

**TRAUMATIC INJURIES OF THE ORGANS
OF THE RETROPERITONEAL SPACE: ADULT AND PEDIATRIC
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I. Introduction

In this chapter we will review the traumatic injuries to the organs of the retroperitoneal space, which will include the pancreas, kidneys, ureters, adrenal glands, abdominal aorta, and the inferior vena cava. Although, the abdominal portion of the esophagus is technically retroperitoneal, as is true of the distal portion of the first part of the duodenum, its second and third parts, and the fourth part with exception of the most distal part, and the ascending and descending colon, they were discussed in the previous chapter, “Traumatic Injuries of the Organs of the Abdominal Cavity: Adult and Pediatric,” for the sake of continuity. The first part of this chapter will be devoted to a discussion of the relevant anatomy of each of the organs. The purpose of this discussion is two fold. First, it will constitute a review of the anatomy for the medical students, pathology residents and forensic pathology fellows, aiding them in understanding the pathophysiologic dynamics involved in traumatic injuries to these organs. Second, since this educational blog may also be used by medicolegal investigators, law enforcement personnel, and attorneys, who have little knowledge of anatomy, such a review will help them to have a better understanding of the relationships between trauma applied to the victim and their injuries. There will also be a discussion, in a general sense, of the mechanism of injury to the retroperitoneal organs when they are subjected to trauma.

II. Relevant Anatomy of the Retroperitoneal Space

A. Overview: The relevant anatomy of the retroperitoneum can be summarized as that portion of the abdomen posterior to the peritoneal cavity extending from the diaphragm to the pelvic inlet. It is separated from the peritoneum anteriorly by the posterior peritoneal fascia and is bounded posteriorly by the transversalis fascia. It contains portions of the esophagus, duodenum, colon and pancreas, as well as the kidneys, ureters, adrenal glands, abdominal aorta, and the inferior vena cava (Figs. 1, 2 & 3).

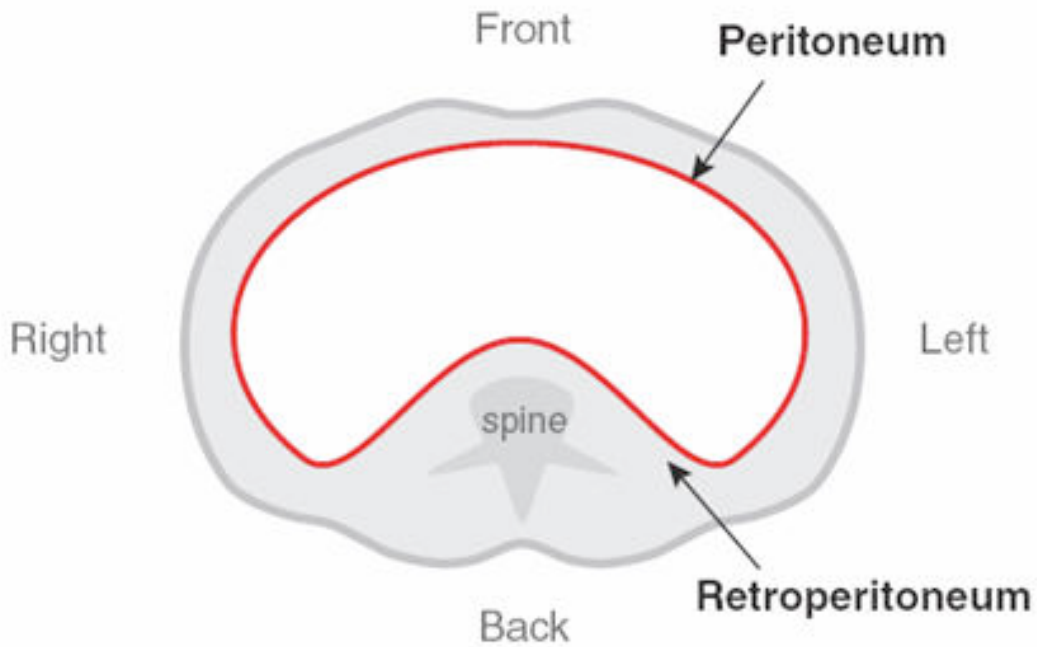


Fig. 1. This is a drawing showing the abdominal cavity lined by the peritoneum and the location of the retroperitoneal space. (Drawing supplied by Christopher Windham M.D.) (Wiki)

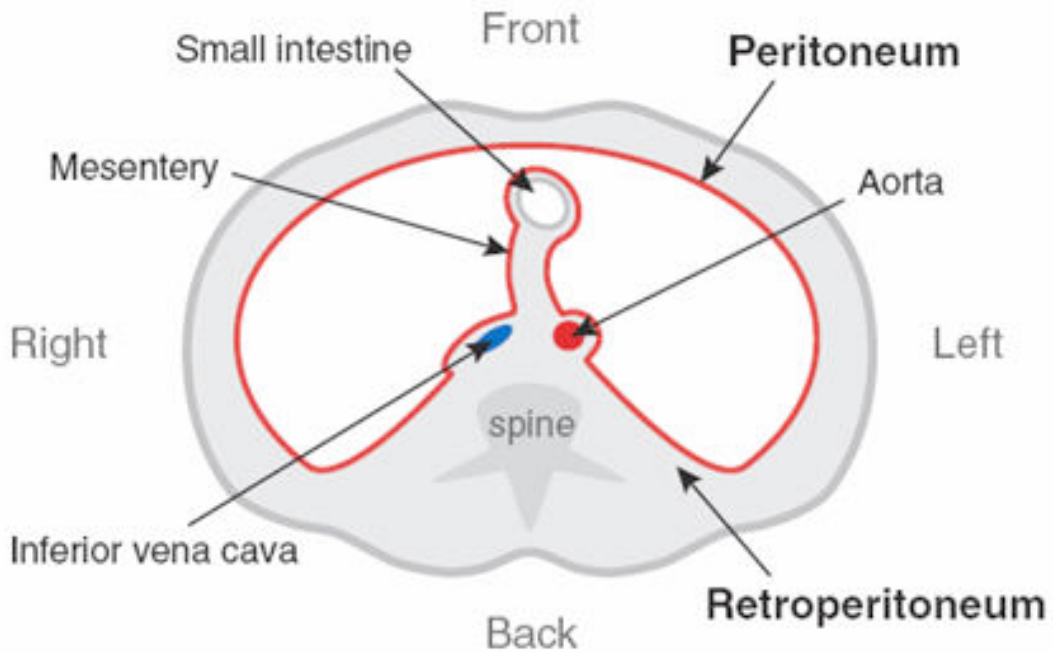


Fig. 2. The above is a drawing showing the location of the inferior vena cava and aorta in the retroperitoneal space. (Drawing supplied by Christopher Windham M.D.) (Wiki)

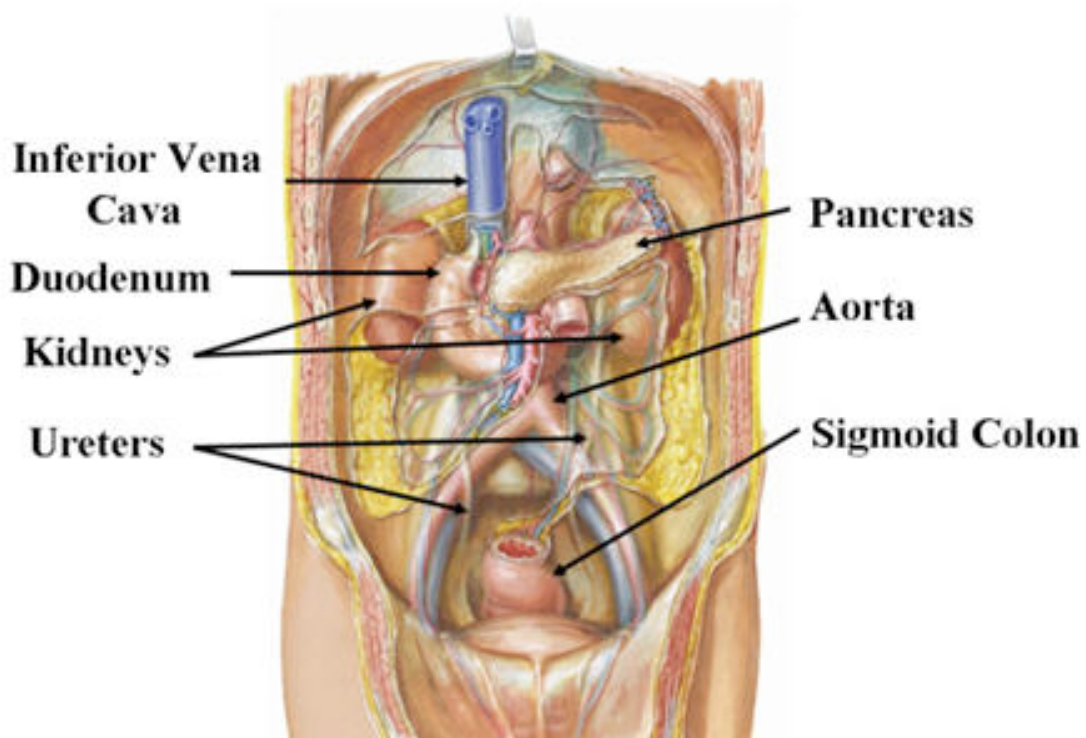


Fig. 3. This is an illustration in which the organs of the abdominal cavity have been removed showing the boundaries and organs of the retroperitoneal space. (Illustration provided by Christopher Windham M.D.) (Wiki)

Traditionally, the retroperitoneum is divided up into several spaces according to their relationships to the fascial layers that surround the kidneys and ureters. In this description the layers of the perirenal fascia enclose a **perirenal space**, which contains the kidneys, upper ureters, adrenal glands, the respective neovascular supply, and the perirenal fat (Fig. 4). The anterior layer of the perirenal fascia is continuous across the midline allowing the right and left perirenal spaces to communicate. This continuation is anterior to the neovascular structures. Anterior to the anterior layer of the perirenal fascia is the **anterior pararenal space** (Fig. 4). This space contains the retroperitoneal segments of the abdominal esophagus; the duodenum, except for the proximal portion of the first part and the most distal portion of the fourth part; the ascending and

descending colon; the pancreas, except for its tail, which is located in the splenorenal ligament; and the root of the mesentery of the small intestine (Fig. 2).

Since only the front and left side of the abdominal esophagus are covered by peritoneum, it is considered retroperitoneal. Embryologically, the abdominal esophagus developed behind the peritoneum thus, it is considered a primary retroperitoneal organ, as is true of the kidneys, ureters, adrenal glands, abdominal aorta, and the inferior vena cava. Those organs, which embryologically developed within the abdominal cavity and thus, suspended by mesentery, but migrated posterior to the peritoneum are considered secondary retroperitoneal organs. This includes the head, neck, and body of the pancreas; the duodenum, except for the proximal portion of its first segment and the most distal aspect of its fourth segment; and the ascending and descending colon. The **anterior pararenal spaces** are also continuous across the midline and are limited posteriorly by the anterior communicating layers of the perirenal fascia and anteriorly by the parietal peritoneum. Behind the posterior layer of the perirenal fascia lies the **posterior pararenal space**, which contains only fat (Fig. 4). These anatomic distinct

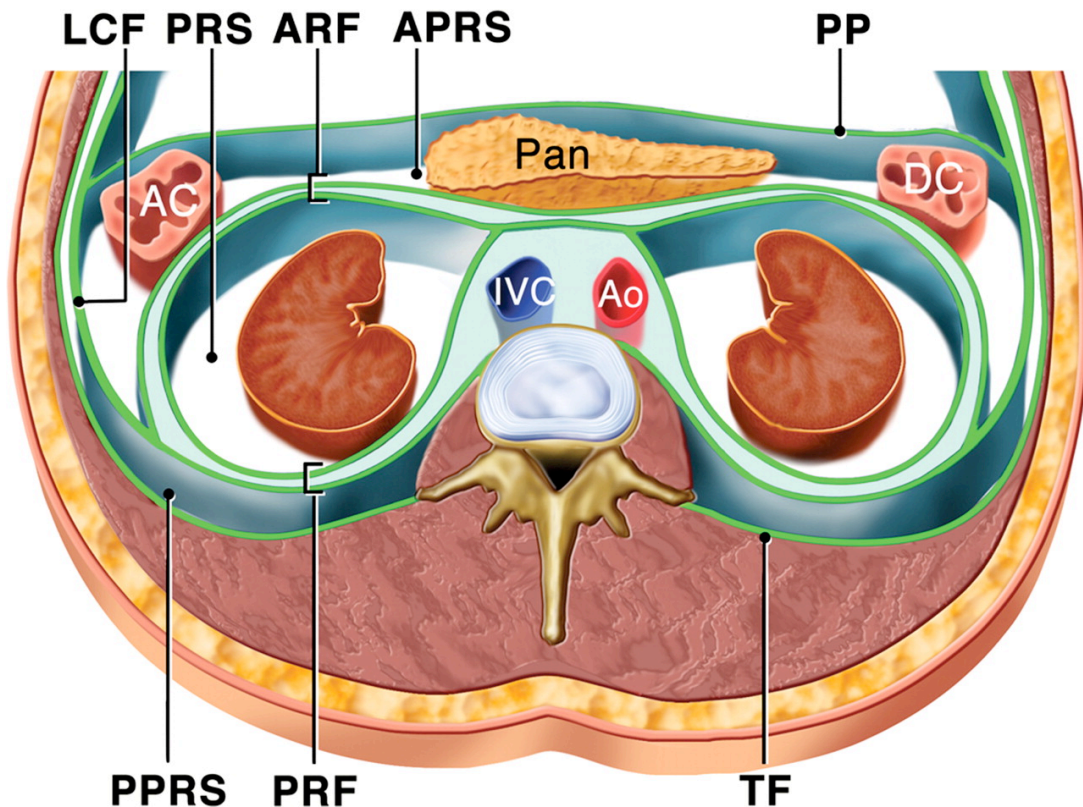


Fig. 4. The above illustration shows the traditional tricompartiment model of the retroperitoneum, which is accordingly divided into the anterior perirenal space (APS), perirenal space (PS), and posterior pararenal space (PPS). The anterior renal fascia (ARF), posterior renal fascia (PRF), and lateroconal fascia (LCF) divide the spaces. (RadioGraphics October 2008 vol. 28 no. 6 1571-1590) (Wiki)

spaces helps to explain why small to moderate amounts of fluid, blood or pus collecting within one or these spaces tends to remain confined to the space in which it is formed. However, recent evidence has demonstrated the fascia separating these spaces is arranged in a laminar fashion and shows considerable variation in the degree of fusion between the lamina thus, there are inter-fascial connections between the spaces. Consequently, retroperitoneal hemorrhage or rapidly expanding fluid collections can spread through these inter-fascial connections (Fig. 5).

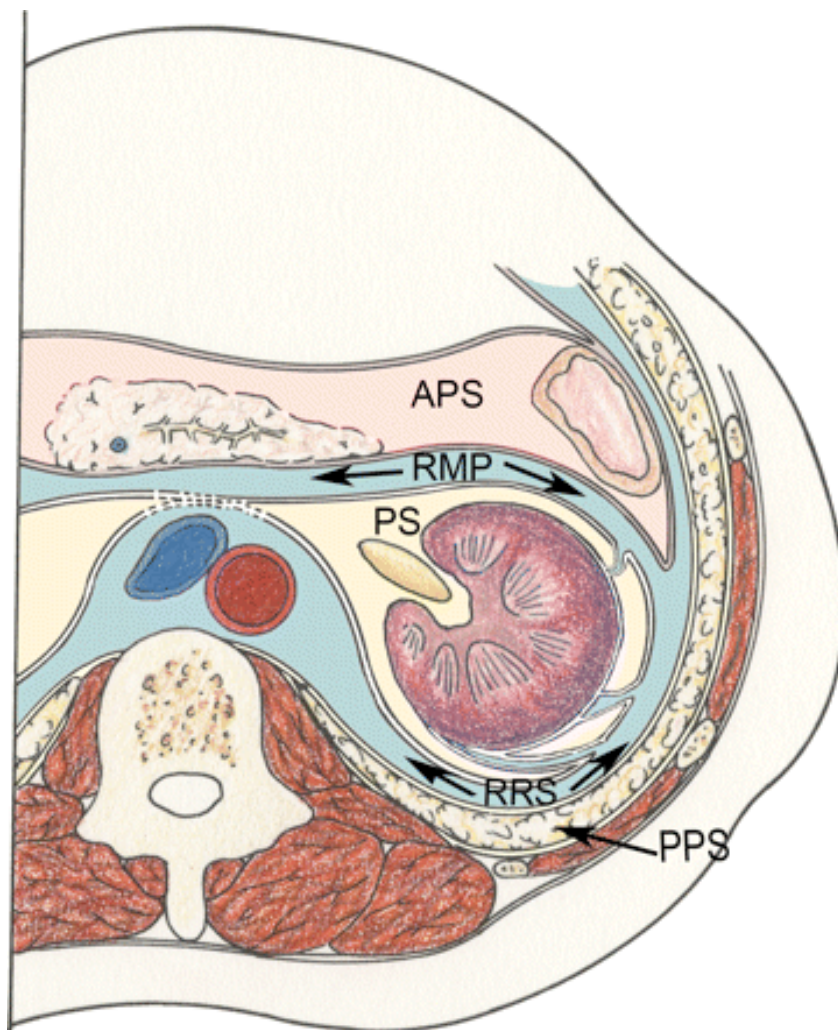


Fig. 5. The above illustration reflects the recently modified tricompartiment model, which reflects the understanding that the perirenal fascial is laminar and variably fused and there are inter-fascial connections between the spaces. The retromesenteric plane (RMP), retrorenal space (RRS), and lateroconal space are potential inter-fascial communications. Perinephric septa run between the renal capsule and the perinephric fascia, allowing subcapsular fluid to communicate with the retrorenal space or retromesenteric plane. APS - anterior pararenal space, PPS - posterior pararenal space, PS - perirenal space. (RadioGraphics October 2008 vol. 28 no. 6 1571-1590) (Wiki)

Below the kidneys, the anterior and posterior pararenal spaces become continuous, forming a single retroperitoneal space. Thus, retroperitoneal fluid or hemorrhage can spread from the abdominal retroperitoneum into the extraperitoneal plevis along the anterior and posterior perirenal fascia, which ultimately combine to form the fascial plane in the iliac fossa. Superiorly, the anterior and posterior perirenal fascia are attached to the diaphragm. This brings us to an important anatomical feature, which needs to be kept in mind. *On the right side, the bare area of the liver is directly connected to the anterior pararenal space. Thus, lacerations involving the bare area of the liver can cause retroperitoneal hemorrhage.*

III. Retroperitoneal Visceral Anatomy

A. Pancreas: The pancreas is a large elongated pink retroperitoneal organ except for its tail occurring at the level of the second and third lumbar vertebrae (Fig. 6).

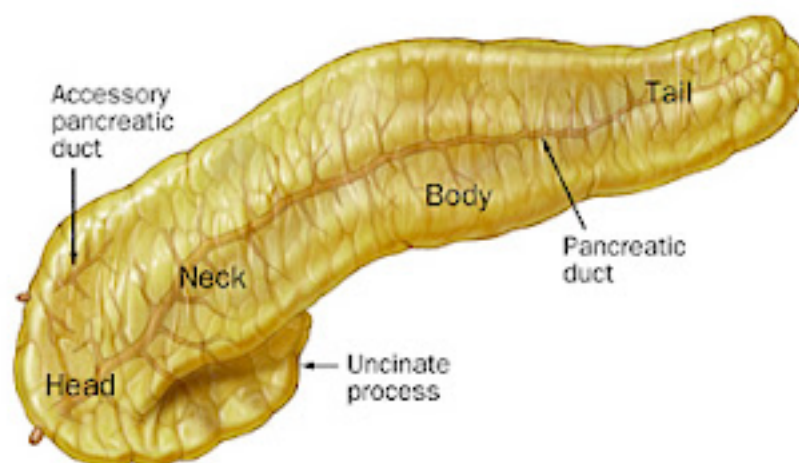


Fig. 6. This is an illustration of the pancreas, which is located transversely across the posterior wall of the abdomen. (Kozzi images) (Wiki)

It lies in the concavity formed by the duodenum, extending behind the peritoneum of the posterior abdominal wall toward the left, reaching the hilum of the spleen (Fig. 7).

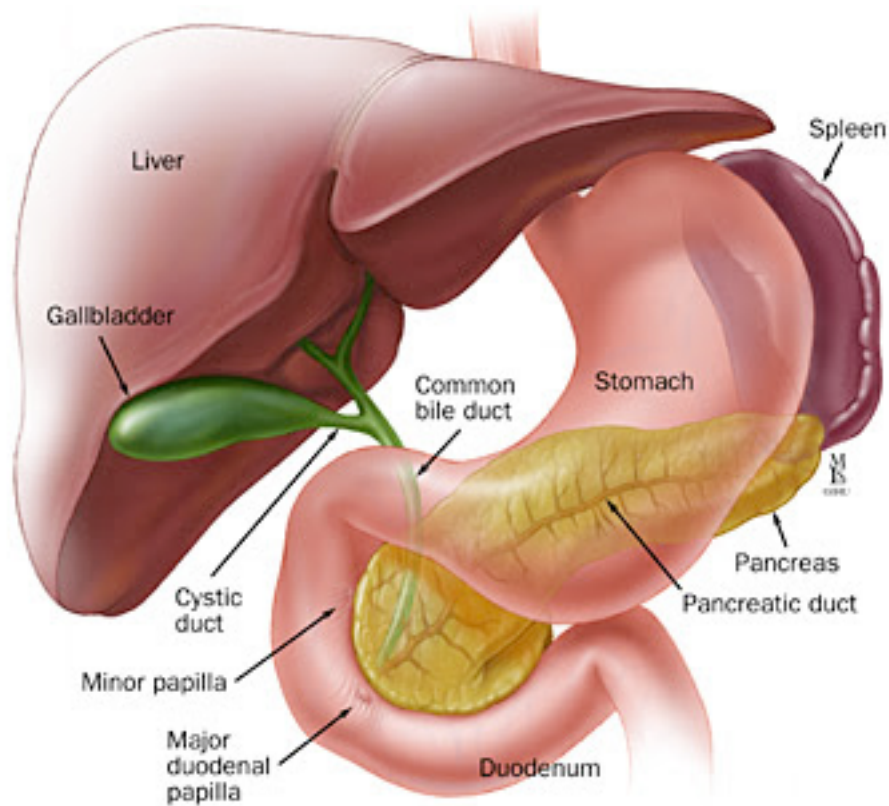


Fig. 7. This is an illustration showing the anatomical relationships of the pancreas. (Kozzi images) (Wiki)

Regarding the surface anatomy, the pancreas lies in the epigastrium and left hypochondriac areas of the abdomen (Fig. 8).

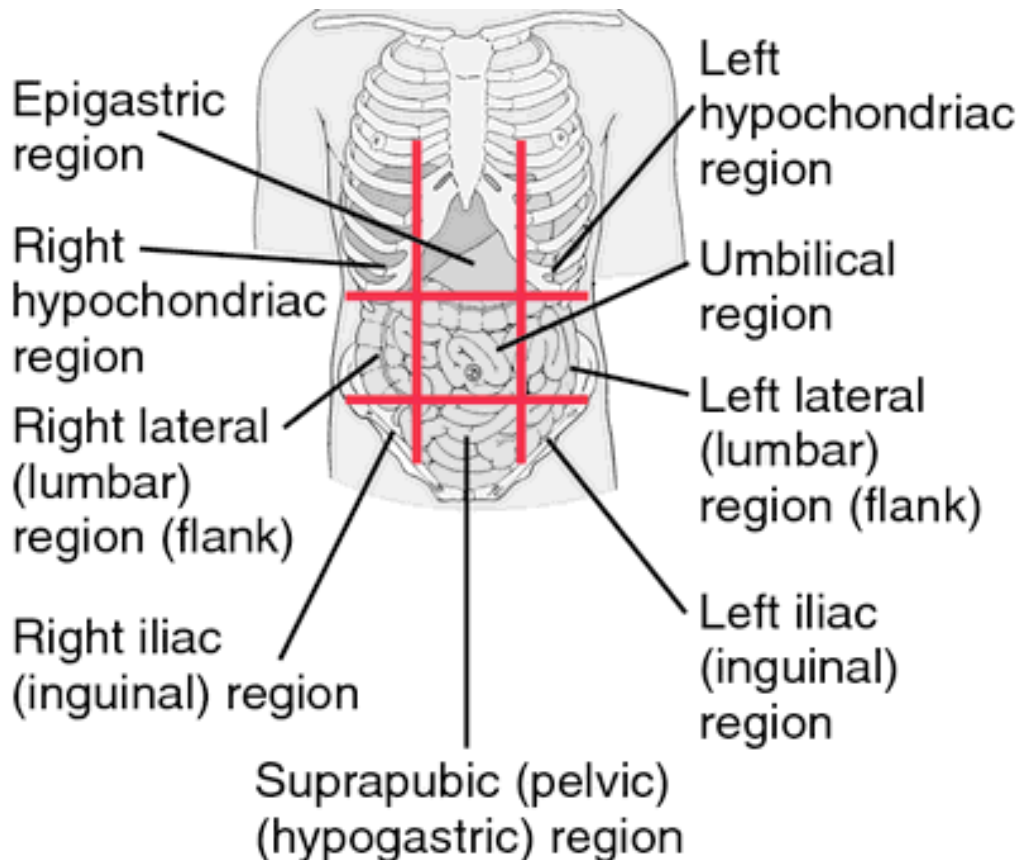


Fig. 8. This is an illustration of the nine quadrants that comprise the surface anatomy of the abdomen. (Wiki)

It is one of two abdominal organs connected to the digestive tract by means of a duct system, the other being the liver. In the adult it measures from 12 to 25 cm in length, varying in weight from 65 to 160 grams with an average weight of 110 grams. It has no definitive fibrous capsule, but is covered by a thin layer of connective tissue from which thin connective tissue septa extend into the gland dividing it into lobules (Figs. 12, 13 & 14). Grossly the pancreas is divided into four anatomic areas: head, neck, body, and tail (Fig. 6).

The head of the pancreas lies within the concavity of the duodenum (Fig. 7), covering the inferior vena cava and the renal veins as they enter it. There is a portion of the head of the pancreas, which is referred to as the uncinata process, that is inserted behind the superior mesenteric vessels (Figs. 9 & 10).

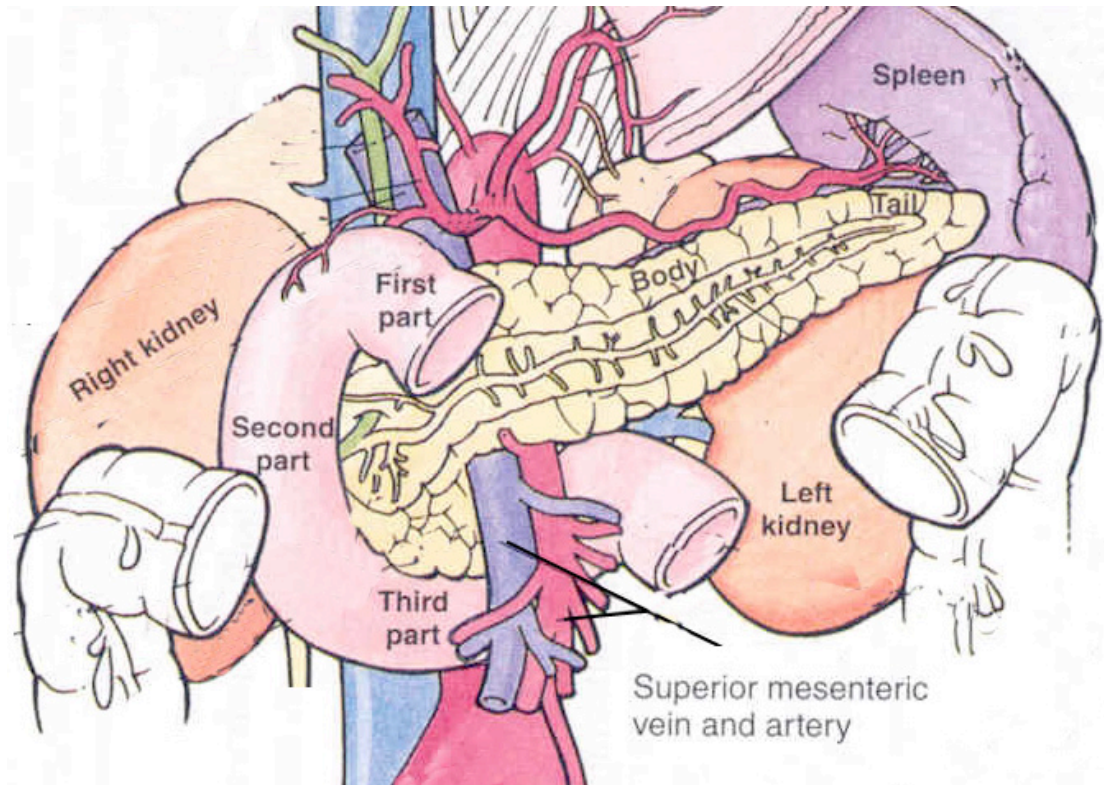


Fig. 9. This illustration shows the superior mesenteric vein (blue) and artery (red) passing between the uncinate process, which is behind these vessels and therefore not seen in this illustration but is seen in Fig. 10, which shows the posterior view, and the head of the pancreas. Remember, when pathological processes involve the head of the pancreas and/or its uncinate process, compression of these vessels can occur. (Wiki)

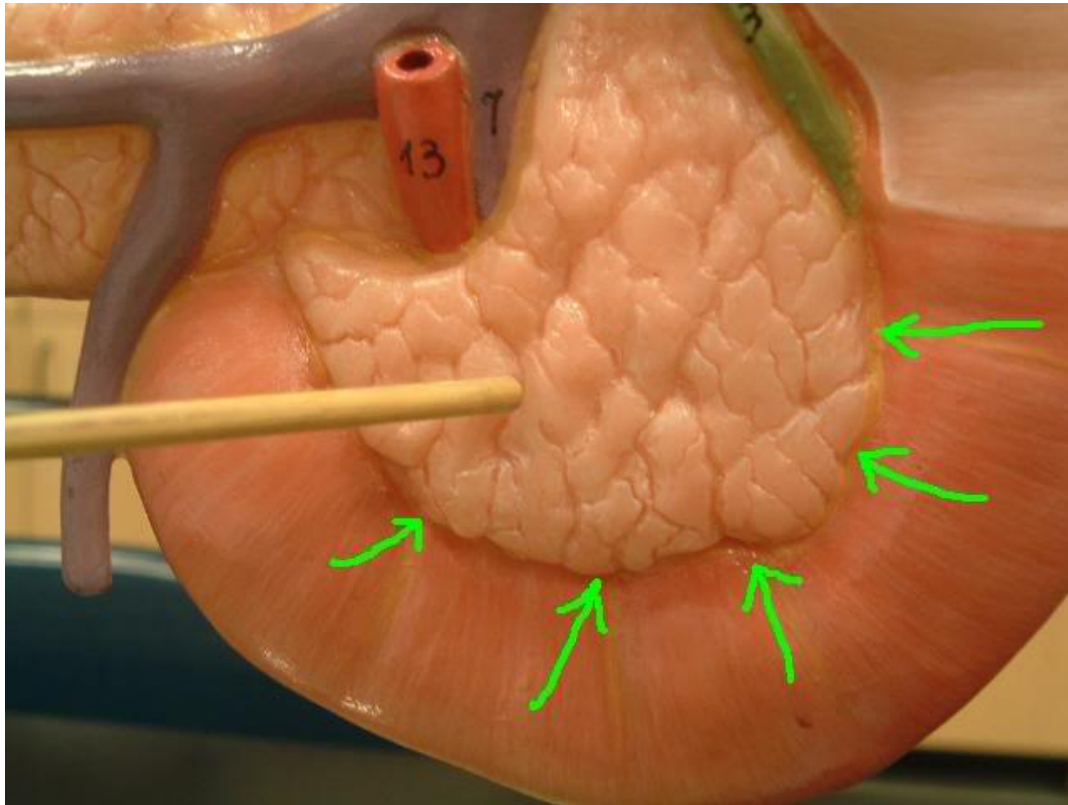


Fig. 10. This illustration shows the posterior view of the uncinete process, which is behind the superior mesenteric artery (13) and vein (7) and is denoted by the pointer. The green arrows show the relationship between the uncinete process and the duodenum. (Wiki)

The neck is a very short constricted segment, which is grooved posteriorly by the superior mesenteric artery and vein (Fig. 9).

The body of the pancreas continues to the left from the neck, passing somewhat upward. It lies in front of the aorta, left renal vessels, left adrenal gland, and the left kidney (Fig. 9).

The tail of the pancreas is usually blunted and turned upward, reaching the hilum of the spleen inferiorly, and is in relation with the left flexure of the colon (Fig. 9).

Within the parenchyma of the pancreas, and traversing its length is a large duct, the pancreatic duct. The pancreatic duct begins in the tail, extending to the head where it unites with the common bile duct at the **ampulla of Vater (major duodenal papilla)**. In the head it is often quite large measuring 2 to 4 mm in diameter. Often there is an accessory pancreatic duct within the head above the main duct, which empties into the

minor duodenal papilla located 2 cm above the ampulla of Vater (Fig. 11). Occasionally, this duct empties directly into the main pancreatic duct.

