anatomic Basis of the Newer Portasystemic Shunt Operations

Selective Distal Splenorenal Shunt

The concept of selective variceal decompression is based on two premises: first, that the splenic and the mesenteric compartments of the portacaval bed are functionally separate; second, that continued hepatic perfusion with mesenteric and portal venous blood enhances the metabolic efficiency of the liver, and prevents or greatly modifies the tendency towards the progressive hepatic deterioration so frequently seen when all the portal blood is diverted away from the liver. The selective shunt is designed to achieve selective decompression of the splenic compartment and the variceal bed without interfering with the flow of mesenteric and portal blood to the liver. In this operation, the spleen is left in situ, the splenic vein is divided, its hepatic end oversewn, and its distal end anastomosed end-to-side to the left renal vein. Variceal decompression is achieved by relieving the pressure in the short gastric and left gastroepiploic branches of the splenic vein which carry the venous return of the stomach to the shunt instead of the esophageal or gastric varices. By maintaining the high venous pressure in the superior mesenteric and portal veins, portal perfusion of the cirrhotic liver is preserved. This is a unique feature which is not shared by any other form of portasystemic shunt. In the selective shunt, it is most important to disconnect the splenic and the superior mesenteric compartments as effectively as possible, in order to prevent or minimize retrograde
flow of portal and mesenteric blood to the decompressed splenic venous bed. In this respect, obliteration of the coronary vein is especially important to prevent retrograde flow of portal blood via this vein to the decompressed variceal bed. The right gastroepiploic vein and the inferior mesenteric vein should also be interrupted because of their free communication with the two compartments of the portal circulatory bed.

**Mesocaval and Mesorenal Interposition Shunt**

Marion and Clatworthy originally described a technique for operative decompression of the portal circulatory bed in which the inferior vena cava is transected and its central end anastomosed end-to-side to the superior mesenteric vein. Because of the morbidity associated with interruption of the inferior vena cava, particularly in adults, a modification of this technique has been recently suggested in which a Dacron graft is interposed between the superior vena cava and the superior mesenteric vein. This mesocaval interposition shunt (M.C.I.S.) is hemodynamically similar but technically easier than the conventional portacaval shunt. The superior mesenteric vein is more accessible than the portal vein, and exposure of the inferior vena cava is less difficult below than above the duodenum, where it is often hidden by an enlarged caudate lobe. Although the graft patency rate in preliminary studies of the mesocaval interposition shunt has been favorable, final assessment of this procedure requires a longer period of observation. In our experience, the mesocaval interposition shunt requires a relatively long graft because of the wide separation between the two veins and because the graft has to be swung around the intervening third part of the duodenum. To overcome these difficulties, we have used an alternative technique in which the graft is interposed between the superior mesenteric vein and the left renal vein. A comparative evaluation of mesorenal versus mesocaval interposition shunt in cadavers, and at the time of operation, has shown that a relatively short graft is needed to cross the gap between the superior mesenteric vein and left renal vein (3.5 cm versus 9.5 cm for the mesocaval shunt). In no instance has it been necessary to lay the graft over or around the duodenum because of the ease with which the duodenojejunal junction can be mobilized, as opposed to relatively fixed third part of the duodenum. Thus, the potential danger of graft kinking by the duodenum or duodenal compression by the graft is eliminated. In addition, the mesenteric anastomosis in the mesorenal shunt is made on the more accessible left side of the vein close to its junction with the splenic vein, whereas in the mesocaval shunt the graft is anastomosed to the right side of the vein at a more peripheral level because of the uncinate process of the pancreas. Based on our anatomic studies, it is estimated that bifurcation or major branching of the superior mesenteric vein would interfere with the proper construction of the mesocaval shunt in approximately one-third of the cases. This is not a problem in the more centrally located mesorenal shunt.
The Unsung Virtues of Ligaments

John V. Basmajian


Modestly laboring behind the scenes, ligaments are often overlooked or disdained while attention focuses on muscles, vessels, and nerves, the dynamic actors on center stage. Yet this attitude - held by surgeons and anatomists alike - is both naive and wrong. Ligaments are often more important than muscles, and in some places where muscles are given the credit, unassuming ligaments carry the entire burden of responsibility. But since their demands are small, we tend to overlook their virtues.

Dynamic and Postural Roles

As one who has devoted a professional lifetime to the electromyographic study of muscles in health and disease, I have become increasingly impressed with the dynamic and postural roles of ligaments. This awareness grew as more and more studies showed inactivity of muscles where my colleagues and I had expected activity. Very early, a principle emerged that stated in its simplest form: Muscles are spared where ligaments suffice. This is true in both postural and dynamic situations; in the pathologic situation, disturbances of ligaments lead to various secondary neuromuscular changes which again create more stir than the loss of function in the ligament.

The quiet and efficient work of ligaments allows for the operation of two laws of muscular function which MacConaill and I proposed.

1. The law of minimal spurt action: No more muscle fibers are brought into action than are both necessary and sufficient to stabilize or move a bone against gravity or other resistant forces, and none are used insofar as gravity can supply the motive force for movement.

2. The law of minimal shunt action: Only such muscle fibers are used as are necessary and sufficient to ensure that the transarticular force directed toward a joint is equal to the weight of the stabilized or moving part together with such additional centripetal force as may be required because of the velocity of the part when it is in motion. That these two laws are valid has been demonstrated clearly by electromyography. In fact, it is the study of muscle that indirectly revealed the role of ligaments.

Contrary to expectation, we found that the vertically running muscles that cross the shoulder and elbow joints are not active to prevent distraction of these joints by gravity. Much more surprising is the fact that they do not spring into action when light, moderate, or even heavy loads are added unless the subject voluntarily decides to flex his shoulders or his elbow and thus to support the weight in bent positions of these joints. Quite often he may do this intermittently or, when uninstructed, from the very onset. But it must be clear that such muscular action is a voluntary action and not a reflex one.
Carlsöö and Guharay confirmed our findings of muscular inactivity in the heavily loaded shoulder and elbow joints - the muscles being biceps, triceps, brachialis, and brachioradialis. In addition, they found that the temperature fell in these muscles, apparently because of a lower oxygen demand.

Even while the muscles were quiescent, our subjects rapidly felt local fatigue. What, then, is fatigue in the heavily loaded limb? Normally, it would be thought of as 'muscular fatigue' but we seen now that this is incorrect. The 'fatigue' that is experienced probably originates from the painful feeling of tension in the articular capsule and ligaments, not from overworked muscles. In fact, as we have seen, the muscles need not be working at all.

An analogous situation occurs in the foot where we found, some years ago, that the muscles that are usually supposed to support the arches continuously were generally inactive in standing at rest. Independently Hicks showed by deduction that the plantar aponeurosis and plantar ligaments were the chief weight-bearers in this position. It would seem, then, that in the normal foot the fatigue of standing is not a muscular phenomenon.

The dual conclusion that articular ligaments suffice to prevent the downward distraction of joints in the upper limb and that fatigue is chiefly a form of pain in the ligaments appears to be of fundamental importance. It not only runs counter to 'common sense' but it is of practical interest, for example, in explaining why dislocations by traction on normal limbs are rare. It should be noted especially that the capsule on the superior part of the shoulder joint including the corachohumeral ligament is extremely thin only when the arm hangs directly downward and the scapula is in its normal position. The special mechanism that includes this ligament together with the supraspinous muscle and the normal slope of the glenoid cavity has been described by me elsewhere. When the shoulder joint is abducted or flexed, however, the capsule is extremely loose and the shoulder joint depends for its integrity on the well-known "rotator-cuff" muscles (see below).

All the experiments reported above finally led to a fascinating one that in turn has led to new ideas. Elkus and I found that healthy subjects suspended by their hands from a trapeze can hold on for less than three minutes even when their fists are kept closed by a special gauntlet. Severe discomfort in the hands (when no gauntlet was used) was the main cause for failure. Action potentials from a large number of muscles were unremarkable and all evidence pointed to a significant ligamentous force rather than muscles preventing articular distraction. Similar EMG studies by Tuttle and myself on apes (gorilla, chimpanzee, and orangutan) confirm these findings.

Quite independent from us, a group in Sweden was arriving at similar conclusions in regard to ligament-sparing from a different type of experiment in cats and man. Thus, when Andersson and Stener greatly increased the tension in the medial ligament of the knee of the cat in specially designed experiments, no reflex muscular contractions appeared in the muscles of the thigh as would have been expected if the usual hypothesis of "ligamento-muscular protective reflexes" were valid. Furthermore, they showed convincingly that the absence of reflex motor effects was not due to the absence of afferent discharges which were well registered from the articular nerves.
Petersén and Stener carried the above experiments forward to human subjects, again using the medial ligament of the knee. Their results were a complete vindication of the conclusions made in the animal experiments described previously. In addition, their work suggests that if injured ligaments are pulled till pain results, muscles do show reflex contraction, but if the torn ligament is then anesthetized, they do not.

Following almost the same line of reasoning, deAndrade, Grant, and Dixon distended human knees with non-irritating plasma (which emphasized the pressure phenomena opposed to pain). There was a definite and even marked inhibition of quadriceps contraction with a depression of motor unit activity. This is undoubtedly a reflex inhibition and helps further to explain the muscle weakness, atrophy, and deformity that follow knee injury and disease.

Freeman and Wyke obtained a definite and chronic drop in reflex postural tonus in cats by cutting the sensory nerve supply of the knee joint capsule. The mechanoreceptors in the joint are involved in reflex muscular activity to maintain posture in quadrupeds; undoubtedly the same mechanisms occur in man as well. All the above observations are surely of great importance in orthopedics and surgery of joint injury; they deserve wide attention.

Specific Functions of Selected Ligaments

Axial Skeleton

Spinal Column

The presence of anterior and posterior longitudinal ligaments, binding the fronts and the backs of the vertebral bodies to one another throughout the length of the column, safeguards the movements of the column as a whole. The vertebral arches possess restraining ligaments also. One series consists of the ligamenta flava, rich in yellow elastic fibers; they stretch between the adjacent laminae of the vertebral arches. Being elastic, these ligaments tend to restore the spinal column to a neutral position after it has been flexed. They also serve, with the laminae, to cover the spinal canal posteriorly and so protect the contained spinal cord. A second series unites adjacent spinous processes as interspinous ligaments. Contiguous with these posteriorly are longer fibers which stretch the length of several spines and are, in consequence, supraspinous ligaments. These have the same effect as the ligamenta flava. Undoubtedly, they relieve the back muscles of considerable work.

The amount of movement permitted between two adjacent bodies also depends on the thickness of the intervening disc, itself a modified 'ligament.' Cervical and lumbar discs are thick, thoracic discs are thin; movements, therefore, are freest in the neck and in the lumbar region, and this must be as true for the joints of the vertebral arches as it is for the bodies.

Ligaments of Atlas and Axis

When the 'No' movement occurs, the skull and atlas move on the axis as a unit and the third joint, which is at the center of the movement, plays an important part. This joint, the median atlantoaxial joint, is a pivot. The dens, projecting upwards from the body of the axis,
rotates in a collar formed by the anterior arch of the atlas and the transverse ligament. A pair of strong, short, check or alar ligaments stretches between the tip of the dens and the skull at the margin of the foramen magnum and serves to limit the rotation of the head. They become taut also in head flexion.

The pivot-and-collar mechanism is completely covered and hidden from view by a broad ligamentous band, the tectorial membrane; it is the upward and expanded prolongation of the posterior longitudinal ligament that binds together the backs of all the vertebral bodies and discs. The tectorial membrane is attached above to the anterior margin of the foramen magnum and offers a smooth and continuous sloping surface for the support of the brain stem and spinal cord.

Anterior and posterior atlantoaxial membranes have similar dispositions to the atlanto-occipital ones. Movements between the skull and the atlas as well as those between the atlas and the axis are, of course, augmented by the flexibility of the remainder of the cervical column.

Upper Limb

Ligaments of Clavicle

Taken too much for granted, the ligaments of the clavicle are truly so important that they determine the very fundamentals of upper limb, and so manual, function. Fortunately, they are seldom seriously injured. The force-transmitting function of the massive coracoclavicular ligament is probably too well known to require emphasis here. The play of forces at the medial end of the clavicle is less well known - and more complicated. The joint surfaces of the sternoclavicular joint are not reciprocally shaped - the "fit" is terrible! Contact of the otherwise ill-fitting surfaces is improved by the existence of a disc. However, this disc, which is really a ligament in disguise, is much more occupied with the duties of absorbing the forces transmitted to the joint along the clavicle from the shoulder region and preventing the clavicle from being driven out of its socket onto the summit of the sternum. The disc can discharge these duties because it is firmly attached - above, to the clavicle; below, to the first costal cartilage. The rarity of dislocation of the joint testifies to the efficiency of the disc in discharging its duties, even when a hole is worn in its middle with advanced age.

Excessive protraction and elevation of the clavicle are prevented by the existence of two structures. One, a ligament, is fundamental; the other, a muscle, is much less important. Immediately lateral to the sternoclavicular joint, the clavicle is bound to the first costal cartilage by the costoclavicular ligament. Being near the center of movement and at first somewhat lax, this ligament allows the lateral end of the clavicle considerable excursion in both protraction and elevation before it finally becomes so taut as to bring further movement to a halt. Lateral to the ligament, acting as a 'dynamic ligament,' the subclavius muscle reinforces the costoclavicular ligament.
Shoulder Joint

As already indicated, the capsule of the shoulder joint plays a vital role in weight-bearing. No other joint has such a loose capsule and is so ill equipped with restraining ligaments. The anterior part of the capsule contains three slight thickenings, the glenohumeral ligaments, which are of doubtful significance. But a ligament with very great significance is the coracohumeral ligament extending from the coracoid process to the greater tuberosity of the humerus. Only its anterior edge may be obvious to the surgeon or anatomist; the remainder blends with, and thickens, the upper part of the fibrous capsule. While this ligament is usually described as becoming taut on lateral rotation (true), it is also taut when the arm hangs vertically because it is part of the superior part of the capsule. It forms a locking mechanism, which, assisted by the supraspinatus muscle, prevents downward dislocation of the humeral head.

When the arm is held directly overhead, the inferior capsule of the shoulder joint becomes extremely taut and then it acts as the restraint. Our experiments indicate that this is effective in the short term with no special fatigue reported in the shoulder region by subjects hanging by their hands. (They let go because of the pain in the hands and forearms.) In the intermediate positions of abduction and/or flexion, whatever security the joint possesses it derives from the rotator cuff muscles that surround and move it, not from ligaments. They pass to their insertions on the two tuberosities immediately adjacent to the head of the humerus. Blending with the capsule and reinforcing it, they deserve the designation of 'dynamic ligaments.' But, as any surgeon can attest, they fail to prevent dislocations in all too many persons.

Elbow and Forearm

Every hinge joint (including the elbow) has a thin loose capsule except at its two sides where the capsule is greatly thickened to form collateral ligaments. The medial (ulnar collateral) ligament is fan-shaped. Fastened to the medial epicondyle above, it spreads to the medial edge of the trochlear notch of the ulna. The anterior part is thickened to form a cord which plays the biggest part in the prevention of abduction at the elbow. The lateral (radial collateral) ligament runs from the lateral epicondyle to the outer surface of the anular ligament and so does not impede supination and pronation. Since the anular ligament grasps the head of the radius firmly in the adult, adduction is effectively prohibited to the radial collateral ligament. (For pedantic reasons, the spelling of "annular" has been changed officially to "anular.")

The fibers of the interosseous radioulnar membrane take a direction downwards and medialwards, a fact which falsely suggests that forces received from the hand by the lower end of the radius are transmitted on their passage up the forearm by the interosseous membrane to the ulna. This function of the membrane has been overemphasized since the hand is usually in a position of pronation when forces are applied to it, and in pronation the interosseous membrane is rather slack. Moreover, there seems to be no good reason why the radius itself should be unable to transmit forces directly to the humerus. The more important use of the membrane would seem to be to increase the area available, both at the front and at the back, for the origins of numerous muscles found in the forearm. Further, the articular
Disc of the inferior radioulnar joint is also a force transmitter in addition to its role of stabilizing the radius and ulna while allowing pronation-supination.

**Carpus and Hand**

In this brief review, space is insufficient to cover the detailed multifarious functions of the many joints and ligaments. A book could be written about them. Only reference will be made to a universal special function of the capsular and interosseous ligaments of the area, viz, their role in producing close-packing. As MacConaill and I have described in detail, every synovial joint contains at least one mating pair of articular surfaces, one of these being male, the other female. When there is more than one mating pair, the joint is 'compound.' When there is an intra-articular disc, the joint is 'complex.'

When one bone articulates with two others within one and the same compound joint, it will have two articular surfaces, one for each of the other two bones. We thus have two mating pairs. For example, the lower end of the radius articulates with the scaphoid and the lunate bones of the radiocarpal joint. Each of these two bones has a male ovoid surface which moves on a corresponding and separate female ovoid surface on the lower end of the radius. Thus we have the two mating pairs that constitute the radioscaphoid and the radiolunate joints. There is an important rule governing the behavior of adjacent mating pairs within any compound joint: No male surface of one mating pair comes into articulation with the female surface of another mating pair. And it is the ligaments that enforce this monogamy!

In all positions except one of any mating pair of surfaces, they fit each other badly, ie, they are not fully congruent. We have called the position in which they are fully congruent the close-packed position. In this special position the female articular surface fits the corresponding (apposed) part of the male surface point-for-point. Moreover, the chief ligaments of the joint are so arranged that they screw the male and female surfaces together in a close-packed position in such a way that they cannot be pulled apart - whence the name. All other positions of a joint are called loose-packed. In the close-packed position we no longer have two bones functionally, only one, because they have been temporarily screwed together. The joints of the carpus and digits are excellent examples of these principles.

**Lower Limb**

**Sacroiliac Joint**

Behind and above the 'synovial' joint of the auricular surfaces a large area of the ilium is rough. Over this area, the sacrum and ilium are not in direct contact; a cleft exists between them. The cleft is filled with a heavy mass of fibers known as the interosseous sacroiliac ligament; behind the cleft, the fibers joining the bones are known as the dorsal sacroiliac ligament. As if even these were not enough, the summit of the crest of the ilium is united to the tip of the transverse process of the fifth lumbar vertebra by a strong band of fibers known as the iliolumbar ligament.

All of this ligamentous mass is under constant strain except when the body is recumbent. On it falls the duty of transmitting the weight of the body above to the hip bone.
By permitting only a minimal movement, the ligaments secure the integrity of the synovial joint. The latter is helpless alone, in spite of the interlocking character of its opposed surfaces.

**Hip joint**

The general functions of the fibrous structures around the hip joint are well described in standard texts. Perhaps it should be pointed out that maximum tautness of the iliofemoral ligament is not achieved until hyperextension is produced. Thus, the hip joint probably is not fully close-packed in the relaxed standing position. This would suggest some instability requiring continuous muscular activity during standing, which occurs appropriately in the iliopsoas muscle.

**Knee and Ankle Joints**

As with the section on the hand above, the many functions of the many ligamentous structures in the knee and ankle cannot be dealt with adequately here. While their structural characteristics - with notable exceptions - have been well covered in texts, their functions have been a matter of guesswork. I believe that many of the roles played by ligaments in the knee and ankle are related to achievement of close-pack. This and other functions require intensive research in these important areas. Descriptions of the fine detail (eg, of tendinous expansions) around the knee have appeared in recent years; important as the new reports are, they remain incomplete.

**Foot**

**Subtalar Joint.** The chief restraining ligaments of this joint are those of the ankle which span the talus to attach to the calcaneus. In addition there is the *interosseous talocalcanean ligament*. It lies in the tarsal sinus immediately in front of the subtalar joint and directly below the very center of the ankle joint. It holds the two bones together and, tightened by any undue movement between them, it limits inversion-eversion of the subtalar joint.

**Talocalcaneoclavicular Joint.** The main mass of the head of the talus projects insecurely in front of the sustentaculum. Here the head is capped by the navicular, between which and the sustentaculum is a triangular interval in which the talus hands its head and reaches its lower limit. A very strong ligament fills this interval and on it the head of the talus rests. This ligament, uniting the sustentaculum to the lower edge of the back of the navicular, is known as the *"spring" ligament* (or *plantar calcaneonavicular ligament*); on its strength and integrity depends, in large measure, the security of the whole foot. Should it be stretched and the interval between the sustentaculum tali and the navicular be widened, the head of the talus sinks in this interval and the foot becomes flat. The spring ligament, at the highest part of the medial longitudinal arch, is at the keystone of that arch. On the medial side of the joint, fibers of the *deltoid* (medial ligament of the ankle joint) reach to the navicular and blend with, and support, the medial edge of the spring ligament; they complete the medial wall of the socket. On the lateral side, the (functionally) much less important *bifurcate ligament* unites the calcaneus and the navicular.
**Calcaneocuboid Joint.** Just as the strong spring ligament binds the navicular to the calcaneus, so the strong, *short plantar ligament (inferior calcaneocuboid ligaments)* binds the cuboid to the calcaneus. Indeed, the two ligaments form an almost continuous structure across this vital region, the one supporting the keystone of the high medial longitudinal arch, the other supporting the keystone of the low lateral longitudinal arch.