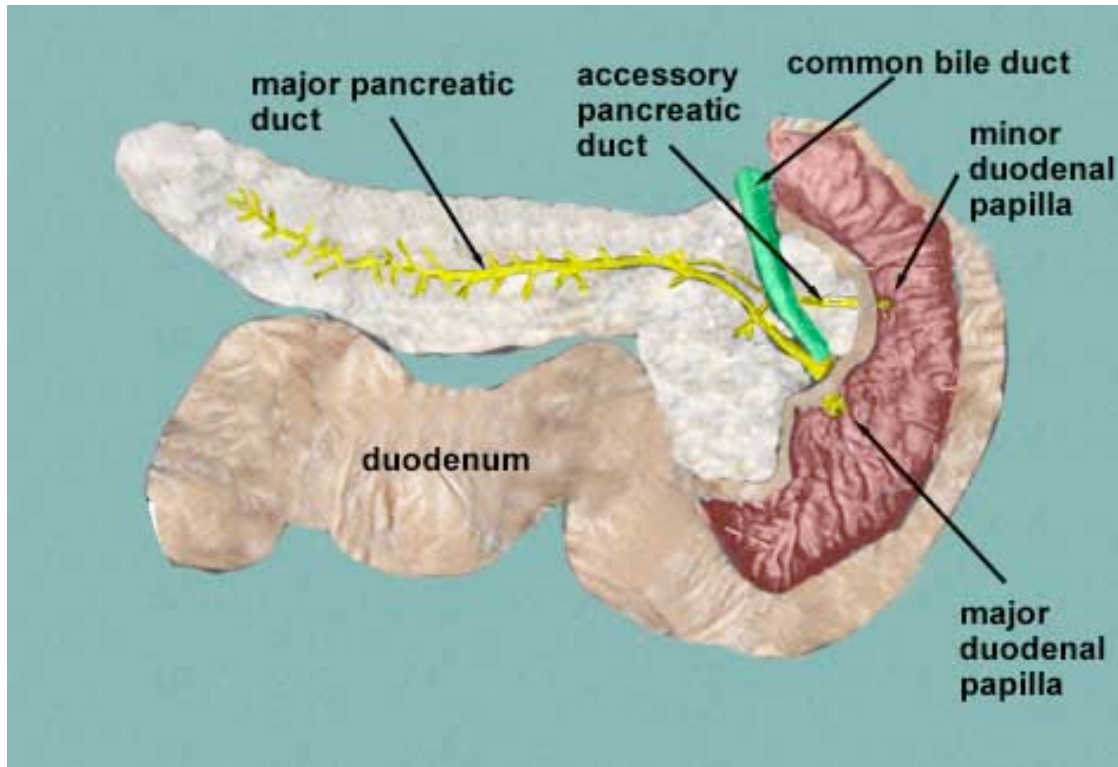


Fig. 11. This is an illustration showing the major pancreatic duct (duct of Wirsung), which provides the pancreatic secretions from the exocrine pancreas to aid in digestion, the accessory pancreatic duct (duct of Santorini), the common bile duct, the major duodenal papilla (ampulla of Vater) and the minor duodenal papilla. The muscular wall of the ampulla of Vater may be thickened, forming the sphincter of Oddi. (Wiki)

The pancreas is an organ, which next to the liver is the second largest gland connected to the duodenum. As a gland it performs two distinct functions. One function is to serve as a digestive organ, which is designated the **exocrine portion** of the pancreas (Figs. 12, 13 & 14). The other function is to play an important part in the control of carbohydrate metabolism, which is referred to as the **endocrine portion** of the pancreas (Figs. 13 & 15). Unlike the liver, in which the exocrine and endocrine functions are carried out by the same cells, the pancreas's exocrine and endocrine functions are carried out by different cell groups.

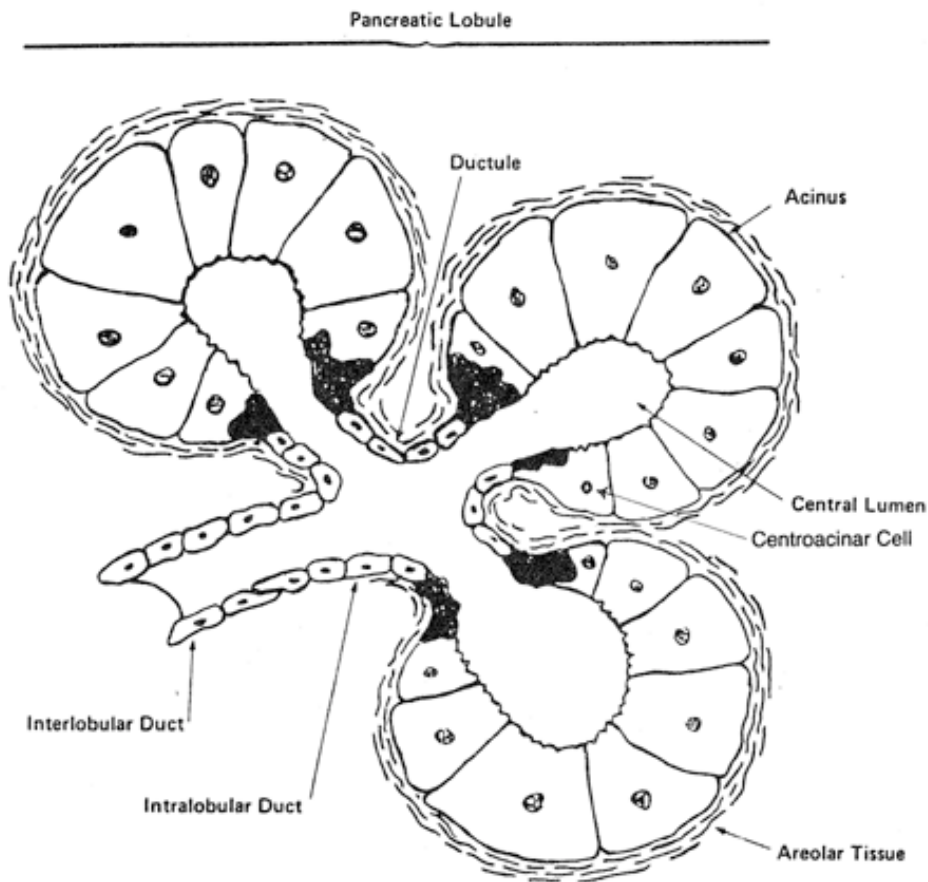


Fig. 12. This is an illustration of a pancreatic lobule composed of clusters of exocrine cells called acini. The exocrine cells are filled with membrane-bound secretory granules. These granules contain digestive enzymes, protease, lipase and amylase, which are released into the lumen as depicted above. From here, the secretions flow into ever-larger intralobular ducts, which eventually collect into the main pancreatic duct, draining directly into the duodenum. Approximately 95% of the secretions within the main pancreatic duct are produced by the exocrine cells of the acini. (Wiki).

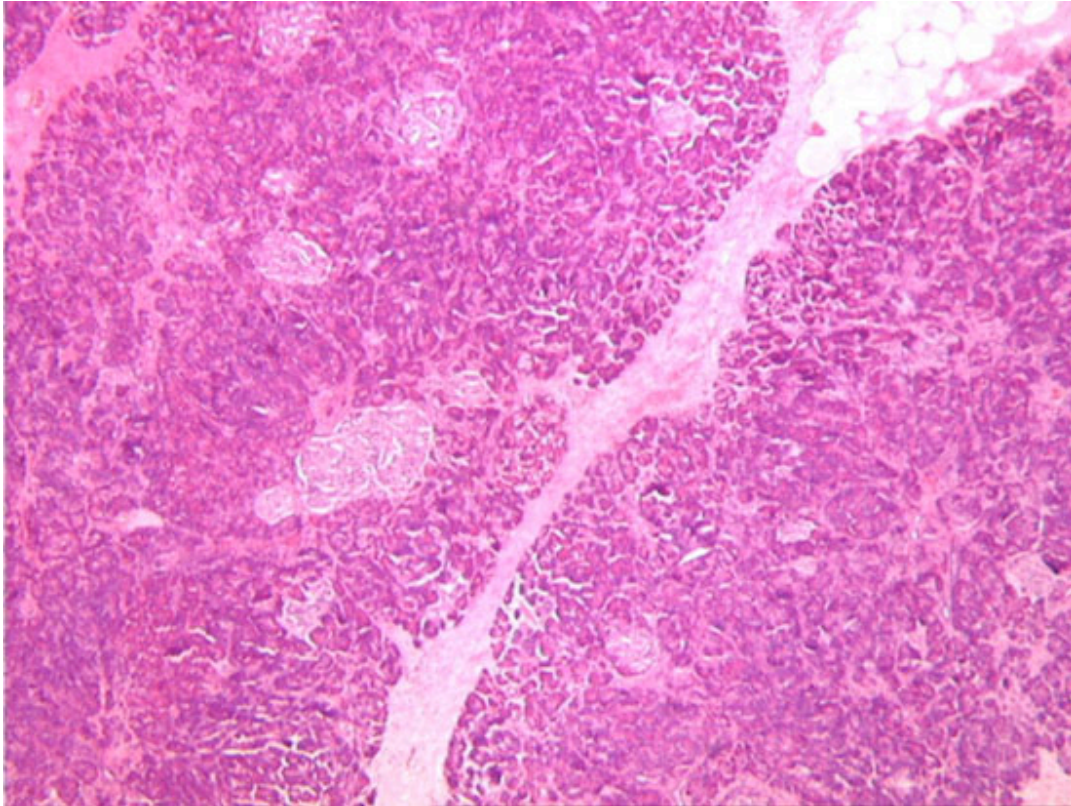


Fig. 13. This is a low-power photomicrograph of an H&E slide of the pancreas. The basophilic exocrine portions of the gland are separated by a connective tissue septum running from top to bottom. Scattered throughout are several pale staining areas called the islets of Langerhans, which are the endocrine portion of the pancreas. These are seen to the left of the connective tissue septa. (Wiki)

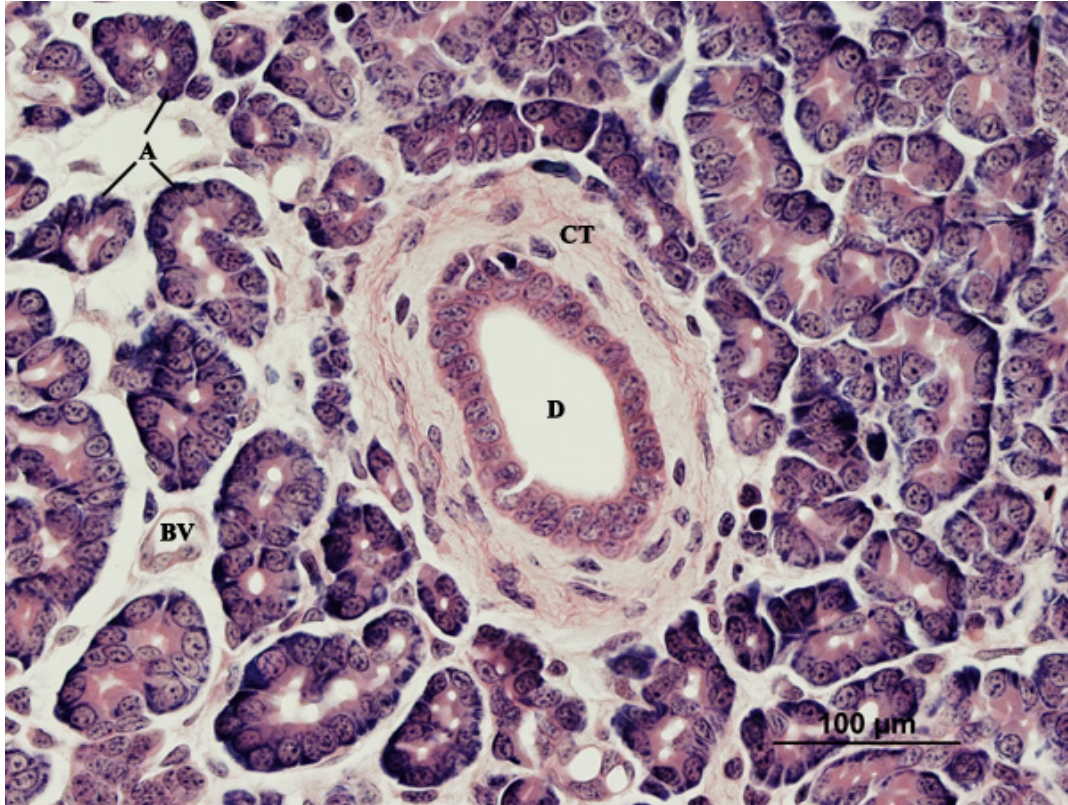


Fig. 14. This is a more high-power photomicrograph of an H&E slide of the exocrine portion of the pancreas. It is composed of acini (A), which secrete digestive enzymes. The lobules of the pancreas are connected by intralobular ducts (D). Abundant connective tissue (CT) is seen surrounding each of the ducts. A small blood vessel (BV) is noted to the left of the duct. (Wiki)

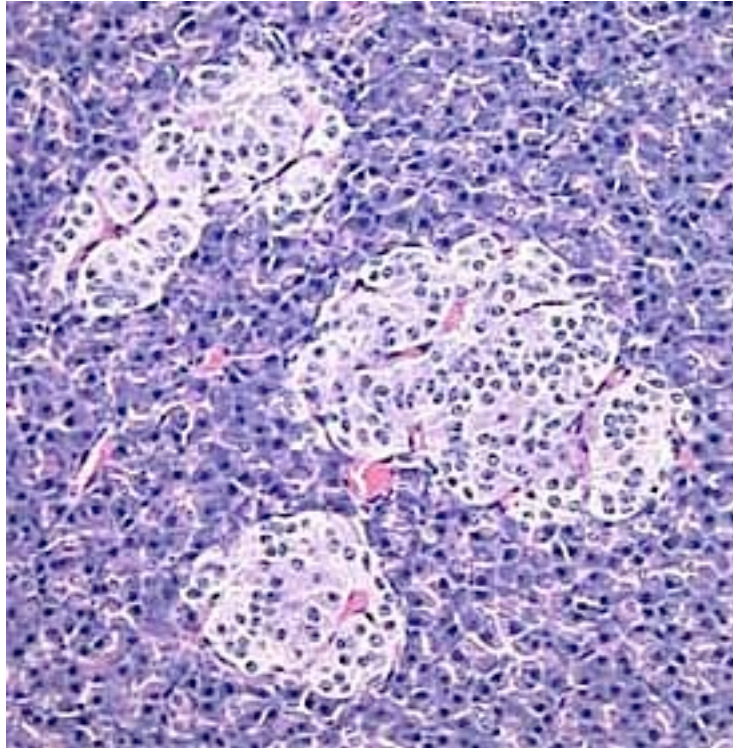


Fig. 15. This is a photomicrograph of a H&E slide of the pancreas showing three pale staining groups of cells called the islets of Langerhans or, more simply, the islets. Human beings have approximately 1 to 2 million islets. The pancreatic islets contain three major cell types, each of which produces a different hormone. The **alpha cells** secrete the hormone glucagon, the **beta cells** secrete insulin and amylin and are the most abundant cells in the islets, and the **delta cells** secrete the hormone somatostatin, which is also produced by other endocrine cells in the body. Along with these hormones the islets also produce other minor hormones.

The islets are richly vascularized thus, giving ready access of their secreted hormones to the circulation. Although, the islets comprise only 1 to 2% of the mass of the pancreas, they receive about 10 to 15% of the pancreatic blood flow. Additionally, they are innervated by the parasympathetic and sympathetic nervous systems, which modulate the secretion of insulin and glucagon. (Wiki)

The exocrine portion secretes pancreatic juice containing digestive enzymes into the duodenum, which contain multiple enzymes for digesting all three major food types: proteins, carbohydrates, and fats. It also contains large quantities of bicarbonate ions, which play a role in neutralizing the acidity of chyme emptied from the stomach into the duodenum. The production of these enzymes is by cell structures call **pancreatic acini**, which are arranged in clusters called lobules (Figs. 12 & 14). Each acinus varies in configuration from rounded structures to short tubules. They consist of a single row

of pyramidal epithelial cells converging toward a central lumen (Figs. 12 & 14). The secreted product of these acini empty into ductules, which ultimately empty into the main pancreatic or accessory pancreatic duct. The exocrine portion secretes approximately 1200 ml of digestive juice per day.

The bicarbonate ions and water are secreted mainly by the epithelial cells of the ductules and ducts that lead from the acini.

The endocrine portion is represented by structures called the **islets of Langerhans**, which are scattered throughout the pancreas as irregular, spheroidal masses of pale staining cells with a rich vascular supply (Figs. 13 & 15). The pancreas has 1 to 2 million islets of Langerhans, each only 0.3 mm in diameter, and organized around small capillaries into which the cells of the islets secrete their hormones. As previously stated, the islets contain three major types of cells, alpha, beta, and delta cells, with the beta cells comprising the greatest number.

The **beta cells** constitute 60% of all the cells of the islets and secrete **insulin** and **amylin**. The **alpha cells** are approximately 25% of the total and secrete **glucagon**. The **delta cells** are about 10% of the total and secrete **somatostatin**. In addition, there is at least one other cell type, the **PP cell**, which is present in small numbers. This cell secretes a hormone of uncertain function called **pancreatic polypeptide**.

The arterial blood supply to the pancreas arises from the **celiac axis** and the **superior mesenteric artery**. The principal arteries from the celiac axis to the duodenum and the pancreas are branches of the **gastrooduodenal artery**, the **anterior superior pancreaticoduodenal** and the **posterior superior pancreaticoduodenal arteries** (Fig. 16). The anterior superior pancreaticoduodenal supplies the head of the pancreas. The posterior superior pancreaticoduodenal supplies the head of the pancreas and the first and second parts of the duodenum. The principal arteries from the superior mesenteric are the **anterior inferior pancreaticoduodenal** and the **posterior inferior pancreaticoduodenal arteries**. Both arteries supply the pancreatic head, uncinata process and the second and third parts of the duodenum (Fig. 16).

The **splenic artery** gives off **pancreatic branches** as it courses along the superior border of the pancreas, supplying the neck, body, and tail (Fig. 16). The pancreas also receives unnamed branches from the superior mesenteric artery, retroperitoneal vessels

and the inferior and superior pancreaticoduodenal arteries.

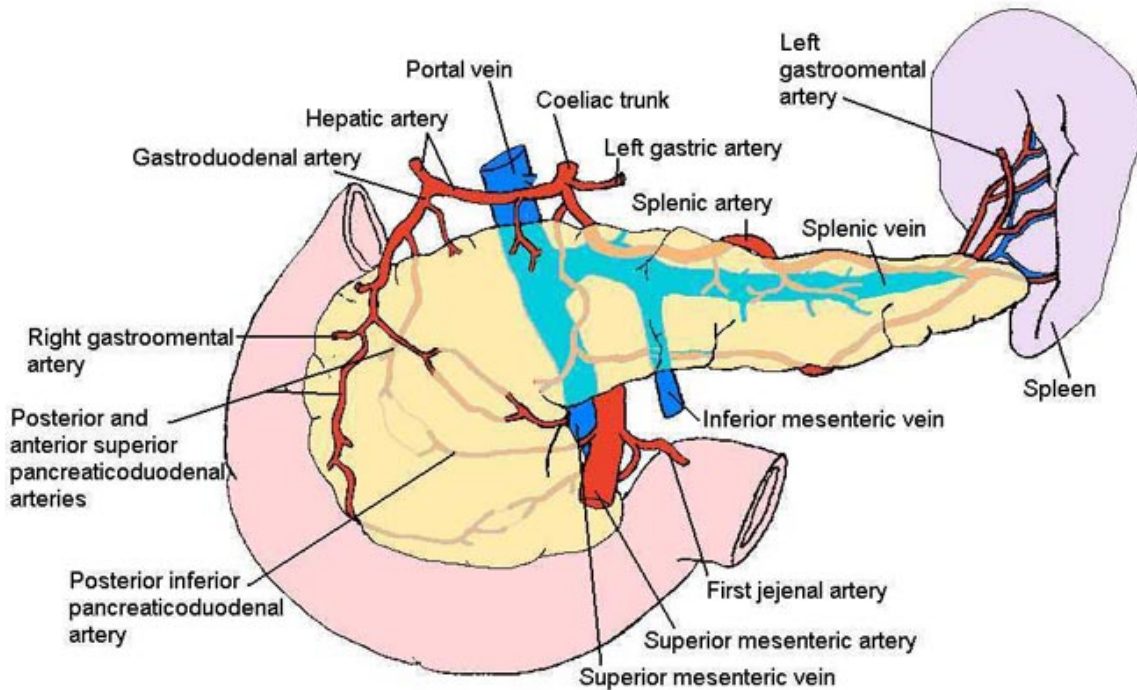


Fig. 16. This is an illustration of the arterial supply and venous vessels of the pancreas. (endocrinesurgeon.co.uk) (Wiki)

The veins of the pancreas and duodenum correspond with the arteries and are usually superficial to them. The **pancreatic veins** ultimately drain into the **portal vein**.

The pancreas contains a network of **lymphatic vessels**. Most of the lymphatic vessels of the pancreas lie within the interlobular septa of connective tissue that separates the pancreas into lobes and lobules. Peripheral extensions of these interlobular lymphatics can be found within the lobules, however, they are few. For the most part, the pancreatic lymphatic vessels follow the blood vessels. There are a few scattered areas in which the intra- and interlobular lymphatic vessels come in close contact with the acini. However, rarely are these lymphatics associated with the islets of Langerhans. The intra- and interlobular lymphatics are similar in structure. Both are thin walled

having an endothelial lining and a delicate component of connective tissue. Both the exocrine and endocrine secretions of the pancreas can enter the thoracic duct via the pancreatic lymph vessels. However, under normal circumstances this avenue is of little importance. The structural relationships between the lymphatic vessels and the pancreatic parenchymal cells is such that the lymphatic vessels do not serve as a significant pathway for the pancreas's secretory products. It appears the lymphatic vessels in the pancreas are primarily for the drainage for substances that, for whatever reason, enter the interstitium. Also, the low flow of lymph through the lymphatic vessels supports the fact that pancreatic lymph is not a functionally important pathway for endocrine secretions to reach the circulation. It appears, the lymphatic system of the pancreas, like that in other organs, is essential in the removal of excess fluid from the interstitium.

The lymphatic drainage of the head of the pancreas follow the blood vessels and drain first into the pancreaticoduodenal nodes, ultimately draining into the pyloric nodes of the gastroduodenal artery above or the superior mesenteric nodes below. The lymphatic drainage of the neck, body and tail follow the blood vessels, ending in the pancreaticolinal nodes along the splenic vessels and in the superior mesenteric nodes. The **nerve innervation** of the pancreas is divided into the exocrine and endocrine portions. The **exocrine lobules** of the pancreas are innervated by **sympathetic** and **parasympathetic fibers**. The **sympathetic fibers** arises from the sixth to tenth thoracic spinal segments with their **preganglionic fibers** forming synapsis in the celiac ganglia (Fig. 17). The resulting **postganglionic fibers** enter the pancreas by following the arteries as a periarterial plexus. The **parasympathetic supply** is from the posterior vagus nerve and the **parasympathetic** component of the celiac plexus (Fig. 18). The supply to the gland is both vasomotor, i.e., arterial (sympathetic) and to the cells forming the acini (sympathetic and parasympathetic).

The innervation of the **endocrine portion** of the pancreas is almost exclusively from the **parasympathetic nerves**. Not only does the parasympathetic system form fine plexuses around the islets, they also form synapses with the neural plexuses innervating the cells of the acini, suggesting a linkage between the neural control of the exocrine and endocrine portions of the pancreas.

Sensory fibers leave the pancreas through both the sympathetic and parasympathetic nerves. The sensory fibers convey the sensation of pain arising from the pancreas. The majority of pain arising from the pancreas is referred to the epigastrium. If the pancreas is involved with an inflammatory process or cancer, which spreads to the retroperitoneum, that pain is conveyed through somatic nerves to the posterior paravertebral region around the lower thoracic spine.

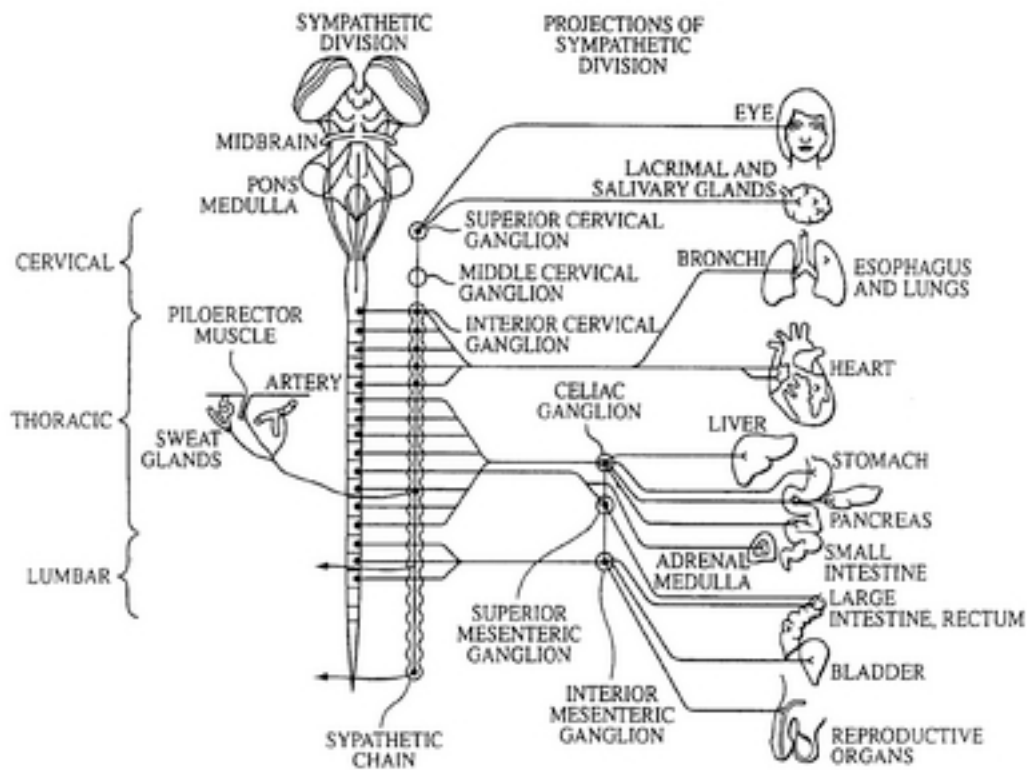


Fig. 16. This is an illustration of the sympathetic nervous systems innervations. (Wiki)

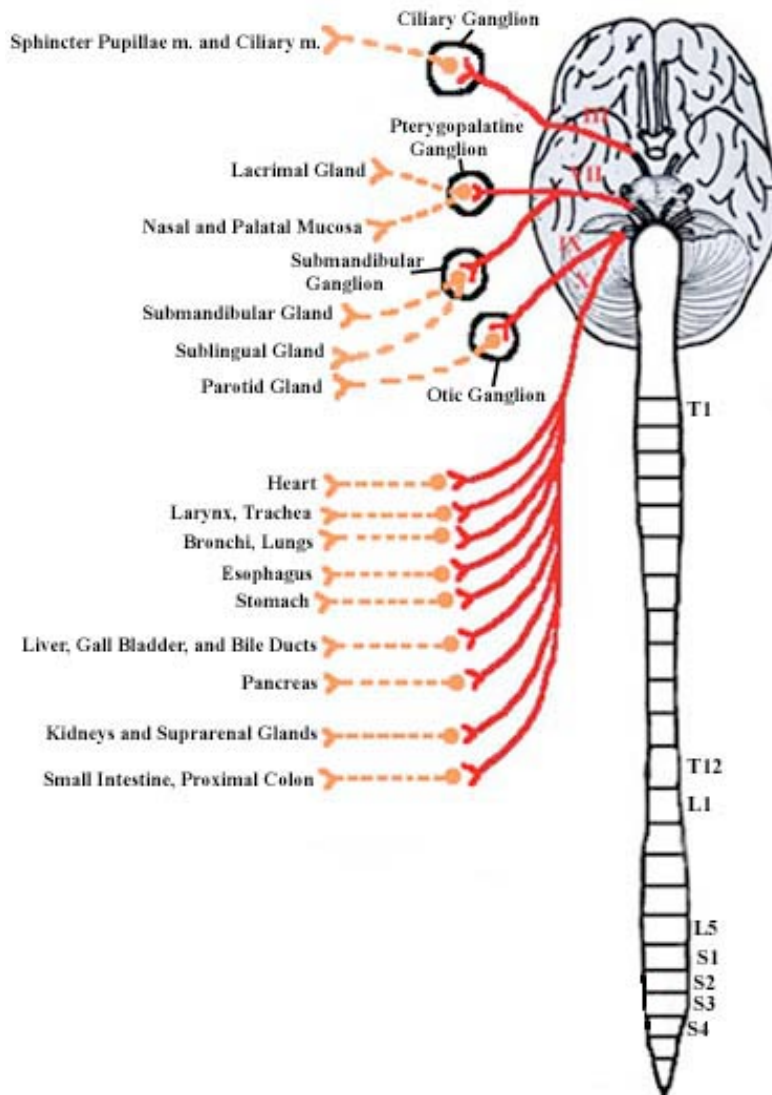


Fig. 17. This is an illustration of the innervations of the parasympathetic nervous system. To review, the cranial segment of the parasympathetic nervous system consist of presynaptic cell bodies in the brainstem and parasympathetic fibers exiting the brainstem in cranial nerves III (oculomotor), VII (facial), IX (glossopharyngeal), and X (vagus). Cranial nerves III, VII, and IX provide parasympathetic innervation to the lacrimal, nasal, palatine and pharyngeal glands; the ciliary muscle of the lens and the sphincter pupillae muscle of the iris; and the parotid, sublingual, and submandibular glands. The parasympathetic nervous system promotes secretion from these glands, causes the pupil of the eye to constrict, and the lens to thicken to accommodate for near vision. The presynaptic parasympathetic fibers from these three cranial nerves synapse in four parasympathetic ganglia. Parasympathetic fibers from cranial nerve III synapse in the ciliary ganglion. Parasympathetic fibers from cranial nerve VII synapse in the

pterygopalatine and submandibular ganglia, and the parasympathetic fibers from cranial nerve IX synapse in the otic ganglion.

Cranial nerve X, the vagus nerve, provides parasympathetic innervation to all the thoracic viscera via cardiac branches, and to the GI tract from the esophagus to the left colic flexure of the colon, including the pancreas, spleen, liver and gallbladder. The parasympathetic innervation to the digestive tract stimulates peristalsis and the secretion of digestive juices. Parasympathetic innervation to the gallbladder causes it to empty. Presynaptic parasympathetic fibers from the vagus nerve synapse in individual cell bodies located on or in the wall of the effector organ, and reach organs in the abdomen via combined autonomic perivascular plexuses.

B. Kidney: The kidneys lie embedded in fat and fibrous connective tissue in a deep retroperitoneal paravertebral gutter formed by the anteriorly projecting lumbar vertebrae and the psoas muscles. This gutter is bounded medially by the psoas muscles. Behind the kidney lies the quadratus lumborum muscle and, at its lateral border, the transversus abdominis muscle (Figs. 18 &19).

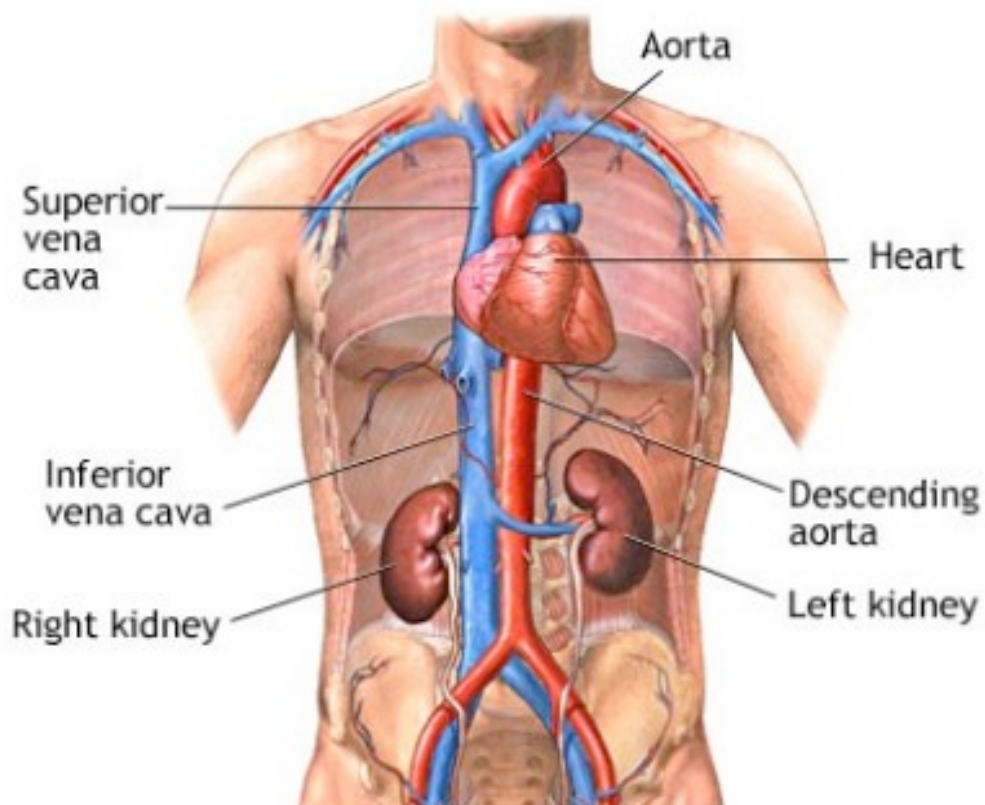


Fig. 18. This is an illustration showing the general location of the kidneys in the retroperitoneal space. (Wiki)



Fig. 19. This is a CT scan showing the location of the kidneys in the paravertebral gutter formed by the anteriorly projecting lumbar vertebrae with the psoas muscles. (Wiki)

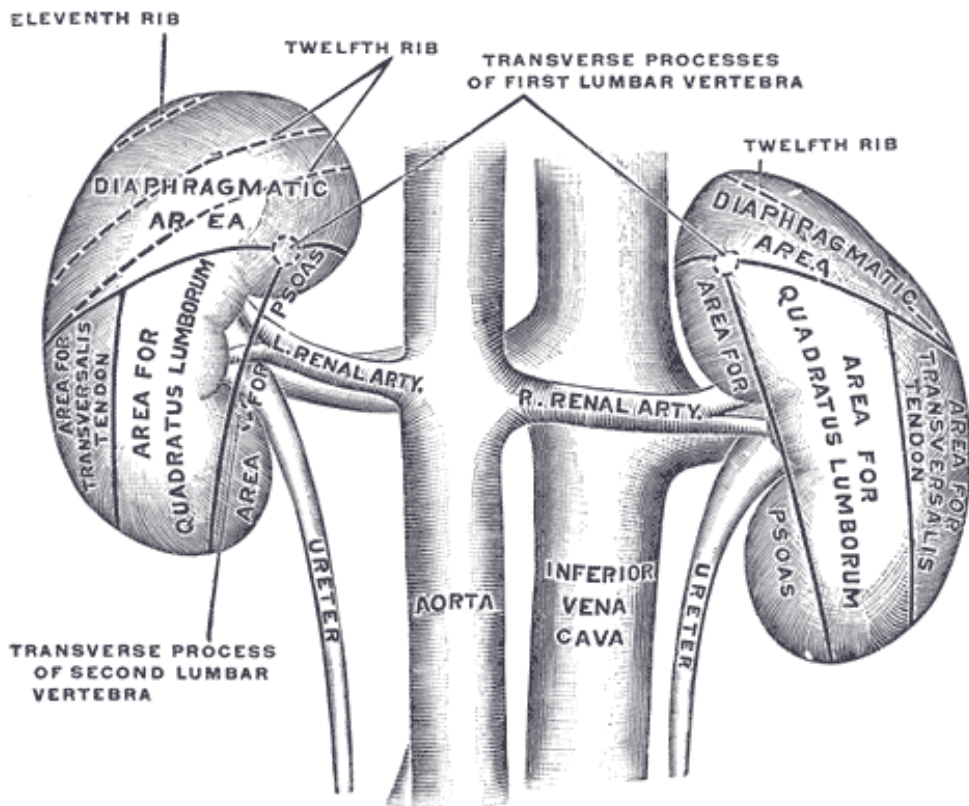


Fig. 20. The above is an illustration showing the relationship of the left kidney to the eleventh and twelfth ribs and the right kidney with the twelfth rib. It also shows the relationships between each kidney and the diaphragms, transversalis abdominis muscles, quadratus lumborum muscles, the psoas muscles and the transverse processes of the lumbar vertebrae. (Wiki)

The superior aspect of each kidney lies on the twelfth rib next to the upper border of the twelfth thoracic vertebra; their inferior aspect lie at the level of the third lumbar vertebra and are further from the median plane than are the superior aspect (poles) (Figs. 20 & 23)

Anterior to the right kidney is the liver above and the colon below. The second part of the duodenum passes in front of its medial border (Figs 21 & 23).

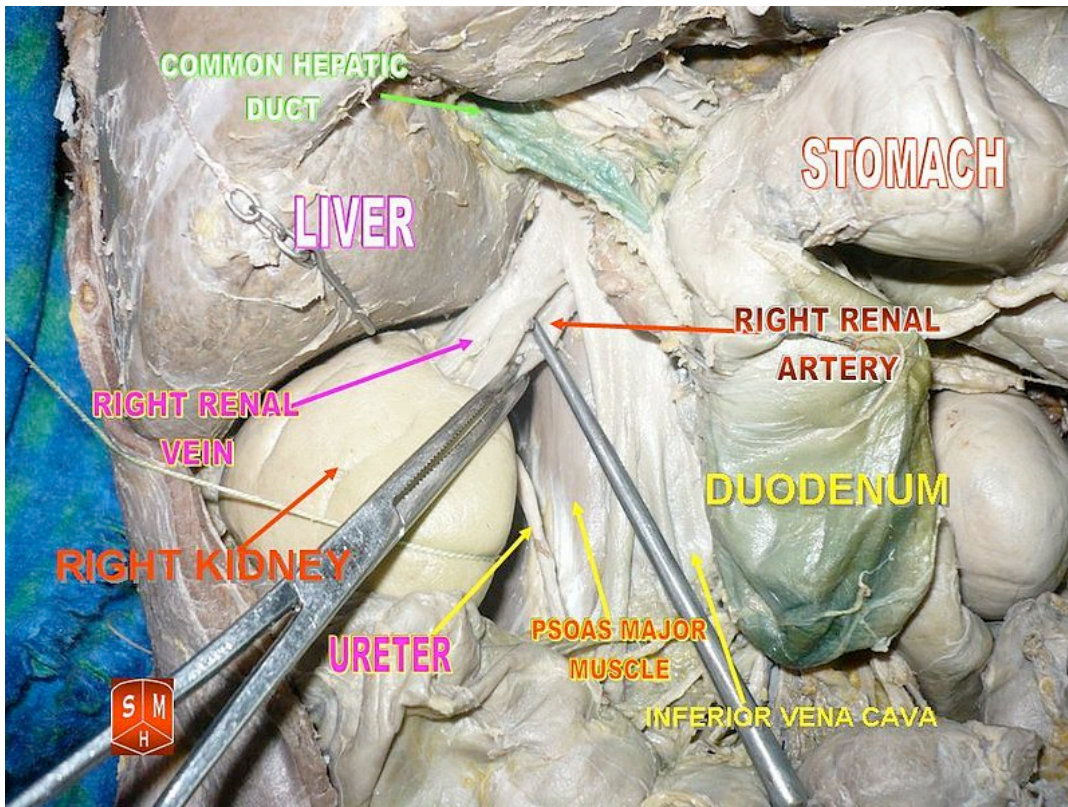


Fig. 21. The above depicts the relationships of the right kidney as seen in a cadaver. (Wiki)

The middle of the left kidney is behind the body of the pancreas, above are the spleen and stomach and below is the beginning of the jejunum (Figs. 22 & 23).

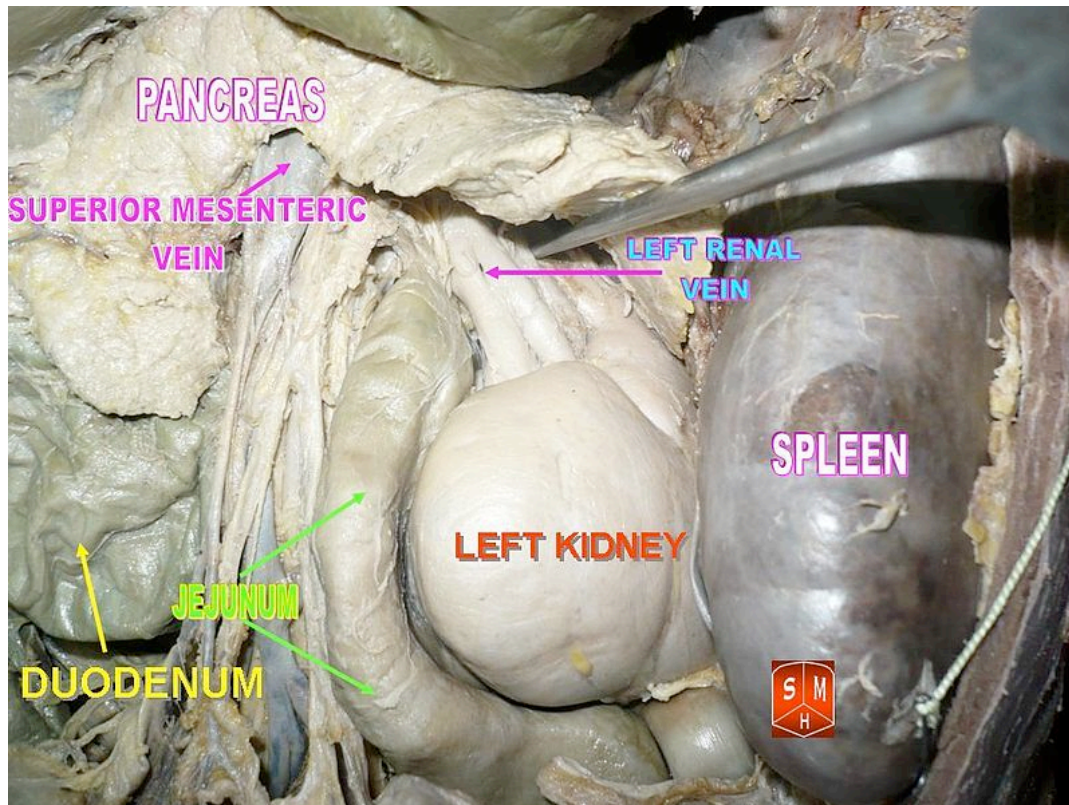


Fig. 22. The above depicts the relationships of the left kidney as seen in a cadaver. (Wiki)

The perirenal fat is enclosed between two layers of perirenal fascia, which unite into the transversalis fascia laterally, immediately beneath the peritoneum.

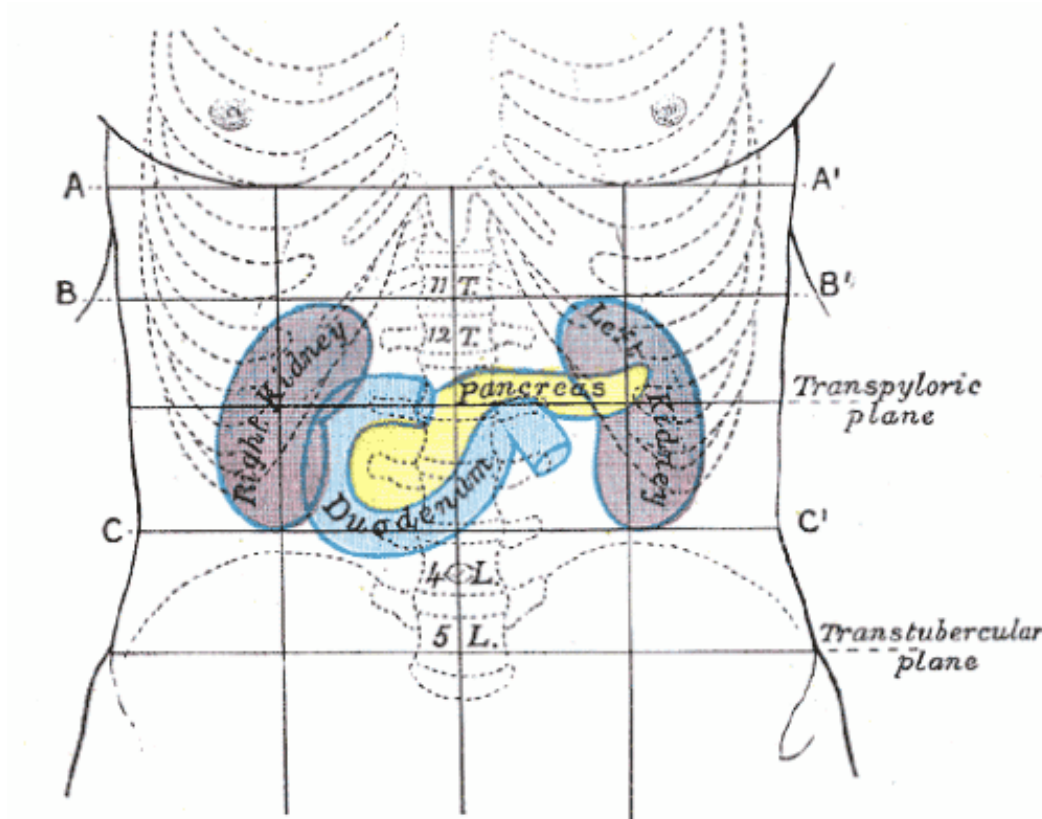


Fig. 23. This is an illustration showing the relationships of the kidneys with the ribs, thoracic and lumbar vertebrae duodenum and pancreas. (Wiki)

The left kidney is usually somewhat longer than the right kidney, and the right kidney is usually slightly lower than the left kidney due to the mass of the liver above it (Figs. 18 & 20). The kidneys are from 10 to 12 cm long, from 5 to 6 cm wide, and about 3 cm thick. The left kidney may be 1.5 cm longer than the right. In the adult male each organ varies in weight from 120 to 170 grams, average 150 grams, while in women the range is 110 to 150 grams, average 135 grams.

The center of the medial border of each kidney has a concavity, that contains the hilum, which is a deep vertical fissure for the entrance of the renal blood vessels, lymphatics, nerve supply, as well as accommodating the renal pelvis, which continues as the ureter (Figs. 24 & 25). The ureter is a large excretory duct, which emerges from the hilum and

courses downward to the urinary bladder, which is situated in the pelvis behind the pubis.

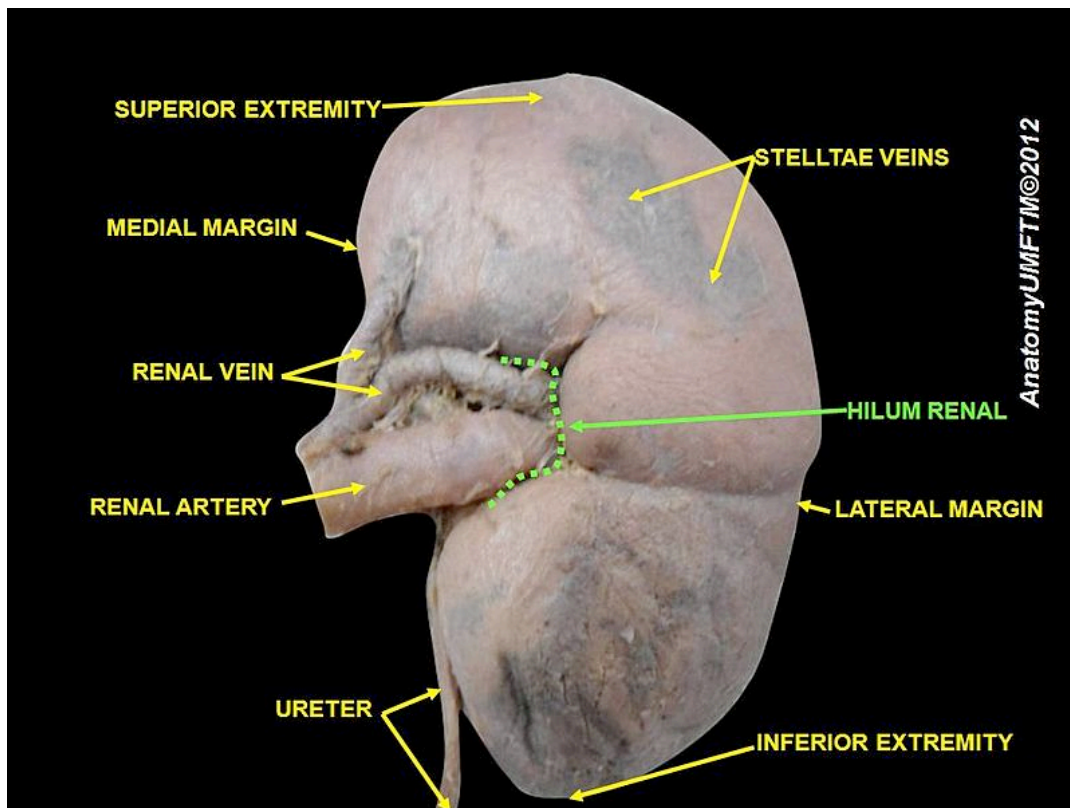


Fig. 24. The above is a cadaver kidney depicting the anatomic margins and the hilum. (Wiki)

Cut Section of Kidney

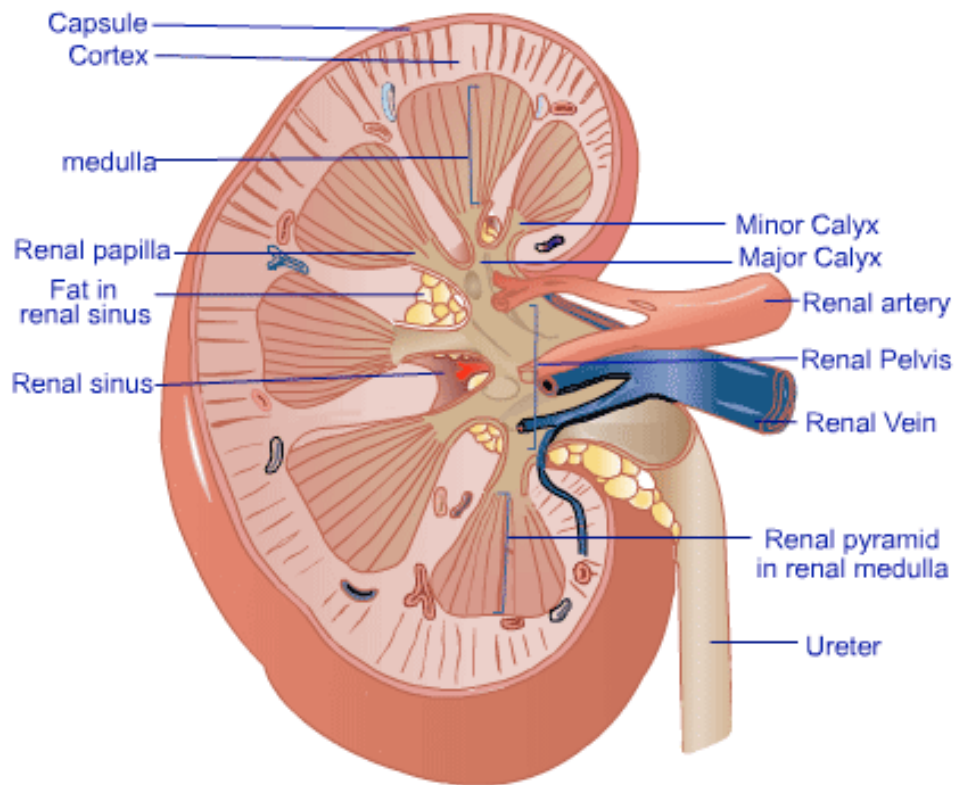


Fig. 25. This is an illustration of a kidney bisected to show general anatomic features. (Wiki)

The anatomical relationships here allow one to allocate the separated kidneys to their correct side. The problem is to know, which is the front surface and which is the back or posterior. This can be done by looking at the hilum, where you will note the renal vein is anterior (in front of) the renal artery, with the artery being posterior (in back of).

When the kidney is bisected it will be seen the hilus immediately expands into a structure called the **renal sinus**. The renal sinus contains fat in which are located two anatomical structures, the **renal pelvis** and **calyces**, and the renal vessels. The renal pelvis is a funnel-shaped expansion of the upper end of the ureter, which sends into the substance of the kidney two or three sizable outpocketings called **major calyces**.

These in turn have a number of smaller outpocketings called **minor calyces** (Fig. 25).

Further examination of the cut surface of the bisected kidney (Fig. 25) will show immediately beneath the **capsule** covering the external surface of the kidney a reddish-brown outer zone, the **cortex** and a darker inner zone, the **medulla**. The cortex has a slight granular appearance due to its high content of structures called **glomeruli**. The medulla has a striated appearance due to the abundant straight tubules and collecting ducts it contains. There are finger-like extensions of the cortex (**renal columns**), which extend to the renal sinus, between sections of the medulla, which are called **medullary pyramids**. Each kidney contains a total of 8 to 18 medullary pyramids. At the apex of each medullary pyramid they taper in to 8 to 18 renal papillae, which project into a minor calyx.

Extending through each renal column is a principal branch of a renal artery, the **interlobular artery**; its name being a reflection of the lobar character of a single medullary pyramid and the cortical tissue on all sides of it. The cortical tissue forming the lateral boundaries of the medullary pyramids is referred to as the **renal columns of Bertin** (Fig. 26).



Fig. 26. The metal probe points to the renal columns of Bertin. The renal columns are an extension of the renal cortex into the medullary region between the renal pyramids. It allows the cortex to be better anchored. Each column consists of lines of blood vessels and urinary tubes and fibrous material. (Wiki)

A medullary pyramid together with the cortical tissue overlying its base (the base of the medullary pyramid is directed toward the overlying cortex, i.e., that portion of the cortex directly beneath the capsule) and covering its sides constitutes a renal lobe.

The functional unit of the kidney is the **nephron**, of which there are two types: 85% are cortical nephrons, which are located entirely within the cortex; 15% are juxtamedullary nephrons, located near the cortex-medulla junction (Fig. 27). The primary function of the nephron is to control the excretion of water, electrolytes, and metabolic waste products. However, it also serves many important homeostatic functions, including: excretion of metabolic waste products and foreign chemicals; regulation of water and electrolyte concentrations; regulation of arterial pressure; regulation of acid-base balance; secretion, metabolism, and excretion of hormones; and gluconeogenesis. Its functions are vital to life and are regulated by the endocrine system through hormones such as antidiuretic hormone, aldosterone, and parathyroid hormone (Fig.27).

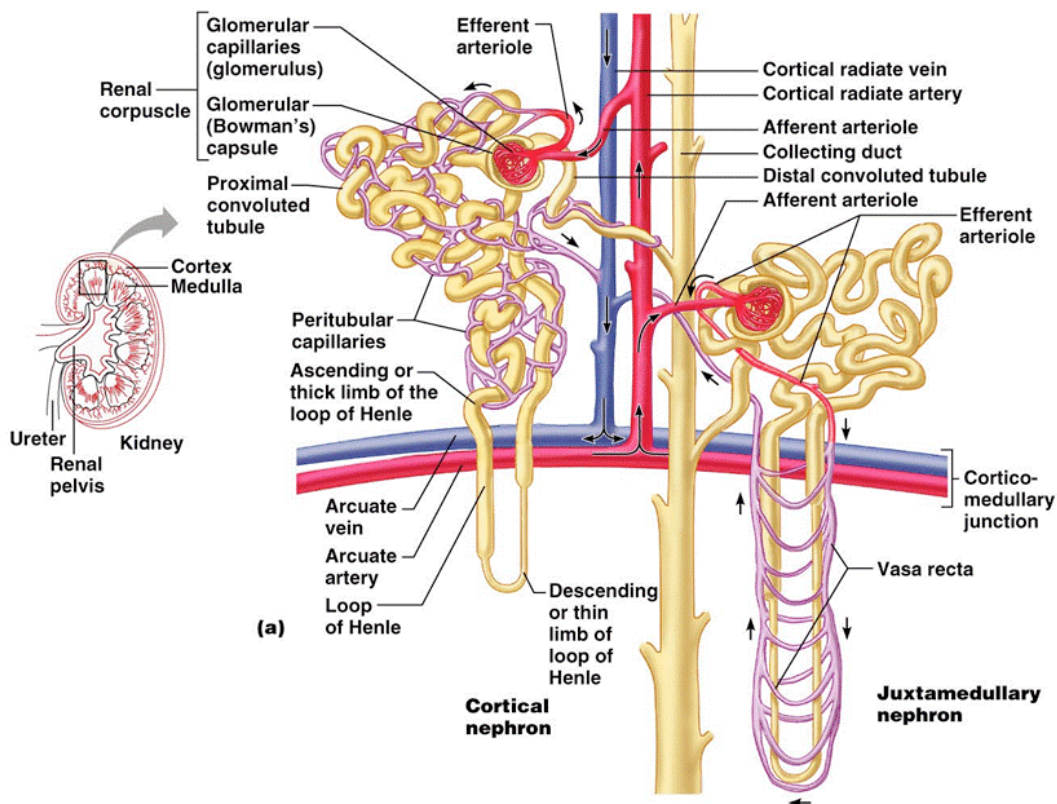


Fig. 27. The above is an illustration of a nephron, which constitutes a functional unit of the kidney. Two elements make up the nephron: the renal tubules and the renal corpuscles. The renal corpuscle contains the glomerulus, the coiled network of capillaries that bring blood into the nephron, and Bowman's capsule, the podlike structure that encases the glomerulus. Blood flow in the renal circulation is subject to high resistance in the afferent and efferent arterioles. The juxtaglomerular apparatus is a structural arrangement between the afferent arteriole and the distal convoluted tubule that forms granular cells and macula densa cells. The pressure of the blood as it enters the glomerulus forces molecules of water, electrolytes, and other substances through the thin glomerular wall into Bowman's capsule. This mixture, called filtrate, collects in the capsule and drains into the renal tubule. Each segment of the tubule reabsorbs different substances from the filtrate as it passes through them. The Proximal tubule is where there is uncontrolled reabsorption and secretion of selected substances. The Loop of Henle establishes an osmotic gradient in the renal medulla that is important in the kidney's ability to produce urine of varying concentration. The Distal tubule and Collection duct are involved in variable controlled reabsorption of Na⁺ and H₂O and secretion of K⁺ and H⁺ occur here. Fluid leaving the collecting duct is urine, which enters the renal pelvis. A second network of capillaries separate from the glomerulus, the peritubular capillaries, entwines the renal tubule to allow the reabsorbed materials to reenter the blood circulation. (midlandstech.edu) (Wiki)

Each kidney contains between 800,000 to 1 million nephrons, although some have suggested as many as 1.5 million, each one capable of performing the above listed functions. The kidney cannot regenerate new nephrons. There is a gradual decrease in the number of nephrons either through aging, disease, or renal injury. After the age of 40, the number of nephrons usually decreases by 10% every 10 years. This loss however is not life threatening because of adaptive changes in the remaining nephrons, which allow them to excrete the right amounts of water, electrolytes, and waste products.

Each nephron is composed of an initial filtering component, the **renal corpuscle** and a **tubule**, which is specialized for reabsorption and secretion (Fig. 27).

The renal corpuscle is the nephron's initial filtering component, it filters out large solutes from the renal tubules for further modification. It is composed of two structures, the **glomerulus** and **Bowman's capsule** (Fig. 28). The glomerulus consists of a tuft of capillaries, which unlike other capillaries in the body, have a high hydrostatic pressure of about 60 mm Hg. It is this high hydrostatic pressure, which provides the driving force for water and solutes to be filtered out of the blood and into the space made by the

second component of the renal corpuscle, Bowman's capsule. It receives its blood supply from an **afferent arteriole**.

Bowman's capsule surrounds the glomerulus. Fluid filtered from the glomerular capillaries flows through a component of Bowman's capsule called **podocytes**. The resulting filtrate is further processed by the second component of the nephron, the **renal tubule** (Fig. 27), which begins as the proximal convoluted tubule.

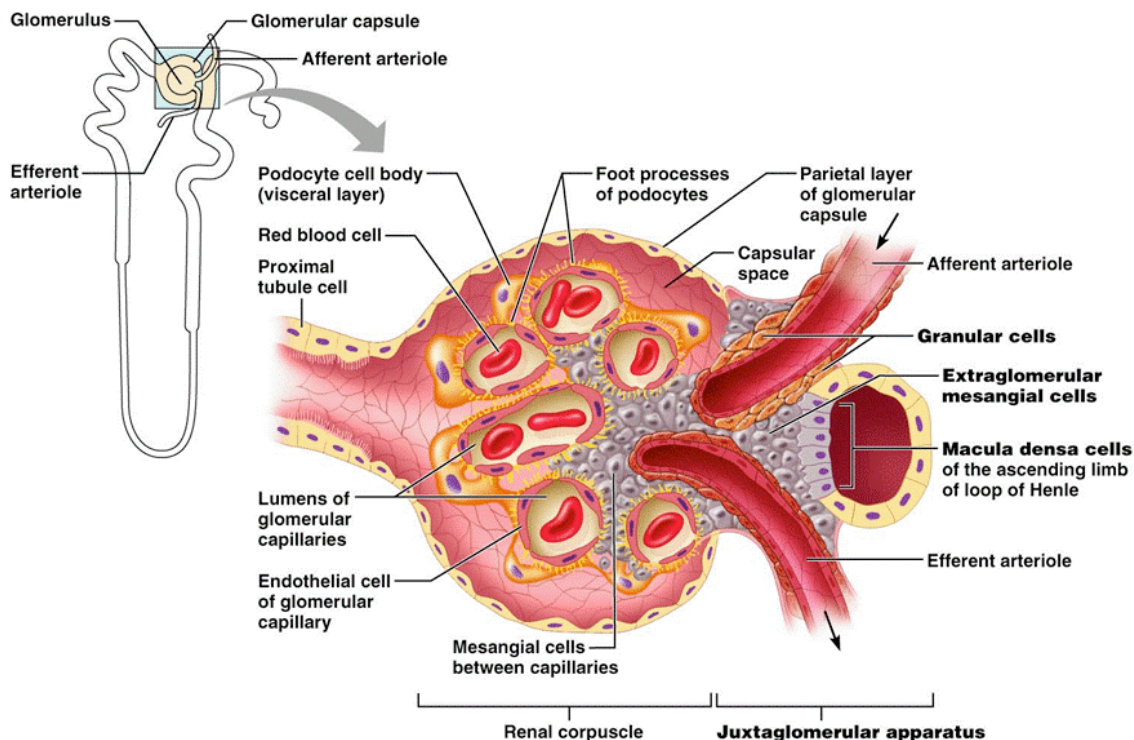


Fig. 28. This is an illustration of a renal corpuscle, which is composed of a glomerulus and Bowman's capsule. The walls of the glomerulus are only a few cells in thickness. The glomerular walls are semipermeable, allowing smaller molecules such as water, metabolic wastes, glucose, and electrolytes to pass through and collect in the capsule (Bowman's capsule) that surround the glomerulus. Together the glomerulus and Bowman's capsule are the renal corpuscle. The fluid and its contents, called filtrate, passes into the tubules of the nephron, which further filter and concentrate the filtrate. The nephron eventually reabsorbs 99% of the filtrate back into the blood; the remaining fluid drains into collecting ducts to move out of the kidneys as urine. The glomerular filtration rate (GFR) is an important measure of kidney function. The GFR of a healthy adult kidney is 120 - 125 ml per minute. (midlandstech.edu) (Wiki)

The renal tubule consist of: **Proximal convoluted tubule**; **Loop of Henle**, which in turn is composed of the **descending limb of the Loop of Henle** and the **ascending limb of the Loop of Henle**; and the **Distal convoluted tubule** (Figs. 27 & 29). After passing through the renal tubule, the glomerular filtrate becomes urine. It is the latter, which then passes into the **connecting tubule** and the **cortical collecting tubule**, which in turn leads to the **cortical collecting duct** and finely the **medullary collecting duct**. The urine then passes through the tips of each medullary pyramid, the **renal papillae**, into the minor calyces, then into the major calyces, followed by the renal pelvis, then the ureter (Fig. 25), which drains into the bladder.

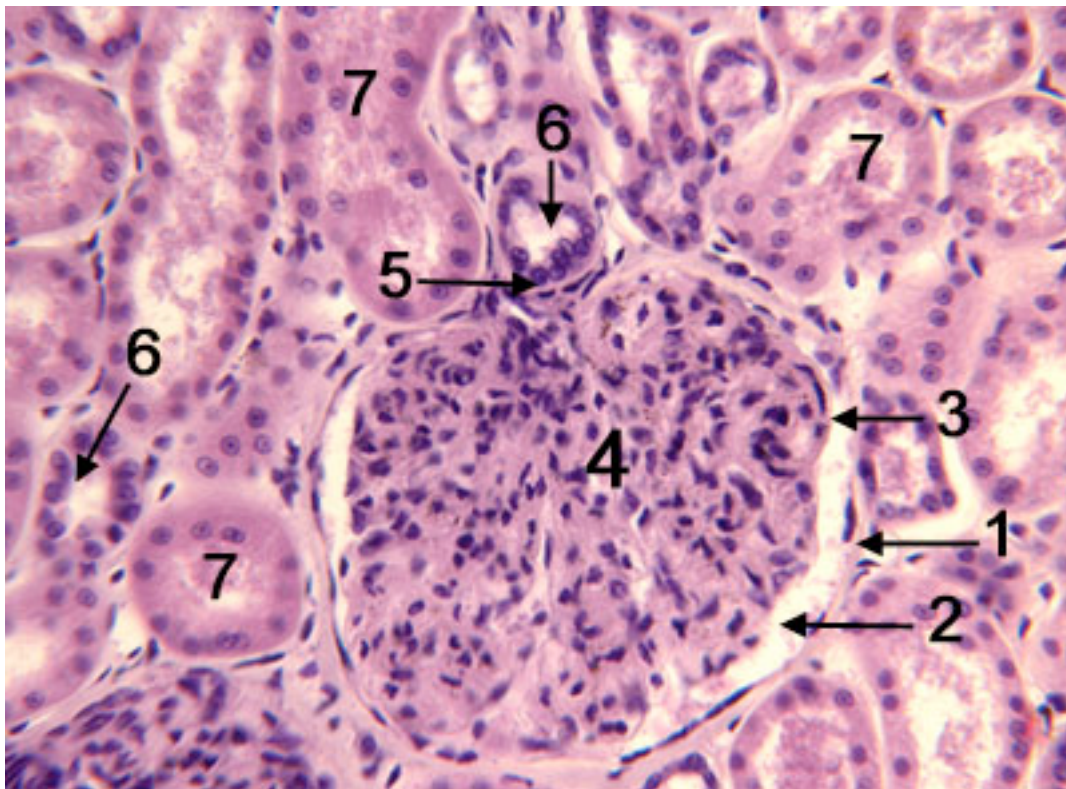


Fig. 29. This is a photomicrography of a kidney showing the following: 1. capsular epithelium, 2. urinary space, 3. glomerular epithelium, 4. glomerulus, 5. macula densa, 6. distal convoluted tubule, and 7. proximal convoluted tubule. (Wiki)

The renal blood supply to the two kidneys is typically about 22% of the cardiac output, or 1100 ml/min.