**A Final Word**

The functional principles of the ligamentous structures require extensive and intensive exploration. But who is to do this? The practicing surgeons who read these pages are at the mercy of outdated textbooks and fragmentary research. Because the functioning ligaments in health is so smooth and trouble free, surgeons cannot realize their profound significance in the presence of joint disease and trauma - even when they themselves are uninjured. Today, this article well may be unique in the surgical literature in its attempt to throw a spotlight on these important supporting actors; if, however, it remains unique, it will have failed part of its purpose, the stimulation of renewed inquiry into the functional role of ligaments throughout the body in health and disease.
The radical neck dissection is a safe, effective therapeutic procedure for eradication of metastatic neoplasia originating in the head and neck region. Utilizing the steps of the radical neck dissections as an outline, we will present specific aspects of neck anatomy requisite to a complete and safe dissection.

**Lymphatics**

The purpose of the radical neck dissection is to remove the lymphatics and the drainage areas of primary cancers originating in the head and neck. Applying this principle of regional lymphatic excision (radical dissection), all cervical lymphatics and, in addition, certain adjacent muscles, nerves, and glands must be sacrificed to facilitate complete extirpation of the disease. A "partial" or upper neck dissection for carcinoma violates the lymph node bearing fields and negates the tenets of the en bloc dissection of cervical lymphatics.

Rouviere has classified the cervical lymph nodes into ten principal groups. They may be further recognized conceptually as a superior horizontal chain arranged about the base of the head and jaw, leading to a vertical chain in the midportion of the neck and another horizontally oriented chain in the supraclavicular region. The groups of nodes in the superior horizontal chain are the submental, submandibular, preauricular (parotid), postauricular (mastoid), and occipital. These nodes receive afferents from the integument of the head and mucous membranes of the nasal and oral cavities and the adjacent nasopharynx, oropharynx, tonsil region, and the salivary glands. Their efferents drain into the vertically oriented deep jugular nodes. This jugular chain, in addition, receives major afferents to its important subdigastric nodes from the deep parotid region, the nasopharynx, and the tonsil. A posteriorly and more superficially oriented vertical group is in relation to the spinal accessory nerve and the external jugular vein. An anteriorly oriented chain drains the cervical viscera and the thyroid gland. The supraclavicular horizontal chain receives afferents from the vertical chains immediately superior, the upper thoracic wall, and the upper extremity.

The primary lymphatic ducts deliver large volumes of lymph and chyle into the venous system, and if injured surgically should be securely ligated. On the right, three terminal lymphatic trunks empty into the junction of the right subclavian and the right internal jugular vein, either directly or indirectly. These trunks are the subclavian trunk from the supraclavicular nodes, the jugular trunk, and the bronchomediastinal trunk. It is unusual for these three trunks to coalesce to form a true right lymphatic duct. On the left, the chief collecting channel of the lymphatic system which drains all lymphatics from below the diaphragm, the left half of the thorax, the left side of the head and neck, and the left upper extremity is the thoracic duct. It may receive the jugular, subclavian, and bronchomediastinal trunks on the left side. Occasionally, however, one or more of these trunks may enter separately.
An en bloc resection of all the lymphatic bearing tissues will therefore require dissection from the inferior margin of the mandible to the clavicle and from the anterior aspect of the trapezius muscle to the muscles covering the cervical viscera. These tissues also lie between the deep investing cervical fascia and the prevertebral lamina of the deep cervical fascia. Of necessity, this en bloc resection includes the sternocleidomastoid muscle, the jugular vein, submaxillary gland, omohyoid muscle, spinal accessory nerve, the external jugular vein, and the tail of the parotid. Usually, the posterior belly of the digastric and the sternohyoid muscles are also included in the resection. If there is involvement in the submental region, the anterior belly of the digastric is taken.

**Incisions**

Cervical incisions should be designed to provide adequate exposure, avoid superimposition of intraoral, pharyngeal, or laryngeal incisions, and follow the natural transverse skin lines of the neck, thus favoring optimum healing and appearance. Particularly in cases which follow upon preoperative irradiation, these incisions should be adequately vascularized and so designed that the flaps are safe from necrosis or delayed healing over the carotid artery. The double transverse incision as described by MacFee is quite safe and meets these criteria admirably. Its upper limb extends at the level of the hyoid in a slight curve from the trapezius anteriorly to approximately midline. The lower incision is transverse 2.5 cm above the clavicle. An alternative incision has a vertical limb along the anterior trapezius and a single transverse limb extending anteriorly in the midportion of the neck. These incisions both avoid the oft used trifurcate incision of Crile and Martin which is so vulnerable to necrosis over the carotid artery. In order to gain access to the intraoral structures, the upper transverse limb of these incisions may be curved and extended to the midline of the lower lip, thus providing a well vascularized flap which can be rotated posteriorly.

The cervical nodes all lie deep to the investing layer of the deep cervical fascia. Therefore, the skin flaps are elevated immediately beneath the thin platysma muscle and just above the investing layer. The primary superficial veins - external jugular, anterior jugular, and facial - lie just beneath the platysma and superficial to the investing layer of the deep cervical fascia.

During elevation of the flaps, the sensory branches of the cervical plexus are divided as they emerge from the posterior aspect of the sternocleidomastoid muscle and penetrate the investing layer of the deep cervical fascia. These include the greater auricular nerve and branches C-2 through C-4. These cutaneous nerves will be divided again as they penetrate at the prevertebral lamina of the cervical fascia at the deep extent of the surgical field. Thus, a segment of them is included in the specimen. These nerves, particularly the greater auricular, are excellent for interposition donor grafts for nerve repairs such as the seventh.

**Root of the Neck**

Removal of the specimen always proceeds in centripetal fashion; in the manner the potential cancer-bearing lymphatics are not violated. The dissection usually beings in the supraclavicular area where the superficial or investing lamina of the deep cervical fascia over the sternocleidomastoid muscle is divided approximately 2 to 3 cm above the clavicle. This layer of fascia completely encompasses the neck and invests the sternocleidomastoid,
omohyoid, and trapezius muscles. It continues upward as the fascia of the parotid and submaxillary gland. The sternal and clavicular heads of the sternocleidomastoid muscle are divided at this level parallel to the clavicle. The carotid sheath, a condensation of the cervical fascia, is entered 2 to 3 cm above the clavicle and the jugular vein is isolated, carefully ligated, and divided after absolute identification of the adjacent vagus nerve and protection of the common carotid artery. The vagus nerve may inadvertently be lifted up with the jugular vein and carelessly included in the ligating sutures. Injury at this level will include those fibers which make up the recurrent laryngeal nerve.

The dissection then continues posterior in the supraclavicular triangle to the anterior border trapezius and down to the level of the prevertebral lamina. The main lymphatic trunks emptying into the junction of the subclavian and jugular veins are preserved; however, if injured they should be securely ligated. The external jugular vein, coursing to the subclavian vein, is ligated and divided and will be reflected upward with the specimen. The transverse cervical artery, a terminal branch of the thyrocervical trunk from the first portion of the subclavian artery, must be divided and included in the specimen along with the accompanying veins and lymph nodes about these vessels. The node-bearing fatty areolar tissue in the posteroinferior aspect of the supraclavicular triangle is included in the en bloc dissection. This posterior inferior limit of the dissection is the junction of the omohyoid and trapezius muscle.

The prevertebral lamina of the deep cervical fascia is well-defined and the specimen is dissected upward based on this fascia. It is important to remember that the brachial plexus, phrenic nerve, subclavian vessels, scalene muscles, and dome of the pleura are all just beneath this lamina. The spinal accessory nerve, the only major nerve that lies above the superficial lamina of the deep cervical fascia, is divided at its entrance to the trapezius muscle and is reflected upward with the specimen along with its accompanying chain of lymph nodes. On occasion the spinal accessory nerve may be preserved if there is no gross metastatic disease in the neck. The vessels not remaining with the specimen are ligated with fine permanent ligatures and are not cauterized because sudden increase in venous pressure from straining or a Valsalva maneuver tends to dislodge the cauterized thrombus leading to bleeding beneath the flaps.

As the dissection proceeds cephalad, the roots of the cervical plexus which emerge from the prevertebral lamina are identified. The phrenic nerve which is the only structure coursing from lateral to medial just beneath the prevertebral lamina is identified along the anterior aspect of the anterior scalene muscle. Its contribution from roots C-3 and C-5 must be accurately identified, and these cervical branches, which are quite substantial, are divided distal to their phrenic nerve contributions. The ansa hypoglossi, composed of the descendens hypoglossi from C-1 and the descendens cervicallis from C-2 and C-3, courses superficial to the carotid sheath to supply the sternohyoid, sternothyroid, and omohyoid muscles and must be divided to gain access to the deeper structures. The posterior aspect of the dissection is carried superiorly on the prevertebral fascia covering the splenius capitis and levator scapulae. Medially the jugular vein and its accompanying nodes are cleared from the carotid artery, vagus nerve, and posteriorly situated sympathetic trunk. The investing tissue over the carotid bulb is infiltrated with local anesthetic to preclude bradycardia and hypotension. Care must be taken lest undue traction be placed on the carotid artery during any of the dissection particularly in the elderly patient. An atheromatous plaque may fracture and loose microemboli of atheromatous material into the cerebral circulation.
The hypoglossal nerve is identified as looping over the external carotid artery beneath the posterior belly of the digastric muscle and the mylohyoid muscle to provide motor supply to the intrinsic and extrinsic tongue musculature, including the styloglossus and hyoglossus muscles.

The superior thyroid and external maxillary arteries are divided, care being taken to preserve the external laryngeal nerve which is just deep to the superior thyroid artery. This nerve originates at the nodose ganglion of the vagus and provides sensory innervation to the mucosa of the larynx and to the cricothyroid muscle. The ranine plexus of veins is in close relationship to the hypoglossal nerve. These veins are ordinarily quite small and inconspicuous. After distal ligation of the internal jugular vein, these ranine veins become engorged and distended. They must be handled carefully and securely lest undue bleeding obscure the field of dissection and lead to inadvertent injury of underlying structures. The external maxillary artery is divided as it approaches the submandibular triangle just beneath the posterior belly of the digastric. The omohyoid muscle is also divided from its origin on the hyoid; thus both bellies of the omohyoid are divided and it is included in the specimen in its entirety.

**Superior Aspect of the Dissection**

The en bloc specimen must be separated from the upper neck and mandible unless additional tissue is to be resected in continuity, such as primary tumors of the tongue, jaw, floor of mouth, or pharynx. The platysma, which is innervated by the lowermost fibers of the facial nerve, is divided along the inferior aspect of the mandible. In the absence of adjacent metastatic disease, the mandibular ramus of the 7th nerve, which supplies motor innervation to the lateral aspect of the lower lip, is identified and preserved. This branch of the facial nerve usually crosses above the posterior facial vein and its course is deep to the platysma. It usually crosses the external maxillary artery and anterior facial vein at or above the inferior border of the mandible. However, on occasion it may be up to 1 cm below the inferior border in relationship to these vessels. Nerve fibers to the platysma are frequently mistaken for the ramus mandibularis but are at a lower level in the neck.

The submandibular triangle is cleaned down to the anterior belly of the digastric muscle. If there is gross clinical disease in this region, the anterior belly of the digastric is divided at its mandibular insertion and reflected with the specimen. The floor of this dissection is the mylohyoid muscle. At the posterior border of the mylohyoid muscle, the external maxillary artery and vein are divided at the level of the lower border of the mandible. The mylohyoid muscle is retracted anteriorly and the lingual nerve, a terminal branch of the mandibular portion of the trigeminal, is identified high under the mandible while the submaxillary gland is retracted downward. Its two small branches to the submaxillary ganglion are divided, which permits removal of the submaxillary gland and the nodes within the submaxillary triangle. These tissues are cleaned down to the hyoglossus muscle and Wharton's duct is ligated and divided. The hypoglossal nerve posterior and inferior to the submaxillary gland is carefully preserved, unless the major portion of the tongue is to be removed to include a primary carcinoma.

Posteriorly the inferior pole of the parotid is divided in line with the lower mandible. The adjacent nodes are reflected downward to be included in the specimen. The posterior
facial vein within the parotid is identified and ligated at this time. The main trunk of the facial nerve is above this level and is not usually encountered. The sternocleidomastoid muscle is divided at its origin from the mastoid process and reflected downward. The posterior belly of the digastric and the stylohyoid, originating from the styloid process, are divided and reflected downward. This gives good access to the jugular vein near its exit from the base of the skull and also makes it possible to clear adequately the high jugular nodes and the subdigastric nodes which are frequently the site of metastatic disease. Prior to division of the jugular vein high in the neck, the spinal accessory nerve is again divided and the vagus and hypoglossal nerves, which also exist the jugular foramen and hypoglossal canals respectively, are carefully identified and preserved. At this point the dissection is complete.

It must be stressed that the specific type of en bloc procedure depends upon the type, location, and histology of the primary tumor. The basic principle, however, is extirpation of the gross regional lymphatic drainage system of a tumor at the time of operation.
First Rib Resection for Neurovascular Syndromes of the Thoracic Outlet

Howard S. Brown, R. Aurelius Smith


During the first half of this century, various syndromes attributed to compression of neural and vascular structures in the anatomic area of the thoracic outlet have been described. These include the cervical rib, scalene anticus, costoclavicular, first dorsal rib, and hyperabduction syndromes. This complex array of subdivisions has engendered some confusion in diagnosis and treatment. Although no unanimity of opinion can yet be said to exist, several authors have more recently attempted to relate these conditions to a common denominator, that of compression of the neurovascular bundle between first rib and clavicle, with or without presence of a cervical rib. Such a unifying concept of the fundamental mechanical factor involved has led to new surgical approaches to the relief of symptoms associated with this syndrome. It is our purpose to describe the surgical anatomy of first rib resection as it relates to the thoracic outlet.

Historical Background

The association of cervical rib with symptoms of arterial and neurologic impairment of the upper extremity was well reviewed as early as 1904 by Eisendrath, who reported 35 cases and credited Holmes Coote with the first resection of a cervical rib for this condition in 1861. John B. Murphy in 1905 added a further case, complicated by aneurysmal dilatation of the subclavian artery, treated by cervical rib resection. That the same symptoms might result from compression of the brachial plexus and the subclavian artery by a normal first rib, in the absence of a cervical rib, was recognized by Thomas Murphy, who performed the first resection of a normal first rib for this syndrome in 1910. A supraclavicular approach was used, with division of the anterior scalene muscle. Similar cases were reported by Brickner and by Telford and Stopford.

A new facet was introduced in 1927 with the report of Adson and Coffey, who emphasized an active role of the anterior scalene muscle in compression of the neurovascular bundle, and advocated scalenotomy without cervical rib resection. Their concept was advanced by Ochsner et al and by Naffziger and Grant, who reported cases without cervical ribs but with identical signs, and recommended anterior scalenotomy as the treatment of choice. The term "anterior scalene syndrome" seemed to have become firmly established.

The lack of uniformly favorable response to this procedure, however, made it clear that other factors might be involved. In 1943 Falconer and Weddell described compression of the subclavian artery and vein in the costoclavicular space, and in 1962 Falconer and Li reported further surgical experience with this "costoclavicular syndrome" of brachial plexus and vascular compression by a vise-like action between the first rib and clavicle. They believed this mechanism to be the major factor in production of symptoms. A supraclavicular approach to first rib resection was used in their cases. This opinion was supported by Brintnall et al who, along with Falconer, advocated a "finger pinch" test for costoclavicular compression at operation. In the presence of a positive test, they felt that scalenotomy or cervical rib
resection would be inadequate without first rib resection. Clagett advised a posterior, thoracoplasty-like approach.

A further term was introduced by Wright in 1945, the "hyperabduction syndrome." He indicated that neurovascular compression might occur either in the costoclavicular space, or between the tendon of the pectoralis minor muscle and the coracoid process.

Finally, "thoracic outlet" symptoms associated with abnormalities of the first rib were reported by White et al in 1945.

The problem of causal factors in these variously described syndromes was well discussed by Walshe et al, whose analysis led them to believe that multiple factors were involved in every case of thoracic outlet rib pressure syndrome. They stressed that an abnormal cervical rib or first rib should be considered as an indication of an abnormal thoracic outlet in general, rather than as a sole cause in itself, and concluded that the concept of the scalenus anticus syndrome was an oversimplification. They also pointed out the vise-like action of the clavicle and first rib upon the adjacent neurovascular bundle.

On the basis of this rationale, attention was focused upon resection of the first rib as treatment of choice. The most popular methods at this time are the transaxillary approach, based on the work of Roos and Sanders, and the infraclavicular approach, based on the work of Gol, Brodsky, Hamlin and Nelson.

**Symptoms and Signs**

The symptoms and signs of the thoracic outlet syndrome have been thoroughly described in a number of publications, and need be only briefly summarized here. Neurologic symptoms are more common than vascular, but the two may occur concurrently. There are usually intermittent paresthesias, pain, or numbness in the ulnar distribution, which may sometimes involve the median nerve as well. These symptoms are worsened by certain movements of the shoulder girdle, which approximate the clavicle to the rib, such as downward and backward pulling of the arm, as in carrying weights, or hyperabduction of the arm. Symptoms may be primarily nocturnal, depending upon sleeping positions. The symptoms may spread to the shoulder, side of the neck, or distal extremity. There may be subjective weakness or fatigability of the extremity. Objective neurologic deficit is relatively uncommon, but there may be ulnar sensory loss or atrophy of the hand muscles of ulnar or median innervation.

Vascular symptoms include arterial insufficiency with pallor, coldness, and ischemic numbness in the hand and fingers, simulating Raynaud's phenomenon in some cases. Fusiform, poststenotic dilatation of the subclavian artery may occur, which may be complicated by thrombosis and embolization to the digital arteries with gangrene. Least commonly, there may be venous occlusion, with edema and cyanosis of the hand.

Provoking factors in the development of symptoms seem to include drooping shoulders, particularly in women, excessive muscular development or postural attitudes in some athletic or occupational endeavors, hypertrophic breasts, hyperextension trauma to the neck, and injury to the shoulder girdle and clavicle.
The paucity of objective findings in many cases leaves one with a clinical diagnosis based largely upon subjective symptoms. The Adson, costoclavicular, and hyperabduction tests are not consistently clearly positive in reproducing clinical symptoms, and may conversely produce some degree of radial pulse obliteration in asymptomatic subjects. They are helpful when the symptoms produced clearly simulate the patient's complaints and correlate with accompanying pulse changes. Reproduction of the neurologic symptoms with the arm abducted to 90 degrees, the hand externally rotated, and the neck extended was found to be the most reliable test by Sanders. In contrast, relief of symptoms when the arm is hyperabducted is often found to occur in patients with cervical disc disease.

Further adjuncts to diagnosis advocated by some are nerve conduction velocity tests, plethysmography, and arteriography. Again, such tests cannot always be expected to be conclusive, depending not only upon the severity of the condition but also upon whether the problem is predominantly neural or vascular compression. Nerve conduction velocity studies across the thoracic outlet would appear to be the most directly measurable objective test for neurologic impingement. However, the normal range for the ulnar nerve conduction velocity across the thoracic outlet as shown by Jebson is 52 to 78 meters per second, with a mean velocity of 61.3 meters per second. The wide range of normal here would seem to limit its value as a specific test for thoracic outlet obstruction, and we have not found its use to be as good a diagnostic tool as reproduction of the patient's symptoms by the Adson, costoclavicular, or hyperabduction tests. We do, however, advocate pre- and postoperative nerve conduction velocity studies across the thoracic outlet as a basis for comparison. Arteriograms and phlebograms are not routinely done. They are indicated only when objective vascular findings are present. Neurologic involvement is more common than vascular in our experience, and we agree with Sanders and Falconer that the brachial plexus may be compressed between the clavicle and the first rib without involvement of either subclavian artery or vein. In many cases, myelography may be necessary to exclude an intraspinal lesion.