The renal arteries arise, one on each side of the aorta, at the level of the upper border of the second lumbar vertebra. Their origin is about 1 cm below that of the superior mesenteric artery. The **right renal artery**, longer and a little lower than the left, passes behind the inferior vena cava, the head of the pancreas, and the second part of the duodenum (Figs. 21 & 30). The **left renal artery** lies behind the left renal vein, the pancreas, and the splenic vein (Fig. 30). The **renal veins** pass lateral-ward anterior (in front of) the arteries (Fig. 30), but at the hilum of the kidneys the arteries and veins break up into segmental arteries (branches), which enter the renal sinus, both anterior and posterior to the renal pelvis. Before dividing into their major branches, each renal artery gives off an **inferior suprarenal artery**, which ascends to the lower part of the adrenal gland and **ureteric branches** to the ureter.

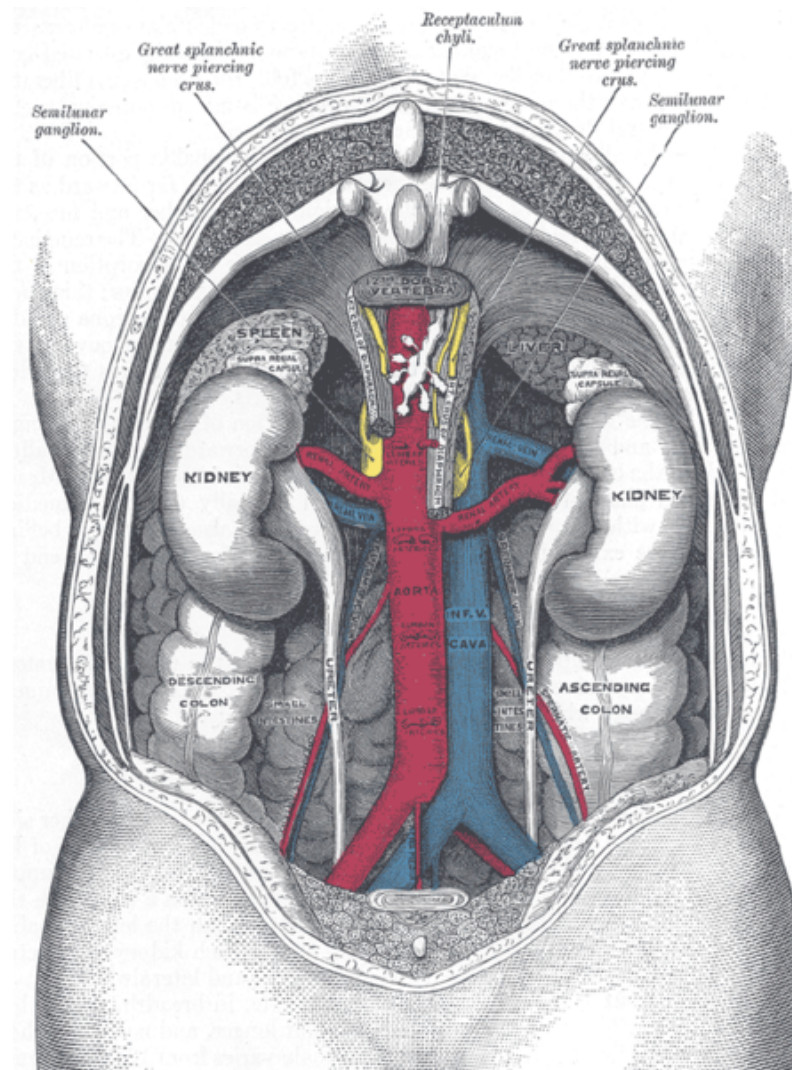


Fig. 30. The above illustration shows the renal arteries branching left and right from the aorta (in red), the inferior vena cava in blue, viewed from behind with the spine removed. (Wiki)

Within the renal sinuses the major branches (segmental arteries) of the left and right renal arteries divide into **anterior interlobar and posterior interlobar branches** (Fig. 31).

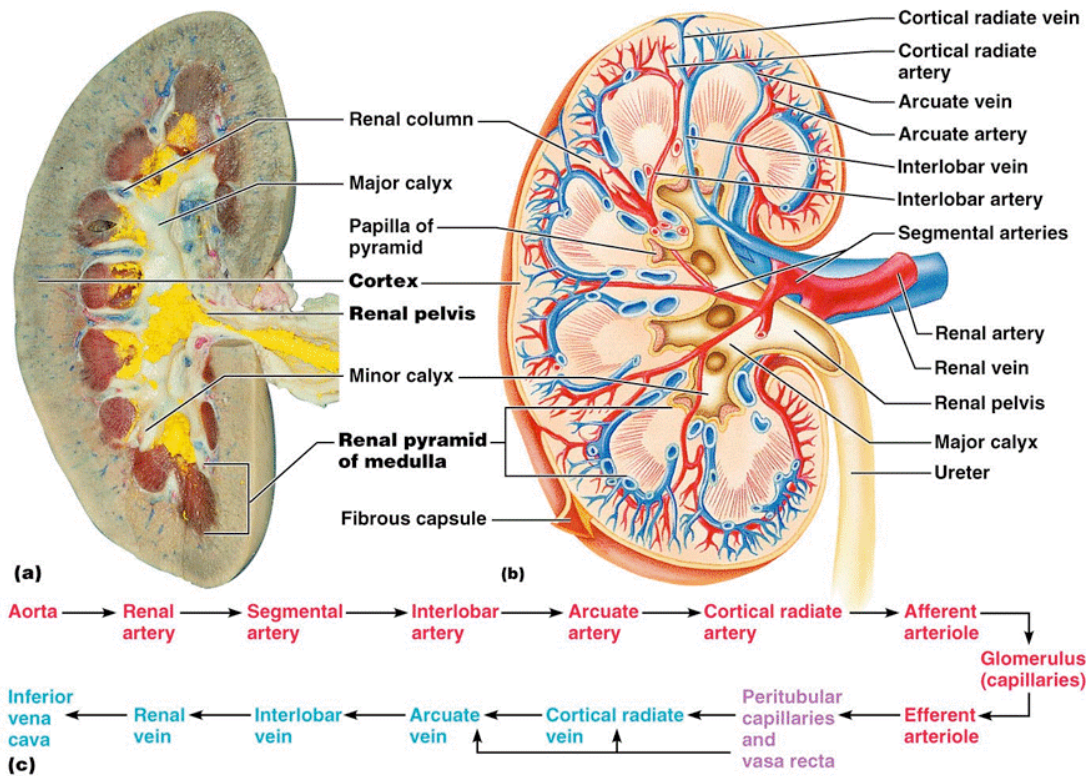


Fig. 31. This illustration shows the blood supply into and out of the kidneys progresses to the cortex through renal arteries to segmental, lobar, interlobar, arcuate, and cortical radiate arteries, and back to renal veins from cortical radiate, arcuate, and interlobar veins. The renal plexus regulates renal blood flow by adjusting the diameter of the renal arterioles and influencing the urine-forming role of the nephrons. (midlandstech.edu) (Wiki)

The anterior interlobar branches are larger and distribute somewhat past the midcoronal plane of the kidney. The interlobar arteries branch to form the **arcuate arteries**, which in turn branch to form the **interlobular arteries (radial arteries)**, which branch to form the **afferent arterioles**, which lead to the **glomerular capillaries**. The distal end of

each glomerulus coalesce to form the **efferent arterioles**, which in turn leads to the formation of a second capillary network, the **peritubular capillaries** that surround the renal tubules (Figs. 27, 31 & 32) Thus, the renal circulation has two capillary beds (networks), the glomerular and peritubular capillaries, which are arranged in series and separated by the efferent arterioles, which help regulate hydrostatic pressure in both sets of capillaries (Fig. 32). The 60 mmHg of hydrostatic pressure in the glomerular capillaries causes rapid fluid filtration, whereas the much lower hydrostatic pressure in the peritubular capillaries, approximately 13 mmHg, allows for rapid fluid reabsorption. Through adjusting the resistance in the afferent and efferent arterioles, the kidneys can regulate the hydrostatic pressure in both capillary networks thus, changing the rate of glomerular filtration, tubular reabsorption, or both in response to changing physiologic demands.

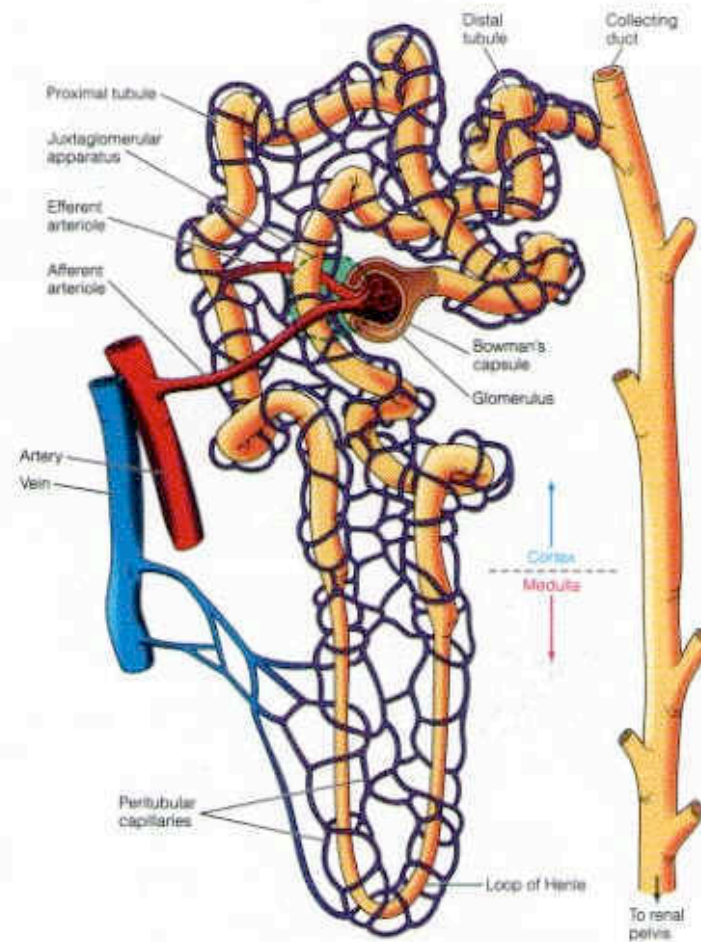


Fig. 32. The above illustration shows the vascular components of the nephron: afferent arteriole-carries blood to the glomerulus; efferent arterioles-carries blood from the glomerulus; glomerulus-consists of capillaries that filters a protein free plasma into the tubular component; and peritubular capillaries-supply the renal tissue involved in exchanges with the fluid in the tubular lumen. (Wiki)

The peritubular capillaries empty into the vessels of the venous system, which run next to the arteriolar vessels. The blood vessels of the venous system progressively form the **interlobular veins, arcuate veins, interlobar veins** and the **renal vein**. Both the left and right renal veins end in the **inferior vena cava** (Figs. 30).

The **lymphatic vessels** of the kidney form three plexuses: one in the substance of the kidney around the renal tubules, under the renal capsule, and in the perirenal fat. Collecting vessels from the intrarenal plexus form four or five trunks, which follow the renal vein to end in the lateral aortic nodes; the subcapsular collecting vessels join them as they leave the hilum. The perirenal plexus drains directly into the lateral aortic nodes.

Innervation of the kidney is both **sympathetic** through the **renal plexus** and **parasympathetic** through the **vagus nerve**. Rami from the celiac ganglion and plexus, aorticorenal ganglion, lowest thoracic splanchnic nerve, first lumbar splanchnic nerve and aortic plexus form a dense plexus of autonomic nerves around the renal artery, the **renal plexus** (Fig. 33).

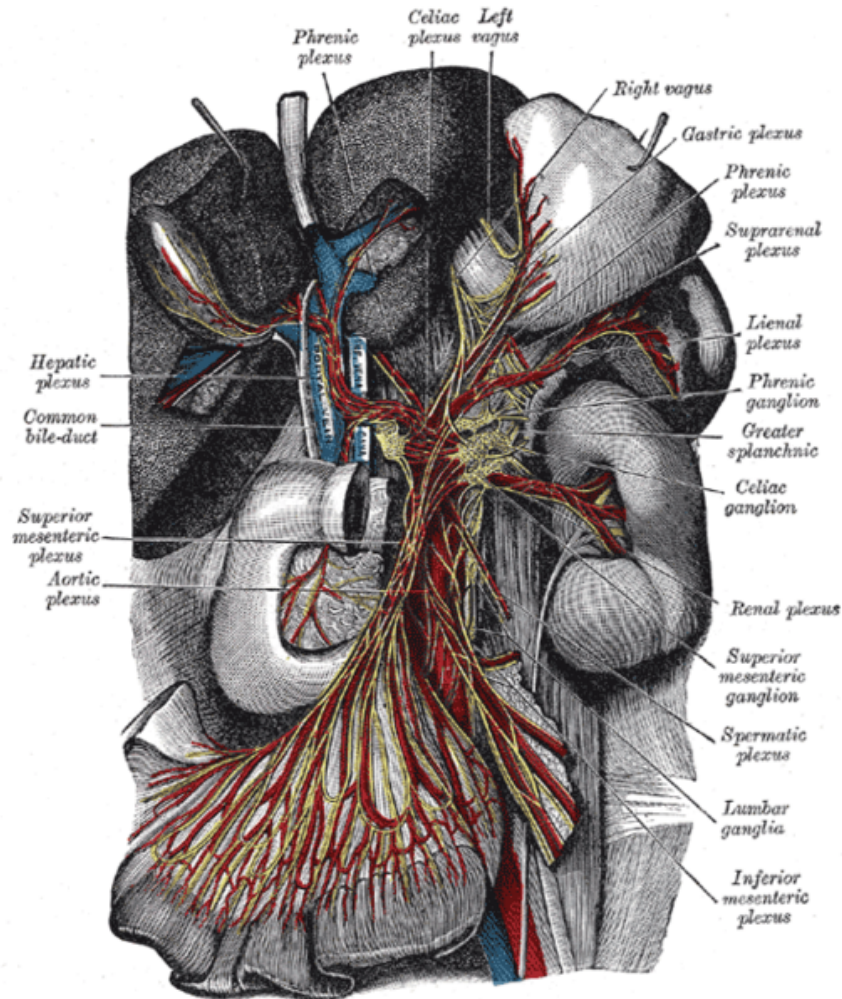


Fig. 33. This is an illustration of the renal plexus, which is formed by filaments from the celiac plexus, the aorticorenal ganglion, the aortic plexus, lower thoracic splanchnic nerve, and the first lumbar splanchnic nerve. The renal plexus is labeled at the center right. (Wiki)

Small ganglia occur in the renal plexus, the largest typically behind the origin of the renal artery. The renal plexus extends into the kidney around the arterial branches and supplies the vessels and renal glomeruli, and especially around the cortical tubules. The renal plexus also sends offshoots to the testicular plexus, the inferior vena cava on the right side, and the ureteric plexuses.

Preganglionic sympathetic fibers pass through the lower part of the sympathetic trunk and the lowest thoracic splanchnic nerve to join the renal plexus. These preganglionic fibers synapse with **postganglionic neurons** in the renal plexus. The

postganglionic fibers are distributed to the kidney along the branches of the renal artery. The sympathetic nerves are vasoconstrictor in action to the renal arteries. **Preganglionic parasympathetic fibers** enter the renal plexus from the vagus nerve. Here, they synapse with **postganglionic neurons**, whose fibers are distributed to the kidney along the branches of the renal artery. The parasympathetic fibers are believed to be dilatory in action (Fig. 34).

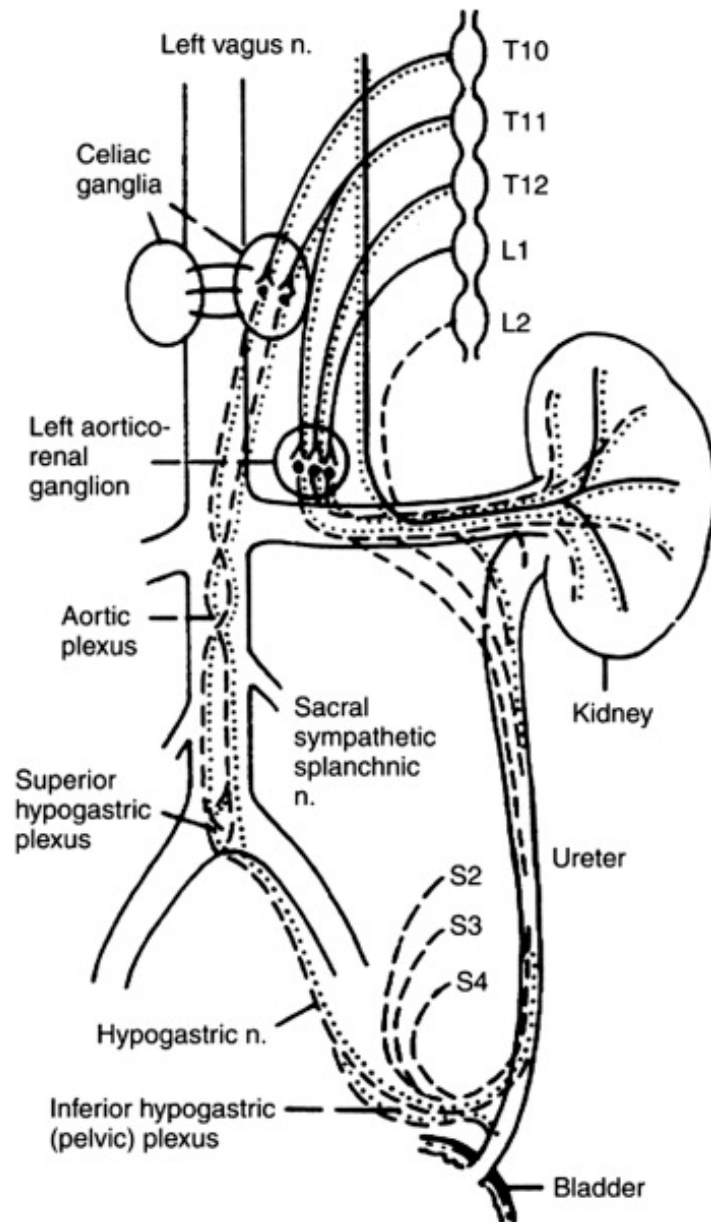


Fig. 34. The above illustration shows the sympathetic, parasympathetic and sensory innervation of the kidney. Sympathetic fibers from the spinal cord run to the sympathetic chain and, thereafter, through the splanchnic nerves synapse in the celiac ganglion. The splanchnic nerves lie medial to the sympathetic trunk over the bodies of the thoracic vertebrae. Solid line: sympathetic component; dotted line: parasympathetic component; dashed line: afferent sensory fibers, which travel with the autonomic nerves. (National Kidney Foundation) (Wiki)

The **afferent fibers** from the kidney probably reach the spinal cord through the tenth, eleventh, and twelfth thoracic nerves (Fig. 34).

Referred renal pain is typically felt as a dull and constant ache in the costovertebral angle just lateral to the sacrospinalis muscle and just below the twelfth rib. This pain often spreads along the subcostal area toward the umbilicus or lower abdominal quadrant. Referred pain can be caused by any renal disease, which produces sudden distention of the renal capsule, such as in **acute pyelonephritis** due to its sudden edema and **acute ureteral obstruction** due to its sudden renal back pressure.

However, it must be understood that many renal diseases are painless because their progression is so slow that sudden capsular distention does not occur. Such diseases are cancer, chronic pyelonephritis, staghorn calculus, tuberculosis, polycystic kidney, and hydronephrosis due to chronic ureteral obstruction.

C. Ureter: The ureters are thick-walled muscular tubes, which represent a continuation of the renal pelvis superiorly. Its peristaltic contraction conveys urine from the kidneys to the urinary bladder. Each adult ureter has a length, which varies between 25 to 30 cm, and a diameter of about 0.5 cm. Due to its thick wall it has a very narrow lumen. The ureters have three points of narrowing, which are of embryological origin, at the ureteropelvic junction, as they pass over the iliac vessels, and at the entry to the bladder wall. Approximately half of the ureter's length is abdominal, and half is in the pelvis (Fig. 35).

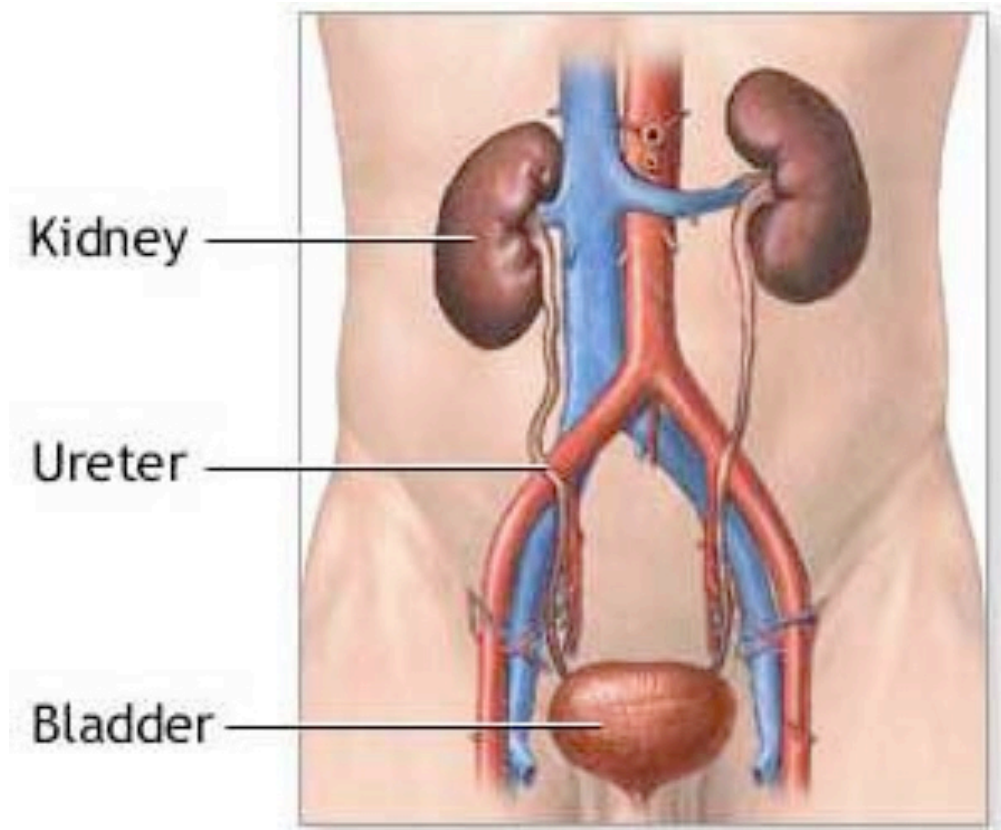


Fig. 35. This is an illustration of the path the ureter takes from each kidney to reach the bladder. (Wiki)

The walls of the ureters consist of an inner mucosa that is continuous with the kidney pelvis and the mucosa of the bladder. It has a double-layered muscularis, and a connective tissue adventitia covering the external surface (Fig. 36).

The ureters have an entirely retroperitoneal course as they run downward and slightly medial to reach the bladder, passing over the psoas major muscle as it does so. The psoas major muscle separates the ureters from the tips of the lumbar transverse processes. The ureters descend on the psoas major muscles until crossing the bifurcation of the common iliac arteries or the beginning of the external iliac arteries where they enter the pelvis. In its course through the retroperitoneal space of the abdomen, the right ureter is crossed anteriorly (in front of) by the right colic and ileocolic vessels, the second part of the duodenum above and the lower part of the root of the

mesentery below. The left ureter is crossed anteriorly by the left colic vessels and the mesentery of the pelvic colon below.

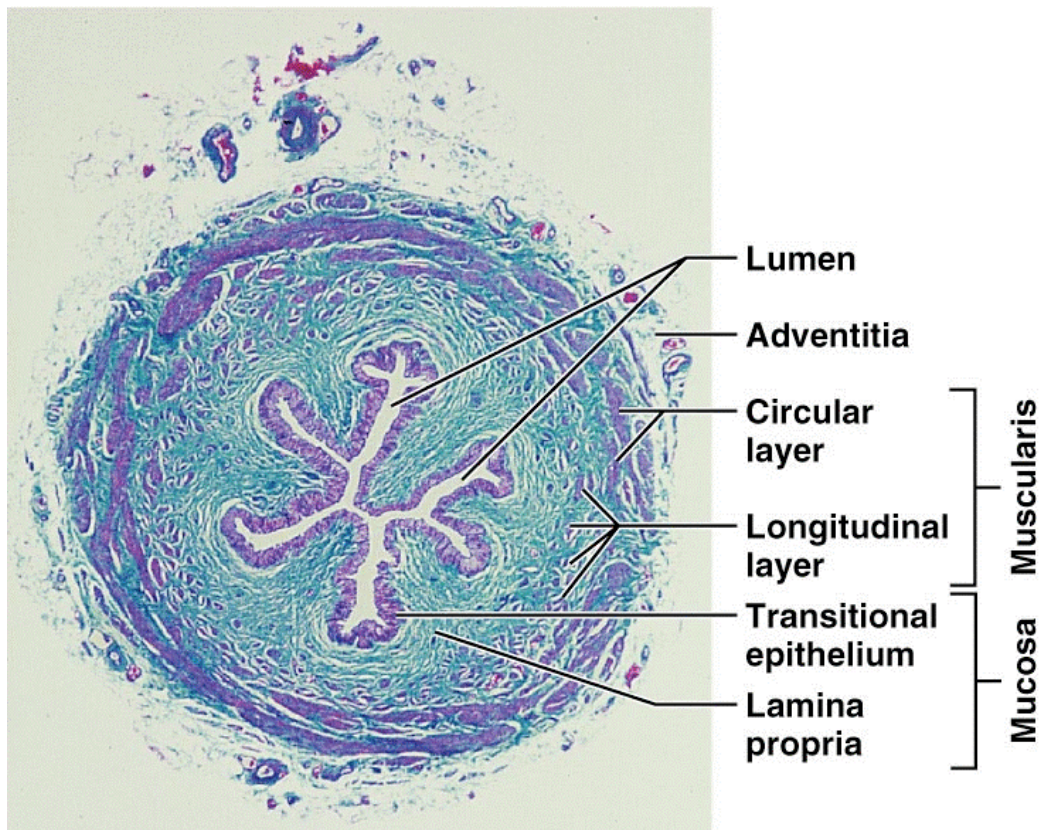


Fig. 36. The above is a photomicrograph of the ureter denoting its various anatomical components. (midlandstech.edu) (Wiki)

Although this chapter concerns the organs of the abdominal retroperitoneal space, for the sake of continuity we will briefly discuss the path of the ureters through the retroperitoneal space of the pelvis. Most of the path in the pelvis is along the lateral wall as it descends in a posteriolateral direction. Opposite the ischial spine and greater sciatic foramen, it turns anteromedially into fibrous tissue above the **levator ani** and within approximately 20 cm reaches the lateral angle of the bladder. As it passes along the lateral wall of the pelvis, it is anterior (in front of) the **internal iliac artery** and the beginning of its anterior trunk. Posterior to the ureter are the **internal iliac vein**, **lumbosacral nerve** and **sacroiliac joint** (Fig. 37).

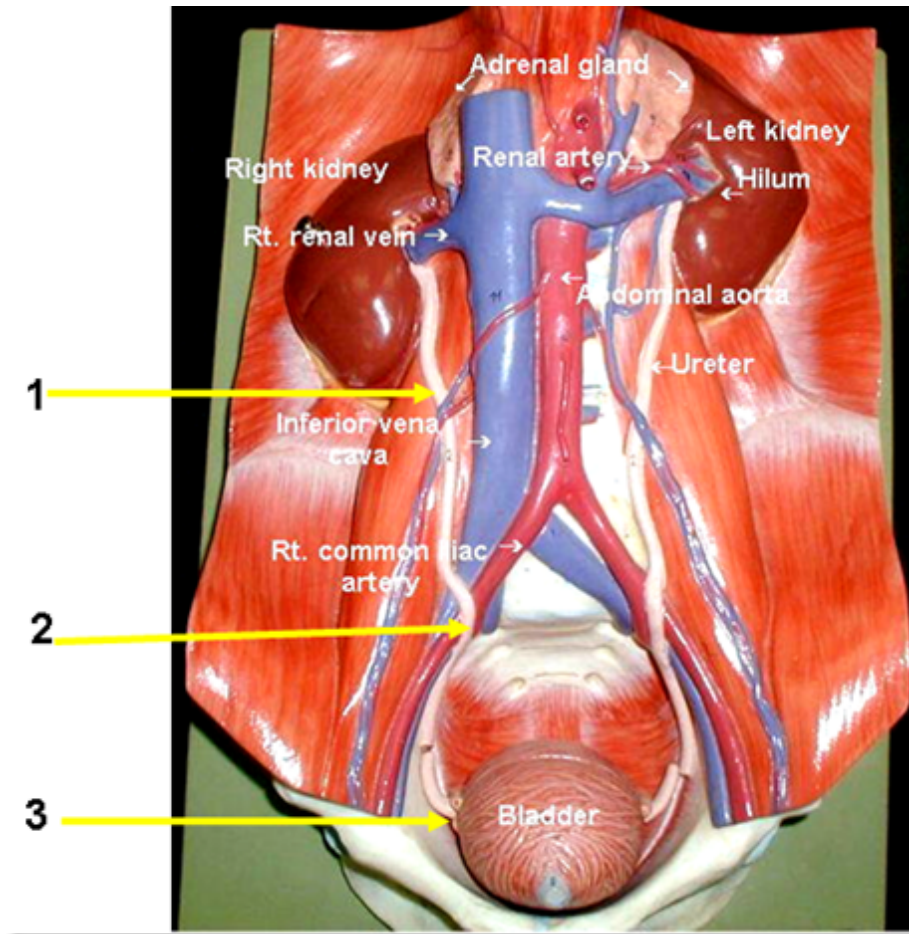


Fig. 37. This is an illustration showing the course of the ureter through the retroperitoneal spaces of the abdomen and pelvis. The numbers apply to the normal areas of anatomic constrictions of the ureter: 1. is where the renal pelvis joins the abdominal part of the ureter; 2. is the point where the ureter crosses the pelvic brim (it enters the pelvis by crossing the region of the bifurcation of the common iliac arteries in front of the sacro-iliac joint); and 3. where the ureter pierces the urinary bladder. (imueos.wordpress.com) (Wiki)

In the male, the ureter passes under the **vas deferens** and anterior to (in front of) the upper end of the **seminal vesicles** to run obliquely through the bladder wall, ending in one angle of the trigone. The trigone is a smooth region of the internal urinary bladder formed by the two **ureteral orifices** and the internal **urethral orifices** (Fig. 38). This

area is very sensitive to expansion and once stretched to a certain degree, the urinary bladder signals the brain of its need to empty.

Urinary Bladder

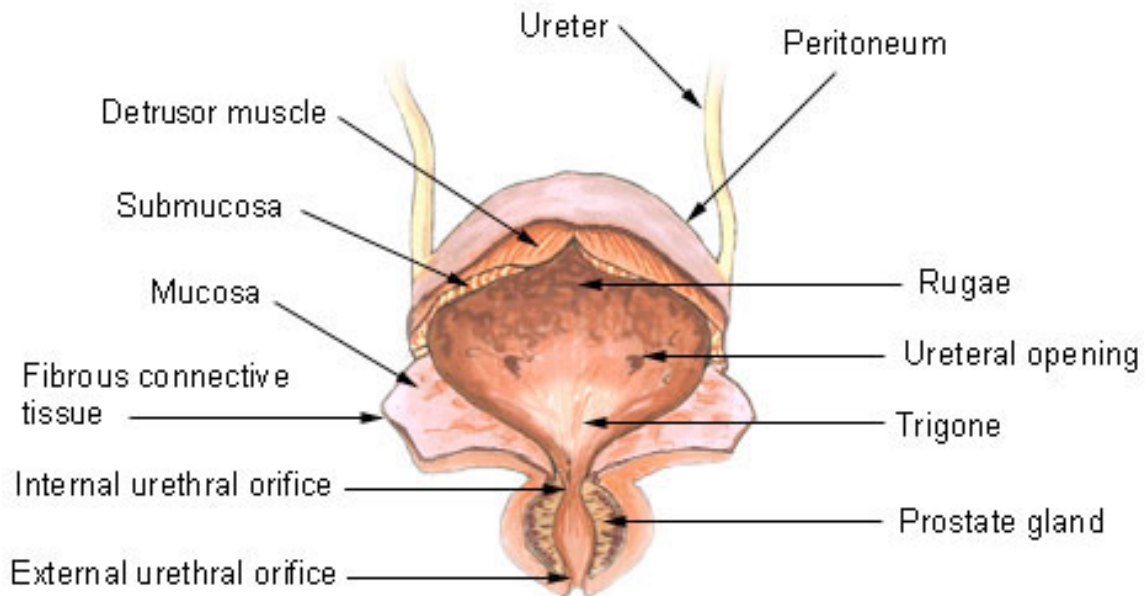


Fig. 38. This is an illustration of the location of the trigone within the urinary bladder. (Wiki)

In the female, when the pelvic part of the ureter passes in front of the internal iliac artery it is directly posterior to the ovary. The final section of the ureter runs below the lower part of the broad ligament, running lateral to the cervix and lateral vaginal fornices and is briefly in front of the vagina just before entering the bladder. The fornices of the vagina are the deepest portions of the vagina, extending into the recesses created by the vaginal portion of the cervix. The word 'fornix' is Latin for 'arch' (Fig. 39).

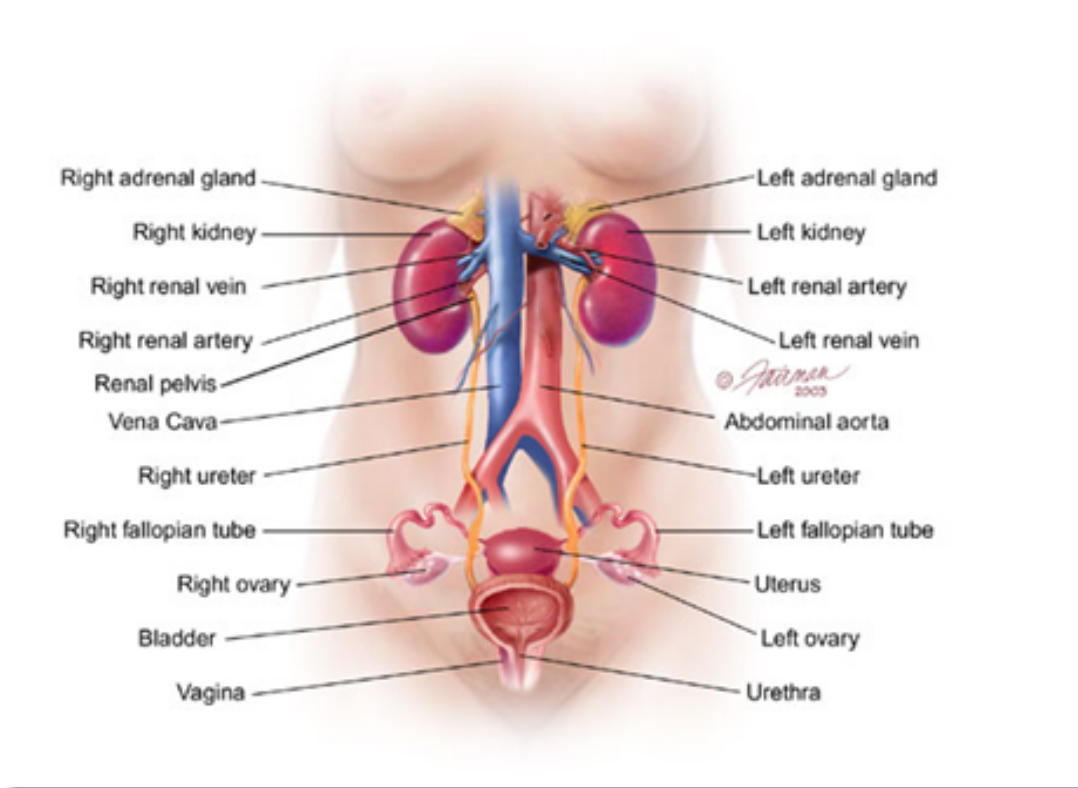


Fig. 39. This is an illustration of the path of the left and right ureters through the female pelvis. (imueos.wordpress.com) (Wiki)

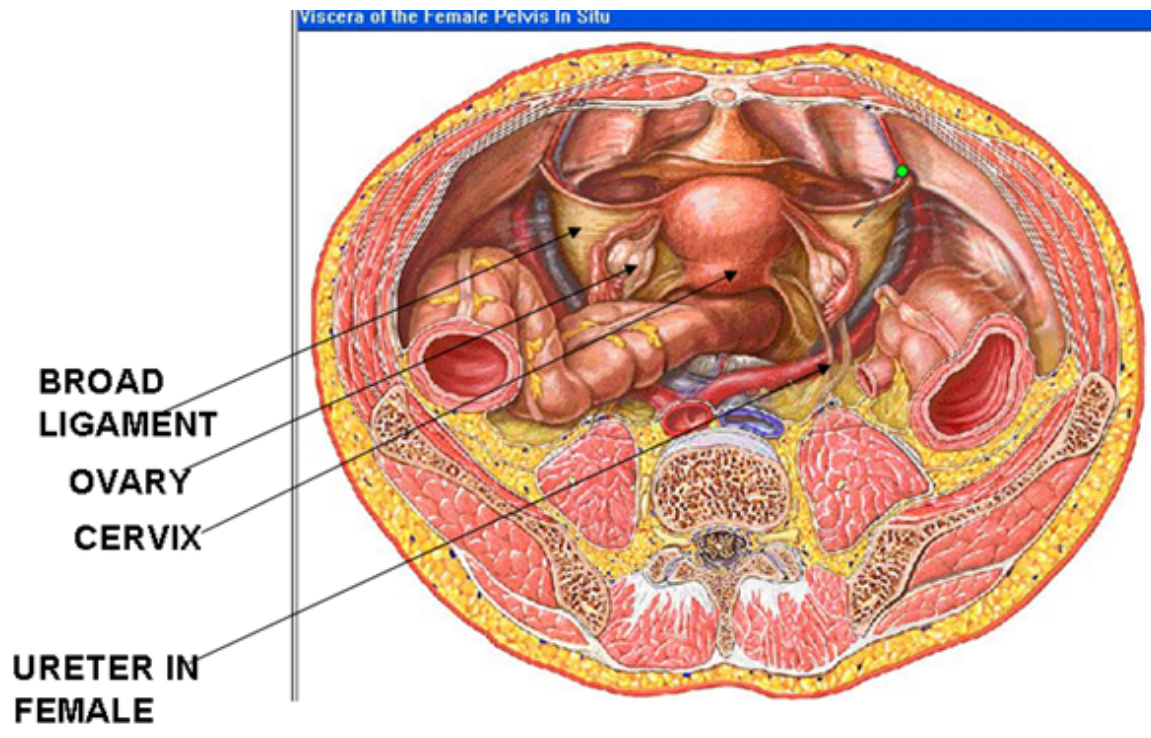


Fig. 40. The above is a cross section of the female pelvis showing how the ureter runs forward through the broad ligament (ligaments that connect the uterus to the lateral pelvic wall), related closely to the cervix of the uterus and uterine artery and forms the floor of the ovarian fossa. (imueos.wordpress.com) (Wiki)

The vascular supply to the ureters is by branches of the renal, gonadal, common iliac, internal iliac, vesical and uterine arteries, and the abdominal aorta (Fig. 41). The abdominal ureter is supplied by vessels, which arise medial to the ureter, while the pelvic ureter is supplied by vessels arising lateral to the ureter.

The venous drainage of the ureter follow the arterial supply.

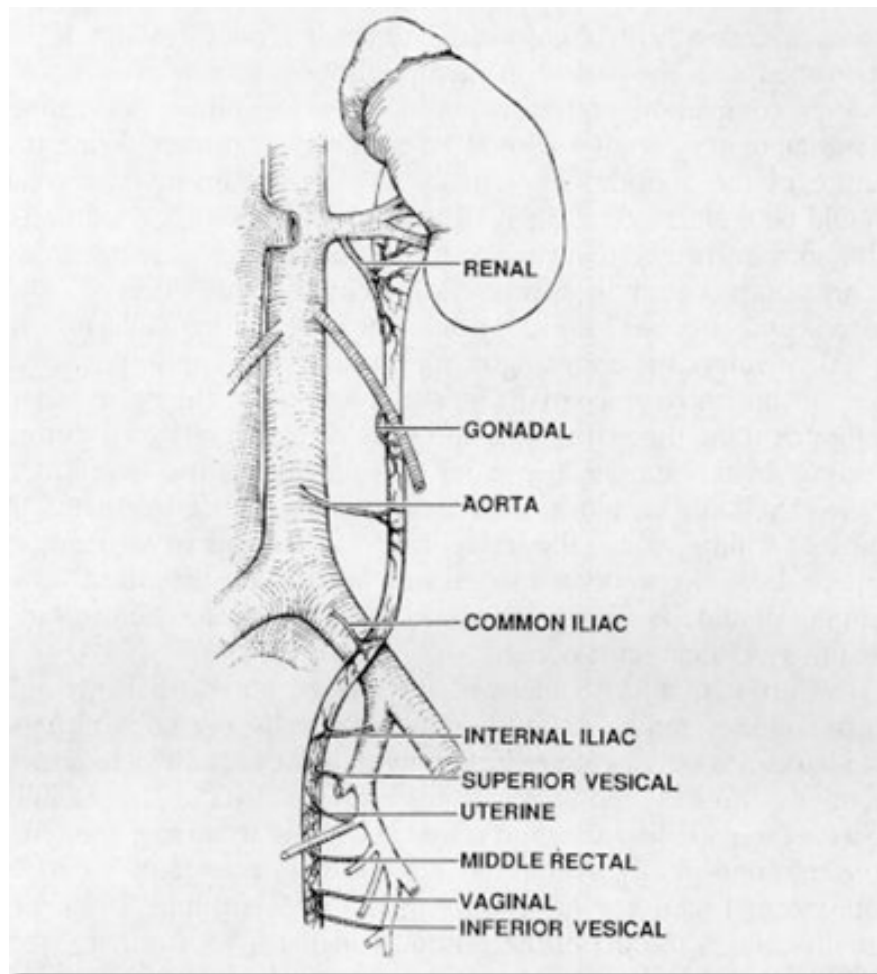


Fig. 41. The ureter receives its blood supply from: renal artery, aorta, common iliac artery, internal iliac artery, superior vesical artery, and the inferior vesical artery. In the female there are two additional vascular systems which contribute, the uterine and vaginal. (imueos.wordpress.com) (Wiki)

The **lymphatic vessels** of the ureter form plexuses in the submucosa, intramuscular and adventitia, all of which communicate with one another. The upper abdominal ureter lymphatics often join the renal lymphatic collecting vessels or will directly enter the lateral aortic nodes. The lower abdominal ureter lymphatics drain to the common iliac nodes, whereas the lymphatics of the pelvic ureter drain to the common, external or internal iliac nodes.

The **innervation** of the ureters is through the **ureteric plexus**, which is derived from the **renal, aortic, superior, and inferior hypogastric plexuses**. The ureteric nerves consist of an irregular plexus in the adventitia of the ureter. This plexus receives branches from the renal and aortic plexuses in its upper part, from the superior hypogastric plexus and hypogastric nerve in its intermediate part, and from the hypogastric nerve and inferior hypogastric plexus in its lower part.

The **sympathetic innervation** of the ureter is derived from T10-12 and L1. The **parasympathetic innervation** is derived from S2-4 (Fig. 34). The full significance of the autonomic innervation of the ureters is not fully understood except for the fact that they conduct afferent sensory stimuli. The ultrastructural features of the human ureter seems to indicate the autonomic nervous system has a minor role in the control of ureteral motility. Furthermore, excised portions of the ureter continue to contract, and denervation of the lower portion of the ureter does not cause reflux to occur.

Afferent pain fibers from the ureter pass back to T11, T12, and L1, L2.

Referred ureteric pain is often severe and spasmodic (renal colic) and is referred to the groin, labia majora, scrotum and the anterior thigh. When referred to the scrotum, the cremaster muscle, which has the same innervation, may reflexly retract the testis.

D. Adrenal glands: Typically, the adrenal glands are not addressed in blunt force traumatic injuries of the abdomen. However, one part of the adrenal gland, the cortex, is essential to life. Interference with its function causes substantive disruption of fluid and electrolyte balance in the body thus, it can play a significant role in determining cause of death.

The adrenal glands are situated slightly anterior on the supramedial aspect of each kidney (Fig. 42). The glands are situated close to the midline, being on either side of the vertebral column. The right adrenal gland is behind the inferior vena cava and the

right lobe of the liver and is in front of the diaphragm and the superior right renal pole. The left adrenal is behind the cardia of the stomach and the pancreas and splenic artery. It is in front of the left leaf of the diaphragm and the upper pole of the left kidney. The two glands are separated medially by the celiac arterial trunk and celiac plexus of nerves. They have a golden yellow color and structurally are divided into two functionally distinct anatomic areas, and outer **cortex** and an inner **medulla**. Functionally, these parts are two separate endocrine glands.

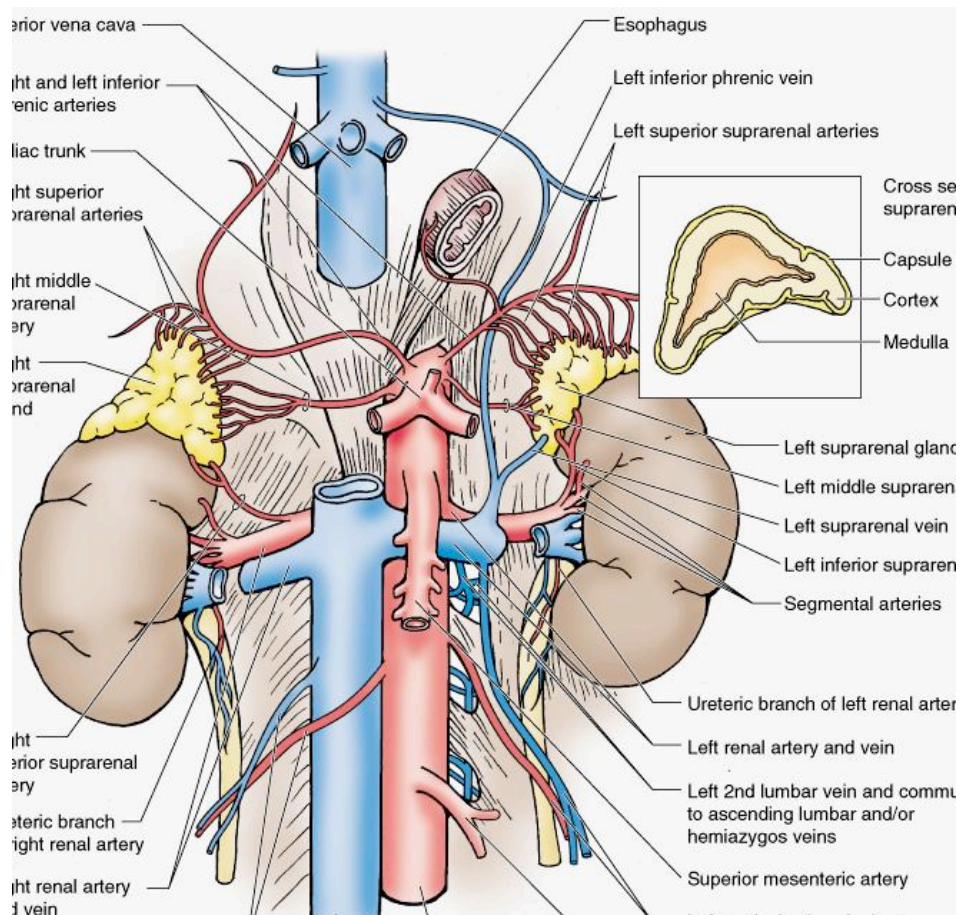


Fig. 42. This is an illustration of the anatomic relationships of both adrenal glands. (RahulGladwin.comImageGallery) (Wiki)

As pointed out, the **cortex** is concerned with maintaining fluid and electrolyte balance. It is also concerned with carbohydrate, protein and fat metabolism, is important in normal body reactions to stress, helps maintain blood pressure and cardiovascular function, helps slow the immune system's inflammatory response, and helps balance

the effects of insulin breaking down sugar for energy. To accomplish these functions the cortex secretes a group of hormones called **corticosteroids** of which there are two major types, the **mineralocorticoids** and the **glucocorticoids**. In addition, small amounts of **sex hormones** are secreted, especially **androgenic hormones**. These hormones are all synthesized from the steroid **cholesterol**, and they all have a similar chemical formulas, however, slight differences in their molecular structures gives them several different functions (Figs 43 & 44).

The **adrenal medulla** is functionally related to the **sympathetic nervous system**; it secretes the hormones **epinephrine** and **norepinephrine** in response to sympathetic stimulation. In many respects the medulla can be thought of as a **postganglionic sympathetic organ**, for it receives only a **preganglionic innervation** (Figs. 43 & 44).

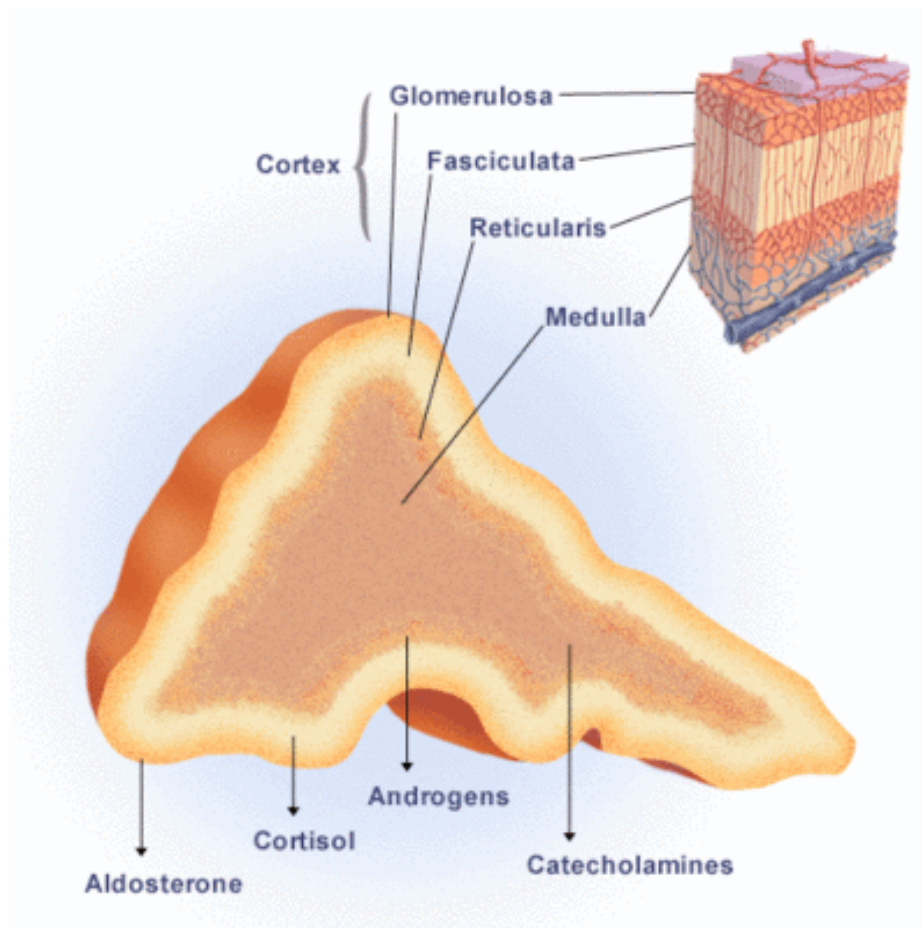
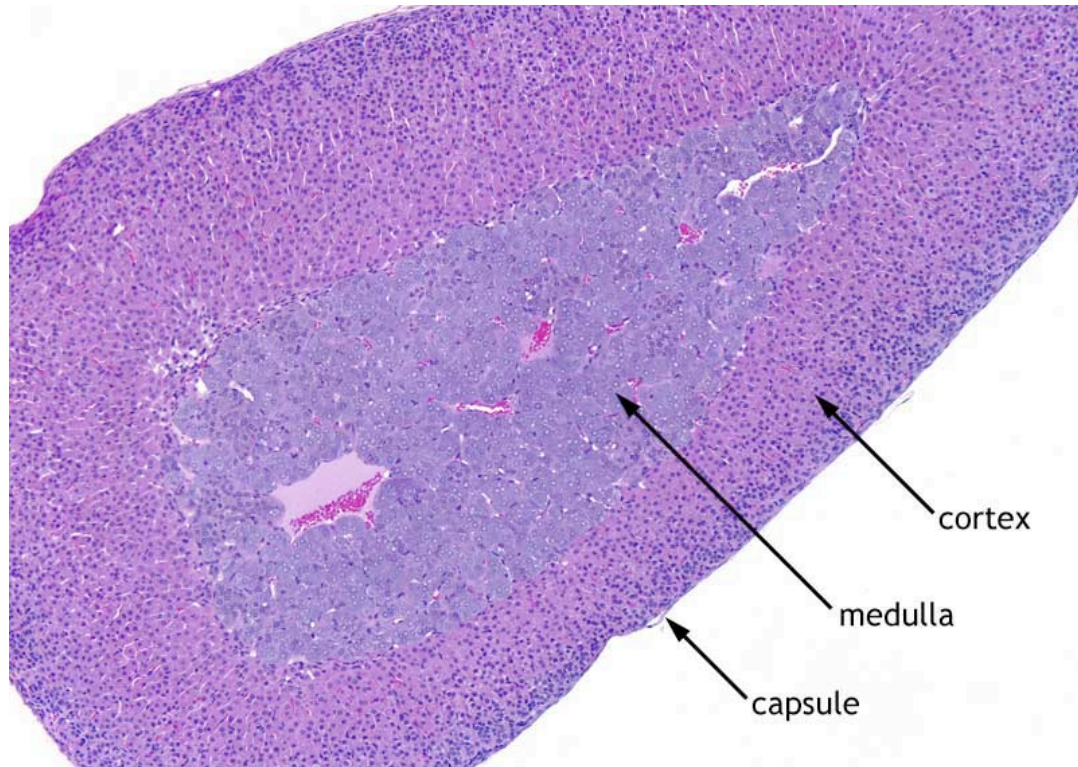


Fig. 43. This is an illustration of the anatomic parts of the adrenal gland, the anatomic parts of the cortex, and where the location of production of the hormones. (Wiki)



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Fig. 44. This is a photomicrograph of a H&E slide showing the components of the adrenal gland. (Wiki)

The **left adrenal** is crescentic in configuration, whereas the **right** is triangular or pyramidal in shape (Fig. 45). They vary in length from 3 to 5 cm, 2 to 3 cm in breadth, and less than 1 cm in thickness. In adults the combined weight of the glands is 10 to 14 gm, the left being a little larger than the right. The medulla contributes approximately one-tenth of the total weight. At birth, the weight of the glands is greater than a few months later, usually amounting jointly to 5 or 6 gm, due to the presence of the provisional cortex. At the end of the age spectrum, there is a slight decrease in weight.

Each gland has a hilum on its anterior surface; from this furrow emerges a single, prominent, suprarenal or central vein. It is typically 5 mm in diameter. On the left side it unites with the phrenic vein and then drains into the left renal vein. On the right side it empties directly into the posterior surface of the inferior vena cava.

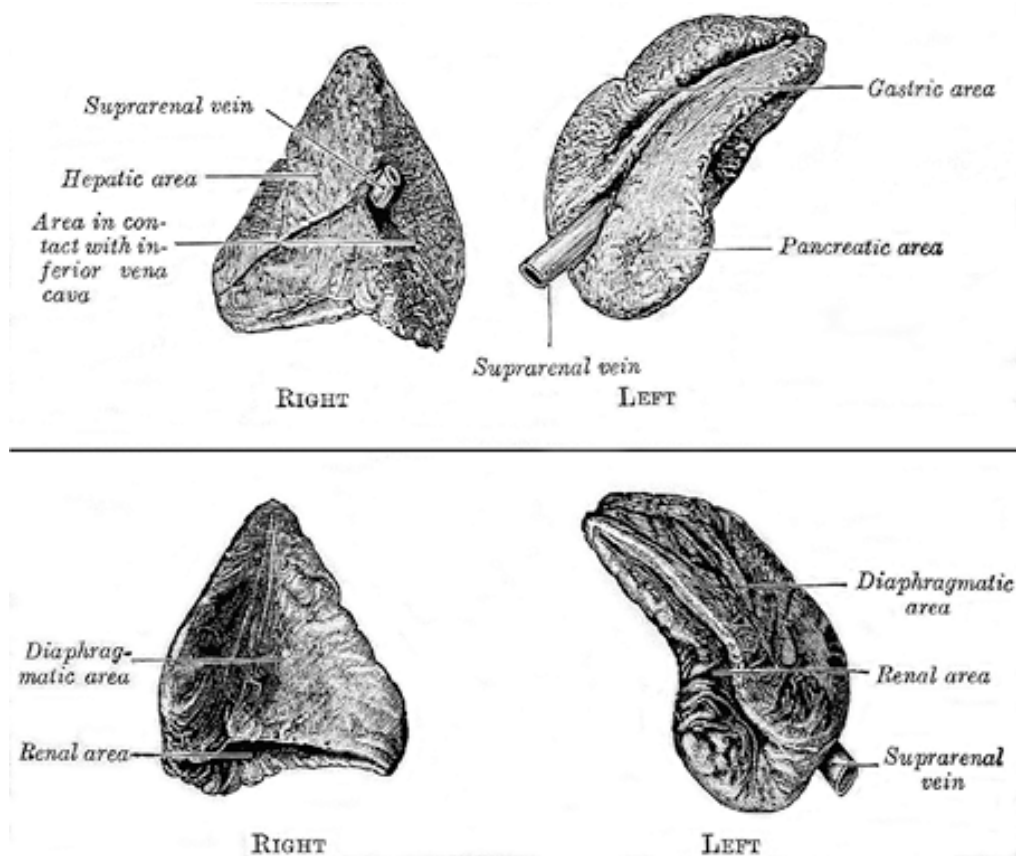


Fig. 45. The upper images represent the posterior surface of the adrenal glands, whereas the lower images represent the anterior surface. (Wiki)

The adrenal glands, even as compared to other endocrine glands, are highly vascular. More blood passes through these glands in relation to their size than through any other organs of the body, with the possible exception of the **thyroid gland** (Figs 42 & 46). Three types of adrenal arteries supply the gland. An average of from six to eight **superior adrenal arteries** are delivered from the phrenic artery. A **middle adrenal artery**, sometimes several, arise from the abdominal aorta, typically, at or above the level of the renal artery. One or more **inferior adrenal arteries** arise from the renal artery.

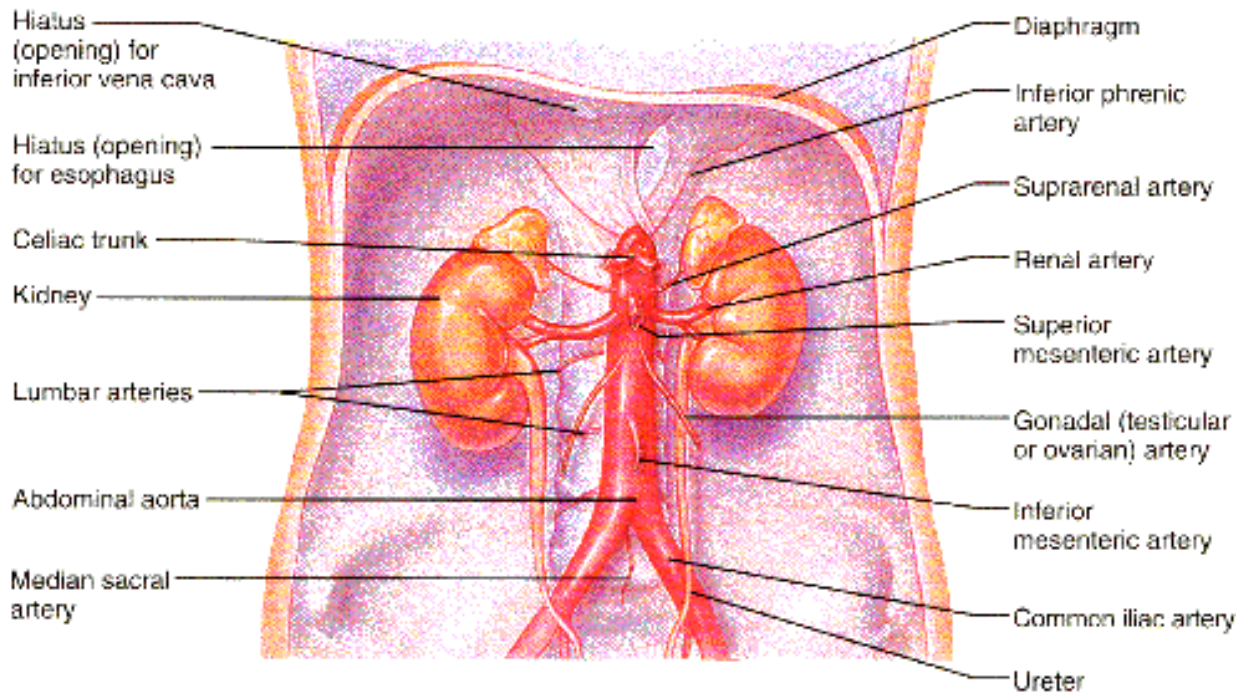


Fig. 46. This illustration shows the blood supply to the adrenal glands. (rci.rutgers.edu) (Wiki)

The **lymphatic vessels** of the adrenal glands arise from a plexus under the capsule and from another in the medulla of the gland. They follow the vessels of the gland, predominantly the suprarenal vein and drain into the lateral lumbar aortic nodes between the level of the celiac artery and origins of the renal arteries. Some lymphatic vessels perforate the diaphragm to reach the posterior mediastinum.

The adrenal glands, relative to their size, have a larger **autonomic nerve** supply than any other organ. On each side, the **adrenal plexus** lies between the medial aspect of the gland and the **celiac** and **aorticorenal ganglia**. The adrenal plexus contains mostly **preganglionic sympathetic fibers**, which originate from spinal cord levels T3-L2, with the greatest number of fibers arising at T9 and T10. The majority of these preganglionic sympathetic fibers take origin from neurons within the **nucleus intermediolateralis pars funicularis (IMLPF)**, with a small portion coming from neurons located in the **pars intercalatus** (Fig. 47). Those preganglionic sympathetic fibers taking origin from the neurons located in IMLPF at T3-T9, reach the celiac ganglia and adrenal plexus through the **greater splanchnic nerve** with the majority not

synapsing on the neurons in the celiac ganglion or adrenal plexus and innervate the adrenal medulla. The preganglionic fibers arising from the neurons located in IMLPF at T10-L2 also contribute to the sympathetic innervation of the adrenal medulla. These preganglionic fibers pass through the sympathetic chain, exit through the lesser splanchnic nerve, again, with the majority not synapsing in the celiac ganglion or adrenal plexus, and innervate the adrenal medulla (Fig. 47). However, some of the preganglionic sympathetic fibers form synapses in the celiac ganglion and ganglion cells in the adrenal plexus. The resulting postganglionic sympathetic fibers represent a small number of sympathetic fibers innervating the adrenal glands. Both the fibers in the greater and lesser splanchnic nerves, as well as the postganglionic fibers taking origin in the celiac and adrenal plexus ganglion cells, synapse on the large medullary chromaffin cells in the adrenal medulla, which in many respects are considered as **postganglionic sympathetic neurons**. Acetylcholine is the transmitter released at the sympathetic preganglionic terminals. The cells which they synapse on then release epinephrine (about 80%) and a small percentage (about 20%) of norepinephrine into the blood stream.