The differential diagnosis includes other neurologic disorders which may mimic the thoracic outlet syndrome, particularly cervical disc disease, cervical tumors, syringomyelia, tumors of the superior sulcus, and entrapment neuropathies of the ulnar nerve at the elbow and the median nerve at the wrist. Thorough competent neurologic evaluation of patients prior to surgery is essential.

Surgical Treatment

Operation is indicated in those patients who fail to respond to conservative measures such as a change in occupational, athletic, or sleeping habits, or to appropriate physical therapy. The appropriate physical therapy concentrates on a thoracic outlet exercise regime which will stretch the anterolateral cervical muscles, anterior shoulder muscles, and pectoral muscles, and will strengthen the posterior shoulder girdle, and in particular the shoulder girdle elevators. Patients are also shown postural correction and are referred to a competent physical therapist to administer this program.

The objective of operative treatment of this syndrome is obviously to relieve thoracic outlet compression. There are three possible sites of compression to be considered. The first rib is common to the two major sites: the scalene triangle, bounded by the first rib, anterior, and middle scalene muscles, and the costoclavicular space bounded by the first rib, clavicle,
and subclavius muscle. The third site, the pectoralis minor space, is thought to be much less significant. Many anomalies of the ribs, clavicle, muscles, or fascial band attachments may be associated with these syndromes; first rib resection will, however, decompress the thoracic outlet through both the scalene triangle and costoclavicular spaces. In addition, the pectoralis minor tendon can easily be divided when carrying out first rib resection when deemed advisable. Sympathectomy can be done through either the transaxillary or infraclavicular approach, but even in the presence of Raynaud's phenomenon, is probably not necessary since removal of mechanical arterial obstruction seems the more important factor in relief.

Transaxillary First Rib Resection

First rib resection is outlined here basically after the method of Roos. We feel that certain surgical instruments are very helpful for this technique. They are as follows: (1) Sauerbruch double action rib rongeur, (2) Bethune or Moure-Coryllos right angle rib shear, (3) Doyen costal elevator, (4) Coryllos raspatory, (5) Overholt periosteal elevator No 1. The patient, under general endotracheal anesthesia, is positioned in a true lateral position with the hips supported and the shoulder of the operative side rotated posteriorly approximately 60 degrees with the arm suspended initially by a sling to an IV pole. It is important that the thorax be supported so that there is no undue stress on the spine. The entire hand, arm, axilla, shoulder, and anterior and posterior chest are prepped. The hand and arm are held by an assistant and wrapped with stockinette while the remainder of the operative field is draped to appropriately expose the axilla. One should not undertake the transaxillary approach without having a separate assistant who does nothing but hold the arm.

An approximate 4 inch incision is made at the lower margin of the axillary hairline and continued subcutaneously directly to the serratus anterior muscle fascia of the chest wall. Once the appropriate subcutaneous plane is reached, blunt dissection is continued up to the first rib. It is important to identify the second intercostal brachial cutaneous nerve as it exits via the second interspace and gently retract it out of the way. Once dissection has been developed up to the first rib, adequate exposure of the thoracic outlet is only possible during elevation of the extremity by an assistant. The hammerlock, as mentioned by Roos, is very important and helpful. The arm should not be elevated for more than 10 minutes at a time in order that undue stress not be placed upon the brachial plexus.

The subclavian muscles tendinous attachment is incised with scissor dissection flush on the first rib, carefully retracting the subclavian vein away from the dissecting scissors. The anterior scalene muscle can be palpated as it attaches to the scalene tubercle, and with the subclavian artery and brachial plexus held away with an appropriate instrument, the anterior scalene muscle is incised adjacent to and flush on the first rib. A nerve root retractor or similar instrument is used to hold the adjacent neurovascular structures away while these muscles are being incised under direct vision. Utilizing either the Coryllos raspatory or Overholt periosteal elevator No 1, the scalenus medius muscle posteriorly and the intercostal muscular attachments are pushed away leaving the periosteum attached to the rib. It cannot be overly emphasized that elevation of the arm for appropriate visualization of the structures involved during this dissection is imperative, and that the neurovascular structures must be visualized at all times and held away from the cutting instruments. The first rib is then transected posteriorly within 1 cm of the transverse process of the first thoracic vertebra using a right angle Bethune or Moure-Coryllos rib shear. This is probably the most difficult part of
the operation and it requires expert elevation of the arm with concomitant displacement of the first thoracic nerve root with a nerve root retractor or other suitable instrument and transection of the rib under direct vision. Sometimes the cut cannot be made smoothly, and when this happens after the initial transection, a segment of the rib can be removed and the remaining portion of the rib attached to the transverse process can be nibbled away with a Sauerbruch double action rib rongeur to leave a smooth surface. This nibbling procedure must also be done under direct vision.

Both Roos and Sanders feel that the removal of the first rib posteriorly is important to prevent future entrapment of the brachial plexus nerve roots with periosteal development. We feel that this particular portion of the operation is better done through the transaxillary approach and, although this certainly gets beyond the area where the clavicle approximates itself to the first rib, we recommend resection of the first rib to within 1 cm of the transverse process of the first thoracic vertebra. The first rib anteriorly is then either disarticulated from its sternocostal junction or transected with a rib shear as done posteriorly, being careful to avoid the subclavian vein. A cervical rib, if present, is divided posteriorly under direct vision immediately after posterior transection of the first rib. We would agree with Roos that if the cervical rib is 2 cm or less in length, resecting it would endanger the adjacent nerve roots and is not necessary. Fibrous bands and cervical ribs attaching to the first rib should be resected as far posteriorly as can be safely accomplished. Following removal of the first rib, hemostasis should be obtained and pleural leaks should be checked for by putting saline in the wound and having the anesthesiologist inflate the lung. If there are any air leaks, this, of course, will be demonstrated by air bubbles in the field. If there is an air leak, we merely insert a catheter into the pleural space and close the wound in layers around it with the catheter being attached to suction during the closing process. Following complete closure of the wound, we then remove the catheter. Patients recover rapidly from this procedure and are usually able to be discharged within 5 days postoperatively. We do not do bilateral rib resection when utilizing the transaxillary approach. The pectoralis minor muscle tendon may be divided where it inserts on the coracoid process of the scapula by freeing up the lateral margin of the pectoralis major muscle for exposure. We do not routinely do this, but some people feel that it is advisable.

Infraclavicular First Rib Resection

The patient is positioned supine with a pad to elevate the scapula and shoulder, with the arm abducted. A muscle-splitting incision approximately 12 cm in length is carried through the pectoralis major muscle to expose the first rib below the clavicle, beginning medially at the costochondral junction. The periosteum is elevated subperiosteally from the rib, and the costochondral junction is removed piecemeal with the Leksell rongeur. The pleura can then be stripped from the posterior surface of the mobilized rib with careful blunt dissection. The rib is next retraced inward, and the neurovascular bundle traversing the costoclavicular space is palpated and retracted with the index finger. With the neurovascular structures thus constantly protected beneath the index finger and the pleura retracted, the rib with its periosteum is rongeured away, scraping off the insertions of the anterior and middle scalene muscles. Posterior to the neurovascular bundle, rib removal is accomplished by feel rather than under direct vision, but usually can be taken back to within 3 to 4 cm of the transverse process without excessive retraction of the neurovascular bundle; and in some asthenic patients, to within 1 to 2 cm of the transverse process. If a cervical rib is present,
it can also be trimmed through this approach. This incision can be extended slightly downward to the deltopectoral junction if the pectoralis minor tendon is to be divided. If a tear occurs in the pleura, it is closed around catheter suction with the lung inflated. The pectoral fascia, subcutaneous tissue, and skin are closed in layers. Bilateral rib resection can be done as a one-stage procedure by this technique.
Anatomic Considerations in Carotid Endarterectomy

Milton F. Bryant


It has been reported that Dr. M. E. DeBakey performed the first carotid endarterectomy in 1953 for atherosclerotic obstruction of the carotid artery. The report in 1954 of Eastcott, Pickering, and Rob on the reconstruction of the internal carotid artery in a patient with intermittent attacks of hemiplegia stimulated many surgeons to consider further the problems of cerebral vascular insufficiency. Our original experiences with the surgical treatment of strokes at Piedmont Hospital were reported in 1959. Considerable experience has now been accumulated which seems to show that approximately 60 to 70 per cent of patients with cerebral vascular insufficiency have stenotic, ulcerative, or occlusive lesions in the extracranial portion of one or more of the four major arteries that supply the brain. Various authors suggest that these data implicate these extracranial lesions as the cause of cerebral vascular insufficiency. Unfortunately, experience has at times proved this assumption wrong, much to the disappointment of the operating surgeon.

Caution in the Selection of Patients for Surgery

Even after 20 years of experience, one must admit that at times the selection of patients for surgery is extremely difficult and, as yet, ill-defined. The initial enthusiasm associated with any new surgical procedure is frequently found to be excessive. This was found to be true for stellate ganglion blocks in the treatment of cerebral artery thrombosis. Enthusiasm for ligation of the internal mammary artery for angina pectoris faded under the eye of careful scientific evaluation. The mere presence of a stenosing or ulcerating atherosclerotic plaque in the internal carotid or vertebral artery does not insure that removal of the plaque will benefit the patient. Even the improvement in these patients following removal of an atherosclerotic plaque may not be entirely related to the operative procedure, as some of these patients improve spontaneously. The ability to tolerate obstruction in one or more of the four major arteries that supply the brain is dependent upon the development of collateral circulation. Many transient cerebral ischemic attacks may be due to distal embolization of thrombotic or atherosclerotic material from an ulcerating plaque and may not be related to the degree of stenosis produced by the plaque. Most physicians agree that our present surgical procedures upon the carotid arteries have a place in the management of cerebral vascular insufficiency; however, further study is needed to determine more precise methods of selecting patients for carotid endarterectomy.

Pathology

For all practical purposes, atherosclerosis is the usual cause of obstructive or ulcerative lesions in the extracranial portion of the carotid arteries. The lesion is usually located in the base of the internal carotid artery and/or carotid bifurcation. The lesion extends 2 to 6 cm into the internal carotid artery; however, at times it may extend to the base of the skull or intracranially. On occasion one encounters embolic obstruction of the carotid artery or obstruction due to arteritis, fibromuscular hyperplasia, or kinking. It has been recognized for some time that atherosclerotic lesions will infrequently accumulate where major arteries
branch. Swirling, eddying, and other flow changes have been offered as the explanation for deposition of atherosclerotic material at arterial bifurcations.

DeBakey and associates have described three patterns of involvement in the carotid arteries - proximal, cervical, and intracranial. The proximal portion of the external carotid artery is also frequently involved. Obstruction to the right internal carotid artery may occur at its origin from the brachiocephalic artery or from direct obstruction at the origin of the brachioccephalic artery from the aorta. In the cervical form the atherosclerotic involvement is in the region of the bifurcation of the common carotid artery. Intracranial involvement occurs in the carotid artery at or distal to the foramen lacerum. Finally, a mixed form of obstruction may occur in which the atherosclerotic lesions involve multiple sites or are diffuse.

Practical Anatomic Considerations in Carotid Endarterectomy

Variations in arterial anatomy are frequent and numerous. The common or standard type anatomy pertinent to the carotid arteries will be stressed with the idea that the operating surgeon will familiarize himself with the many possible variations. Exposure of the carotid bifurcation can be made by a short, curved, transverse cervical incision along the lines of Langer. This results in a cosmetically attractive scar. Most surgeons feel an oblique incision along the anterior border of the sternocleidomastoid gives easier and better exposure of the anatomic structures. The thin, quadrangular platysma muscle lies beneath the paniculus adiposus and is separated from the external layer of the cervical fascia by loose areolar tissue. The main cutaneous branches of the cervical plexus and the external jugular vein lie beneath this muscle layer. All of these structures must be sectioned; however, no significant functional deficit results. The platysma muscle is supplied by the cervical branch of the facial nerve. Variation in the muscular development of the platysma is frequent. In making the cervical incision one must be certain that the supramandibular and inframandibular branches of the facial nerve are protected from injury in order to avoid drooping of the lower side of the face. A number of the superficial sensory branches of the cervical plexus are necessarily sectioned. This produces varying degrees of numbness medial to the incision, but this problem corrects itself in 3 to 6 months as nerve regeneration occurs.

Variations of the Aortic Arch

The branches of the aortic arch are the brachiocephalic, left common carotid, and left subclavian arteries. The brachiocephalic artery is 3.5 to 5 cm in length and rises to the level of the right sternoclavicular joint to bifurcate into the right common and right subclavian arteries. The left common carotid artery is usually the second branch of the aortic arch, although it may arise from the brachiocephalic (formerly named the innominate) artery.

The exact incidence of aortic arch variations is not known, but in view of the radical modifications which the original symmetric arrangement of the aortic arches undergo during embryologic development, it is not surprising that variations from the usual adult plan frequently occur. In the adult, the aortic arch measures approximately 28 mm in diameter and, after giving off its three main branches, the diameter averages 23 mm. Opposite the origin of the left subclavian artery is a ligament, the ligamentum arteriosum, that connects the concavity of the arch to the proximal portion of the left pulmonary artery. Between the left subclavian artery and the ligamentum arteriosum there is, always in the newborn infant and
occasionally persisting in the adult, a constriction of the arch - the isthmus aortae. This may be succeeded by a dilatation named the aortic spindle. Extreme narrowing of the arch at the isthmus is known as coarctation of the aorta. Many variations in branching of the aortic arch occur. At one extreme, the usual three separate origins from the aortic arch occur and, at the other extreme, all branches arise from a single main stem called the brachiocephalic artery. All sorts of combinations may occur. The right dorsal aortic root may persist so that the supply to the descending aorta is double. One arch may differ from the other in caliber. A double aortic arch may form a ringlike constriction around the trachea and esophagus necessitating surgical intervention. The left aortic root may disappear leaving a right aortic arch and this may or may not be associated with situs inversus. On occasions there may be persistence of the right aortic arch resulting in complete reversal of the arch branches. In these instances, the right subclavian artery passes dorsal to the trachea and may result in esophageal compression. Abnormalities of partitioning of the primitive ventral aortic trunk may occur with resulting various degrees of aortic and pulmonary artery narrowing. This may go so far as complete obstruction to the lumen of either the ascending aorta or the pulmonary artery. A persistent ductus arteriosus may permit these infants to survive. This is in distinct contrast to the persistence of an unclosed ductus arteriosus in an individual with an otherwise normal vascular system. Surgical intervention for this condition, with ligation of the ductus arteriosus, was successfully carried out for the first time by Gross and Hubbard in 1938. Vascular surgeons operating upon the carotid arteries must be familiar with and prepared to interpret any of these anatomic variations. The exact vascular anatomy for each individual is usually first brought to light by the arteriographic studies performed upon patients with symptoms suggestive of cerebral vascular insufficiency. Insufficient knowledge of anatomy and embryology may lead to erroneous interpretation of arteriograms and a possible catastrophic surgical end result.

Common Carotid Arteries

The common carotid arteries are paired vessels that lie within the anterior cervical triangle on the prevertebral fascia. They are from 8 to 12 cm in length and extend from the sternoclavicular articulation to the superior border of the thyroid cartilage where they divide into the external and internal carotid arteries. The point of bifurcation varies, but in 85 percent of individuals it occurs within 2.5 cm of the top of the thyroid cartilage. The common carotid arteries are contained in the carotid sheath which houses the internal jugular vein and the vagus nerve. The jugular vein may lie ventro- or dorsolateral to the common carotid artery while the vagus nerve lies dorsal and between the two vessels. The ansa cervicalis, with its superior and inferior rami along with the branches to the strap muscles, is located in the anterior part of the carotid sheath and should be protected from injury. At times one or more of the branches to the strap muscles may hinder exposure and these branches can be sacrificed without significant residual. In exposing the common carotid arteries the crossing middle thyroid veins are tied and sectioned.

Dorsal and medial to the common and internal carotid arteries are the retropharyngeal space and the prevertebral fascia with its enclosed cervical sympathetic trunk and ganglia. Some surgeons feel that the superior cervical sympathetic ganglia should be removed at the time carotid artery surgery is performed. The superior ganglion is easy to expose and requires only a few minutes to remove. Removal of the ganglion produces Horner's syndrome, which causes the patient minimal problems, or none at all.
**External Carotid Artery.** The external carotid artery is the smaller (in adults it is of equal size) of the two branches into which the common carotid divides. Eight independent branches usually arise from the external carotid artery before it divides into its two terminal branches - the maxillary and superficial temporal arteries. Terminal branching occurs posterior to the neck of the mandible. One usually ligates the superior thyroid artery permanently or temporarily when performing a carotid endarterectomy, as this provides better exposure. The ascending pharyngeal artery is described as the second branch of the external carotid artery but it may be the first branch and, at times, it may actually arise from the carotid crotch or from the proximal portion of the internal carotid artery. This vessel may be ligated to gain exposure. Better exposure can also be obtained by tying and cutting the facial and lingual veins. The hypoglossal nerve is located beneath the facial vein, on the surface of the external carotid artery, and one must protect this nerve from injury or hemi-atrophy of the tongue will ensue. The sternocleidomastoid artery arises from the posterior side of the external carotid artery and then hooks over the loop formed by the hypoglossal nerve and follows the accessory nerve into the sternocleidomastoid muscle. By ligating and sectioning the sternocleidomastoid artery one can push the hypoglossal nerve superiorly and protect it from injury.

**Internal Carotid Artery.** The internal carotid artery originates from the common carotid artery opposite the superior border of the thyroid cartilage and, as mentioned above, in adults the external and internal carotid arteries are of equal size. The proximal portion of the internal carotid artery is dilated to form the carotid sinus which is innervated by the carotid sinus nerve. This nerve arises from the glossopharyngeal nerve at the base of the skull and descends between the internal and external carotid arteries to innervate the carotid sinus. Blockage of the sinus nerve with a local anesthetic prior to any dissection or manipulation of the carotid bulb region is essential in order to prevent unwanted baroreceptor reflexes. We have not noted any permanent elevation in blood pressure from dividing one or both carotid sinus nerves. It is felt that numerous other baroreceptor nerves resume the function of the carotid sinus nerves. The carotid body, a chemoreceptor, is 2 to 3 mm in diameter and can be seen in the region of carotid bifurcation. If necessary this structure can be removed without producing any known permanent effects. The internal carotid artery is usually located dorsolaterally to the external carotid artery and this may cause some confusion due to nomenclature - internal and external. The internal carotid artery ascends vertically lying on the prevertebral fascia, with the internal jugular vein lying laterally. When the carotid bifurcation is high, exposure can be gained by sectioning the ligamentous portion of the stylohyoid muscle and the ligamentous portion of the posterior belly of the digastric muscle. With low carotid bifurcation, exposure may be helped by cutting the superior belly of the omohyoid muscle. At times the internal carotid artery becomes elongated and tortuous and may actually buckle or kink upon itself and interfere with blood flow. The kinking may be congenital or acquired and surgical correction for kinking may at times be necessary. The vagus nerve descends dorsolaterally to the internal carotid artery. Medial to the artery, the superior laryngeal nerve courses in a medial and inferior direction to the larynx and may be injured if careful dissection is not carried out. It is felt that injury to the superior laryngeal nerve is the most common and serious nerve injury produced by surgeons performing carotid endarterectomies. The cervical portion of the internal carotid artery has no named branches, although the ascending pharyngeal artery has frequently been noted to arise from this vessel. At the base of the skull the glossopharyngeal, vagus, accessory, and hypoglossal nerves exist just dorsal to the artery. As the internal carotid leaves the cavernous sinus it gives of the
ophthalmic artery, then the anterior cerebral, middle cerebral, posterior communicating, and chorioid arteries.

Summary

Patients with cerebral vascular insufficiency can frequently be treated and benefitted by carotid endarterectomy. Experience has now been accumulated which shows that approximately 60 to 70 per cent of patients with cerebral vascular insufficiency have stenotic, ulcerative, or occlusive lesions in the extracranial portion of one or more of the four major arteries that supply the brain. Careful diagnostic studies must be performed in order to properly select patients for surgery. As with any operative procedure it is essential that the operating surgeon have a thorough knowledge of the embryology and anatomy along with an understanding of the anatomic variations that may be encountered in the operative field. At Piedmont Hospital we refuse to accept the old adage which propose that "It is futile to write anything new anatomically because it has all been said before." New operations, new approaches, and new knowledge keep anatomy in a live and dynamic state.
Surgical Anatomy of the Deep Fascia of the Neck

K. Nandy


Perhaps one of the most controversial subjects in the human body is the study of the deep cervical fascia. The description given by some morphologists about this fascia as a distinct continuous entity should be considered a myth. The students of anatomy are often confused by the diverse description and above all its variable development in different subjects. Nevertheless, the clinicians have always considered this to be important information because of its influence on the course of certain diseases of the neck.

The description given in the textbooks on the subject has been gathered through gross dissection on cadavers, histologic sections, injection of dye into fascial spaces, and also by clinical observation of certain diseases of the neck.

**Investing Layer**

Perhaps the most conspicuous part of this deep fascia is the one that surrounds the neck deep to superficial fascia and platysma, commonly known as the investing layer. This layer is attached posteriorly to the spines and supraspinous ligaments of the cervical vertebrae. Traced anteriorly it splits to enclose trapezius and sternomastoid muscles but remains united in between the two muscles, forming the fascial roof of the posterior triangle. At the anterior border of sternomastoid the two layers again unite and become continuous with the similar fascia of the opposite side forming the fascial roof of the anterior triangle. At the upper border it is attached to occipital, temporal, and mandibular bones, and splits in two places enclosing parotid and submandibular glands. At its lower border the fascia attaches to the sternum, clavicle, and scapula, and encloses suprasternal space (Burns) above the jugular notch and supraclavicular spaces above the middle-third of the clavicle.

**Pretracheal Fascia**

This is commonly described as the visceral fascia attached above thyroid and cricoid cartilages of the larynx. Traced downward it descends deep to the infrahyoid muscles and in front of the trachea and splits to enclose the thyroid gland. This layer has been traced through the inlet of the thorax into the superior mediastinum where it may blend with the fibrous pericardium of the heart. The part of this fascia between larynx and thyroid gland is often thickened and is referred to as the suspensory ligament of the thyroid gland (ligament of Berry). This may account for the up and down movements of the thyroid gland during deglutition.

**Prevertebral Fascia**

This layer has been described as extending between the sternomastoid muscles of both sides lying deep to the pharynx and esophagus and in front of the prevertebral muscles. The fascia also constitutes a fascial carpet for the muscles in the floor of the posterior triangle.
The fascia over the lower part of the posterior triangle is prolonged over the axillary vessels and the cords of the brachial plexus like a sleeve and is known as the axillary sheath.

**Buccopharyngeal Fascia**

This may be considered to be part of the prevertebral fascia lying in front of it, being separated from it by the retropharyngeal and retroesophageal spaces.

**Fascial Spaces**

The different layers of the deep cervical fascia enclose a number of spaces in between them. The lining of the spaces may not only prevent spread of infection from one area to another but also determine the direction of spread of infection. The various fascial spaces are as follows: (1) visceral compartment, (2) neurovascular compartment (carotid sheath, (3) retropharyngeal and retroesophageal spaces, (4) muscular compartment, (5) parotid space, (6) submandibular space, (7) suprasternal space (Burns), and (8) supraclavicular space.

**Visceral Compartment.** This space is bounded by the pretracheal layer anteriorly and the prevertebral fascia posteriorly, extending from the base of the skull above to the superior mediastinum below. Surrounded by loose connective tissue, the contents of this space are mainly the trachea, esophagus, and thyroid gland. Grodinsky and Holyoke described the anterior and posterior parts of this space as pretracheal and retrovisceral spaces. These authors stressed that these spaces are in continuity with each other and infection in these spaces may be transmitted to the mediastinum by anatomic pathways.

**Neurovascular Space (Carotid Sheath).** This space is probably enclosed by the pretracheal and prevertebral layers under cover of sternomastoid muscle extending from the base of the skull to the root of the neck. It contains common and internal carotid arteries, internal jugular vein, and vagus nerve, with arteries, vein, and the nerve occupying separate compartments.

**Retropharyngeal and Retroesophageal Spaces.** Between the prevertebral and buccopharyngeal fascia, these spaces extend downward from the base of the skull behind the pharynx and esophagus to the superior mediastinum. It may be emphasized here that these spaces constitute one continuous space behind the pharynx and esophagus, containing loose areolar tissue and retropharyngeal lymph nodes.

**Muscular Compartment.** The space is bounded by the investing fascia anteriorly and the pretracheal fascia posteriorly, and mainly contains the infrahyoid muscles, such as the sternohyoid, sternothyroid, and omohyoid.

**Parotid Space.** The parotid space contains the parotid gland, lymph nodes, facial nerve, retromandibular vein, and the external carotid artery and its terminal branches. The space is enclosed by the two layers of investing fascia in the interval between the mastoid process of the temporal bone and the ramus of the mandible. The space is usually completely occupied by the parotid gland and associated lymph nodes and the space may therefore be considered a clinical one, rather than an anatomic one. The thick fascia surrounding the space
undergoes considerable stretching during parotitis and this may account for the pain and discomfort.

**Submandibular Space.** This space is below the body of the mandible and enclosed by the two layers of the investing fascia and the submandibular fossa of the mandible. The space contains the submandibular salivary gland and lymph nodes and is separated from the parotid space by the stylomandibular ligament.

**Suprasternal Space (Burns).** The investing layer splits into two layers above the jugular notch of the sternum enclosing this space. It contains the sternal head of the sternomastoid, anterior jugular veins and jugular venous arch, and sometimes a lymph node.

**Supraclavicular Space.** This space is between two layers of the investing fascia above the middle third of the clavicle and contains the lower part of the external jugular vein, transverse cervical vein, and suprascapular vessels and supraclavicular nerves.

**Applied Anatomy**

The knowledge of the deep cervical fascia is considered important to clinicians, for it may prevent the spread of infection from one area to another by providing a fascial barrier. An infection in the visceral compartment may easily find its way into the superior mediastinum by the help of gravity and may produce serious complications. On the other hand an abscess formed behind the prevertebral fascia due to caries of cervical vertebrae may extend laterally behind the fascia and point at the posterior border of the sternomastoid muscle, or follow the axillary sheath to point at the axilla, or perforate the prevertebral layer to bulge through the posterior wall of the pharynx (retropharyngeal abscess).

With the advent of antibiotics and the ease of controlling infections, the practical importance of the deep cervical fascia has been considerably reduced. An excessive emphasis on the minute details of the disposition of the fascia is therefore trivial. Nevertheless, the knowledge of the fascial layers and spaces is often helpful to the clinician in understanding certain disease processes in the neck. Finally, the study of the disposition of the fascial layers at the dissection table should be considered as a worthwhile exercise and may provide the first step toward the learning of the intricate anatomy of the neck.
The term "great arteries" refers to the major intrathoracic arterial structures and includes the aorta - ascending aorta, aortic arch, and descending thoracic aorta - and its major branches, the innominate, left common carotid, and left subclavian arteries; and the pulmonary artery trunk with its two branches. The success of any surgical procedure upon these vessels and the ease with which the procedure will be performed depends to a great degree on the operator's thorough knowledge of their courses and relationships and how to obtain good exposure of these vascular structures. The purpose of this communication is to review briefly the gross surgical anatomy of these arteries, their main embryological malformations, and the best possible surgical approaches to them.

Aorta

The aorta arises from the left ventricle at approximately the level of the third left sternocostal joint. It first courses upward, forward, and to the right to approximately the level of the right second sternocostal joint, then arches obliquely to the left and posteriorly to a point just to the left of the vertebral column at the level of the lower border of the fourth thoracic vertebra. From this point on, it descends caudally and slightly to the right to penetrate the diaphragm at the level of the twelfth thoracic vertebra.

The ascending aorta is completely encased within pericardium and lies posterior and to the right of the pulmonary artery trunk. The right pulmonary artery is directly posterior to it and the superior vena cava lies immediately on the right. The only branches of the ascending aorta are the right and left coronary arteries which arise from the respective sinuses of Valsalva.

The arch of the aorta lies behind the lower half of the manubrium. Its right posterior aspect is related to the trachea, esophagus, left recurrent laryngeal nerve, and thoracic duct, as well as several nerves to the deep cardiac plexus that descend with the trachea. The left anterior surface is covered by the left mediastinal pleura and is crossed by four nerves, the left phrenic, left vagus, and two cardiac nerves. Within the concavity of the arch lie the main pulmonary trunk and its two branches, the ligamentum arteriosum and the left recurrent laryngeal nerve as it curves under the arch from its origin from the left vagus nerve. The superior aspect of the aortic arch usually gives rise to three branches, the innominate, left common carotid, and left subclavian arteries. The arch and the origins of its branches are crossed anteriorly by the left brachiocephalic vein which descends obliquely from left to right.

The descending thoracic aorta is continuous with the arch and begins at the lower level of the fourth thoracic vertebra. As it descends caudally, it first lies on the left side of the bodies of the fifth through seventh thoracic vertebrae. Below this level, the aorta lies directly anterior to the remaining thoracic vertebrae. Throughout this course, the thoracic duct andazygous vein lie on its right and the hemiazygous veins on its left posterolateral side. In its upper portion it crosses posterior to the root of the left lung and lies to the left of the
esophagus. Since the aorta angles slightly to the right in its descent to the abdominal cavity, and the esophagus angles slightly to the left, the latter structure comes to lie to the left of the aorta near the diaphragm. The branches of the descending aorta are the bronchial, esophageal, and intercostal arteries, as well as small branches to the pericardium and diaphragm.

**Branches of the Aortic Arch**

The innominate (brachiocephalic) artery is the first branch of the aortic arch. It arises behind the center of the manubrium, ascends cephalad in a course which is slightly oblique to the right, and divides behind the right sternoclavicular joint into its branches, the right subclavian and right common carotid arteries. Its total length is approximately 5 cm. The trachea lies immediately posterior to it and the left brachiocephalic vein crosses directly anterior to its origin.

The left common carotid arises immediately after the innominate artery and is followed by the left subclavian artery, and both ascend cephalad behind the left sternoclavicular joint. They originate from the more posterior portion of the aortic arch and follow a semispiral course to reach the left neck. In the groove between these two branches, the left vagus descends to reach the aortic arch and behind them are the left recurrent laryngeal nerve, thoracic duct, and left side of the trachea and esophagus. The left brachiocephalic vein crosses in front of both arteries but is closely related only to the left common carotid due to the more posterior origin of the left subclavian.

**Pulmonary Artery**

The main pulmonary artery trunk originates from the infundibular portion of the right ventricle and passes cephalad and slightly to the left. It is approximately 2 inches long and most of its length is within the pericardium. Shortly after leaving the investment of the pericardium and within the concavity of the aortic arch, it bifurcates into its two branches, the right and left main pulmonary arteries. The pulmonary trunk lies anterior to the left of the aortic root, anterior to the left coronary artery, and anterior medially to the left atrial appendage.

The right and left pulmonary arteries course directly transversely from their origin from the pulmonary artery trunk, thus resembling the horizontal portion of the letter "T". The right pulmonary artery, which is slightly longer, lies directly posterior to the ascending aorta and superior vena cava and the left pulmonary artery lies directly anterior to the descending aorta. The right and left mainstem bronchi and inferior tracheobronchial lymph nodes lie directly posterior to both pulmonary arteries and thus separate them from the esophagus. The ligamentum arteriosum, a fibrous band of tissue which represents the remnants of the ductus arteriosus, runs from the left pulmonary artery to the concave surface of the arch of the aorta just distal to the origin of the left subclavian artery and around it the left recurrent laryngeal nerve makes its loop.

**Congenital Anomalies of Great Arteries**

The above description of the aortic arch and its branches represents the normal embryologic development of the left aortic arch and its branches, which is estimated to be
encountered 83 per cent of the time. In addition, various anomalies of the great arteries have been seen. These include anomalies of the left aortic arch, the right aortic arch, and double aortic arch.

The presence of these various anomalies can be explained by the location at which, during the embryonic life, interruption of the embryonic arches occurs in the Edwards hypothetical double aortic arch.

**Left Aortic Arch**

Left aortic arch with aberrant right subclavian artery is considered to be the most common malformation of the aortic arch, one in every 200 persons. In this anomaly, the right subclavian artery arises as the fourth branch from the aortic arch and, crossing behind the esophagus, extends to the right arm. Usually the patients with this anomaly are asymptomatic but symptoms due to esophageal compression have occasionally been observed during childhood and in adults with aneurysm of this anomalous artery. This malformation is formed as a result of interruption of the right aortic arch in the hypothetical double aortic arch between the right common carotid and the right subclavian arteries.

Left aortic arch with right descending aorta results from the interruption of the embryonic right aortic arch in the hypothetical double aortic arch between the right subclavian artery and the descending aorta. The branching of the vessels of the arch in this anomaly is normal, and it is a very rare malformation and results in no airway or esophageal problems.

**Right Aortic Arch**

The right aortic arch anomalies can be divided into five types. Type I occurs when, in the hypothetical double aortic arch, interruption takes place in the embryonic arch between the descending aorta and left ductus arteriosus. This results in a persistent right aortic arch giving off first the innominate artery, then the right common carotid, and finally the right subclavian artery. The left innominate artery crosses the midline over the trachea and then branches to the left common carotid and left subclavian arteries. This anomaly does not form a vascular ring and is not accompanied by symptoms of compression of the trachea and esophagus. It is almost always associated with cyanotic congenital heart disease.

Type II right aortic arch results from the interruption of the left embryonic arch in the hypothetical double aortic arch between the left subclavian artery and the ductus arteriosus. Again the right aortic arch gives off first the left innominate artery, then the right common carotid, and finally the right subclavian artery. The left innominate artery crosses the midline over the trachea and forms, with the left ligamentum arteriosum, a vascular ring which may be symptomatic.

Type III right aortic arch gives off first the left common carotid, then the right common carotid, the right subclavian, and finally an aberrant left subclavian artery from a posterior aortic diverticulum. The ligamentum arteriosum originates from the left subclavian at its origin forming a vascular ring which may cause significant compression symptoms. This anomaly results from interruption of the left embryonic arch in the hypothetical double aortic arch between the left subclavian and the left common carotid arteries.
Type IV right aortic arch occurs when interruption of the left embryonic arch in the hypothetical double aortic arch takes place between the ascending aorta and the left common carotid artery. The right aortic arch gives off first the right common carotid artery, then the right subclavian, and finally an aberrant retroesophageal left innominate artery. A left ligamentum arteriosum joins the pulmonary artery to the descending aorta at the origin of the left innominate artery, thus forming a vascular ring, which may be symptomatic.

Type V right aortic arch gives off first the left common carotid artery, then the right common carotid, and last the right subclavian artery. The left subclavian artery does not originate from the aorta but it is connected only to the pulmonary artery by the ligamentum arteriosum. The occurrence of this anomaly can be explained when interruption of the left embryonic arch occurs in the hypothetical double aortic arch at two places, between the left common carotid and left subclavian arteries and between the descending aorta and the left subclavian artery. The patients with this malformation, when symptomatic, have symptoms of subclavian steal syndrome.

Double Aortic Arch

When both aortic arches persist, the anomaly of double aortic arch occurs with the right common carotid and subclavian arteries arising independently from the right arch and the left common carotid and left subclavian arteries arising from the left arch. Depending upon the patency of either of the two arches, the double aortic arch can be classified according to two types.

In Type I double aortic arch, both arches are patent and form a constricting ring around the trachea and esophagus, commonly producing symptoms of compression of these two structures, but this anomaly may go unrecognized and be discovered during incidental aortography. Due to the fact that it frequently produces symptoms of tracheal compression, this malformation is the most important of all vascular ring anomalies.

Type II double aortic arch is the anomaly which has both aortic arches in continuity, but one of them, almost always the left one, is partially atretic. The atresia may occur between the descending aorta and left ductus arteriosus, between the ductus arteriosus and left subclavian artery, between the left common carotid and left subclavian artery, or between the ascending aorta and left common carotid artery, or between the ascending aorta and left common carotid artery, resulting in the following respective anomalies. Regardless of the location of the atresia, a diverticulum of the descending aorta at the point of junction of the two arches is always present, as well as a complete ring around the esophagus and trachea, resulting commonly in compression of these structures.

Cervical Aortic Arch

It is a very rare anomaly in which the ascending aorta arises normally from the left ventricle and extends higher so that the aortic arch is located in the neck. The origin of the arch vessels in this anomaly varies - it might be normal, or the external and internal carotids might originate separately from the arch and the right subclavian artery may be aberrant, or the left may originate as a last branch of the right aortic arch from the retrosternal segment of the aorta. Symptoms of tracheal and esophageal compression may be present in the cases
with cervical aortic arch and all of them have a pulsatile mass in the neck which can be mistaken for aneurysm of the innominate, subclavian, or carotid artery.

**Coarctation of the Aorta**

Coarctation is constriction of the aortic lumen of varying degree and length and occurs anywhere along the aorta, but in 95 per cent of the subjects it is a localized stenosis just distal to the subclavian artery near the site of the ligamentum arteriosum. When the coarctation is located below the ductus arteriosus, it is referred to as a postductal coarctation and when it is located above the ductus arteriosus, it is referred to as a preductal coarctation. The ductus may remain open or closed in each type, although it is more likely to be open in the preductal coarctation. The coarctation is characterized by deformity of the aortic media involving the superior, anterior, and posterior wall, frequently at the origin of the descending aorta, forming a curtain-like infolding of the wall which causes eccentric stenosis of the lumen. The remainder of the aorta may be normal or a segment, commonly the isthmus, may be hypoplastic. The development of the aortic hypoplasia may be explained by the relationship of the coarctated aortic segment to the aortic insertion of the ductus arteriosus. ie, when the ductus arteriosus enters the aorta distal to the coarctation, blood flow during fetal life is through the ductus arteriosus to the descending aorta. Thus, due to the diminished blood flow through the aortic isthmus and arch, varying degrees of hypoplasia of these segments occur. This phenomenon is not expected to occur when the ductus arteriosus enters the aorta proximal to the coarctation.

In the patients with coarctation of the aorta, varied degrees of collateral circulation are formed between the branches of the subclavian arteries - vertebral, thyrocervical, costocervical, internal mammary, and the intercostal arteries - which may result in considerable bleeding during surgical repair of the lesion. Occasionally, an anomalous artery arising from the back of the aorta immediately proximal to the coarctation, the so-called Abbott's artery, is encountered in these patients, which courses medially beneath the aortic arch or the left common carotid artery which may be as large as 1 cm in diameter and aneurysmally dilated. This vessel with the aneurysmally dilated and fragile intercostal arteries can make the dissection for repair of coarctation of the aorta hazardous.

Coarctation of the aorta usually manifests with heart murmur, hypertension of the upper extremities and its complications, and congestive heart failure, particularly during infancy.

**Surgical Exposure of the Great Arteries**

The ascending aorta, the main pulmonary artery trunk, the first portion of the aortic arch, the innominate artery, and the left common carotid artery are best exposed through midsternotomy incision, which can be extended into the neck if needed to expose the common carotid arteries. For concomitant exposure of the second and third portion of the subclavian arteries, the incision should be extended in the supraclavicular fossa parallel to the clavicle, the median portion of the clavicle should be resected, and the sternocleidomastoid, sternohyoid, sternothyroid, and scalenus anticus muscles should be divided. During this maneuver, special attention should be paid not to injure all the vital structures located in this
area, especially the phrenic nerve which is lying over the latter muscle and, on the left side, the thoracic duct as it joins the left subclavian vein.

Exposure of the ascending aorta with the innominate artery or with the aortic arch, left common carotid, and left subclavian arteries can also be gained through an ipsilateral anterior thoracotomy through the fourth intercostal space and a midsternotomy extending from the level of the fourth intercostal space to the sternal notch, with an ipsilateral supraclavicular incision and excision of the medial portion of the clavicle. This incision, the "trap door," exposes all these vascular structures but is more taxing to the patient and to the surgeon alike.

The best exposure for the first portion of the subclavian artery, such as is needed for the creation of a subclavian to pulmonary artery shunt, is best accomplished by a lateral thoracotomy through the fourth intercostal space. Exposure of the proximal portion of the descending aorta for repair of coarctation of the aorta is best done through a posterolateral thoracotomy through the fourth intercostal space; exposure of the whole descending aorta, such as will be needed for aneurysmectomy, is best accomplished through a posterolateral thoracotomy but through the fifth intercostal space or the bed of the fifth rib. In mobilizing the descending aorta, care should be exercised to protect all the neighboring structures, esophagus, trachea, etc, and particularly the intercostal veins, because after injury their medial end retracts medially and far to the right and is very difficult to gain control; the intercostal arteries, especially in the presence of coarctation of the aorta, when they are thin and aneurysmally dilated; and the recurrent laryngeal nerve during the division of the ligamentum arteriosum.