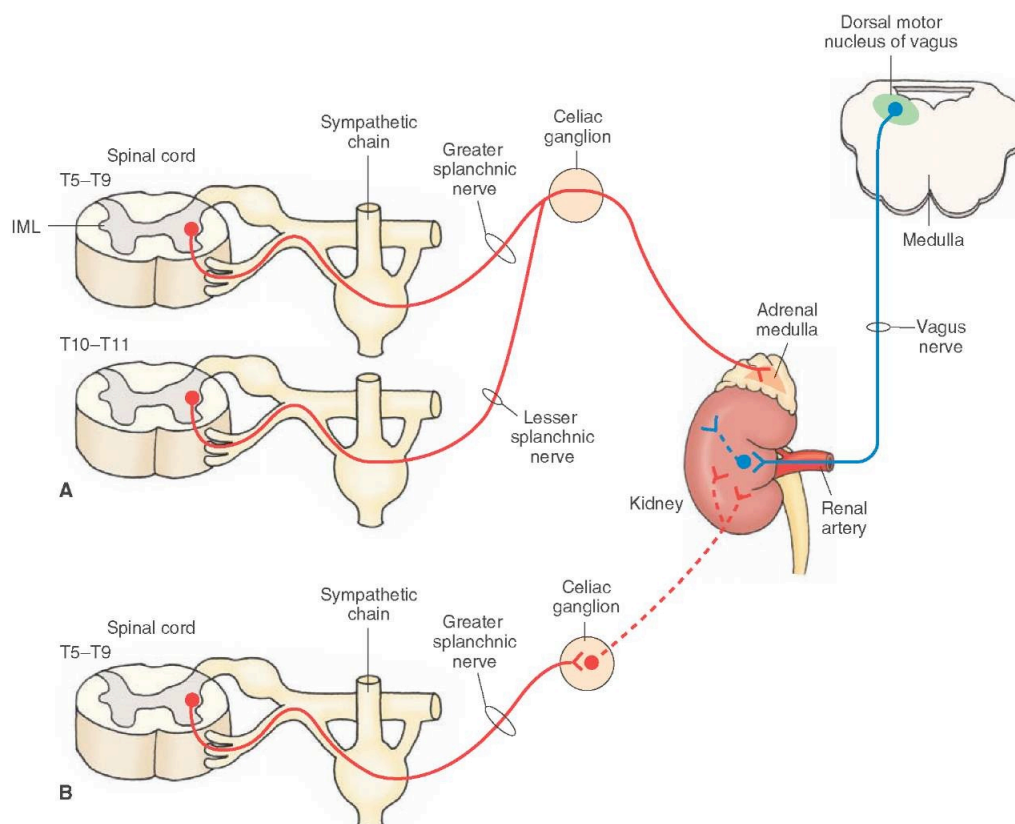


Fig. 47. The above figure illustrates the autonomic innervation of the adrenal gland and kidney. Red = sympathetic nervous system; Blue = parasympathetic nervous system. Solid lines = preganglionic fibers, dotted lines = postganglionic fibers. T = thoracic spinal segment; IML = intermediolateral cell column (intermediolateralis pars funicularis in text). (what-when-how.com/neuroscience/the-autonomic-nervous-system-integrative-systems-part-3) (Wiki)

The conventional line of thought is that steroid secretion by the cortex is hormonally controlled. However, there is substantial evidence that neural activity can directly affect endocrine organs including the adrenal cortex. This was documented through demonstrating terminals containing synaptic vesicles next to cortical cells. It appears there is some form of integrated neural and hormonal vascular circuit between the innervation of the cortex and medulla.

Although, both the guinea pig, and to a lesser extent the rat, have demonstrated a clear **parasympathetic innervation** to the adrenal medulla, there is no evidence, using the marmoset as a representative of primates, of parasympathetic innervation of the adrenal medulla in humans. It is true that both the cortex and medulla also contain **acetylcholinesterase positive axons** with some of these forming synapses with ganglion cells in the **zona fasciculata** and **reticularis**. It is also true the principal neurotransmitter for the parasympathetic system is **acetylcholine**. However, the first synapse in the sympathetic chain is mediated by nicotinic receptors, which are physiologically activated by acetylcholine as pointed out above.

The **afferent nerve supply** from the adrenal medulla is significant. The neuronal cell bodies of these afferent nerves lie within the **dorsal root ganglia** and the **vagal sensory (nodose) ganglia**.

E. Abdominal aorta: The aorta enters the abdomen in front of the lower part of the body of the twelfth thoracic vertebra, just to the left of the midline, by passing through the **aortic hiatus** of the diaphragm. It continues downward in the **retroperitoneal space** for 10 to 12 cm, ending in front of the fourth lumbar vertebra, slightly to the left of the midline, by dividing into the common iliac arteries. It measures about 2 cm in width as it passes through the aortic hiatus. It diminishes considerably in size after giving rise to the **celiac arterial trunk** and the **superior mesenteric artery**, and then it retains a fairly uniform caliber of 1.75 cm in width to its termination. The level of its termination in

the common iliac arteries is marked on the anterior surface of the abdomen as the **intercrestal line**, which is a line extending across the highest level of the iliac crests, usually crossing the L4/L5 disc, at a point 2 to 3 cm below and slightly to the left of the umbilicus (Figs. 46 & 48).

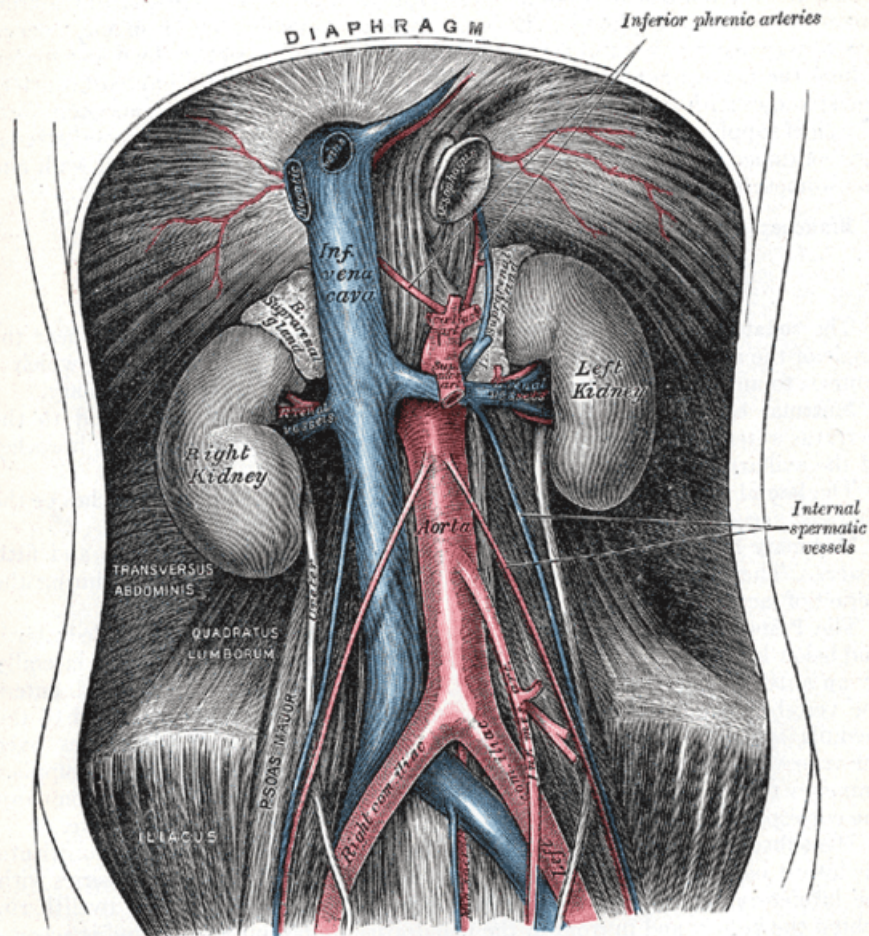


Fig. 48. This is an illustration of the abdominal aorta and its branches. (Wiki)

Posteriorly, the abdominal aorta rest on the lumbar vertebrae, the intervertebral discs, and the anterior longitudinal ligament. The left lumbar veins cross behind it to reach the inferior vena cava. In the superior part of the abdominal aorta, the azygos vein, the cisterna chyli and the thoracic duct, the right crus of the diaphragm, and the right celiac ganglion are to the right of it. In its inferior aspect the inferior vena cava is to the right (Fig. 48). On the left of the aorta are the left crus of the diaphragm, the left celiac

ganglion, and the left lumbar chain of nodes. It is crossed by the left renal vein (Fig. 48).

The branches of the abdominal aorta listed in the order in which they arise are: phrenic arteries, celiac trunk, middle adrenal arteries, first lumbar arteries, superior mesenteric artery, renal arteries, testicular (or ovarian) arteries, second lumbar arteries, inferior mesenteric arteries, third lumbar arteries, fourth lumbar arteries, middle sacral artery and the common iliac arteries (Figs. 49 & 50).

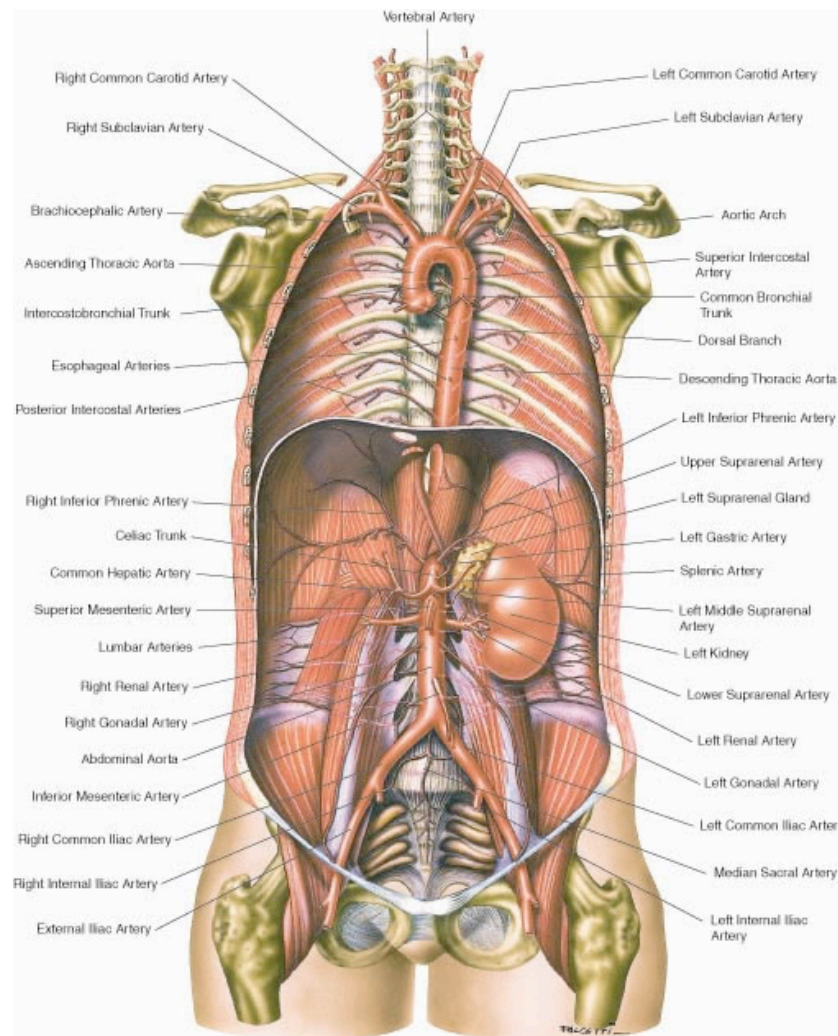


Fig. 49. This illustration shows the branches of the thoracic and abdominal aorta. (Wiki)

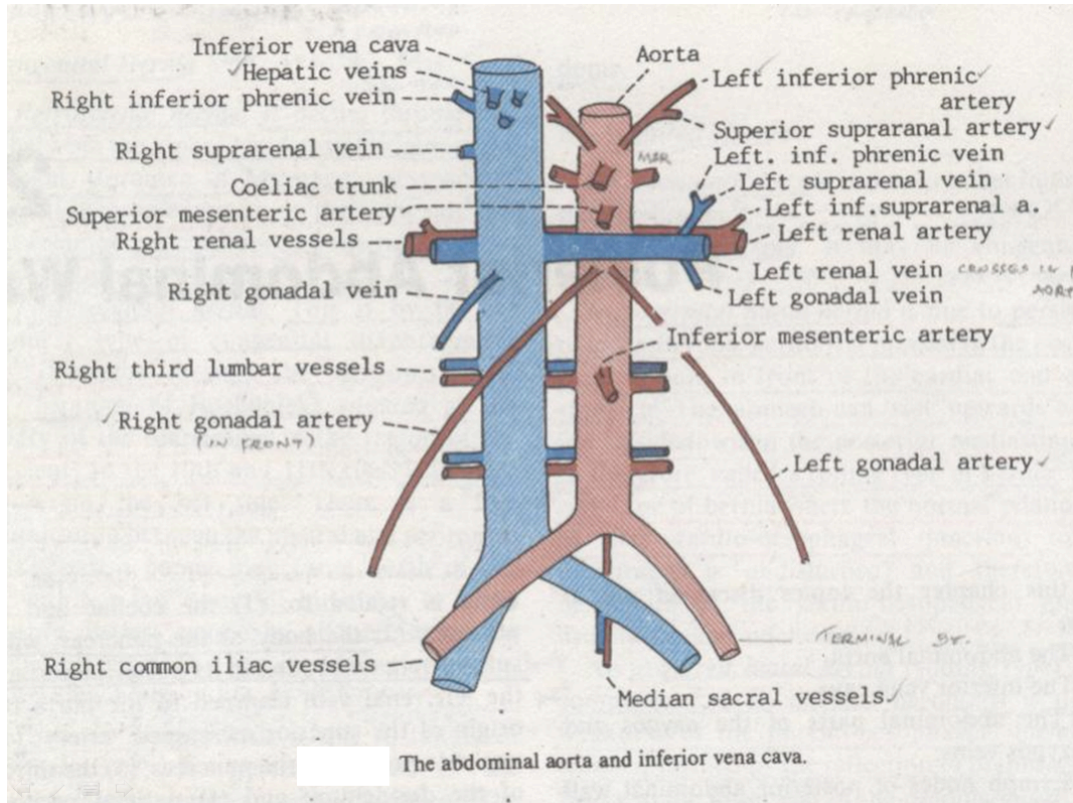


Fig. 50. This illustration shows the branches of the abdominal aorta and inferior vena cava. (Wiki)

Regarding the neural regulation of blood flow in the aorta, we will discuss in general the neural regulation of the circulation.

The nervous system controls the circulation almost entirely through the **autonomic nervous system (ANS)**. The most important component of the ANS in the control of circulation is the **sympathetic nervous system**. The other component of the ANS, the **parasympathetic nervous system**, helps regulate the circulatory system through its regulation of heart function.

In most tissues all vessels are innervated by the sympathetic nervous system except the capillaries. The innervation of the small arteries and arterioles allows sympathetic stimulation to increase resistance to blood flow and thus, decrease the rate of blood flow through the tissue.

The innervation of the medium and large sized arteries and veins, makes it possible for sympathetic stimulation to decrease the volume of these vessels.

Concerning the abdominal aorta, it receives its sympathetic innervation primarily from L1 and L2. Stimulation of these sympathetic nerves leads to decrease in the diameter of the abdominal aorta. This reduction in diameter is through the contraction of the smooth muscle in the wall of the abdominal aorta.

F. Inferior vena cava: The inferior vena cava is formed anterior and to the right side of the fifth lumbar vertebra 2.5 cm below the **supracristal plane**. The supracristal plane is an anatomical transverse plane at the upper most part of the pelvis, the iliac crest. It is usually at the level of the L4 vertebra. The supracristal plane passes through the umbilical region and the left and right lumbar regions (Fig. 51).

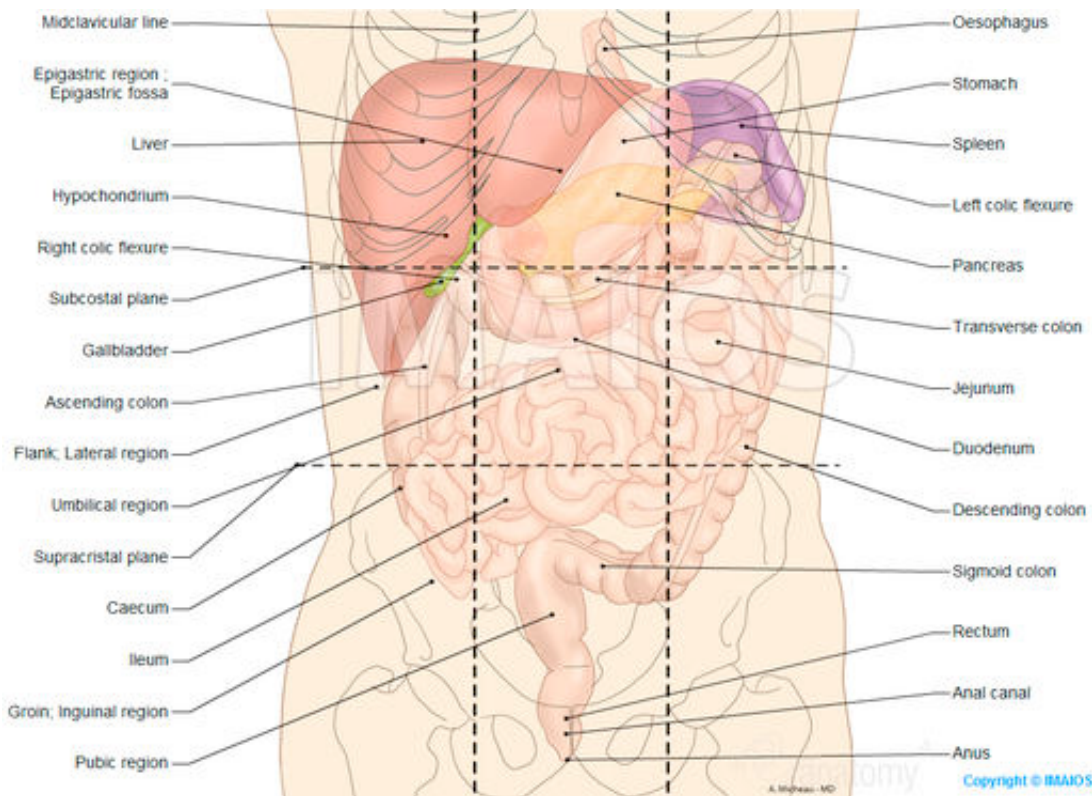


Fig. 51. This illustration denotes the nine anatomic surface regions and the contained organs within each region as well as the various planes of the body including the supracristal plane. (imaios.com) (Wiki)

The inferior vena cava is formed by the junction of two **common iliac veins**, which collects venous blood of the lower limbs and the non-portal blood of the abdomen and pelvis (Figs. 48 & 50). The portal blood is that blood, which is carried by the **portal**

vein. This is venous blood from the gastrointestinal tract, except the lower part of the anal canal, from the spleen, and from the pancreas.

The vena cava is approximately 20 cm in length, ascending in the **retroperitoneal space** 2.5 cm to the right of the midline. At the level of the liver it is contained within a deep groove on its posterior surface. It passes through the diaphragm in its most anterior opening, between its median and right 'leaves', then passing through the pericardium posteriorly to enter the inferior posterior part of the right atrium (Fig. 52).

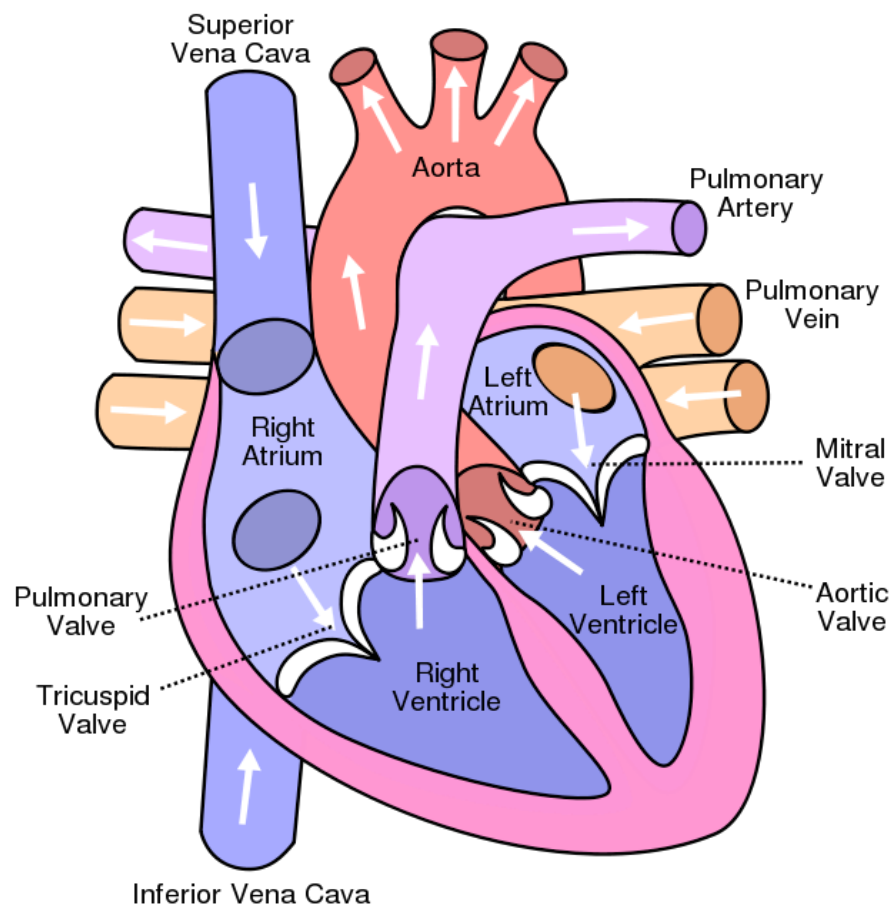


Fig. 52. This illustration shows the entrance of the inferior and superior vena cava into the heart. (Wiki)

In the **retroperitoneal space** it lies to the right of the abdominal aorta (Figs. 48 & 50), the right crus of the diaphragm, and the caudate lobe of the liver. It begins below and posterior to the right common iliac artery, but is more anteriorly placed at the level of the kidneys where the left renal vein crosses the aorta anteriorly (Figs. 48 & 50).

The inferior vena cava receives veins, which correspond with the arterial branches of the abdominal aorta except for those veins, which drain into the portal vein. From below upward, the inferior vena cava receives the lumbar veins, the right testicular (or ovarian) veins, the renal veins, the right suprarenal vein, the right phrenic vein, the anterior portion of the left phrenic vein and the hepatic veins. It also receives a few small tributaries, including one or two direct from the ureter. The left phrenic vein is often double. Its posterior portion joins with the left suprarenal vein to become a tributary to the left renal vein. The anterior portion of the left phrenic vein passes in front of the esophageal hiatus to empty into the inferior vena cava (Fig. 53).

The **innervation** of the inferior vena cava has been discussed in a general sense under the innervation of the abdominal aorta.

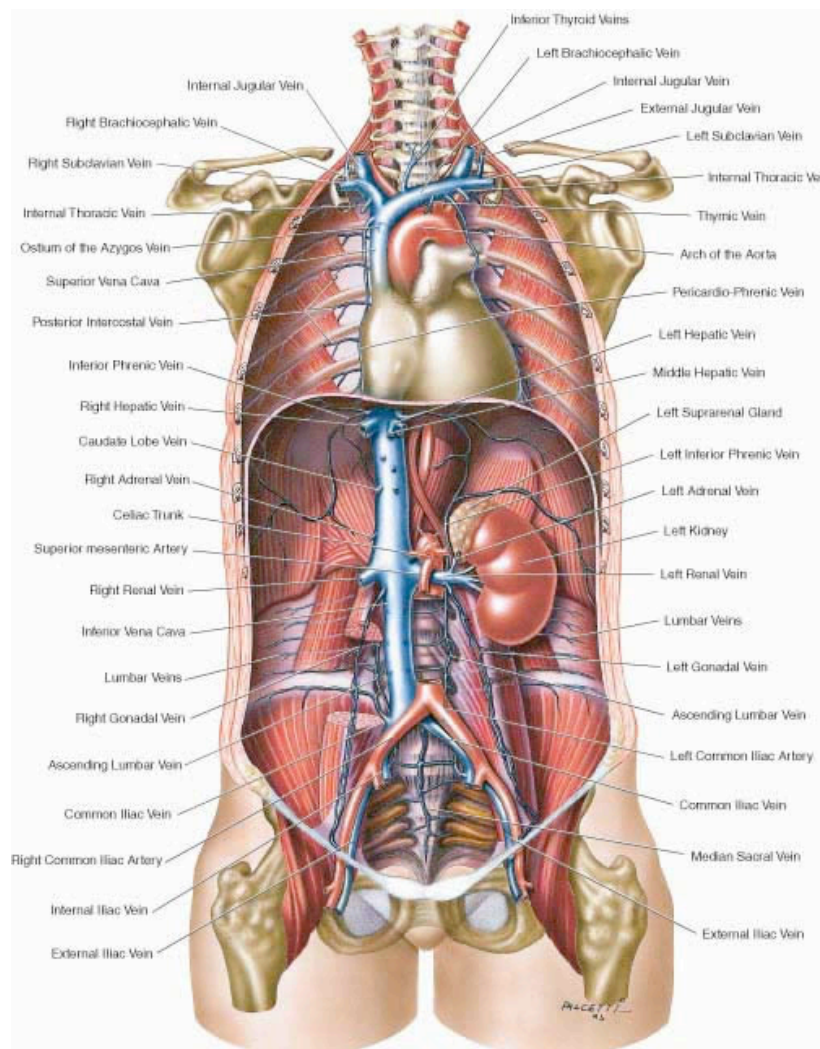


Fig. 52. The above illustration depicts the veins which drain into the inferior vena cava. (Wiki)

IV. Traumatic Injuries of the Organs of the Retroperitoneal Space of the Abdomen: Adult and Pediatric

A. General Comments: Retroperitoneal injuries occur in approximately 12% of those cases involving trauma to the abdomen. It is not uncommon for physical examinations, laboratory test, peritoneal lavage, focused ultrasonography and conventional CT scans to fail to detect evidence of retroperitoneal injury even when it is significant. However, the introduction of multi-detector CT scan has markedly changed the clinicians ability to diagnose even subtle retroperitoneal injuries. The multi-detector CT scan provides a high degree of accuracy when used to evaluate trauma cases with a reported sensitivity approaching 100% for the identification of active bleeding, as well as bowel, mesenteric, kidney, adrenal gland and pancreatic injuries in the initial assessment of traumatic abdominal injuries.

B. Overview of Mechanisms of Blunt Force Injury to the Retroperitoneal Space:

Typically, the mechanism of blunt impact injuries to the organs of the abdomen is attributed to “crush,” which is an oversimplification of the biophysics of traumatic injury to the chest or abdomen. Although, crush injury is one of the mechanisms of injury to the organs of the chest or abdomen, there are three other mechanisms, all of which involve the ‘propagation of waves’ (stress, shock, and shear waves) through the body. It is the motion of the chest or abdominal wall, which generates waves that propagate through the body and in the process transfer energy to internal sites, such as the organs of the retroperitoneal space. It is these waves of energy, which account for the pathology at sites within the abdominal cavity and the retroperitoneal space, which are some distance from the point of impact. Thus, what is perceived as a minor displacement of the body wall may produce serious injury in the retroperitoneal space if the impact to the abdominal wall was of high velocity. The biophysics of impact injury to the abdomen was discussed in considerable detail in the previous chapter, “Traumatic Injuries of the Organs of the Abdominal Cavity: Adult and Pediatric,” pages 89 to 92.

C. Pancreas: Pancreatic trauma is relatively uncommon, occurring in less than 5% of patients with major abdominal injuries, and typically representing a small portion of the extensive abdominal injuries. Rarely, it is the only major lesion. It has been suggested it is the tenth most injured organ compared to other organs (e.g., brain, spleen, liver, etc). The first reported case of pancreatic trauma was by Travers in 1827, with the diagnosis being made at autopsy. In 1903 Mikulicz was able to identify only 45 cases of pancreatic trauma reported in the literature. He observed that all 20 patients not operated on died, and 18 of 25 (72%) operated on survived. In 1930, Stern reviewed 62 reported cases, 30 of which were treated successfully, and 32 patients died.

Culotta *et al.* reported on wartime development of the treatment of pancreatic injuries in 1956. They found 5 cases from the Civil War with a 80% mortality; 5 cases from British Troops from World War I with a 80% mortality; 62 cases from World War II with a 56% mortality; and 9 cases from the Korean conflict with a 22% mortality. What must be kept in mind is that during the Civil War and World War I, treatment was nonoperative. The first reported case of successful end-to-end suture of a transected pancreas was reported by Newton in 1929, however, it was not until World War II that operative intervention as more of a routine for pancreatic trauma was introduced.

The incidence of pancreatic trauma in patients sustaining blunt or penetrating trauma is typically represented as being 1 to 2%. However, in 1971, Jones and Shires reported on a 20 year study, which showed an increasing frequency of pancreatic injuries. For example, the incidence of pancreatic injury reported in 1968 by DiVincenti *et al.* was 1.6% in a study that covered the period 1951 to 1966. However, Berni *et al.*, reported in 1982, the incidence of pancreatic injury to be 3.5% in a study that covered the period 1971 to 1981. It is believed the underlying reason for this increase is the more frequent motor vehicular accidents, as well as more frequent criminal behavior with the use of sharp pointed instruments, such as knives.

In 1989, Clancey published a review of pancreatic trauma in which he reviewed a total of 17 studies involving 1,984 patients. He found pancreatic injury due to penetrating trauma in 73% of cases and blunt force trauma in 27%. Associated injuries were given in 12 of the studies involving 1,606 patients. The associated injuries were as follows: liver (42%); stomach (40%); major vascular systems (35%); thorax (31%); colon and

small bowel (29%); spleen (25%); CNS, skeleton, and extremities (25%); and duodenum (18%). Nine of the studies showed one or more associated injuries in 58 to 98%.

There are two major types of pancreatic injury, **penetrating** and **non-penetrating**.

The two primary **penetrating injuries** are missile and stab wounds. The most common type of penetrating injury to the pancreas is a missile wound (Fig. 53). Typically, with missile injuries, the missile also passed through the liver, duodenum, or stomach before perforating or penetrating the pancreas. Although, it would be unusual for the pancreas to sustain singularly a missile or stab wound, such wounds are likely to be accompanied by life threatening bleeding due to the proximity of major vascular structures to the pancreas. For example, the body of the pancreas overlies the aorta, inferior vena cava, renal veins and the right renal artery. The **splenic vein** courses along the posterior surface of the body of the pancreas before joining the **superior mesenteric vein** to form the **portal vein** behind the neck of the pancreas. The superior border of the body of the pancreas contains the **splenic artery** (Figs. 16, p 17 & 54). The inferior border of the pancreas contains or is in proximity to the **inferior pancreatic artery**.

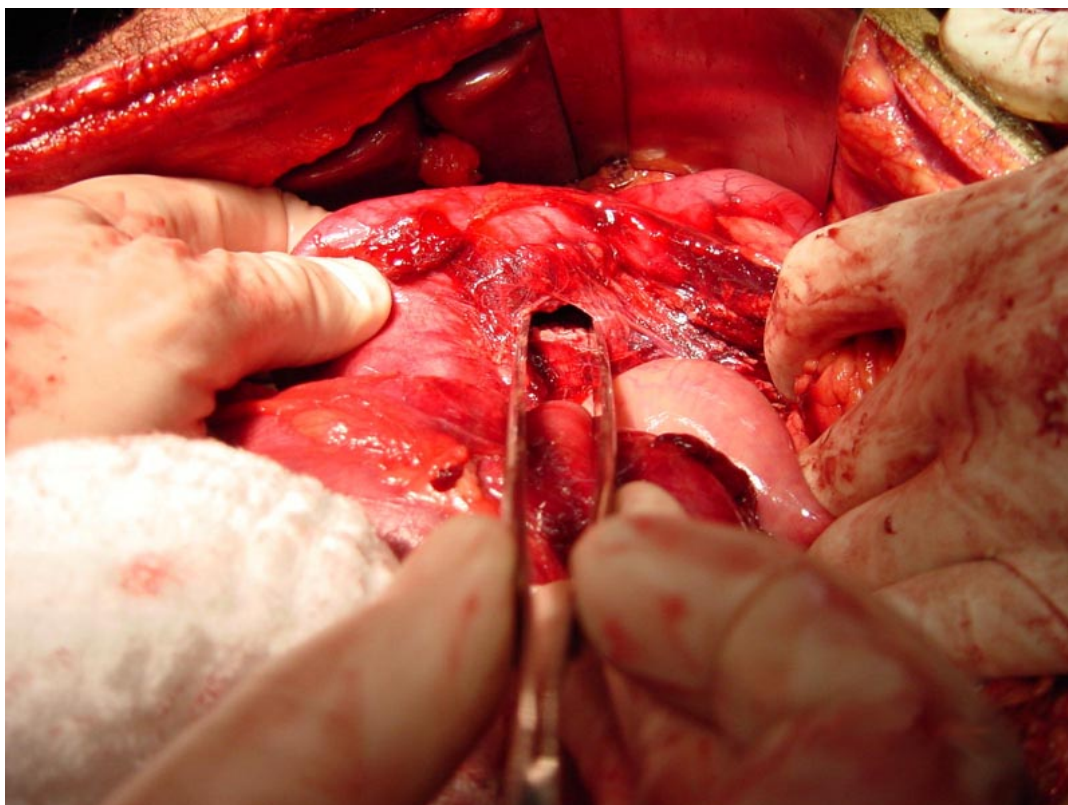


Fig. 53. The above is a gunshot wound of the head of the pancreas. (Andreal Amatuzzi, University Hospital of Cajuru, Brazil, Trauma.Org) (Wiki)

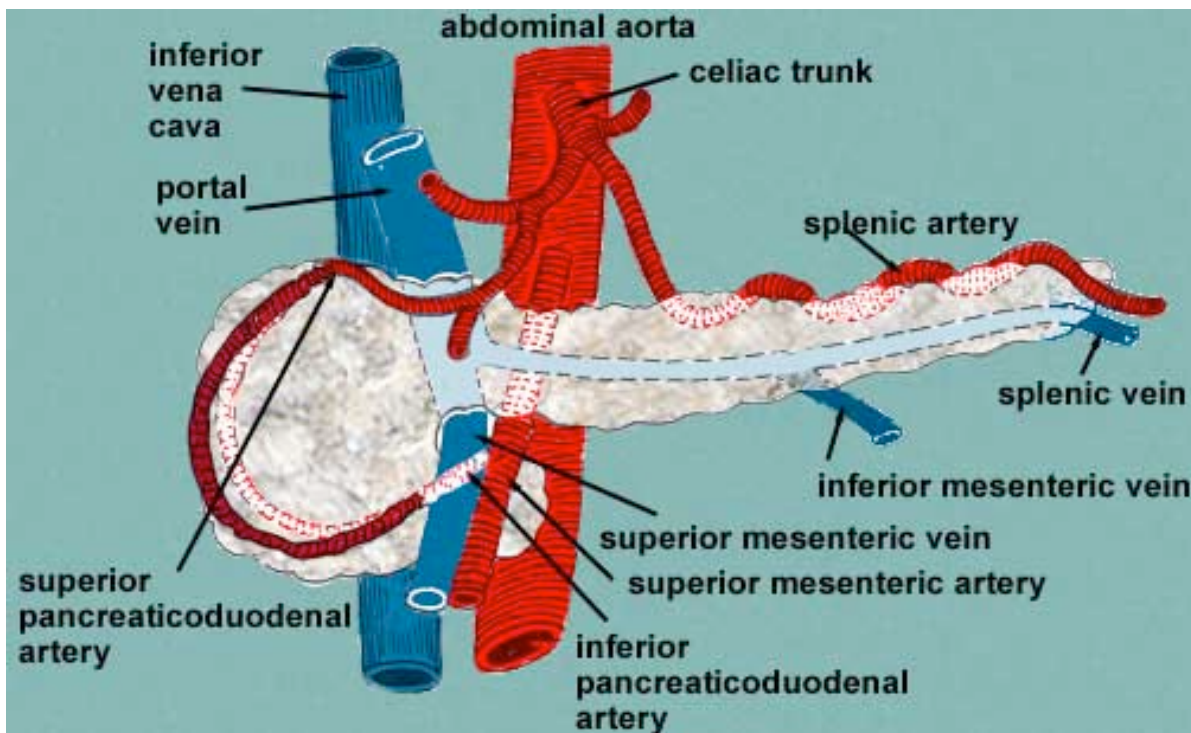


Fig. 54. This illustration shows the proximity of many major blood vessels to the pancreas. (Wiki)

There are occasions in which the victim may survive the immediate effects of a missile or stab wound, but subsequently die due to the development of a peritonitis, which may be generalized or localized to the lesser omental bursa. Typically, the peritonitis is associated with chalky white precipitates (plaques) of fat necrosis due to fatty acids saponifying with alkaline salts to form soaps. This type of tissue destruction is technically referred to as enzymatic fat necrosis, which is due to the escape of pancreatic secretions, and is virtually diagnostic of acute hemorrhagic pancreatic necrosis (Figs. 55 & 56). If not appropriately treated these penetrating injuries of the pancreas can be followed by the development of intraabdominal or extraabdominal (enterocutaneous) fistula.