The surgical repair of aortic arch anomalies is best accomplished through a left posterolateral thoracotomy through the fourth intercostal space. In the case of double aortic arch, the smaller of the two should be divided. The division of the arch should be performed distally to the origin of the common carotid artery so that this vessel will be left to receive blood from the ascending aorta rather than from the descending aorta, where the orifice at the point of junction of the arch to the descending aorta might be small. In the case of an aberrant subclavian artery, ligation of this vessel should be done at its origin from the aorta, whereas symptoms of trachea compression by an anomalously originating innominate artery can be relieved by lifting this vessel against the chest wall after its adventitia is sutured to the sternum. Because of the possibility of trachea collapse with the loss of external support following surgery for relief of the airway compression by an aortic arch anomaly, dissection around the trachea should be minimal so that sufficient support tissue is left around it.
The Surgical Knee

John M. Roberts


The knee is currently a target area of enhanced interest to the surgeon who treats disorders of the musculoskeletal system. The popularity of sports, particularly football, justifies the sometimes acute concern with continuing function of an athlete's knee, which is often threatened by a valgus-producing force on the rotated limb above the fixed foot. In-depth studies by specialists in sports medicine have increased knowledge of the anatomy and biomechanics of the knee. The development of more efficient techniques of internal fixation and the emerging concept of early mobilization in the treatment of joint fractures have caused traumatologists to reassess the elements of priority in the function of the knee. The increasing geriatric population and the governmental approval of a bonding substance for implants have been factors in the rapid evolution of the techniques of partial or total replacement arthroplasties for the arthritic knee. While the rationale for certain surgical adventures may be somewhat empirical, the common goal of any reparative or reconstructive procedure is restoration of function commensurate with the patient's age, occupation, and lifestyle. This review is conceived as another look at the anatomy of the knee, both to refresh the memory of the arrangement of structural components and to perceive this anatomy in the context of the surgeon's attempts to improve or preserve function.

Stability and Motion

Strength and motion are features important for function of the knee joint. The degree of stability and mobility required by a specific individual is determined by functional demand. Simply stated, the weight-bearing knee should not buckle or give way. Although motion of the knee is primarily hinge-like around a transverse axis, abduction-adduction movements around a sagittal axis and rotation movements around a longitudinal axis can and do occur synchronously. A rather constant sequential pattern of motion in all three planes occurs in a given subject's knee during walking. Kettelkamp et al have shown that the tibia extends, abducts, and externally rotates on the femur just before or at the beginning of stance phase. The tibia flexes, adducts, and internally rotates during early swing phase. Kettelkamp et al used an electrogoniometer to demonstrate the following physiologic ranges of motion during walking: flexion-extension was 65 to 70 degrees, abduction-adduction approximately 10 degrees, and internal-external rotation approximately 15 degrees. Up to 110 degrees flexion of the knee is optimal for arising from a sitting position in a chair without pushing up with one's arms. Less than full extension of the knee will often result in a noticeable limp. The "screw-home" mechanism is defined as external rotation of the tibia on the femur which occurs during the final degrees of extension of the knee. Helfet relates the "screw-home" motion to a tracking mechanism imposed by the shapes of the femoral condyles. Both stability and mobility depend upon the shape, integrity, and interrelationships of the various bony, cartilaginous, ligamentous, and muscular components.
Femoral Surface

The knee is the largest joint in the human body. The apposing ends of both femur and tibia flare abruptly to produce large articular surface areas for wider distribution of force. The distal femur enlarges to become the medial and lateral condyles. If the femur is viewed from the distal end, the condyles are confluent with a narrow transverse diameter in their anterior portions but widen to frame the intercondylar notch by their posterior positions. The height of the lateral condyle is greater, but the medial condyle is curved so that the distance from anterior to posterior border is longer. Schematically, the distal end of the femur is triangular in shape. Standing on such a surface gives tripod-like stability. Stability is also enhanced by distinct flattening of the anterior portion of the articular surface of the lateral femoral condyle when seen from the lateral view. In the standing position, with the knee extended, this flattened portion is in contact with the lateral tibial plateau and the lateral meniscus. Because external rotation of the tibia relative to the femur has occurred with extension and because the transverse diameter of the contacting anterior border of the femoral condyles is foreshortened, a variable amount of the lateral tibial plateau is uncovered. The lateral border of the lateral femoral condyle crosses the central part of the tibial plateau and may act as a wedge with valgus-producing force on the knee to cause a depressed lateral tibial plateau fracture. The contour of the medial femoral condyle is more consistently rounded when viewed from the medial aspect, but Kettelkamp and Jacobs have found by radiocontrast methods that the total area of contact between the femur and tibia in the medial compartment was 1.6 times greater than the area of contact in the lateral compartment during the first 35 degrees of flexion of the knee. The same study also demonstrated that portions of the menisci were included in the contact areas, suggesting that the menisci do function in part as weight bearing structures. Brantigan and Voshell have observed that the menisci form cushion-like bumpers between femur and tibia during hyperextension and hyperflexion. The same authors observed that 90 degrees of hyperextension would occur with all ligaments and menisci intact if the anterior portions of the femoral condyles were excised. This emphasizes the fact that the shape of the distal femur is a major factor in providing stability to the knee.

Upper Tibial Surfaces

The articular surface of the upper end of the tibia is surprisingly variable in contour. Fairbank has demonstrated this graphically by constructing models of the upper tibial surfaces based on serial tracings of specimens in various planes. The lateral tibial plateau is decidedly convex when viewed from both the anterior and lateral aspects. The surface of the medial plateau is saddle-shaped with a mildly convex elevation in the central portion, dipping into a concavity anteriorly and laterally. These surfaces on the upper end of the tibia are not entirely congruent with the femoral condyle surfaces. Brantigan and Voshell have shown that the space between the peripheral borders of the medial femoral and tibial condyles is 0.5 mm and the space between the peripheral borders of the lateral condyles is 3.5 mm after the menisci are removed. The menisci complement the tibial surfaces to present a combined tibial-meniscal surface which is congruent with the femoral condyles. During knee motion in the sagittal plane (flexion-extension), the menisci move forward and back with the upper end of the tibia. Last has pointed out that the menisci tend to move with the distal end of the femur during rotation movements when the knee is flexed. The menisci also move outward or centrifugally at the time of axial impact on the knee joint and thereby help to dissipate a threatening degree of force on the articular cartilage at the time of landing from a jump or
fall. The menisci and cruciate ligaments form a "figure-of-eight" configuration when viewed from above. The posterior end of the lateral meniscus is connected across the intercondylar eminence with the anterior end of the medial meniscus by Barkow's ligament. The posterior end of the lateral meniscus is also connected to the femoral attachment of the anterior cruciate ligament near its insertion into the medial surface of the posterior portion of the lateral femoral condyle. The posterior horn of the lateral meniscus is also attached to the medial femoral condyle by the ligaments of Humphrey and Wrisberg which embrace the posterior cruciate ligament. Both menisci are attached to the upper tibia by the circumferential coronary ligament. The anterior ends of the menisci are connected by the transverse ligament of the knee. The capsule is attached to the rims of both menisci except in the posterolateral corner of the knee joint where the popliteus tendon intervenes between the capsule and the lateral meniscus. The popliteus muscle inserts in part into the posterior horn of the lateral meniscus. During initial flexion from the extended position, the popliteus muscle pulls the lateral meniscus posteriorly out of the way of the lateral femoral condyle. The complex mooring system of the menisci has been outlined to emphasize their stability despite their ability to move out of the way of the potentially grindstone action of the rolling and gliding femoral condyles.

**Anterior Cruciate Ligament**

The anterior cruciate ligament extends from the medial surface of the posterior portion of the lateral femoral condyle downward through the intercondylar notch to the anterior portion of the intercondylar eminence. The intercondylar eminence is a triangular area on the upper tibial surface which tilts downward slightly from the tibial spines to the anterior rim of the upper tibia. In a child, the ligament may be stronger than the cartilage and bone to which it attaches distally. An avulsion fracture of the intercondylar eminence may result from a hyperextension injury. The posterior cruciate ligament extends from the posterior intercondylar eminence upward to attach to the lateral surface of the medial femoral condyle in the intercondylar notch. The upper insertion of the posterior cruciate ligament is adjacent to an area on the articular surface of the medial femoral condyle which may become necrotic in a condition of adolescence called osteochondritis dissecans. This anatomic relationship has caused some to think that osteochondritis dissecans may be a traumatic lesion. The distal attachment of the posterior cruciate ligament may cause an avulsion fracture of the posterior intercondylar eminence with forceful hyperextension of the knee. Brantigan and Voshell have found that the cruciate ligaments remain tight in all degrees of extension and flexion of the knee. With flexion of the fibers of the posterior cruciate ligament wind on the longitudinal axis of the ligament to shorten somewhat in adjustment to the closing points of attachment of the town ends. The anterior and posterior cruciate ligaments wind upon each other with internal rotation of the tibia upon the femur. Helfet has described the cruciate ligaments as guide ropes for rotary movement of the knee. If both collateral ligaments are transected in a fresh amputation specimen, nearly 180 degrees of external rotation of the tibia and the femur is possible. The anterior cruciate controls anterior gliding and the posterior cruciate posterior gliding of the tibia on the femur with the knee flexed. Frankel quotes Morrison to have shown that the force on the anterior cruciate is 35 points and that on the posterior cruciate is 74 pounds during walking.
**Extensor Mechanism**

The knee joint is straightened by the extensor mechanism which consists of the quadriceps muscle, the patella, and the patellar ligament. Last has described the quadriceps tendon as a trilaminar structure. Near its insertion into the upper pole of the patella, the anterior third of the conjoint tendon represents the tendon of the rectus femoris muscle, the middle third the confluent tendons of the vastus medialis and vastus lateralis, and the posterior third the tendon of the vastus intermedius. The distal portion of the vastus medialis actually inserts into the medial aspect of the proximal two-thirds of the patella rather than into the conjoint or central tendon. Lieb and Perry have called this portion of the vastus medialis the vastus medialis obliquus. The line of muscle fibers in the vastus medialis obliquus makes an angle of 50 to 55 degrees with the longitudinal axis of the quadriceps muscle as a whole. The vastus medialis obliquus functions primarily to prevent lateral subluxation of the patella by counteracting the lateral pull of the vastus lateralis. Quadriceps atrophy often occurs rapidly following knee joint injury or surgery. The atrophy is most apparent in the region of the vastus medialis obliquus. There is a characteristic extensor lag of approximately 15 degrees. These findings have led observers in the past to conclude that the function of the vastus medialis obliquus is to produce the final 15 degrees of extension. Lieb and Perry have refuted this conclusion, demonstrating in experimental models that the final 15 degrees of extension is produced by the quadriceps muscle as a whole and that the extensor lag is due to generalized weakness of the entire muscle.

**Patella**

The articular surface of the patella faces the trochlear surface of the anterior and distal end of the femoral condyles. The articular surface of the patella is divided by a shallow keel into a larger lateral facet and a smaller medial facet. Hughston has shown that lateral posture and/or tilting of the patella may predispose to either subluxation or chondromalacia of the patella. The patella is connected to the tibial tubercle by the patellar ligament, completing the extensor mechanism. The length of the patellar ligament determines the position of the patella relative to the distal femur when viewed from the lateral aspect. Hughston has emphasized that the patella should lie between the anterior projections of lines traced over the roof of the intercondylar notch and the central third of the distal femoral epiphyseal plate respectively on a lateral x-ray (Blumensaat's lines). Malposition of the patella proximal to the upper line is called patella alta. Malposition of distal to the lower line is called patella baja. Kaufer has shown that the patella improves the efficiency of the extensor mechanism by increasing the quadriceps moment arm. This is the perpendicular distance between the patellar tendon and the axis of knee flexion which lies in the region of the posterior portions of the femoral condyles. After patellectomy, a 30 per cent increase in quadriceps force may be required to extend the knee fully.

**The Capsular Checkrein**

Hyperextension of the knee by the quadriceps mechanism is prevented by a complex network of capsule, ligament, and tendon insertions which might be called a capsular checkrein. Nicholas has described this as a large flexible half-sleeve extending from the midline of the medial surface of the knee around posteriorly to the midline of the lateral surface of the knee around posteriorly to the midline of the lateral surface of the femur and
tibia. This structure relaxes during flexion, which permits increased range of passive internal and external rotation as pointed out by Hallen and Lindall. Rotary movements of the fixed tibia are controlled by the hamstring muscles which insert on both the medial and lateral aspects of the upper tibia. The capsular checkrein may be divided for convenience into three parts: medial, posterior, and lateral.

**Medial Portion**

The medial portion of the capsular checkrein is composed of the joint capsule, medial collateral ligament, and insertion of the semimembranosus muscle. The superficial portion of the tibial collateral ligament extends from the medial epicondyle of the femur to a point approximately a handsbreadth below the joint line on the medial surface of the tibia. The anterior or leading edge of this ligament remains tight in all degrees of flexion and extension. Above the joint line the superficial portion of the tibial collateral ligament is fused with the deep portion of the medial collateral ligament, which is actually a thickening in the medial joint capsule. Below the medial meniscus, the superficial portion of the tibial collateral ligament is separated easily from the deep portion of the ligament. Between the joint line and the distal insertion there is often a bursa. The anterior edge of the tibial collateral ligament is directed anteriorly from proximal to distal insertions which reinforces the action of the anterior cruciate ligament in restricting forward gliding of the tibia on the femur. With flexion of the extended knee the anterior edge of the tibial collateral ligament moves slightly posteriorly but remains tight. The posterior oblique extension of the tibial collateral ligament has been described by Hughston and Eilers as a distinct entity which they call the "posterior oblique ligament." It originates from the adductor tubercle and courses distalward and posteriorly to fuse with the posterior capsule of the knee joint, to attach to the posterior horn of the medial meniscus, and to extend over the medial limb of the distal insertion of the semimembranosus. This structure has been found by Hughston and others to be critical in maintaining stability against anteromedial rotation of the tibia on the femur. Since it is often torn by a valgus-producing force on the rotated knee, care should be taken to obtain adequate repair in the posteromedial corner following such injury. The deep portion of the tibial collateral ligament extends from the epicondyle of the femur downward to the rim of the meniscus and is continued to the medial surface of the tibia just below the articular border as the coronary ligament of the meniscus. Evidence for describing the deep portion of the collateral ligament of the meniscus. Evidence for describing the deep portion of the collateral ligament as a separate entity is the fact that abduction stress may produce tears of the two parts of the collateral ligament at different levels. O'Donoghue has urged that it is important to restore continuity to the deep portion of the collateral ligament, an important stabilizing structure of the knee. The semimembranosus insertion reinforces the capsule checkrein in the posteromedial corner. One part of the tendon inserts underneath the superficial part of the tibial collateral ligament on the medial aspect of the upper tibia. The remaining three medial hamstrings (sartorius, gracilis, and semitendinosus) insert in a group as the pes anserinus or "goose's foot." Last has pointed out that the superficial portion of the tibial collateral ligament may represent phylogenetically a degeneration of the hamstring-like distal insertion of the adductor magnus. Kennedy and Fowler have mounted fresh amputation specimens on a sophisticated stress machine which causes in sequence rupture of the medial capsular ligament, the superficial tibial collateral ligament, and the anterior cruciate ligament with external rotation of the knee beyond 45 degrees. Slocum and Larson have emphasized that external rotation is controlled by the concerted action of the posteromedial capsule, superficial
tibial collateral ligament, anterior cruciate ligament impinging on the medial aspect of the lateral femoral condyle, the posterior third of the medial meniscus, and the vastus medialis.

**Posterior Portion**

The posterior portion of the capsule checkrein consists of the posterior capsule of the knee joint reinforced by the insertions of the semimembranosus and popliteus muscles and the posterior cruciate ligament. The semimembranosus has a multifocal insertion: the medial and posterior aspect of the tibia just below the articular rim, the posterior horn of the medial meniscus, the posterior capsule, and the posterior oblique extension of the tibial collateral ligament. The extension upward and laterally across the popliteal aspect of the capsule is called the oblique popliteal ligament. The popliteus muscle originates from the posterior surface of the upper tibial metaphysis and inserts into the lateral femoral condyle below the fibular collateral ligament and into the posterior horn of the meniscus. As mentioned before, it draws the lateral meniscus posteriorly as it internally rotates the tibia during the first few degrees of flexion from the fully extended position. Kaplan has demonstrated an inconstant structure called the flabella-fibular ligament which extends from the proximal end of the fibula upward to the flabella when the latter is present. The extension of the posterior edge of the fibular collateral ligament across the tendon of the popliteus is called the arcuate ligament. This ligament has been considered by some to be a third insertion of the popliteus muscle into the head of the fibula.

**Lateral Portion**

The lateral portion of the capsular checkrein consists of the iliotibial band, the fibular collateral ligament, the popliteus muscle, the biceps femoris insertion and the lateral capsular ligament. The lateral capsular ligament is a thickened portion of the joint capsule which extends from the lateral aspect of the femoral condyle just above the joint line to the lateral aspect of the tibia just below the joint line. It is attached to the rim of the lateral meniscus except where the tendon of the popliteus muscle intervenes in the posterolateral corner. The fibular collateral ligament has been considered to be phylogenetically a degenerative portion of the peroneus longus muscle. The fibular collateral ligament extends from the lateral femoral epicondyle downward to the upper end of the fibula. This ligament relaxes with flexion of the knee and allows adduction and external rotation, as demonstrated by Brantigan and Voshell. Other stabilizing structures on the lateral aspect of the knee are the iliotibial tract and the biceps tendon. Kaplan has described the anatomy of the iliotibial tract which is an aponeurotic structure extending downward over the lateral aspect of the thigh from a yoke-like motor comprised of the tensor fascia lata anteriorly and the gluteus maximus posteriorly. A contracted iliotibial tract was often indicated in the youngster with chronic anterior poliomyelitis as a deforming force producing genu valgus and external tibial torsion. The iliotibial band inserts into a prominence on the anterolateral aspect of the lateral tibial plateau called Gerdy's tubercle. Marshall et al have described an intricate relationship between the insertion of the biceps femoris and the lateral collateral ligament. The superficial portion of the tendon of insertion extends over the lateral collateral ligament anteriorly to the deep fascia of the peroneal compartment posteriorly to the fibula and deep fascia of the calf. The middle portion of the insertion of the biceps femoris loops around the distal third of the fibular collateral ligament. Active contraction of the biceps femoris muscle would thereby take up slack in the relaxed fibular collateral ligament when the knee is flexed. The deep portion of
the insertion of the biceps femoris extends underneath the fibular collateral ligament forward
to Gerdy's tubercle. Fibers from the deep portion of the biceps femoris tendon become
confluent with the iliotibial band and take up slack in the band when the knee flexes.

The many interrelated structures of the capsular checkrein function almost as a single
unit. It is difficult to demonstrate the specific contribution of each muscle insertion in an
experimental model, but it can be assumed that the muscles do reinforce the overall structure
during various movements of the knee in vivo. The posterior capsule functions not only as
a checkrein against hyperextension but also helps to control rotation and abduction-adduction.
Any traumatic violation of this structure in part or in whole invites consideration of repair.
Complete disruption of the posterior capsule is usually associated with anterior dislocation of
the knee, which may cause serious injury to the popliteal artery or peroneal nerve. The
integrity of the capsule checkrein is vital to normal function of the knee joint.

Surgical Approaches

Many specific surgical approaches to the knee joint have been described with
correspondingly specific procedures in mind. These have been described concisely in various
textbooks and review articles. The various approaches are summarized.

The median parapatellar approach is the most utilitarian method. The incision begins
several fingerbreadths above the proximal pole of the patella. It is centered over the medial
border of the conjoint quadriceps tendon. The incision extends downward in curvilinear
fashion over the medial border of the patella and along the medial edge of the patellar
ligament. The superficial fascia and retinaculum is incised in line with the incision. The
quadriceps tendon is incised between the conjoint portion and the tendon of insertion of the
vastus medialis. After opening the synovium the patella may be reflected lateralward to
expose the articular surfaces of the distal femur, upper tibia, and patella. Better access to the
tibial collateral ligament and posteromedial corner of the joint capsule may be obtained by
extending the incision posteriorly at its distal end as described by O'Donoghue. Hughston and
Eilers have emphasized that surgical treatment of internal derangement of the knee requires
adequate exposure. A second incision may have to be made either over the posterior aspect
of the medial compartment or over the lateral aspect of the knee to supplement the initial
approach. The synovium, capsule, extensor retinaculum, subcutaneous tissues, and skin should
be closed appropriately in layers. Often early mobilization of the knee with an active exercise
program is desired. It is possible to start isometric quadriceps exercises early in the
postoperative period if the median parapatellar approach is used.

Summary

Satisfactory results from surgical treatment for disorders of the knee joint depend upon
familiarity with the functional anatomy of this region. Significant elements in providing
stability to the knee are the shapes of the opposing bone ends and menisci, the extensor
mechanism, and the capsular checkrein. The concept of grouping specific anatomic parts into
functional units is not only an aid to the memorizing process but a way of emphasizing
restoration of function as the goal of surgical treatment of the knee. Surgical approaches
should be planned to avoid unnecessary violation of the functional unit but should afford in
every case adequate exposure of pathology.
Surgical Anatomy of the Hip

Joseph H. Dimon

The hip joint is a deep joint surrounded by large and powerful muscles necessary for its proper function. Essential neurovascular structures lie in front and in back of the hip. For man's normal daily activities the hip is perhaps the most important joint that he possesses. It is estimated that when one takes a step on the leg in normal walking, forces are transmitted across the hip approximately 2.5 times the body weight to balance the forces acting about the acetabular-femoral pivot point. It is amazing that the hip joint performs so well for so long and so uncomplainingly, in most instances. Occasionally, this mechanism develops problems requiring the surgeon’s attention, and this article directs itself to these occasions.

There are many excellent texts which present the detailed anatomy and the surgical anatomy of the hip joint. This article will emphasize general principles that will be of practical help should a surgical approach to the hip become necessary.

General Principles

Always think in terms of areas, muscle function, and critical neurovascular structures. How can one safely surgically approach the hip, for whatever reason necessary, with minimal trauma to important musculo-skeletal and neurovascular structures?

The hip should always be draped free so that the leg can be moved about, unless the surgery is being done for reduction and fixation of a fracture of the femoral neck or intertrochanteric area which may require reduction, positioning, immobilization, and nailing on a fracture table. If the hip is being surgically approached for other reasons (eg, biopsy, incision and drainage, arthrotomy, synovectomy, arthroplasty, etc), always drape the leg free as the ability to move the joint about will greatly facilitate the identification of anatomically safe and dangerous areas and allow their utilization and protection.

Dissect in line with muscle planes rather than cut across them. If it is necessary to detach muscles, do so at their tendinous insertions or bony origins (subperiosteal at origins). Retract muscles away from the hip joint toward their nerve and blood supply, thus preventing damage and assuring continued function. Always remember the general location of critical structures - the sciatic nerve (posterior), femoral nerve and vessels (anterior) - and retract these away from the hip joint with adjacent structures protecting them (muscle and soft tissues). Remember, flexing the knee and extending the hip will relax tension on the sciatic nerve and straightening the knee and flexing the hip will relax tension on the femoral nerve and vessels.

In order to employ these principles in the exposure of the hip joint, I feel it is necessary to have at least two long and gently curved Cobra retractors, as these tend to find their way instinctively around the periphery of the capsule of the hip joint, allowing protection and gentle retraction of important structures.
Front of the Hip (Anterior Approach)

**Sartorius muscle.** The sartorius muscle runs diagonally across the front of the hip from lateral (anterior superior spine) to medial (inserts medially in the upper aspect of the tibia). Its nerve supply comes in from medially (femoral); it can therefore be detached from its origin (anterior superior iliac spine) and retracted medially.

**Rectus Femoris Muscle.** The rectus femoris muscle lies over the front of the hip joint on top of the capsule and courses straight down the leg from its origin at the anterior inferior iliac spine (straight head) and anterior superior aspect of the acetabulum (reflected head). Nerve supply comes in medially and distal to the hip joint (femoral); it can therefore be detached from its origin and reflected distally and medially.

**Iliopsoas Muscle.** The iliopsoas muscle is the most powerful hip flexor. It sweeps over the pelvic brim across the front and medial aspect of the hip, forming a strong tendon which dives down below the inferior capsule and inserts into the lesser trochanter of the femur. In addition, its muscular fibers fan out and insert distally into the shaft of the femur. The neurovascular bundle (femoral nerve, artery, and vein) lies on top of this muscle beneath the sartorius muscle, and its nerve supply enters in the pelvis medially; the iliopsoas can therefore be retracted from in front of the hip joint medially along with the femoral neurovascular bundle.

**Important Points to Remember in Execution of the Anterior Approach**

Think about mobilization, protection, and retraction of the hip flexors and the femoral nerves and vessels. All are retracted medially as a general rule.

**Surgical Approach**

The patient is placed in a supine position with a small roll or towel under the sacral area on the operative side to life the hip slightly anteriorly. The hip is draped free.

An anterior incision is made, gently curving down over the anterior aspect of the iliac crest in front of the hip.

Palpate the interval between the tensor and sartorius, ie, a hand's breadth distal to the anterior superior iliac spine. Open this interval and a fatty layer will be encountered. The circumflex vessels can be felt as a leash when one passes the finger distally in this interval, and they can be ligated if a wide anterior exposure is going to be necessary. The sartorius origin can be detached from the anterior superior spine and the sartorius gently retracted medially (toward its nerve supply). The tensor origin can be subperiosteoally reflected and retracted posteriorly (toward its nerve supply).

The dissecting finger can then sweep under the edge of the iliopsoas as it emerges from the pelvis, and by directing the finger posteriorly and distally beneath the anterior aspect of the hip, one can mobilize the iliopsoas and its tendon medially along with the femoral nerves and vessels. A Cobra retractor can then be inserted between the medial aspect of the hip and the iliopsoas with the femoral nerves and vessels, thus protecting them.
A Cobra retractor can similarly be placed around the superior capsule of the hip underneath the abductors through the interval identified between the tensor and sartorius, thus protecting the abductors of the hip.

The rectus femoris as it crosses the hip can be detached from the anterior inferior iliac spine and reflected distally and medially (toward its nerve supply) with or without an underlying flap of hip capsule as one prefers.

Vital structures and functional muscles are now protected, exposing the anterior aspect of the hip and the joint can be opened as widely as one desires by incision into or excision of the capsule or both.

**Side of the Hip (Lateral Approach)**

**Tensor Muscle.** The tensor muscle and fascia arises from the iliac crest, fuses with the gluteus maximus posteriorly, and courses over the lateral aspect of the hip and thigh. The nerve to the tensor comes in from behind, just below the iliac crest; the tensor can therefore be split vertically as far as necessary in its midportion (not critical from a standpoint of function) and retracted anteriorly and posteriorly as long as the vertical cut stops two fingerbreadths below the crest of the ilium (nerve will not be severed).

**Gluteus Medius and Minimus.** These major abductors run from the wing of the ilium and insert into the greater trochanter. The anterior edge of the abductor group (gluteus) inserts anteriorly in the greater trochanter so that its leading edge runs almost transversely in front of the hip. Don't be fooled; this *is* the anterior edge of the abductors. The nerve supply and blood supply for the major abductors comes from posteriorly and superiorly; the abductors may therefore be detached at their insertion into the greater trochanter (with a portion of the trochanter to allow firm reattachment) and retracted up and back. If they do not retract easily, the piriformis and superior external rotators may be sectioned at their insertion into the trochanter with no great concern. The hip may be approached in front of or behind the abductors without detaching the trochanter by retracting the abductors and using the position of extreme external rotation (to get to the front of the hip) and internal rotation (to get to the back of the hip). This exposure, without detaching the trochanter and abductors, is more difficult, however.

**Vastus Lateralis Muscle.** The vastus lateralis muscle covers the lateral aspect of the femur from just below the greater trochanter distally. Its nerve supply comes from anteromedially (femoral); it can therefore be subperiosteally reflected forward and medially to expose this area of the femur as necessary. From a practical standpoint it can be split and retracted anteriorly and posteriorly, if you wish. This, in theory, might denervate and devascularize the posterior reflected portion of the muscle, but in clinical practice it seems to make no difference. Because of the anatomy, however, I prefer to retract the muscle anteriorly toward its nerve and major blood supply.
Important Points to Remember in Execution of the Lateral Approach

The abductors should be preserved, and the femoral neurovascular bundle protected as it lies in front of the hip (retracted anteromedially). The sciatic nerve must be protected as it lies posteriorly behind the hip (retract it posteromedially).

Surgical Approach

The lateral approach to the hip may be utilized when the hip is draped free as for biopsy, arthrotomy, arthroplasty, etc, or when the hip cannot be draped free as with the reduction and nailing of fracture on the fracture table. I will first discuss the lateral approach when used for the immobilized hip.

In this particular case the hip is not draped free, but a large area is blocked off with drapes after appropriate preparation. A straight incision is made over the lateral aspect of the hip, and the tensor is incised in line with its fibers. It is retracted, exposing the vastus lateralis muscle covering the upper one-third of the femur laterally. The vastus lateralis is then incised along its posterior aspect just in front of the linea aspera and subperiosteally reflected anteriorly. A Cobra or Bennett retractor is placed under this muscle to protect the posterior structures including the sciatic nerve. This exposes the upper lateral aspect of the femur, and appropriate drill holes, guide wire and nails, and/or appliances can be inserted with proper x-ray control for internal fixation of hip fractures.

Now, let us discuss the lateral approach to the hip with the leg draped free. A lateral incision is made over the trochanter of the hip, and this can be swung posteriorly, dividing the fibers of the gluteus maximus if one wishes, or slightly anteriorly in the interval between the tensor muscle where it blends with the maximus. I usually prefer the latter. This interval is opened, exposing the greater trochanter of the hip, often obscured by the trochanteric bursa which is dissected off the trochanter so that one can better identify the structures. A Cobra retractor is then placed posterior to the hip around the inferior aspect of the capsule through the external rotators, and this protects the sciatic nerve posteriorly. The examining finger then slides along the anterior aspect of the hip under the brim of the pelvis beneath the flexors, femoral vessels and nerves, and a Cobra retractor is inserted here and gently retracted to protect these anterior structures. The greater trochanter can then be osteotomized and reflected superiorly with the attached abductor muscles, incising the insertion of the piriformis and upper external rotators into the trochanter posteriorly, if necessary, to mobilize the trochanter. The trochanter, with its abductors, is then retracted superiorly exposing the underlying capsule. Vital structures are protected by the various retractors. The capsule can then be opened and/or excised and the hip further inspected as necessary.

Back of Hip (Posterior Approach)

The posterior aspect of the hip is covered superficially by the gluteus maximus. It can be split in line with its fibers as its nerve and blood supply arborize medially from its undersurface. Split the maximus in the lower one-third if possible.

Sciatic Nerve. The sciatic nerve is in the layer of fat between the gluteus maximus and the external rotators of the hip. To locate this layer, punch a Kelly or other clamp through
the substance of the gluteus maximus, open this hole by opening the clamp, separate the muscles with your fingers, and the fatty layer will appear underneath the gluteus maximus. The sciatic nerve lies in this layer and can be palpated here with the examining finger and retracted posteriorly and medially away from the hip joint. Retracting it in this direction decreases the stretch on the nerve, as do flexing the knee and extending the hip.

**External Rotators.** The external rotators lie across the back of the hip in close relation to the posterior capsule. These may be cut at their insertion into the femur and greater trochanter and retracted posteriorly and medially (toward their nerve and blood supply).

**Important Points to Remember in Execution of the Posterior Approach**

Think about identification, protection and retraction of the sciatic nerve which lies underneath the maximus and on top of the external rotators.

**Surgical Approach**

The patient is placed on the opposite side and turned forward about 30 degrees. The hip and leg are draped free. An incision is made along the inferior border of the gluteus maximus in line with its fibers, and this may be extended down the side of the hip into the tensor fascia area if a wide exposure is necessary. The gluteus maximus fibers are split, and the fatty layer beneath the gluteus is identified and the sciatic nerve palpated with the finger. The nerve is always retracted gently posteriorly and medially to protect it. The gluteus maximus is then further split distally down into the tensor fascia area, if necessary. A Cobra retractor can then be placed around the superior aspect of the hip capsule beneath the abductors, thereby protecting them. A second Cobra is placed inferiorly underneath the capsule through the substance of the external rotators, exposing the external rotators overlying the posterior capsule of the hip at that level. The external rotators are then incised at their insertion into the trochanteric area of the hip and are retracted medially toward their nerve supply, thereby exposing the posterior capsule of the hip. The capsule can then be incised for exposure of the hip joint itself.

**Medial Approach to the Hip**

The medial aspect of the hip may be approached by going through the adductor area. This approach is rarely used by the general surgeon or orthopedist. It allows one to get down to the area of the lesser trochanter for biopsy and/or release of the psosas tendon at its insertion. It allows only limited access to the hip, and then with difficulty.

If one elects to use this approach, the patient is placed supine with the hip and leg draped completely free. The leg is flexed and externally rotated. A linear incision is made over the aspect of the thigh in line with the adductor muscles. The interval between the adductor brevis and longus anteriorly with the adductor magnus posteriorly is bluntly separated with a clamp, retracting the adductor brevis and longus forward and the adductor magnus posteriorly, and the femur is palpated with the examining finger. Two Cobra retractors are placed subperiosteally about the femur, retracting the vital structures forward and backward. This exposes the area of the lesser trochanter, the insertion of the iliopsoas tendon, and the distal inferior capsule of the hip joint. Exposure is limited, making this
approach best reserved for biopsy of the area of the lesser trochanter and/or release of the iliopsoas tendon.

**Philosophy**

In studying hip surgery under Doctor Otto E. Aufrane, an internationally known hip surgeon, at the Massachusetts General Hospital, I learned to respect and follow the following principles. Handle all tissues gently; release traction frequently to restore intermittent blood supply; and always keep in mind where the critical vital structures are or should be. Appreciate the fact that a previously operated hip's anatomy may be different from normal. Study the problem and be thinking of it far in advance of the surgical procedure. Considerable thought and reflection on the problem will pay off at the time of surgery. These principles have superbly withstood the test of clinical application.

**Conclusion**

The principles taught in this particular article are simply broad ideas and generalities that should be kept in mind. This paper does not purport to be a substitute for detailed and thorough review of the specific approach that one selects when exposing the hip joint. Heeding these principles, however, may reduce the possible problems that one might encounter in surgically investigating this interesting and deeply situated joint.
Muscle Transposition Flaps for Coverage of Lower Extremity Defects

Anatomic Considerations

Stephen J. Mathes, John B. McCraw, Luis O. Vasconez

Full thickness skin loss in the lower extremity presents special difficulties to the surgeon and his patient. Soft tissue in juxtaposition to the wound is usually remarkably thin and poorly vascularized. Moreover, the base of the wound very often is exposed, dead, infected bone. These chronic, persistent, economically debilitative ulcerations, traumatic, vascular, metabolic, or infectious in origin, classically are managed by split thickness skin grafts or distant flaps. In 1966, Ralph Ger suggested the use of muscle as a transposed flap as a practical single stage technique to accomplish coverage for these difficult ulcerative lesions.

The principle of muscle transposition to provide cover for areas of tissue loss in the lower extremity is now well established. Critical to successful, safe, effective transfer of such a muscle flap is a precise working knowledge of the anatomy of the muscle under consideration. A number of criteria based on muscle anatomy and function have been defined to evaluate the potential of a muscle for its use as a transposition flap.

To be suitable for transposition a muscle must have a belly of reasonable size. One determinant of the extent of rotation is muscle length. The site of the principal arterial source and venous return equally influence the feasibility of the use of a particular muscle as a flap. If the vascular pedicle is located close to the origin, the muscle can then be extensively rotated with safety. In cases of multiple arterial muscular branches, the branches in the proximal third of the muscle must be dominant or the muscle will not survive as an immediate transposition flap. Under these circumstances a delay procedure would be prudent.

A lower extremity muscle used in transfer can obviously no longer serve its original locomotor function. Consequently, there should be an ample number of synergistic muscles to compensate for this deletion of muscular function. Preservation of motor innervation is not an absolute requirement in the muscle flap since muscular contractions in the rotated position serve no function. In fact, in some circumstances the eventual decrease of denervated muscle bulk well may be a desirable feature.

Brash's anatomical study of the lower extremity offers excellent data on the site of motor nerve entry into the muscles of the lower extremity. Data concerning the site and variability of the arterial supply of the lower extremity muscles have not been as well documented. While exploring the possibilities of muscles for use in transfer procedures, therefore, we found it necessary to perform detailed vascular dissections on amputated limbs. Dissections on ten such above knee amputations with the vasculature injected with colored latex for precise identification and localization of arterial muscle blood supply has provided these data. Results of this anatomic study on muscles in the lower extremity which have potential for use as a muscle flap are presented.
**Gastrocnemius**

The gastrocnemius is a superficial posterior leg muscle composed of medial and lateral bellies that merge midway in the leg to form a common tendon. The longer medial belly may be utilized to cover defects in the proximal third of the tibia. The muscle can be exposed by a midposterior incision. Our dissections disclosed that each belly receives a single large artery derived from the popliteal artery. These branches penetrate the muscle above the level of the tibial epicondyles. Loss of the gastrocnemius is easily compensated for, since plantar flexion can be performed by the soleus, flexor digitorum longus, flexor hallucis longus, tibialis posterior, and plantaris muscles.

**Soleus**

This superficial posterior muscle has a broad muscle belly admirably suited for transfer procedures. It lies anterior to the gastrocnemius and is best exposed through a posteromedial incision. It inserts as part of the Achilles tendon and must be sharply separated at this level for use in transfer procedures. If transposed, it can cover defects in the middle third of the leg. In our dissections, the dominant arterial vessels to the muscle were branches from the peroneal artery and a large proximal branch from the posterior tibial artery. There were two or three more inferior branches derived from the posterior tibial artery. The more proximal branches were consistently present, allowing safe transfer. The most distal branch can be sacrificed and the muscle transposed safely on the more proximal vascular pedicle. Following transfer, the plantar flexion of this muscle would be preserved by the other strong plantar flexors mentioned above. Except in rare circumstances, the soleus and gastrocnemius have not been utilized in the same lower extremity since plantar flexion would be seriously impaired in such cases.

**Flexor Digitorum Longus**

The flexor digitorum longus is a deep posterior leg muscle with a moderate size belly. Since this muscle is immediately posterior and adjacent to the soleus muscle, it is best used for supplementary coverage with the soleus muscle to correct defects in the middle third of the leg. In our dissections there were constant arterial branches entering the proximal third from the posterior tibial artery. However, there was also a distal third arterial branch derived from the posterior tibial artery. Sacrifice of this distal branch in transfer might be critical in certain cases. The function of phalangeal flexion would be preserved by the flexor digitorum brevis.

**Peroneus Longus**

The peroneus longus is a lateral leg muscle with a belly suitable in size for transfer for coverage of defects in the lateral middle or distal third of the leg. Exposure may be gained through a posterior lateral incision. The source of arterial blood supply to this muscle is the peroneal artery. One or two arterial branches enter the more proximal portion of the muscle mass. Plantar flexion and foot eversion would persist with deletion of this muscle function provided the foot flexors and the peroneus brevis muscle remained intact.
Peroneus Brevis

A second lateral leg muscle is the peroneus brevis. Its smaller belly is located inferior and adjacent to the peroneus longus. It represents a muscle for use with the peroneus longus for coverage of large defects in the lateral inferior third of the leg. In our dissections, the arterial supply was consistently a branch from the peroneal artery entering the proximal portion of the muscle. When used along with the peroneus longus muscle in transfer, the foot would lose eversion. Such a loss in most circumstances would generally be an acceptable disability.

Abductor Hallucis

The abductor hallucis muscle is a medial posterior intrinsic muscle of the foot. It has an adequate muscle belly for coverage of defects in the area of the medial malleolus. The muscle can be reached by a medial transverse incision in the foot. In our dissections, the arterial source originates from branches of the medial plantar artery. Consistently there was a large proximal branch. Variably, two to three smaller branches entered along the muscle belly. With transfer of this muscle, its function of great toe abduction and foot spring would be lost.

Abductor Digiti Minimi

This muscle is a lateral posterior intrinsic muscle of the foot. This muscle mass may be transposed to provide coverage of defects about the lateral malleolus. Access to the muscle is easily accomplished through a lateral horizontal incision in the foot. In our dissections, the arterial supply for this muscle is consistently from one branch derived from the lateral plantar artery. This branch enters close to the origin of the muscle belly. Some loss of abduction and flexion of the fifth toe would be expected following this muscle's transfer.

Flexor Digitorum Brevis

The flexor digitorum brevis muscle is a posterior intrinsic muscle of the foot. It has a muscle belly which, when folded posteriorly, can provide coverage of defects overlying the calcaneus and heel. The muscle is exposed through a midplantar incision. In our dissections, the muscle has dual blood supply from an arterial branch of the medial plantar artery along the midbelly and from an arterial branch from the lateral plantar artery proximal near its origin. This second source of arterial supply appears constantly and should be adequate to preserve the muscle circulation with transfer. Following transfer of this muscle, lateral phalangeal flexion would be preserved by the action of the flexor digitorum longus.