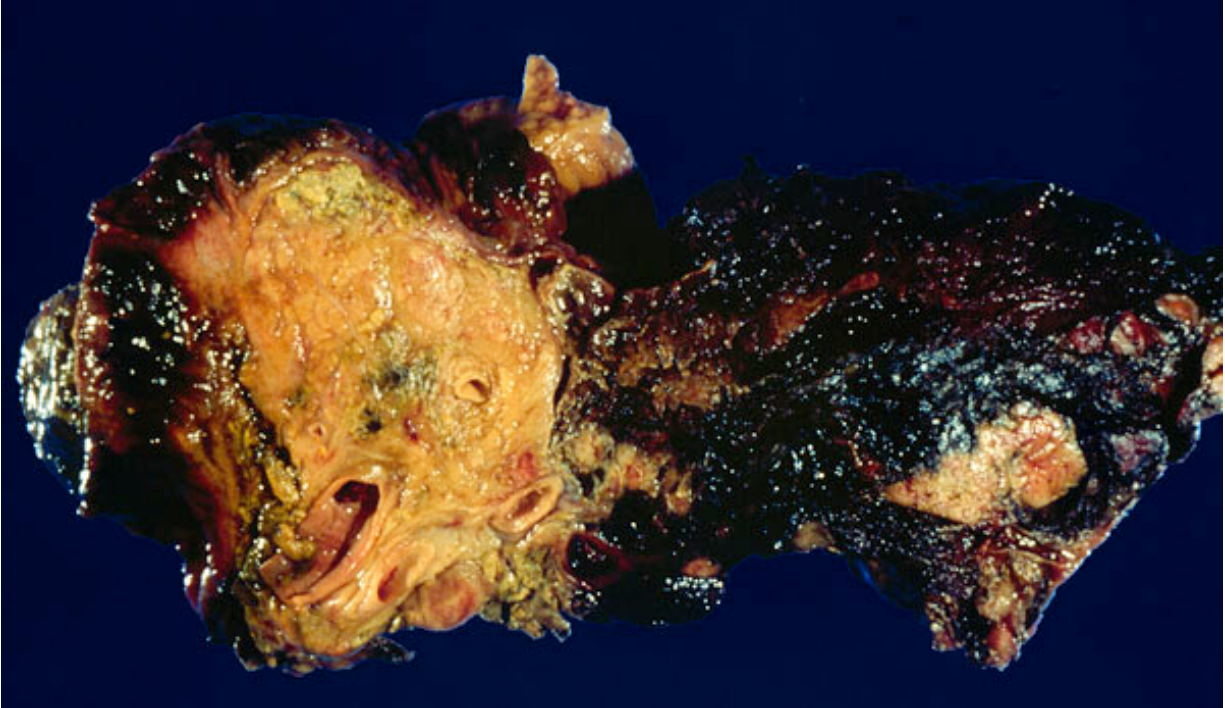


Fig. 55. This is an example of hemorrhagic pancreatitis. The black area is hemorrhage and the yellow is fat necrosis. Cellular injury to the pancreatic acini leads to the release of powerful enzymes, which damage pancreatic and peripancreatic fat leading to the production of soaps, which appear as the soft, chalky white areas seen in the above photograph. (uaz.edu.mx) (Wiki)

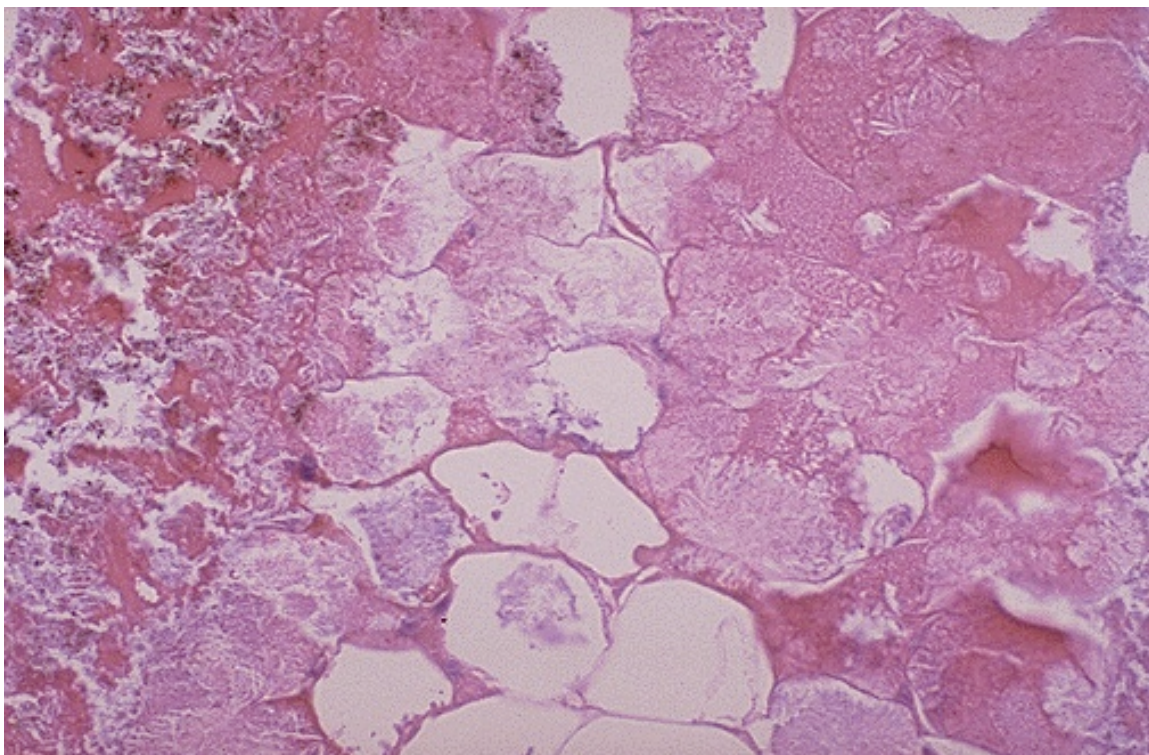


Fig. 56. The above is a photomicrograph of fat necrosis next to the pancreas (peripancreatic). There are some remaining steatocytes (fat cells) in the left side of the image, which are not necrotic. The necrotic fat cells are on the right. They have vague cellular outlines, have lost peripheral nuclei, and their cytoplasm has become a pink amorphous mass of necrotic material. (Wiki)

Non-penetrating injuries of the pancreas include **lacerations, contusions** and **transections**. The retroperitoneal location of the pancreas protects it in most instances of blunt force abdominal trauma. Typically, blunt force injuries to the pancreas are the result of direct impact or deceleration injury, and are often associated with injuries to the liver, spleen, duodenum, stomach, kidneys, and the adrenal glands. Although, blunt force traumatic injury limited to the pancreas is uncommon, such injuries do occur, and may not show any evidence of contusions or other injuries to the abdominal wall. Non-penetrating pancreatic injury usually results from severe anterior-to-posterior force vectors, compressing the head of the pancreas against the second or third lumbar vertebrae, with injury typically occurring to the left of the superior mesenteric artery and vein. In adults, transection of the pancreas typically occurs in line with the superior mesenteric vein at the neck of the pancreas.

The pathophysiology that results from a severe localized force applied to the epigastrium is the result of substantive crush effect, which leads to intraparenchymal hemorrhage with disruption of the pancreatic parenchyma and ductal system. This crush effect causes the leakage of pancreatic enzymes into the disrupted parenchyma with its subsequent digestion as well as the adjoining tissues. This leads to a collection of fluid containing pancreatic enzymes, hemolyzed blood and necrotic debris, which in turn incites a severe inflammatory reaction in the adjoining tissue. It is this inflammatory reaction which leads to encapsulation of the collection of fluid by fibrous tissue to form a cyst. This typically takes 4 to 6 weeks. In approximately 50% of these cyst there is a communication with a pancreatic duct. Since such cyst lack an epithelial lining and instead are lined by fibrous tissue with scattered plaques of hemosiderin embedded in the fibrous lining, the prefix “pseudo” is applied to the cyst, hence the name “pseudocyst” (Fig. 57).

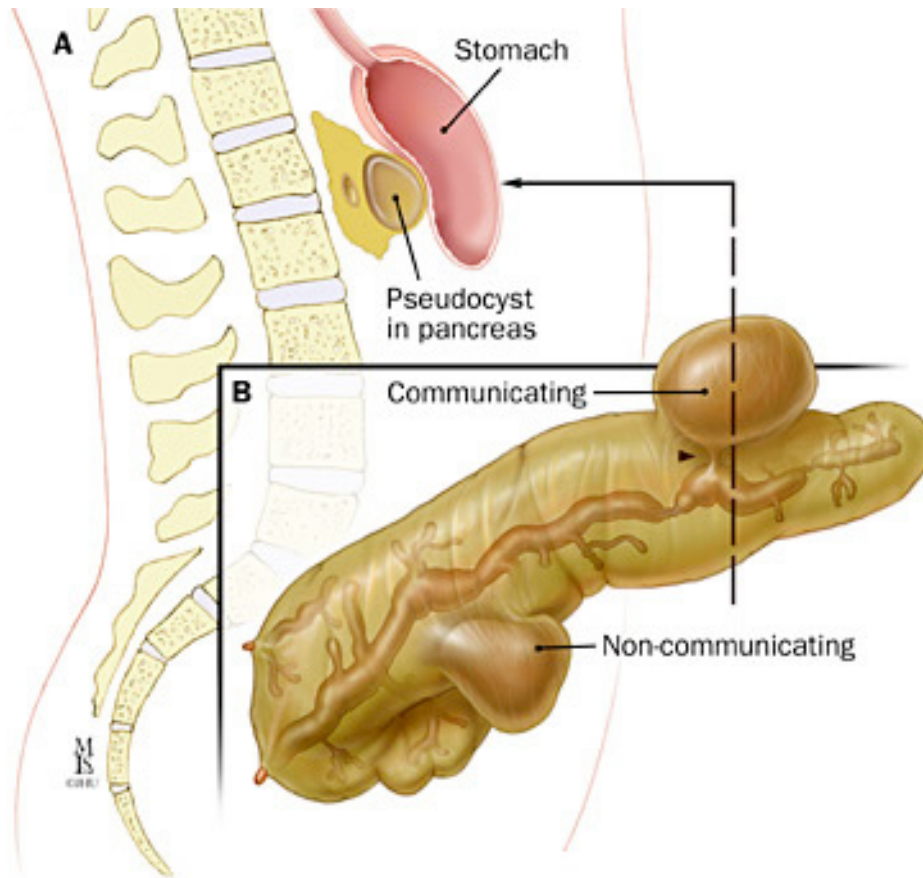


Fig. 57. The above is an illustration of a pseudocysts in the pancreas. A. shows the compression of the adjacent stomach as the pseudocyst increases in size. B. shows two pseudocyst, the upper is referred to as communicating in that it is connected to a pancreatic duct, the lower is not connected to a duct, hence, it is noncommunicating. (Wiki)

Pseudocyst account for approximately 75% of cysts of the pancreas. Pseudocyst are usually solitary, although they can be multiple and may be found in the parenchyma of the pancreas or, more commonly, involve the lesser omental bursa or lie in the retroperitoneum between the stomach and liver, or between the stomach and the transverse colon. They also can be found in the subdiaphragmatic position. Having said this, it is important to remember they can be found anywhere from the groin to the mediastinum and even the neck, having ascended in the retroperitoneum through the diaphragmatic hiatus into the mediastinum. Typically these cyst range in size between 2 to 30 cm. If the pseudocyst are small (less than 4 to 6 cm) they can resolve spontaneously. It is believed approximately 50% of all pseudocyst resolve

spontaneously and thus are managed conservatively. However, the larger pseudocyst will require some form of treatment due to the potential of complications (Figs. 57, A & 58).



Fig. 58. This is a photograph taken at the time of surgery to treat a pseudocyst, which is the large dark cavity immediately to the right of the center. Pancreatic pseudocysts are usually a complication of pancreatitis (Fig. 59), although in children they frequently occur following abdominal trauma. Men are affected 5 to 10 times more than women. (Wiki)

Pseudocyst can rupture leading to shock as well as hemorrhage. They can become super-infected leading to an abscess and ultimately sepsis. If they are particularly large they can compress other organs. Although our discussion has centered on the induction of pseudocyst due to trauma, pseudocyst usually arise in a setting of **acute pancreatitis** often in the background of chronic alcoholic pancreatitis, however, in children they frequently follow abdominal trauma (Fig. 59).

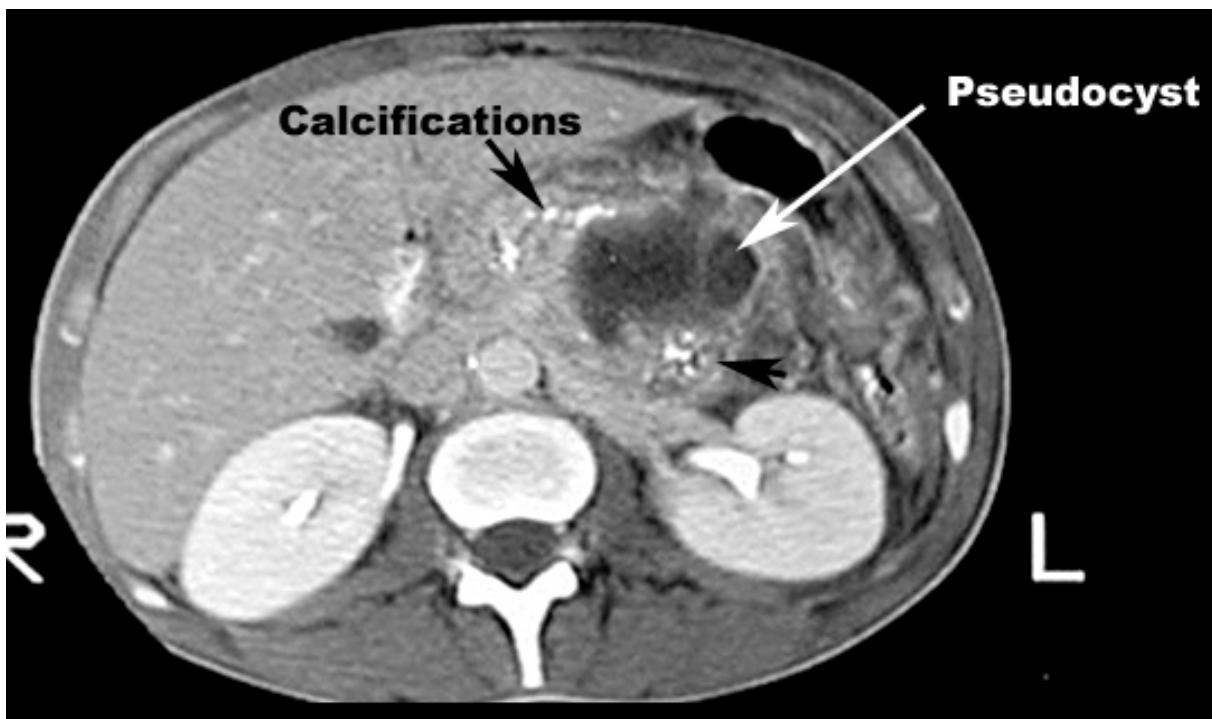


Fig. 59. The above is a CT scan of a patient with chronic pancreatitis with pseudocyst (white arrow) and calcifications (black arrow). (Wiki)

As has been implied, not all cyst in the pancreas are pseudocyst. There are also **congenital cyst**, and **neoplastic cyst**. **Congenital cyst** are believed to be the result of anomalous development of the pancreatic ducts. These pancreatic cyst may be associated with cysts in the kidney and liver, and thus part of the pathological picture of **polycystic disease**. These cyst are usually unilocular, thin-walled, and range from microscopic lesions to 5 cm in diameter. They are lined by cuboidal epithelium or, if the intracystic pressure was high, by flattened epithelium. They are enclosed by a thin fibrous capsule, without evidence of hemosiderin laden macrophages, and are filled with clear serous fluid and not hemorrhagic necrotic fluid often rich in pancreatic enzymes as in pseudocyst Figs. 60, 61, & 62).

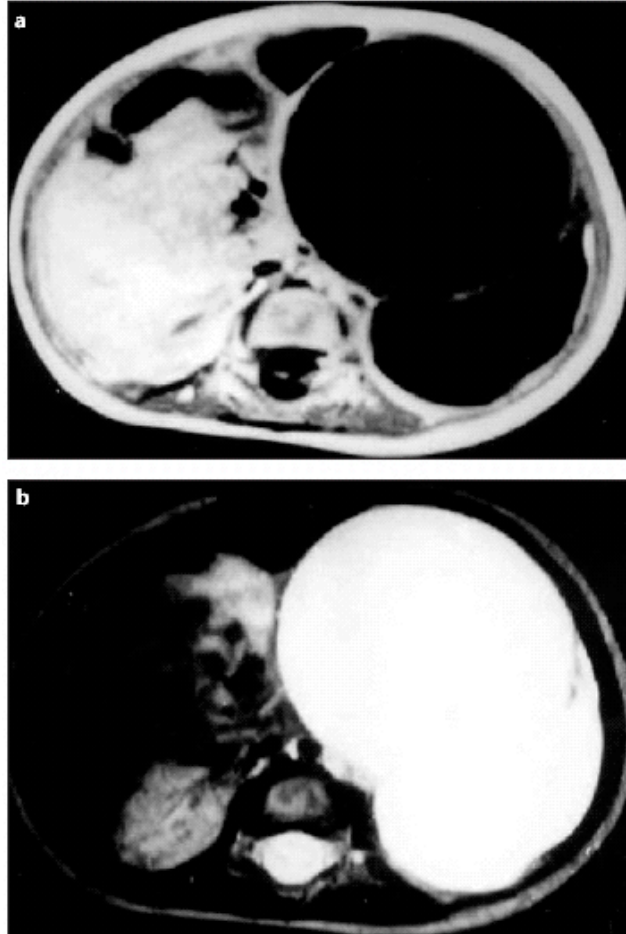


Figure 1. a, b. Transverse T1- (a) and T2-weighted (b) MR images show a lobulated mass in the left upper quadrant of the abdomen, hypointense on T1- and hyperintense on T2-weighted sequences.

Fig. 60. This is a CT of a pancreatic cyst seen in a newborn girl. (Geneve Foundation for Medical Education) (Wiki)

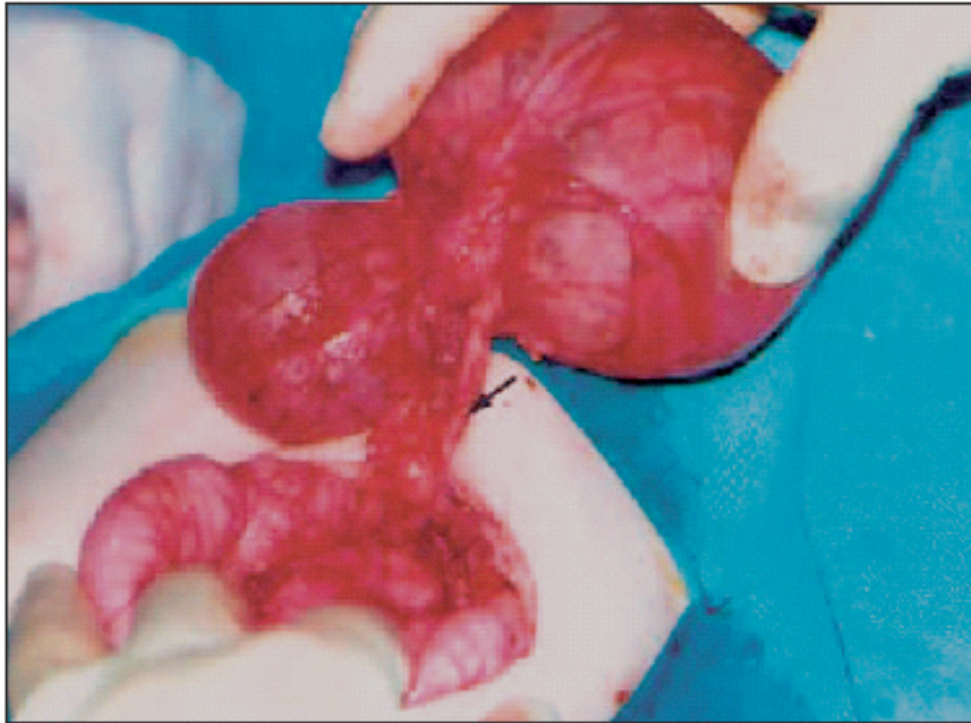


Figure 2. During the surgery, a bilocular cystic mass showing continuity towards the tail of the pancreas was observed (*arrow: tail of the pancreas*).

Fig. 61. This is the cyst seen of CT in Fig. 60. (Geneve Foundation for Medical Education) (Wiki)

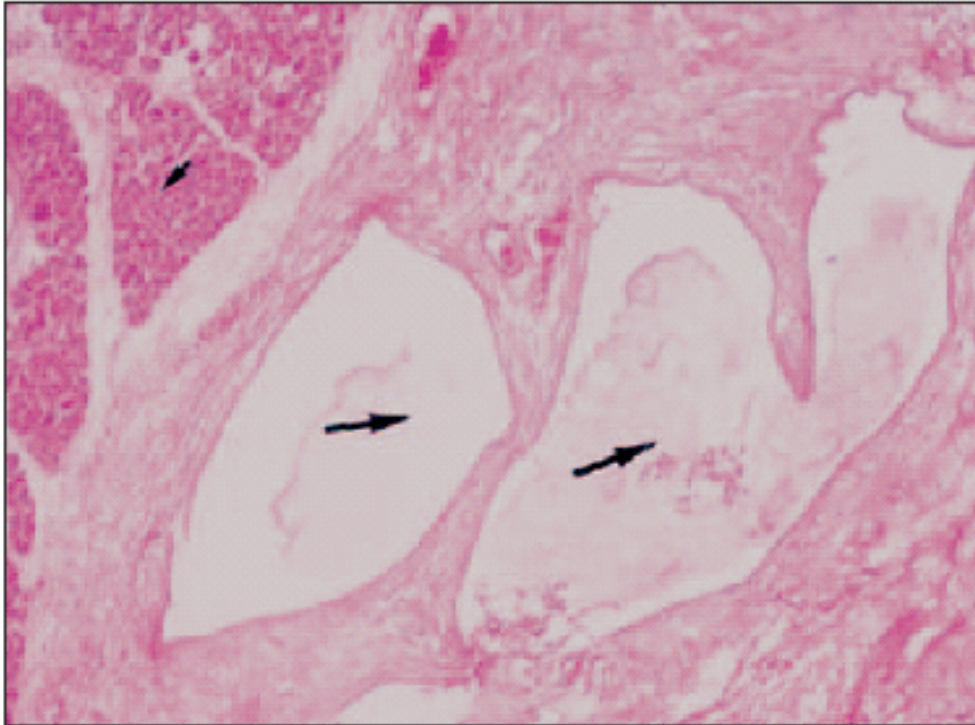


Figure 3. Normal pancreatic tissue (*small arrow*) and cystic areas (*large arrows*) coated with cuboidal epithelial tissue in various sizes were observed during histopathological examination (HE x40).

Fig. 62. This is a photomicrograph of the cyst seen in Figs. 60 and 61. (Geneve Foundation for Medical Education) (Wiki)

Neoplastic cyst represent approximately 5 to 15% of all pancreatic cyst. Cystic neoplasms constitute less than 5% of all pancreatic neoplasms. Some of these neoplastic cyst are benign, such as the **serous cystadenoma**, although there is a rare serous microcytic carcinoma, whereas cyst, such as a **mucinous cyst**, can be benign

(**mucinous cystadenoma**) (Figs. 63 & 64) or malignant (**mucinous cystadenocarcinoma**).

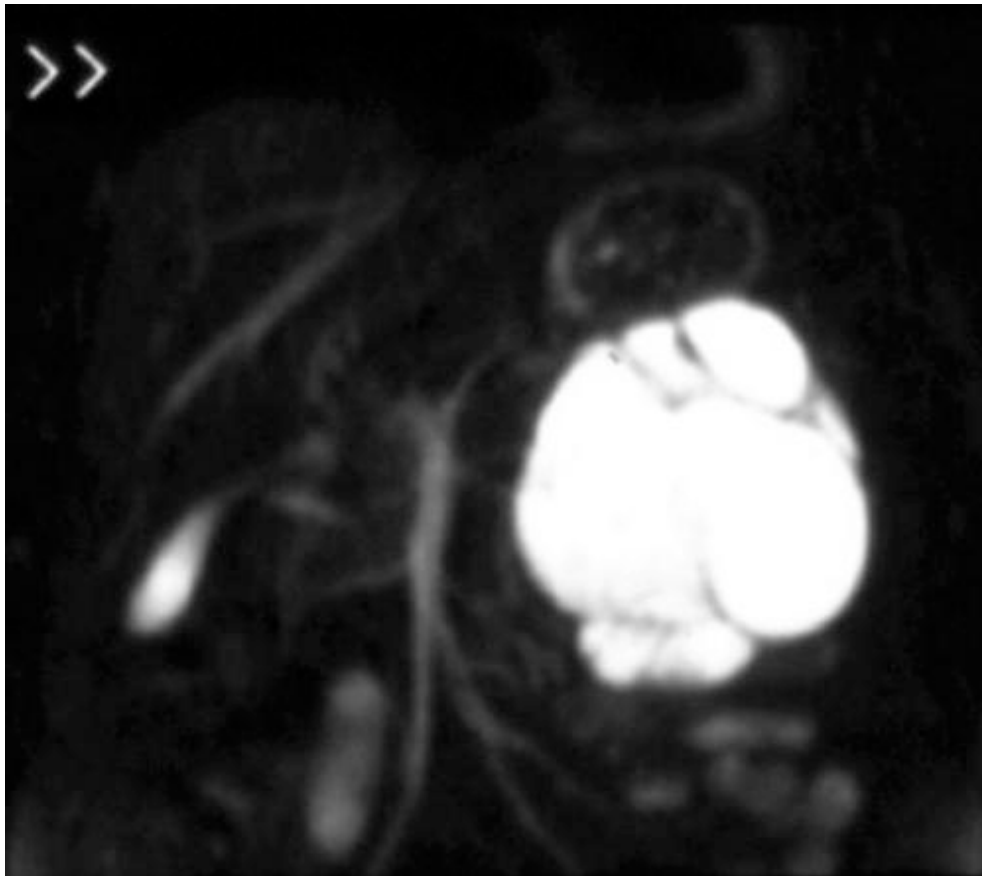


Fig. 63. This is a CT scan of a mucinous pancreatic neoplasm showing a macrocystic pancreatic mass with cystic components located in the tail. (Alfonso Ragozzino, Mariano Scalione, Department of Radiology, Cardarelli Hospital, Napoli, Italy) (Wiki)

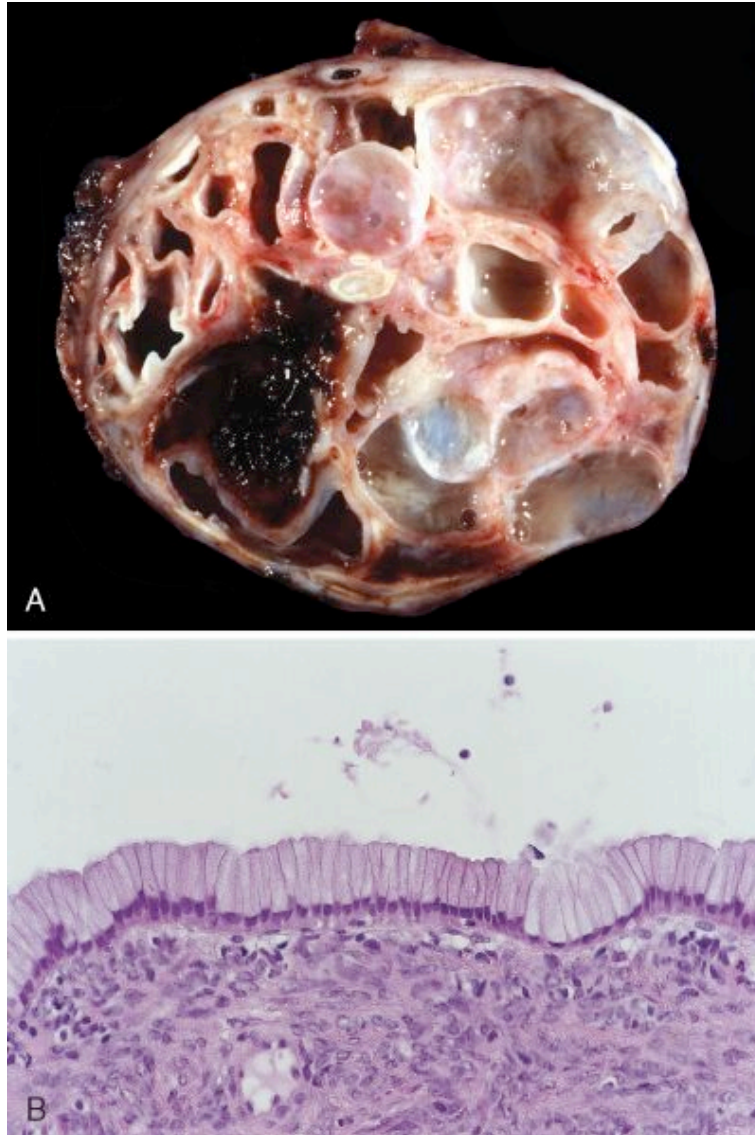


Fig. 64. The image in A represents a cross-section through a mucinous multiloculated cyst in the tail of the pancreas. The cysts are large and filled with tenacious mucin. B, represents a photomicrograph of the cysts, which shows they are lined by columnar mucinous epithelium, with an underlying dense stroma. (akramania.byethost11.com) (Wiki)

Lacerations of the pancreas often cause profuse bleeding due to the proximity of arteries and veins, which have been described above (Figs. 16, p 17 & 54). Such hemorrhage is copious, extending into the retroperitoneal space and often from there into the peritoneal cavity through the foramen of Winslow. The foramen of Winslow

(epiploic foramen) is the passage of communication between the greater omental bursa (peritoneal space) and the lesser omental bursa, which is anterior to the pancreas and duodenum, but posterior to the stomach (Figs. 65 & 66). It provides an avenue of retroperitoneal bleeding to enter the peritoneal cavity.