Summary

The latex injection technique, even in extremities amputated for vascular disease, can provide anatomic specimens that yield information about the precise localization of arterial supply to muscles. Dissections in such specimens have provided the necessary surgical anatomic knowledge for safe, effective, one-stage muscle transposition procedures. The vascular anatomy of muscles suitable for transposition flaps in the leg and foot has been
described to aid the surgeon whose practice includes the care of patients with skin and soft tissue losses.
The Index Finger - A Unique Creature of Habit

Robert W. Crow

The forefinger serves to exemplify the precise movements of the human hand. It distinguishes itself as the first of four digits with its independence of flexion and unique ability to extend alone. When active it is the initiator of grasp and a complement to the thumb in pinch. When disturbed, in contrast, it can impede hand motion and serve as a barrier to the same functions that it helps when intact.

It shall be the purpose of this paper to discuss the anatomy of this digit and then allude to the more common problems found with it, accompanied by a brief discussion of the accepted methods of restoration for each of these insults.

Range of Motion

The index finger is capable of a full range of motion. It can flex and extend at the distal and proximal interphalangeal joints as well as at the metacarpophalangeal joint. In addition, it has the ability to abduct and adduct at the latter joint which makes it a "diarthrodial multi-axial joint." The metacarpal of this finger is usually the longest of all the metacarpals in the hand. Its base is forked and opens in the central portion for articulation with the lesser multangular. The two sides of this fork articulate radially with the greater multangular and ulnarily with the styloid process at the base of the third metacarpal. The dorsal aspect of the radial articulation serves as the site of insertion of the extensor carpi radialis longus tendon. This unit with its companion, the extensor carpi radialis brevis, which inserts into a similar site on the adjacent third metacarpal, serves as a stabilizing element of the wrist. In severe injuries to the index finger requiring ray amputation, this insertion serves as the proximal line of division of the metacarpal in order to preserve the transverse arch of the hand as well as the stability of the wrist. The ulnar side of the base of this metacarpal is the site of insertion of the interosseous ligament which joins the bases of the second and third metacarpals. The radial volar aspect of the base is also the site of part of the origin of the adductor pollicis muscle, sharing in this capacity with the third metacarpal and the greater and lesser multangular as well as the capitate.

The shaft of the second metacarpal is somewhat flattened, especially on the dorsal surface, but in most other respects is not unlike the remaining metacarpals. The head is large and somewhat irregular while the articular surface is longer on the volar than on the dorsal surface. This latter plateau is a site for the beginning of the capsule of the metacarpal phalangeal joint.

Phalanges

Phalanges do not differ markedly from those of adjacent digits and, therefore, will not be discussed in detail except for their connection with the overlying flexor and extensor mechanisms as well as the surrounding soft tissue which will be alluded to in detail below.
**Extensor Mechanism**

The extensor mechanism is somewhat unique in this finger. The common extensor tendon, the extensor digitorum communis, sends limbs to all four digits of the hand. It arises from the lateral humeral epicondyle as well as the intermuscular septum and then passes through the hood of the fourth dorsal compartment ulnar to Lister's tubercle to insert into the dorsum of the proximal phalanx. This pattern is essentially duplicated in all digits but, uniquely to the index finger, the extensor indicis proprius, originating from the posterior surface of the ulnar as well as the interosseous membrane, runs distally with this common extensor and passes anteriorly to it as it extends through the synovial sheath. It then deviates medially to insert on the ulnar side of the slip of the common extensor into the proximal phalanx. This unique characteristic provides the index finger with the ability to extend itself independently of the adjacent fingers. It also provides an excellent tendon to be used for transfer when thumb extension is deficient or absent and cannot itself be repaired. From the proximal phalanx distally the extensor mechanism of the index finger is not unlike that of the other fingers and divides into a central slip which inserts into the middle phalanx and then extends via two lateral bands which join the lumbrical and interosseous muscle to run distally to terminate at the proximal end of the terminal phalanx. The insertion here is just proximal to the matrix of the nail and this structure is frequently injured concurrently with the tendon in disruptions of this distal insertion.

**Intrinsic Muscles**

The intrinsic muscles which aid the extensor tendons are somewhat different in the index finger than in adjacent digits. One limb of the muscle inserts into the lateral tubercle of the proximal phalanx and the second inserts into the dorsal hood without contribution to the lateral slip of the first lumbrical. This difference allows the first dorsal interosseous to flex the proximal phalanx more effectively in grasp, especially in the thenar web. Because of the absence of the first dorsal interosseous to join the tendinous insertion of the first lumbrical, it does not help in extension of the middle and distal phalanges, as is the case in the other three adjacent digits. This function is supplied fully by the lumbrical muscle and the volar interosseous muscle, which inserts into the dorsum of the middle and proximal phalanges and then joins the terminal phalanx through the lateral band as previously described.

**Flexor Mechanism**

The flexor mechanism of the index finger is not unlike those of the adjacent fingers. The position of the superficial tendon at the wrist is almost always dorsal to those of the third and fourth fingers and usually shares this position with the superficial tendon to the fifth finger. The flexor digitorum profundus runs with the long tendons to the other digits and its function and mode of action are common to each of them.

**Blood Supply**

Blood supply is furnished by branches of both the radial and ulnar vessels. Distal to the pisiform the ulnar artery divides into two branches. The larger remains superficial and joins a smaller branch of the radial artery to form the superficial palmar arch. This arch then
gives rise to the common volar digital vessels which then give origin to the proper digital artery on the ulnar aspect of the index finger.

The smaller branch of the ulnar artery joins the deep branch of the radial artery and proceeds to pass between the heads of the first interosseous to supply the first metacarpal. The proper radial digital vessel most often arises from this branch, and in amputations of the index finger this vessel must be divided distal to the metacarpal artery or the digital vessel to the thumb which also arises at this site may be jeopardized.

The venous drainage is supplied by both a superficial and a deep system. The superficial veins provide the majority of the drainage of the finger. The dorsal digital vessels pass along the sides of the finger and join one another by communicating vessels. The digital vessel on the radial side of the index finger and the venous channels from the thumb provide the radial portion of the dorsal venous drainage on the thenar aspect of the hand as they merge to form the cephalic vein which then proceeds proximally. The volar digital veins accompanying the corresponding arteries on each side of the finger arise from the deep system and connect to the superficial vessels by oblique intercapitular veins. In neurovascular island transfers these venous components should be protected and transferred, as well as the accompanying arteries, to provide adequate drainage for the pedicle.

The lymphatic supply to the index finger largely follows the venous supply and is thus composed of both a superficial and a deep component. The lymph drainage from the finger follows the course of the cephalic vein along the radial aspect of the forearm and empties into the thoracic duct. This is important in cases of infection or tumor involving the index finger, as often the first sign of distal involvement is the presence of a node in the deltopectoral triangle in contrast to nodes in the epitrochlear region which appear much sooner in diseases on the hypothenar aspect of the hand.

**Innervation**

Sensation of the index finger is supplied by components of the radial, ulnar, and median nerves. Innervation of the volar aspect is provided by the digital branches of the median nerve. These also extend to the dorsum to provide sensation to the distal one and one-half phalanges on that surface. Because of this circumferential and almost constant pattern, the index finger is an excellent site to determine interruption of sensation supplied by the median nerve. The dorsal sensation for the proximal portion of the mid and proximal phalanges is supplied by the superficial branch of the radial nerve and its accompanying digital branches.

Motor innervation is supplied by components of each of the three nerves as well. The long flexor tendons are both median innervated while the intrinsic musculature is supplied by both median and ulnar nerves. The latter is important in isolated ulnar nerve injury at the wrist for only the interosseous muscles are denervated while the lumbricals remain intact in the index and midfinger, providing a means of preserving metacarpophalangeal flexion and interphalangeal extension, a function which is usually lost in the ulnar two digits in the same kind of injury. The common extensor tendon and indices proprius are supplied by the radial nerve as are the other finger and thumb extensors.
Summary

The index finger has now been described in its anatomic relationship to the hand as well as in its unique capacity as the "digit of individualization." It is an essential part of the fine precise motion of pinch and grasp when it is intact and can substitute for the thumb when needed in cases of severe injury or amputation. When injured, however, the index finger is a significant barrier in the same capacity that it functionally aids, and despite reconstructive attempts is sometimes subjected by mutual decision of both the physician and the patient to amputation. The index finger is truly "a unique creature of habit".
The Anatomic Basis of Vascular Compression of the Duodenum

John T. Akin, John E. Skandalakis, Stephen W. Gray

Compression of the third or fourth part of the duodenum by the superior mesenteric artery or one of its branches is the anatomic basis for many cases of duodenal obstruction. Various names have been applied: superior mesenteric artery syndrome, Wilkie's disease, and arteriomesenteric ileus. Fluctuation in reported prevalence and the frequent association with neurosis have produced occasional skepticism as to its objective existence. We believe it to be a real disease entity with an anatomic basis best described by the name vascular compression of the duodenum.

Rokitansky is credited with first describing duodenal compression by the superior mesenteric artery. By 1908, Laffer in this country was able to collect 217 cases including four of his own. Interest lagged until 1927 when Wilkie reported 75 cases of his own. Not all of these older cases were well documented. Barner and Sherman critically reviewed the subject in 1963 and found only 281 cases that they considered acceptable.

Embryology

A brief review of the development of the small intestine will explain how the superior mesenteric artery and the duodenum reach the positions in which they are found in the adult abdomen.

The superior mesenteric artery of the adult, together with the celiac axis and the inferior mesenteric artery, is a remnant of the arterial part of the primitive vitelline circulation which supplies the embryonic yolk sac with blood. Originally paired and segmentally arranged, the pairs have fused and their number reduced to three by the 6th week of development. The superior mesenteric artery extends past the intestine and into the yolk sac as long as the organ persists in the umbilical cord. It is the extension that supplies blood to a persistent Meckel's diverticulum.

Up to the middle of the 5th week the midgut is contained within the body of the embryo. Late in the 5th week it begins to elongate faster than does the embryo itself, and pushes out into the umbilical cord. At the same time the herniated intestinal loop rotates 90 degrees counterclockwise so that the future duodenum lies to the right of the embryonic superior mesenteric artery, and the future colon lies to the right. With the return of the elongated intestines to the abdomen during the 10th week, the proximal portion of the loop, the small intestine, returns first. As a consequence, the duodenal portion passes to the left, beneath the superior mesenteric artery. The colon returns soon afterward, so that the transverse portion lies in front of both the duodenum and the artery. Growth of the colon continues after the intestines return to the abdomen. The cecum, at first under the liver, eventually descends to the right lower quadrant of the abdomen. The adult position of the intestines is usually reached only after birth and ends with the fusion of the ascending and descending mesocolon with the abdominal peritoneum.

It should be emphasized that patients with vascular compression of the duodenum have normally rotated intestines. Cases are known in which compression is the result of intestinal
malrotation with grossly altered vascular and intestinal relationships. These rare cases are not included in our discussion of the syndrome.

Surgical Anatomy

One of the consequences of man’s erect posture is that the superior mesenteric artery leaves the aorta at an acute downward angle rather than at a nearly right angle as in quadrupeds. It is through this vascular angle, between the aorta and the superior mesenteric artery, that the third or fourth portion of the duodenum passes. The posterior limb of the angle is formed by the vertebral column and the paravertebral muscles as well as by the aorta; the anterior limb is formed by the superior mesenteric artery in the mesentery and sometimes by one of its first two branches, the middle and right colic arteries, in the overlying transverse mesocolon. The duodenum is suspended in the angle by the ligament of Treitz. The uncinate process of the pancreas and the left renal vein occupy the narrowest part of the angle above the duodenum. These relationships are shown diagrammatically in sagittal section and in anterior view. The duodenum usually crosses the vertebral column at the level of the third lumbar vertebra and the superior mesenteric artery usually arises from the aorta at the level of the first lumbar vertebra. The suspensory muscle of the duodenum (ligament of Treitz), consisting of smooth muscle and fibrous tissue, connects the duodenal flexure and the right crus of the diaphragm.

Variations of the Ligament of Treitz

Haley and Peden showed that the attachment of the ligament of Treitz is variable. It may be attached to the duodenojejunal flexure only, to the flexure and the fourth and even the third parts of the duodenum, or the fourth and third parts of the duodenum only. We have also seen the ligament formed by multiple, separate divisions. Attachment to the flexure and the fourth part is the most frequently encountered. It has been suggested that there is hypertrophy of the smooth muscle fibers in the ligament of Treitz in patients with the clinical syndrome of vascular compression of the duodenum.

Variations in the Level of the Duodenum

Haley and Peden showed that the attachment of the ligament of Treitz is variable. It may be attached to the duodenojejunal flexure only, to the flexure and the fourth and even the third parts of the duodenum, or the fourth and third parts of the duodenum only. We have also seen the ligament formed by multiple, separate divisions. Attachment to the flexure and the fourth part is the most frequently encountered. It has been suggested that there is hypertrophy of the smooth muscle fibers in the ligament of Treitz in patients with the clinical syndrome of vascular compression of the duodenum.

Variations in the Level of the Duodenum

In most patients the duodenum crosses the vertebral column at the level of the third lumbar vertebra. In the remainder, more often in women, it crosses at a lower level. In a few it may cross at the level of the second lumbar vertebra. In such cases the ligament of Treitz may be shorter than usual, bringing the duodenum higher into the angle between the superior mesenteric artery and the aorta. A short ligament does not always raise the level of the third
part of the duodenum but may, instead, raise only the flexure, increasing the angulation of the distal fourth part of the duodenum. Either the third or fourth part of the duodenum crosses the vertebra, although in about 1 per cent of individuals the duodenojejunal junction lies to the right of the spine.

Crossing the spine at a lower level might seem to allow more room for the duodenum, but this is not necessarily the case. The spine turns anteriorly at the lumbar curve, reaching its most anterior position at the fourth lumbar vertebra so that the space between the anterior and posterior limbs of the angle is no greater at the level of the fourth lumbar vertebra than at the level of the third. This lumbar curvature is usually greater in women than in men.

The Superior Mesenteric Artery and Its Branches

The superior mesenteric artery arises from the aorta at the level of the first lumbar vertebra in most individuals. This level may vary from that of the twelfth thoracic vertebra to that of the disc between the second and third lumbar vertebrae, but 75 per cent will arise between the upper one third of the first lumbar vertebra and the disc between the first and second lumbar vertebrae. Its course and relation to structures posterior to it are shown. A groove on the anterior surface of the duodenum sometimes indicates the track of the superior mesenteric artery. The authors have observed this groove.

At the inferior border of the pancreas, the superior mesenteric artery gives off the middle colic artery. This vessel lies in the transverse mesocolon and crosses the third part of the duodenum in its normal position. The right colic artery to the mesocolon arises at the level of the inferior border of the duodenum; it may or may not cross the duodenum. Either the superior mesenteric artery itself or the middle colic artery may compress the duodenum; the right colic artery can rarely be implicated.

Some measurements of the normal angle formed by the superior mesenteric angle and the aorta have been made in cadavers and, by radiography, in living patients. Three of the living patients had clinical evidence of duodenal compression which was confirmed by surgery in two. The published measurements are shown.

Discussion

It is apparent from these relationships that the duodenum may become compressed by relatively minor alterations of the normal anatomy. Such alterations may be produced by extrinsic or intrinsic factors.

Extrinsic Factors

Prolonged abdominal compression can decrease the aortomesenteric angle. A body case is an occasional source of such compression. Such vascular compression of the duodenum has been called the "cast syndrome" by Dorph.

Prolonged bed rest in the supine position results in continuous, unrelieved pressure on the superior mesenteric artery and the duodenum by the weight of the overlying transverse colon.
**Intrinsic Factors**

Progressive lordosis or scoliosis may decrease the distance between the lumbar vertebrae and the superior mesenteric angle.

Marked, rapid weight loss may result in loss of fat so that the superior mesenteric artery impinges more directly on the duodenum.

Most cases of vascular compression of the duodenum cannot be explained so readily. A high duodenum with a small aortomesenteric angle may leave adequate room for the duodenum in childhood, but the space may become inadequate as the duodenum grows with age. More affected patients are in the first three decades of life than in any other comparable period. The greater lumbar curvature in women, perhaps increased by child bearing, may be reflected in the greater number of affected women over 30 years of age.

We suggest that the duodenum fits so exactly into the vascular angle that apparently normal variations of the aortomesenteric angle, the level at which the duodenum crosses the vertebral column, the length and attachment of the ligament of Treitz, or the degree of lumbar spinal curvature may all predispose to duodenal compression. With a high duodenum the third or fourth part may be compressed by the angle formed by the superior mesenteric artery and the aorta. With a short ligament of Treitz the angulation of the duodenojejunal attachment may contribute to the obstruction. With a low duodenum, the compression will be between the superior mesenteric or middle colic artery and the lumbar vertebrae or the paravertebral muscles. Increasing lordosis of age will contribute further to the danger of compression. We believe there is an anatomic basis for vascular compression of the duodenum which must be differentiated from anorexia nervosa.
A congenital diverticulum of the intestine is a continuous threat to the health of its possessor. If it does not become inflamed or ulcerate, it may produce obstruction. If these hazards fail to materialize, neoplastic changes may appear in later life. Only a few such diverticula remain silent throughout a long life.

The embryogenesis of three varieties of congenital diverticula is described here. There are some other diverticula of doubtful congenital origin and many of demonstrable acquired origin. These are usually smaller and less dramatic, both in actual size and pathology.

Of the three types discussed, only Meckel's diverticulum can be considered common. The other two are rare enough that the possibility of their presence is usually overlooked until revealed at operation or necropsy.

**Meckel's Diverticulum**

The most common intestinal anomaly is the persistent of Meckel's diverticulum, a vestigial structure arising from the terminal ileum. It is present in about 2 per cent of the population and is usually said to be more frequently found in males than in females. This is because more males than females suffer from Meckel's diverticulum disease. The sex bias disappears when the diverticulum is reported incidental to surgery or autopsy. This is illustrated in a short series of our own.

The diseases that call attention to the diverticulum are obstruction, ulceration, inflammation, neoplasm, or umbilical symptoms. Most of the sex difference is caused by the greater frequency of obstruction caused by Meckel's diverticulum in male children.

Meckel's diverticulum represents the persistence of an embryonic structure that normally disappears in the 5th week of gestation, or soon afterward, leaving no trace. It is formed early in development as the connection between the primitive gut and the yolk sac and is first known as the vitelline duct. Just before it separates from the gut, its epithelium shows the start of crypt formation, and mucous granules appear in some cells. This suggests that in normal development the potential for growth and differentiation of this tissue is present but is suppressed. If the suppression mechanism fails to operate, the tissue not only persists but grows and develops at much the same rate as the normal ileum.

Whether a blind-ending diverticulum or a patient omphaloileal fistula is left depends upon the length of the unsuppressed portion of the duct. If the whole of it is withdrawn into the abdomen with the return of the intestines during the 10th week, there will be a diverticulum. It its tip remains in the cord, ligation of the cord at birth will result in a fistula. Very rarely only a distal segment of the duct escapes suppression, and a cyst, umbilical sinus, or umbilical polyp results. Although these latter anomalies are related to Meckel's diverticulum, they are outside the scope of this discussion.
From its developmental origin, Meckel's diverticulum must arise from the antimesenteric border of the ileum, although some diverticula may curl around the ileum and adhere to the mesentery. Diverticula that arise from the mesenteric border are not Meckel's.

Meckel's diverticulum may be the same diameter as the ileum or it may be smaller. Most diverticula will be from 1 to 5 cm long but a few may be much longer. About one-half will be found between 46 and 91 cm from the ileocecal valve. An average distance may be considered to be 40 cm in an infant and 50 cm in an adult. Not less than 5 feet of ileum should be examined in order not to miss a Meckel's diverticulum. There are, of course, limits; we do not believe diverticula arising from the jejunum or the colon to be of Meckelian origin.

In about 25 per cent of Meckel's diverticula the tip is attached by a fibrous cord to the abdominal wall at the umbilicus. The cord represents either the distal vestigial portion of the diverticulum or the fibrosed portion of the superior mesenteric artery beyond the normal ileum. Such attachment increases the danger of obstruction from volvulus around the axis of the diverticulum.

Although Meckel's diverticulum arises from that portion of the intestine destined to become ileum, patches of gastric, pancreatic, duodenal or colonic mucosa in various combinations are common. Gastric mucosa is of special interest because, if it has fundic glands (which secrete hydrochloric acid), symptomatic ulceration of the diverticulum or the adjacent ileum will occur. Gross found gastric mucosa in 54 per cent of 130 diverticula examined histologically. Some writers have placed it as high as 80 per cent. Most of this ectopic mucosa contained fundic glands.

Obviously many Meckel's diverticula remain silent throughout life, but their anatomy predisposes them to some specific lesions.

**Intestinal Obstruction.** A diverticulum attached to the anterior abdominal wall may cause volvulus of the ileum. One not so attached may become the leading point of an intussusception.

**Ulceration.** Hydrochloric acid secreting glands of ectopic gastric mucosa will, if present, produce ulceration.

**Inflammation.** Meckel's diverticulum, like the appendix, may become inflamed.

**Neoplastic Disease.** Nothing in the anatomy or embryology of the diverticulum suggests a cause for the high incidence of neoplasia. More carcinoid tumors and leiomyosarcomas are found in Meckel's diverticulum than would be expected from its size and frequency of occurrence. Curiously, both tumors are found predominantly in males.

**Internal Diverticula**

Internal diverticula produce no visible outpouching of the intestine. The diverticula are contained in the mucosa of the wall (intramural) or hang nearly free in the intestinal lumen (intraluminal). The external appearance of the intestine is that of simple intrinsic obstruction. Both types of internal diverticula are the result of developmental errors in the 5th to 8th
weeks of embryonic life. In both cases the diverticulum, at first small, increases slowly in size, producing first chronic obstruction and eventually acute obstruction in postnatal life.

Starting about the 5th week, the embryonic epithelial tube which represents the future gut elongates faster than does the body of the embryo. In the 6th week the increasing length causes the intestine to herniate into the umbilical cord. Such growth requires rapid multiplication of epithelial cells to cover the increasing surface. Cell division becomes so exuberant that more epithelial cells are produced than are required; the original single layer of cells becomes many layered to the extent that the lumen is, in some places, occluded by epithelial cells. Such occlusion does not take place in all parts of the intestine or, perhaps, in all embryos. It is an incidental result of epithelial proliferation rather than a necessary stage in gut development.

In addition to filling the primary lumen of the intestine, the epithelial cells also bulge into the antimesenteric mesenchyme forming bud-like diverticula. These buds are more frequently observed in the duodenum and the ileum than in the jejunum. Over 40 have been counted in a single embryo.

Normally, as growth of the gut slows, fewer cells divide and vacuoles appear in the thick epithelium; these vacuoles coalesce to restore the intestinal lumen, and the diverticular buds, projecting into the mesoderm, are absorbed. The epithelium is again a single layer by the 10th week.

If the lumen is not restored normally, a pocket may remain in the epithelium, or a solid plug of epithelium may occlude the entire lumen. There is some evidence that mesenchyme is stimulated to grow into such areas of solid epithelial cells, thus establishing a blood supply and a connective tissue core. The pocket will thus become a diverticulum or even a completely closed cystic "duplication" within the intestinal wall. It will be separated from the normal lumen by a septum of connective tissue lamina propria covered on both sides by intestinal mucosa. The mucosa of the diverticulum will correspond to that of the normal intestine at the same level.

Should a solid epithelial plug persist, it will also develop a connective tissue core. Such a septum is often complete, and presents as membranous septal atresia shortly after birth. Sometimes the septum is incomplete, with a central aperture large enough for intestinal contents to pass, but presenting enough resistance that a pocket forms in the valve-like septum. Such a pocket will gradually develop into an elongated sac. This sac is an intraluminal diverticulum. Such diverticula tend to enlarge until chronic or acute obstruction develops.

In some cases, recanalization of the intestinal lumen occurs before the transitory diverticula in the peripheral mesenchyme mentioned above have been absorbed in the growing intestinal wall. The diverticula thus become permanent. They usually remain intramural. If they continue to grow with the intestine they will be true, congenital intestinal diverticula.
Dorsal Intestinal Diverticula

In contrast to Meckelian diverticula that always arise from the antimesenteric border of the intestine, there is another group of diverticula that arises from the mesenteric border. This group includes short diverticula that lie in the mesentery as well as those, more aptly termed duplications, that are several feet in length. The most dramatic are those that arise from the duodenum or jejunum, pass through the diaphragm, traverse the thorax, and attach to a cervical vertebra. These dorsal enteric diverticula or duplications are not restricted to the small intestine but may arise from any portion of the alimentary tube. We will confine ourselves to those arising from the small intestine.

Dorsal diverticula may arise by developmental accident during the 3rd week of embryonic life. Before the notochord appears the embryo consists of two cell layers; ectoderm, from the midline of which will form the neural tube, and endoderm, which will form the future alimentary tract. With the forward growth of the notochord during the 18th to 21st day, the endoderm separates from the ectoderm but becomes attached to the growing notochord. At about the 24th day the mesodermal notochord separates from the endoderm. At this time the dorsal elements of the embryo (ectoderm and notochord) are growing cranially faster than is the endoderm. If, during this shearing movement, the notochord has not completely separated from the endoderm, a thin band of endodermal cells will be pulled craniod and dorsad, forming a bridge between the two structures. Should the endodermal band remain attached at both ends, then it will reorganize itself into a tubular structure similar to embryonic gut. The epithelium will induce the surrounding mesenchyme to form the typical connective tissue and muscular coats, and the result will be a giant diverticulum passing from the intestine and attaching to the vertebra. At the point of attachment, one or more vertebral bodies may show anterior spina bifida.

Much more frequently, the connecting band becomes attenuated and breaks, the portion attached to the gut forming a diverticulum passing craniod in the mesentery. If it is long it will usually be called a duplication. The mucosa of the diverticulum or duplication may be that of any gastrointestinal segment. Gastric mucosa is often present at the cranial end, and eventually causes ulceration.

Ulceration, inflammation, and obstruction of the normal channel by distention of the diverticulum are the usual indications of the anomalous structure.

Excision of these diverticula on the mesenteric side of the gut is more difficult than excision of antimesenteric diverticula. The blood supply to the normal channel passes over the diverticulum so that removal of the diverticulum or duplicated segment requires removal of an equal length of normal intestine. In a few cases a common wall between the diverticulum and the normal gut can be extirpated.

Other Diverticula

The developmental mechanisms described above do not account for all intestinal diverticula. Some of these may be of congenital origin, or may occur at points of congenital weakness of the intestinal wall. The exact etiology of many such diverticula is still obscure.
and often controversial. Only the three groups described above can be certainly attributed and well understood developmental defects.
Surgical Embryology of the Exstrophy-Epispiadas Complex

Samuel S. Ambrose, David P. O'Brien

Among the developmental anomalies seen in children, few has as broad a spectrum as the exstrophy-epispadias complex which encompasses numerous abnormalities arising in the hind-end of the embryo. Table 1, the scheme proposed by Marshal and Muecke, depicts the developmental relationships of the variations within the complex. One can readily appreciate the multifaceted nature of a syndrome which can produce deformities as mild as spade penis or patulous rectum on the one hand, while involving severe, complex derangements of the urinary, genital, alimentary, and musculoskeletal systems on the other hand.

Table 1. Developmental Relationships of the Variations Within the Exstrophy-Epispiadas Complex

Classical Exstrophy

Ectopia viscera abdominalis
    with mesonephric duct
Cloacal exstrophy
    with imperforate anus and fistula
    with rectal prolapse
    with inadequate anus
Male
    Epispadias
        with incontinence,
        penopubic
        with continence,
        penile
        with continence,
        balanic
    Spade penis only
Female
    Epispadias with
        incontinence
    Subsymphyseal
    epispadias
    Patulous urethra
Male and female
    Superior fissure
    Duplicate exstrophy
    Musculoskeletal deformity only.

Abnormalities found within the exstrophy-epispadias complex are exotic. The most common condition is classical exstrophy, or ectopia vesicae, in which there is separation of the lower abdominal wall and absence of the anterior vesical wall with protrusion of the posterior bladder wall. According to Campbell, exstrophy occurs once in every 40 to 50,000
live births, but in an autopsy series of over 19,000 children, he found the incidence to be 1 in 1002, suggesting the relationship of the anomaly to early death. As one progresses outward from classical extrophy in Table 1, the anomalies occur with decreasing frequency. The rare condition of cloacal extrophy, for instance, occurs in approximately 1 in 200,000 live births. Table 2 (also from Marshall and Meucke) shows the relative frequency of variations within the extrophy-epispadias scheme in a series of 72 cases. The purpose of this review is to discuss the embryogenesis of the aforementioned anomalies, a subject which has been a source of conjecture and controversy, and to present the clinical manifestations of several of the anomalies within the complex.

Table 2. Variations of Exstrophy-Epispidias in a Series of 72 Patients

<table>
<thead>
<tr>
<th>Variety</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spade penis only</td>
<td>1</td>
</tr>
<tr>
<td>Epispadias with continence</td>
<td></td>
</tr>
<tr>
<td>Balanic only</td>
<td>2</td>
</tr>
<tr>
<td>Penile</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>8</td>
</tr>
<tr>
<td>Epispadias with incontinence</td>
<td></td>
</tr>
<tr>
<td>Subsymphyseal</td>
<td>1</td>
</tr>
<tr>
<td>Penopubic</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Classical exstrophy</td>
<td>40</td>
</tr>
<tr>
<td>Cloacal exstrophy</td>
<td>2</td>
</tr>
<tr>
<td>Superior vesical fissure</td>
<td>1</td>
</tr>
<tr>
<td>Duplicate exstrophy</td>
<td>1</td>
</tr>
<tr>
<td>Characteristic musculoskeletal deformity</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>72.</td>
</tr>
</tbody>
</table>

Embryology

Exstrophy of the bladder has not been found in its embryologic state, due perhaps, as suggested by Patten, to the low incidence of the anomaly. It is also known that the human embryo does not pass through a stage of development which corresponds to exstrophy or epispadias, suggesting that the theory of developmental arrest is not the mechanism responsible for these anomalies. Numerous other theories have been proposed. The "Berstungs Theorie" states that anterior rupture of the embryonic bladder is caused by abnormal retention of fluid. This does not, however, take into account the commonly associated anomalies of the genitalia, musculoskeletal, and intestinal tracts.

To understand the remaining theories which are based on embryologic etiologies for the anomalies, a brief review of the normal pattern of development in the cloacal region, as described by Patten, is necessary. Between the 2nd and 3rd week after fertilization, just caudal to the primitive streak, the apposition of ectoderm and endoderm takes place in the midline without the ingrowth of mesoderm. This area, the early cloacal membrane or cloacal plate, can be easily recognized. By the 4th week the caudal area has elongated and recurred so that
the cloacal membrane occupies a position ventrally, caudal to the body stalk, forming the ventral wall of the urogenital sinus. The paired primordia of the genital tubercle are also present at this time, located just lateral to the upper border of the cloacal membrane. These primordia rapidly enlarge and by the end of the 5th week are fused in the midline superior to the cloacal membrane to form the genital tubercle. This is normally accompanied by a growth of mesoderm toward the midline, thereby lengthening the area between the body stalk and the cloacal membrane. At the same time, the urorectal folds have grown medially and caudally toward the primitive perineum, dividing the urogenital sinus from the rectum.

Patten and Barry theorized that caudal displacement of the paired primordia of the genital tubercle to the point where the urorectal fold divides the primitive cloaca into urogenital and anal components is responsible for the derangement of epispadias. With fusion of the paired genital primordia in the midline, the urogenital portion of the cloaca would be in a cephalic position in relation to the genital tubercle and the anal portion would be just caudal to the genital tubercle, creating the situation for epispadias when the urogenital portion of the cloacal membrane undergoes its normal rupture. This also creates the dorsal grooving of the corpora cavernosa. If the primordia of the genital tubercle were located even further caudally, the ventral abdominal wall in the area would be unreinforced by mesoderm, that is, the infradibulical area of the anterior abdominal wall would be covered only by the urogenital portion of the cloacal membrane and with the normal rupture of this membrane the characteristic exostrophic appearance of the bladder would occur, extending from the base of the penis to the umbilicus. This would also account for the lack of fusion of the pubic arch in its normal location. The more caudally the paired primordia of the genital tubercle arise, the larger the defect created with rupture of the cloacal membranes. If the genital tubercle arose at the same level or caudal to the area of the anal portion of the cloacal membrane, rupture of this portion would leave the bowel exostrophic also. One can appreciate that in Patten and Barry's scheme, varying the caudal displacement of the paired primordia of the genital tubercle can account for many variations of the exostrophic situation.

Marshall and Muecke, while collecting numerous cases of exstrophy and epispadias, found several deformities previously not included as part of this anomalous complex but appearing to fit into the sequence as milder forms of epispadias and/or exstrophy. They theorized that if the genesis of abnormalities were one of division or nonunion starting at the end of the urinary tract and progressing upward, then lesser degrees of epispadias would be more common. They found, however, the reverse to be the case. That is, abnormalities which appear to be located at the vesical outlet, with vesical exstrophy and epispadias, account for some 90 per cent of the cases. They also noted that the defect in the exstrophy-epispadias complex was always midline and longitudinal in character and theorized that if the cloacal membrane itself was overdeveloped or persistent beyond the normal stage of regression, a urogenital opening larger than normal might be expected. An abnormally large and/or persisting cloacal membrane would act as a wedge to the developing structures of the lower abdominal wall. This would leave the involves structures normal but separated. They also affirm that the mesodermal derivatives of all structures in the area of an exstrophy are intrinsically well developed and the bones abnormal only to the degree that might be expected from a wedge effect.

Marshall and Muecke rebut the Pattent and Barry theory by suggesting that if the paired primordia of the genital tubercle are displaced caudally, one might expect, with more
severe degrees of exstrophy, to find the corpora located on the perineum or dissociated from their bony attachments (which rarely occurs). Following the wedge effect of the large or persistent membrane, an excessive or perhaps premature split of the cloacal membrane extending toward the body stalk would lay open the underlying vesical primordia, the internal surface of the cloacal membrane being the ventral wall of the cloaca, with this wall becoming the anterior portion of the bladder. With varying degrees of split of the cloacal membrane, one can see that this theory not only explains exstrophy but also the division of the corpora giving rise to the epispadiac groove usually found in males or the bifid clitoris in females. For epispasias alone, the degree of persistence or overdevelopment of the cloacal membrane would be less, resulting in less separation and more minor deformities. For the more severe abnormalities, such as cloacal exstrophy, one would have to vary only the precise time at which the abnormal cloacal membrane divides as well as the extent of the division in relation to the closure of the urorectal septum. With earlier division and more extensive split, a more marked defect would occur. If split of the abnormally developed cloacal membrane occurred prior to the time of complete formation of the urorectal septum, some stretching of the septum would occur and an abnormally patulous rectum would result. If eversion of structures lying under the cloacal membrane occurred prior to the completion of the urorectal septum, an imperforate anus could result, or at various stages in development of the urorectal septum a rectovaginal or rectovesical fistula could occur. With split of the cloacal membrane prior to the time the urorectal septum has divided the cloaca, the vesical primordia would be left lateral to the exstrophic bowel, an arrangement frequently seen in cloacal exstrophy.

**Experimental Evidence**

Considerable support for the wedge-effect theory of Marshall and Muecke is given by the excellent embryologic experiment performed by Muecke on chick embryos. An inert plastic graft was inserted into the area of the cloacal membrane primordia in chicks at 48 to 52 hours of incubation. With this plastic graft simulating a persistent cloacal membrane and acting as a wedge, chicks with varying degrees of infraumbilical defects were produced. The size of the graft in relation to the of the defect was insignificant, that is, many of the defects produced were much larger than the size of the graft alone. Chicks who underwent the same operative procedure but who did not have insertion of the graft were normal. The exstrophic chicks, as is the case in man, had all structures present but deformed by the infraumbilical defect. This was the first experimental production of exstrophy, an anomaly which is normally not seen in the chick and rarely, according to Muecke, in mammals except for man. This may be due to abnormalities produced in structures surrounding the cloacal membrane. These structures, such as the allantois, perform vital functions in the chick and some mammals but not in man. This would, as Muecke notes, account for the increased incidence of exstrophic defects in man and the paucity of cases in other animals.

**Clinical Manifestations**

The clinical manifestations of entities within the exstrophy-epispadias complex have been alluded to in the previous discussion of embryogenesis. A brief discussion of several of these anomalies along with the presentation of illustrative cases is germane to this review.
Epispadias

Epispadias is the opening of the urethra on the dorsum of the penis or absence of the upper wall of the urethra. In its mildest form, balanitic epispadias, the urethral opening is located dorsally on the glans or at the coronal sulcus with a groove extending distally onto the glans. The intact portion of the urethra proximal to the meatus is located dorsally in the penis, and there may be slight dorsal chordee. This is probably the most common type of incomplete epispadias in the male, according to Campbell.

In penile epispadias the urethra can open anywhere on the dorsal shaft of the penis proximal to the coronal sulcus. The spade-like penis is grooved dorsally and covered with urethral mucosa from the tip of the glans to the urethral meatus. The prepuce usually hangs ventrally. In this particular case, retraction of the distal meatal verge revealed a second opening which, when catheterized, led into the bladder as did the dorsal opening. A voiding cystogram revealed a complete duplication of the urethra, which occurs relatively frequently in epispadias and may be complete or incomplete.

In complete epispadias or first degree exstrophy, the urethra is open to the bladder neck and is usually associated with incontinence.

In the female, epispadias is classified by Campbell as clitoris (in which only a bifid clitoris is present), subsymphyseal, or complete. Incontinence is present in the complete form and associated deformities, such as separation of the labia and pubic bones, occur in varying degrees.

According to the Marshall and Muecke scheme, the embryogenesis of epispadias would involve a shorter persistence or less overdevelopment of the cloacal membrane, resulting in less symphyseal separation and few, if any, infraumbilical defects.

Coincident urinary anomalies are common with epispadias and, in fact, with all abnormalities of the hindgut complex. These include upper urinary anomalies such as agenesis of one kidney, as shown on the excretory urogram. Note also the wide separation of the pubic bones in this particular case. Vescicoureteral reflux has been shown to occur in up to 75 per cent of patients with all degrees of epispadias, making it imperative that all of these patients undergo thorough urinary tract evaluation and reconstruction, if necessary, prior to repair of the epispadias.

Treatment of epispadias depends on the degree of the defect and associated anomalies. Those patients without incontinence usually have satisfactory results, while the amelioration of incontinence may be difficult by present reconstructive means.

Exstrophy

Exstrophy of the bladder, in which the anterior wall of the abdomen and bladder are absent, was the most common defect in Marshall and Muecke's series of 72 patients, accounting for 55 per cent of the deformities. Gross and Cressan, in reviewing a 20-year experience of epispadias and exstrophy, found 18 cases of epispadias and 80 cases of exstrophy. The degree of exstrophy may vary from only a small fissure superiorly to complete
exstrophy, which is the rule. There is epispadias and wide separation of the pubic bones. The figure shows this deformity as it occurs in the male with the posterior bladder wall protruding above the spade-like penis. The pubic bones are splayed and the scrotum may be cleft or bifid and separated. Cryptorchism is common and vesicoureteral reflux occurs in most patients. Concomitant findings in the female are also shown. Note also the patulous and prolapsing rectum due to stretching and weakening of the urorectal septum by early splitting of the cloacal membrane in the embryologic period.

**Cloacal Exstrophy**

Cloacal exstrophy, or vesicointestinal fissure, is one of the most extensive lesions in the exstrophy-epispadias complex. It fortunately occurs rarely, once in approximately 200,000 live births, according to Tank and Lindenauer. The clinical manifestation is characterized by exstrophy of the bladder, which is bivalved on either side of the midline. A ureteric orifice occurs in each bladder half. Between the two hemibladders is an area of intestinal mucosa which is the exstrophic ileocecal area. The distal opening of the ileum lies at the cephalic margin and the proximal opening of a blind hind and/or tail gut at the caudal margin. There may be one or two vermiform appendices opening into the exstrophic cecum. An omphalocele is frequently present above the exstrophic bowel, and atresia of the small intestine is common. The pelvic bones are widely separated and the anus is imperforate. Genital deformities occur in all cases. In the male the penis is usually bifid with each pubic rami. The testes are normally undescended. In females the clitoris is usually divided and the majority have duplex vagina and bicornuate uteri.

Urinary tract anomalies occur in over 50 per cent of these cases while spina bifida and myelomeningocele are commonly associated anomalies.

The embryogenesis of this defect would encompass early extensive splitting of the abnormal cloacal membrane before the urorectal septum has horizontally divided the cloaca. Eventration of the dorsal wall of the intestinal primordia occurs and the vesical primordia are left as paired halves on either side of the intestinal exstrophy.

Survival with this defect was not reported until 1960 and resulted from surgical relief of small bowel obstruction, correction of omphalocele, and recognition and treatment of excessive fluid loss from the small bowel. Surgical repair encompasses major reconstructions of the intestinal, urinary, and genital tracts. Several successes have been accomplished in recent years.