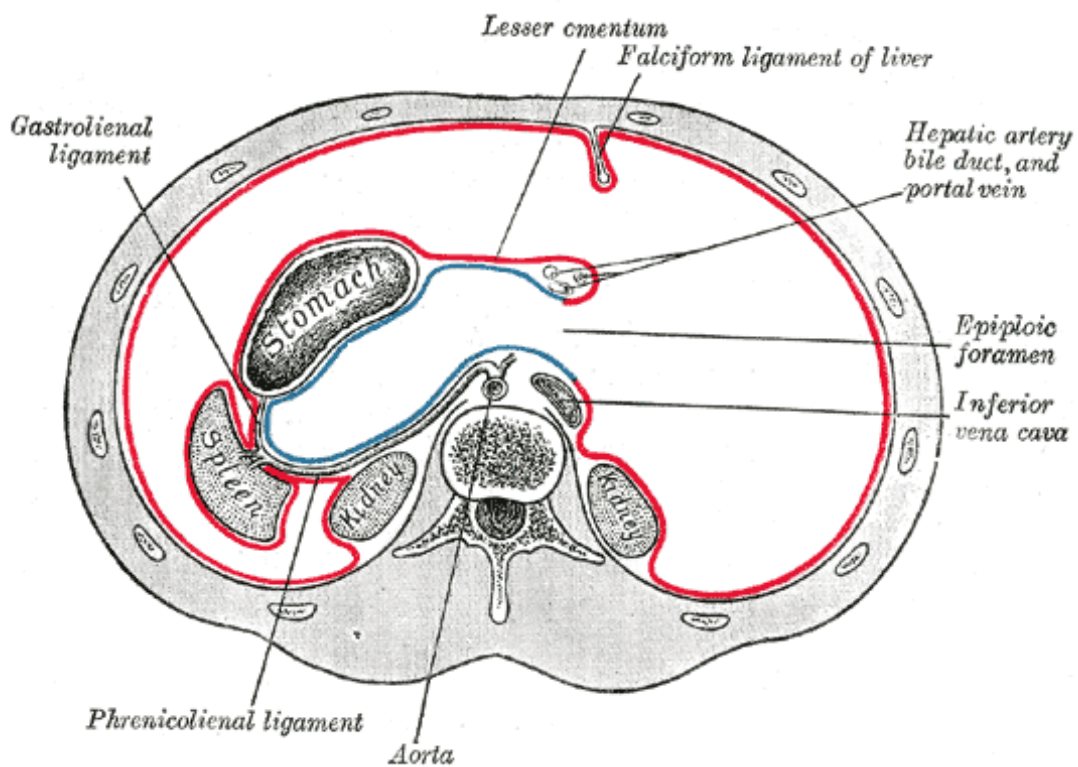


Fig. 65. The above is an illustration showing the location of the Epiploic foramen (foramen of Winslow). The cavity lined by the blue line is the lesser omental bursa. The cavity lined in red is the peritoneal cavity. (Wiki)

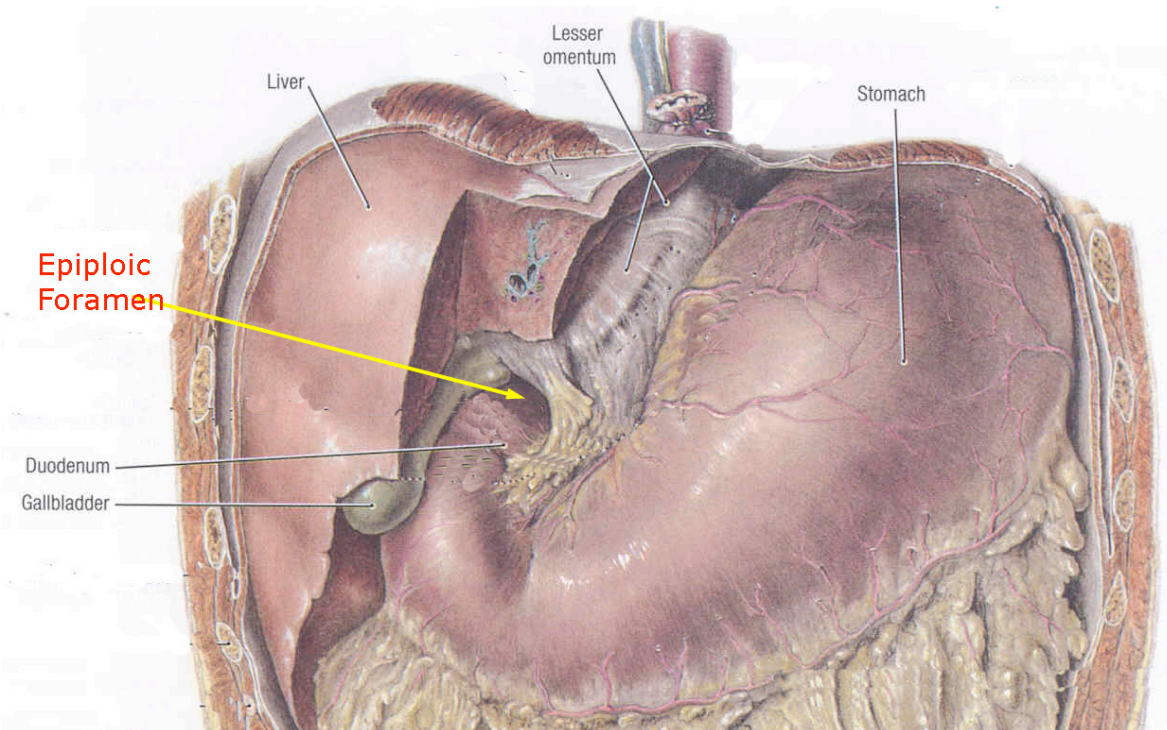


Fig. 66. This is another illustration showing the location of the Epiploic foramen. (Wiki)

If the laceration involves the pancreatic duct system (Figs 67, 68 & 72), it can lead to the development of acute pancreatitis and fat necrosis (Figs. 55 & 56) followed by chemical peritonitis (Figs. 69 & 70). What should be understood is severe pancreatic injury, either penetrating or non-penetrating, can result in mesenteric fat necrosis and a chemical peritonitis. The fat necrosis may remain localized to the retroperitoneal space near the pancreas. However, the peritonitis may be generalized or limited to the lesser omental bursa (Figs. 65 & 66).

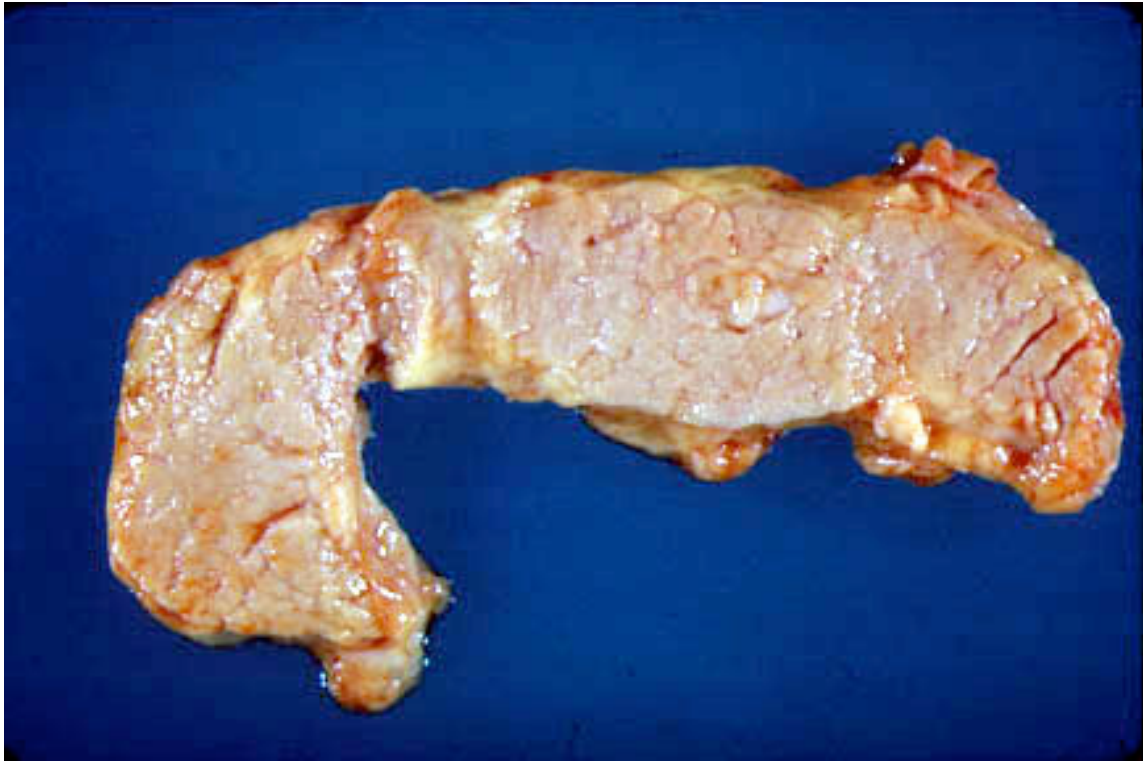


Fig. 67. This image shows a laceration involving the tail of the pancreas. (Wiki)

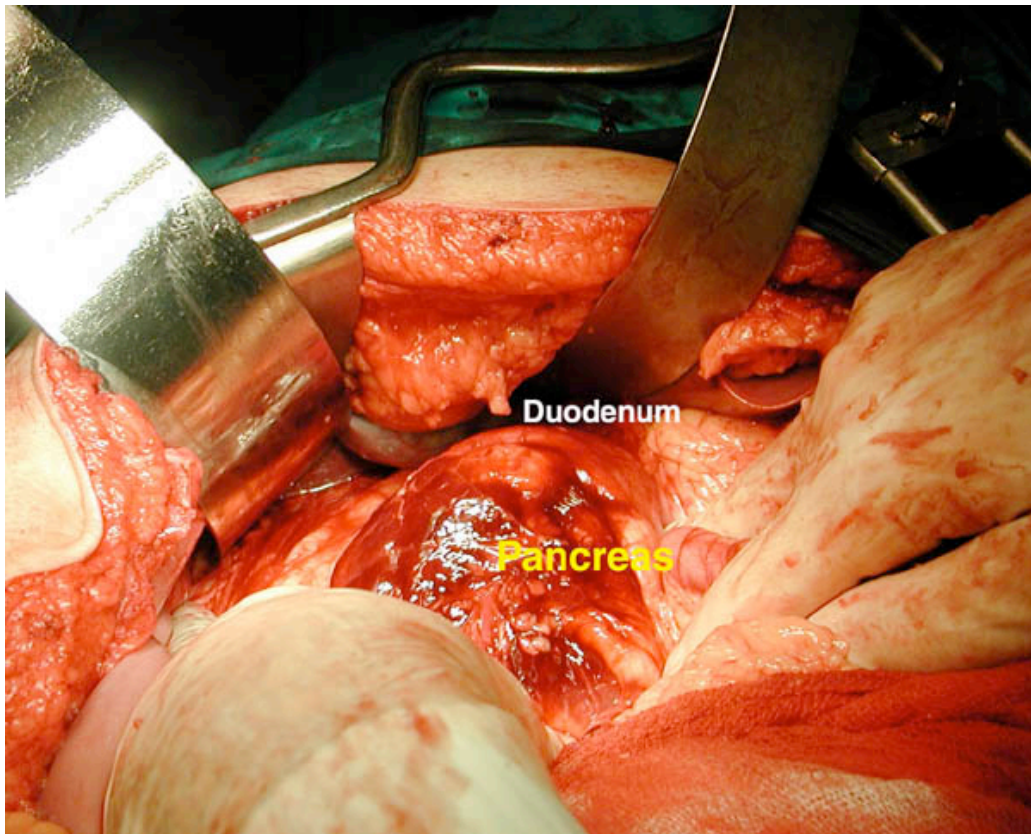


Fig. 68. The above is a photograph taken at the time of surgery on a male car crash victim with closed abdominal trauma from the steering wheel. He sustained a duodenopancreatic trauma with transection of the uncinete process of the pancreas, as well as a longitudinal disruption of the superior mesenteric vein. (Trauma.org, Karim, London, UK, June 17, 2008) (Wiki)



Fig. 69. The above is a picture taken at the time of autopsy of a patient who died with a chemical peritonitis. (Wiki)

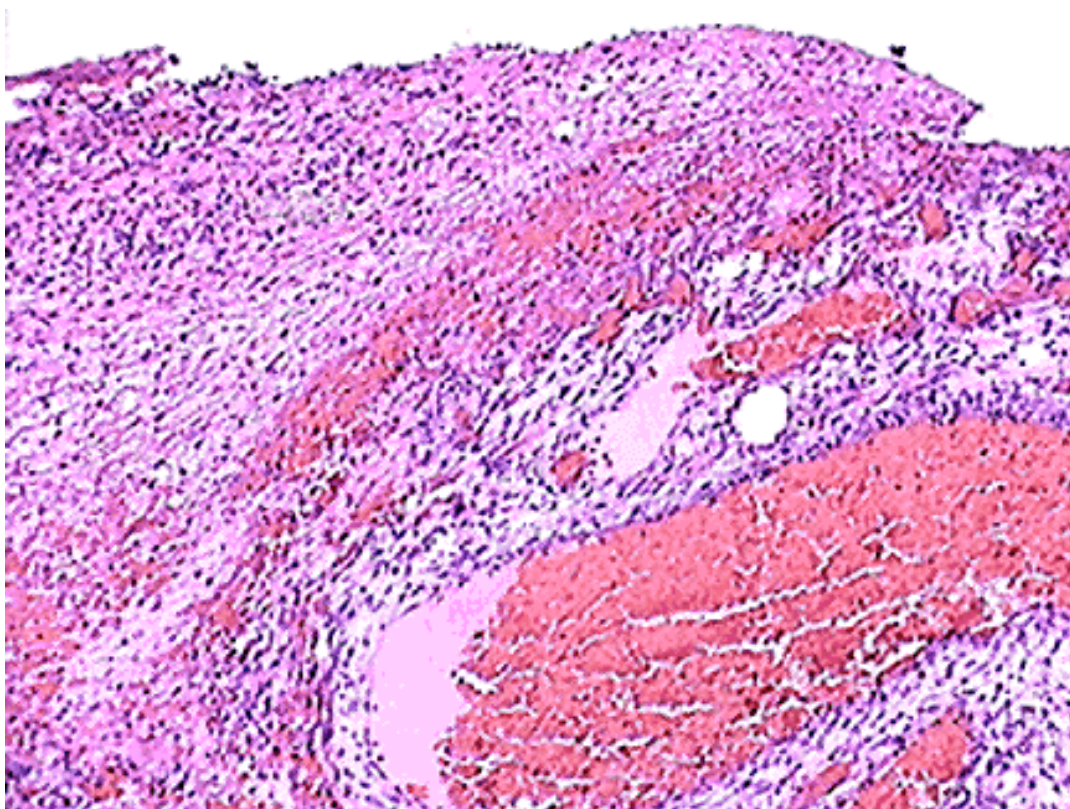


Fig. 70. The above is a photomicrograph of a patient with an acute peritonitis. It shows a prominent acute inflammatory infiltrate with markedly congested vessels. (Image Contrib. by: UCHC) (Description by: T.V. Rajan, M.D.) (Wiki)

Contusions of the pancreas may be focal or diffuse (Figs 71 & 72). Such contusions may lead to thrombosis of the superior mesenteric and splenic arteries or veins causing pancreatic and splenic infarcts. In 1958, McKay, Baggenstois and Wollaeger reported on 32 cases of grossly visible infarcts of the pancreas, five of which involved one-quarter or more of the gland. They demonstrated that this is a distinct entity from acute pancreatitis, with nearly all the instances being secondary to vascular disease, most often polyarteritis nodosa or malignant hypertension. In two of their cases large infarcts were caused by thrombosis of the superior mesenteric and splenic arteries (Fig. 73).

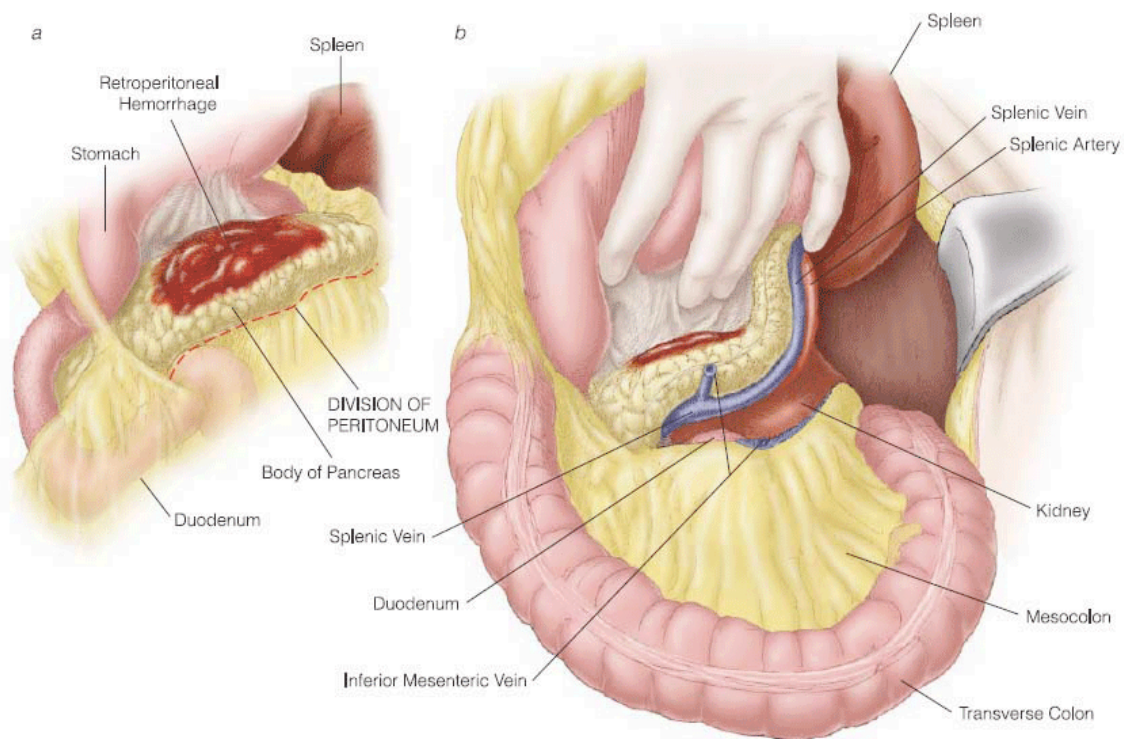


Fig. 71. This illustration shows a contusion of the body of the pancreas. Shown is the appearance of a contusion of the posterior surface of the body of the pancreas overlying the vertebral column (a). The second part of the illustration (b) shows the anterior surface of the body of the pancreas. (acssurgery.com) (Wiki)

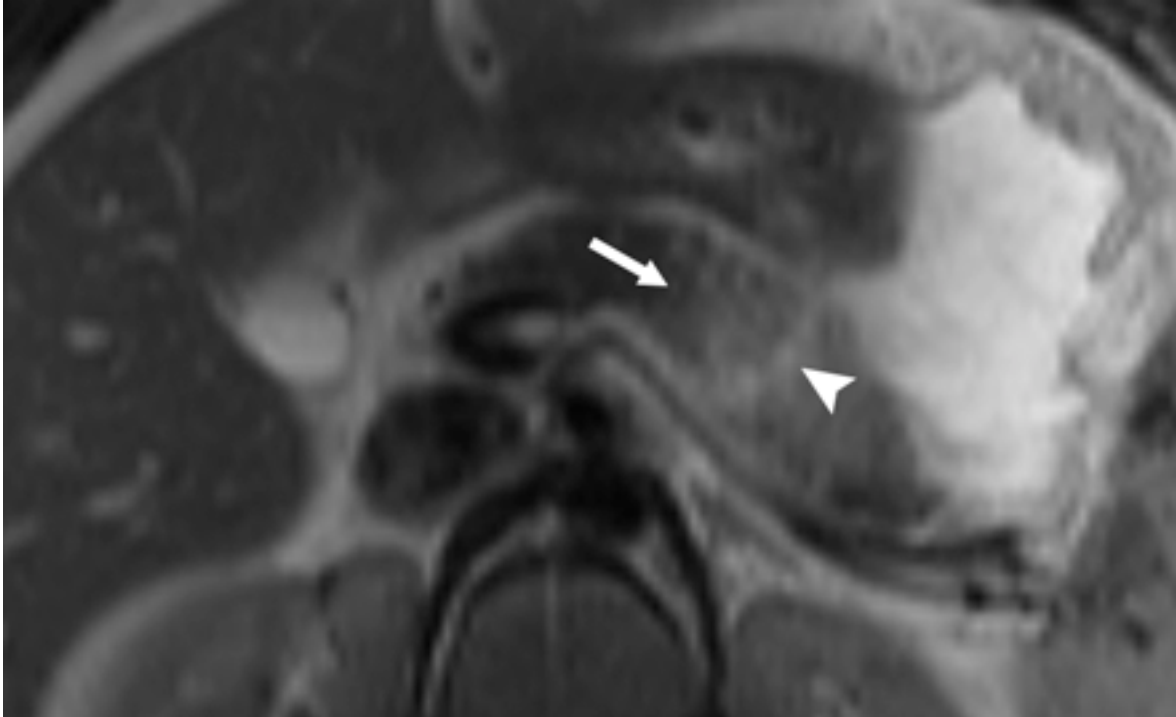


Fig. 72. This is an axial T2-weighted MR image which shows the contusions to the pancreatic body (arrow) and the distal transection (arrowhead). (RadioGraphics) (Wiki)



Fig. 73. The above image is an example of thrombus (dark red elongated structures) in the lumen of the pancreaticoduodenal vessel of the pancreas. There is a spherical tumor mass, approximately 2 cm in diameter, light brown in color in the head of the pancreas. Immediately next to this tumor mass is a hemorrhagic appearing area, which represents a hemorrhagic infarction with fat necrosis. (cai.md.chula.ac.th/chulapatho) (Wiki).

The infarcts are typically pale and well delineated with a peripheral zone of hemorrhage. In some cases this zone is extensive and larger than the infarct itself, and may extend into the necrotic tissue, obscuring it as in Fig. 73. Larger infarcts tend to have a soft center and may liquefy. Typically, these infarcts are multiple. A retroperitoneal or splenic abscess may develop secondary to such vascular damage.

The American Association for the Surgery of Trauma developed an injury scale to grade trauma to the pancreas. The foundation of this grading system is the integrity of the pancreatic duct. Their grading system for pancreatic organ injury scale is as follows:

Grade I: Minor pancreatic contusion or laceration without pancreatic duct injury.

Grade II: Major pancreatic contusion or laceration without pancreatic ductal injury.

Grade III: Distal pancreatic transection or pancreatic parenchymal injury with ductal injury.

Grade IV: Proximal pancreatic transection (to right of superior mesenteric vein) or pancreatic parenchymal injury, not involving the ampulla.

Grade V: Massive disruption of the pancreatic head.

The mortality of patients with pancreatic traumatic injuries is determined by several factors. One of the factors is the mechanism of injury. For example, the mortality associated with shotgun wounds to the lower chest or upper abdomen in one study was 50.6%. However, that from all other types of penetrating trauma was from 7.1% to 23.1%. It appears that patients with penetrating trauma are more likely to have associated injuries. Also, the number of associated injuries affects mortality. For example, patients with zero to one, two to three, and four or more associated injuries have a mortality of 2.5%, 18.6%, and 29.6% respectively.

Mortality is also determined by the location of the pancreatic injury. The overall mortality associated with pancreatic injury to the head, body, and tail were 21.9%, 17.7%, and

9.8% respectively. It is important to remember the proximity of substantive vascular structures related to the head, neck, and proximal body of the pancreas as shown in Figs. 16, p 17 and 54. Also, the clinical finding of a systolic blood pressure of less than 90 is associated with an increase in mortality in those with a pancreatic injury.

The type of operative intervention in patients with pancreatic injury is also related to mortality. For example, total pancreatectomy and pancreatic duct re-anastomosis have mortality rates of 100% and 50% respectively. Those undergoing pancreaticoduodenectomy with a Roux-en-Y anastomosis to the distal pancreas had a mortality of 38.2%. Suture and drainage of the pancreas was associated with a mortality of 19.3%. Patients who underwent drainage only had the lowest mortality of 10.9%. Patients not operated on had a mortality of 89.7%. *What must be kept in mind here is that patients requiring more extensive surgery have more serious injuries, hence, they will have a higher mortality.*

Lastly, complications also play a role in determining mortality rates associated with pancreatic injuries. For example, hemorrhage due to major vascular injury and hemorrhagic pancreatitis are associated with mortality rates of 98% and 86% respectively. Sepsis has a mortality rate of 58%. Abscesses, non-hemorrhagic pancreatitis, pseudocyst, and pancreatic fistula were associated with mortality rates of 30%, 29%, 17%, and 14% respectively. *Pancreatic fistulas is the most common complication occurring in 21.2% of cases.*

Perioperative diagnosis of pancreatic trauma using serum amylase: As has been previously discussed, perioperative diagnosis of retroperitoneal trauma and more specifically pancreatic trauma is difficult. From a laboratory test standpoint there are currently no reliable perioperative test to identify acute injury to the pancreas. One of the laboratory test in which there has been great interest is **serum amylase**.

Amylase is produced by the pancreas and the salivary glands that make saliva. Saliva is a clear, alkaline, somewhat viscid secretion from the parotid, submaxillary, sublingual, and smaller mucous glands of the mouth. Saliva serves to moisten and soften the food, keeps the mouth moist, and contains α -amylase, a digestive enzyme which converts starch into maltose. Saliva also contains mucin, serum albumin, globulin, leukocytes, epithelial debris, and potassium thiocyanate.

The pancreas secretes multiple enzymes for the digestion of the three major types of food: proteins, carbohydrates, and fats. The pancreatic enzyme for the digestion of carbohydrates is pancreatic α -amylase, which hydrolyzes starches, glycogen, and most other carbohydrates except for cellulose, to form mostly disaccharides and a few trisaccharides. The disaccharides and trisaccharides are converted by other enzymes to glucose to supply the body with energy. Technically, all amylases are glycoside hydrolases, which act on α -1,4-glycosidic bands. *What is important to remember is the α -amylases are calcium metalloenzymes thus, they are completely unable to function without calcium.*

The α -amylase secreted by the pancreas is almost identical in its function as the α -amylase of saliva, except that it is several times more powerful. Consequently, within 15 to 30 minutes after the chyme empties from the stomach into the duodenum and mixes with the pancreatic secretions, virtually all the carbohydrates have been digested. The use of serum amylase level as a diagnostic test for pancreatic injury has been of considerable interest since 1943, when Naffziger and McCorkle reported elevated serum amylase levels in nine patients who suffered pancreatic injury. In more recent studies the serum amylase has been reported to be significantly elevated in 10% to 91% of patients with pancreatic trauma. One of the most significant studies regarding serum amylase determinations in blunt abdominal trauma was by Olsen, who reported in 1973 on 179 such patients. He found elevations of serum amylase in only 36 patients (20%). Of these 36 patients, 3 (8%) had pancreatic injury, 2 (6%) had small bowel injury, 19 (53%) had other intraabdominal injury, and 12 (33%) had no intraabdominal injury, which led him to conclude that elevated serum amylase level is not diagnostic of pancreatic injury.

To underscore Olsen's conclusion, besides acute pancreatitis, which may be associated with increased levels of serum amylase, so may cancer of the pancreas, ovaries and the lungs; cholecystitis; severe gastroenteritis; infection of the salivary glands, such as by mumps or a blockage of the salivary duct; intestinal obstruction; pancreatic or bile duct blockage; perforated ulcers; tubal pregnancy and macroamylasemia. Drugs may also increase serum amylase, such as asparaginase, aspirin, birth control pills, cholinergic medications, ethacrynic acid, methyldopa, opiates and thiazide diuretics.

Thus, it appears a serum amylase determination seems to be of little value in an acutely injured patient for the diagnosis of pancreatic injury.

Some have suggested that the determination of increased levels of amylase in peritoneal lavage fluid has possible value in assessing pancreatic injury. At present, it is felt the determination of significant amounts of amylase in lavage fluid, greater than 100 IU per liter (normal range is 23 to 85 IU per liter, although some laboratories give a range of 40 to 140 IU per liter), correlates with the presence of an intraabdominal injury, but is not specific for pancreatic injury.

Today, multi-detector computed tomography provides the most accurate assessment of those patients with suspected pancreatic injury. In addition, endoscopic retrograde pancreatography or magnetic resonance pancreatography should be considered for the assessment of the integrity of the pancreatic duct in the setting of suspected pancreatic injury.

Pediatric Injuries: Pancreatic injury is relatively uncommon in children, occurring in 2 to 5%, some suggesting as high as 10%, in cases of blunt force trauma to the abdomen. Although, it is the fourth most commonly injured solid abdominal organ in children, injury to the pancreas is uncommon in this age group. This is not to say, and as has been previously discussed, children are not susceptible to abdominal injuries, which they are. What makes children susceptible to abdominal injuries is their anatomical differences as compared to adults. In children, the abdomen begins at the level of the nipple. Due to the child's small pliable rib cages and undeveloped abdominal muscles, there is little protection provided for the upper abdominal organs consequently, the liver, spleen, and kidneys are often damaged by relative minor impacts. Abdominal injuries are almost three times as common as thoracic injuries in children, but are much less likely to be associated with a fatal outcome if treated optimally.

The pancreas lies over L2 and L3 in the epigastrium and is thus susceptible to compression by a direct blow. Such compression often leads to a release of pancreatic enzymes due to acinar or ductal rupture. The release of these enzymes into the pancreatic tissue, as has been previously discussed, results in its necrosis accompanied by a pronounced inflammatory reaction. As in the adult, this mass of necrotic tissue is engulfed by a fibrous capsule forming a pseudocyst. Both pancreatitis

and pancreatic pseudocyst occurring in an infant or child are most often due to trauma accidental or non-accidental. Such a pseudocyst may form within days of the injury, or take several weeks.

In children under 3 years of age, pancreatitis is highly suspicious for abuse. Also, a pancreatic cyst occurring in an infant or child, without a history of pancreatitis, should be assumed to be post traumatic. In a report by Merten and Carpenter in 1990, they found abdominal trauma causes in more than 50% of reported cases of pseudocyst in children, with 30% of these showing concurrent evidence of abuse (Figs. 74, 76 & 77). Approximately half the cases of acute pancreatitis in childhood are idiopathic.

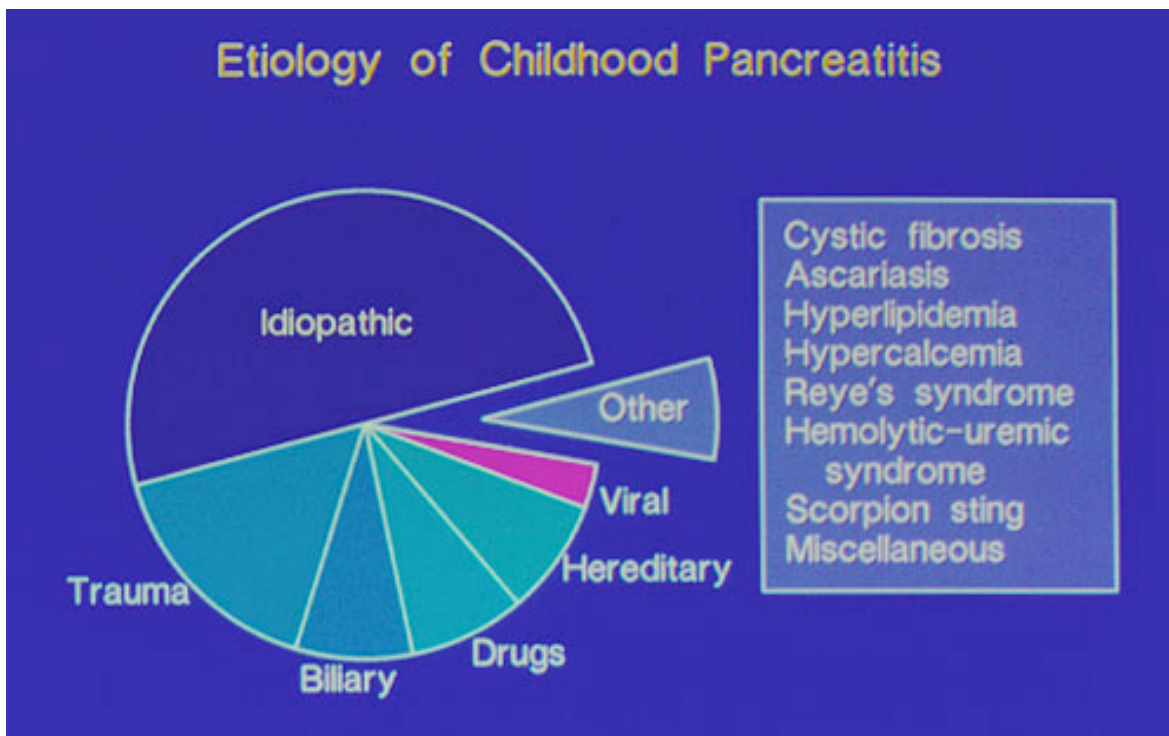


Fig. 74. Trauma such as from bicycle handlebar injuries make up the largest proportion of identifiable causes. Biliary problems such as those associated with gallstones and anomalies of the biliary tract make up the largest group. It is possible that hereditary pancreatitis is less common than represented because cases of hereditary pancreatitis are more likely to be reported than other forms of childhood pancreatitis. Examples of drugs causing pancreatitis include valproic acid and sulfasalazine. As illustrated in the "other" group, many other conditions can be associated with pancreatitis, such as Pancreas Divisum (Fig. 75). (top5plus5.com/Childhood%20pancreatitis.html) (Wiki)

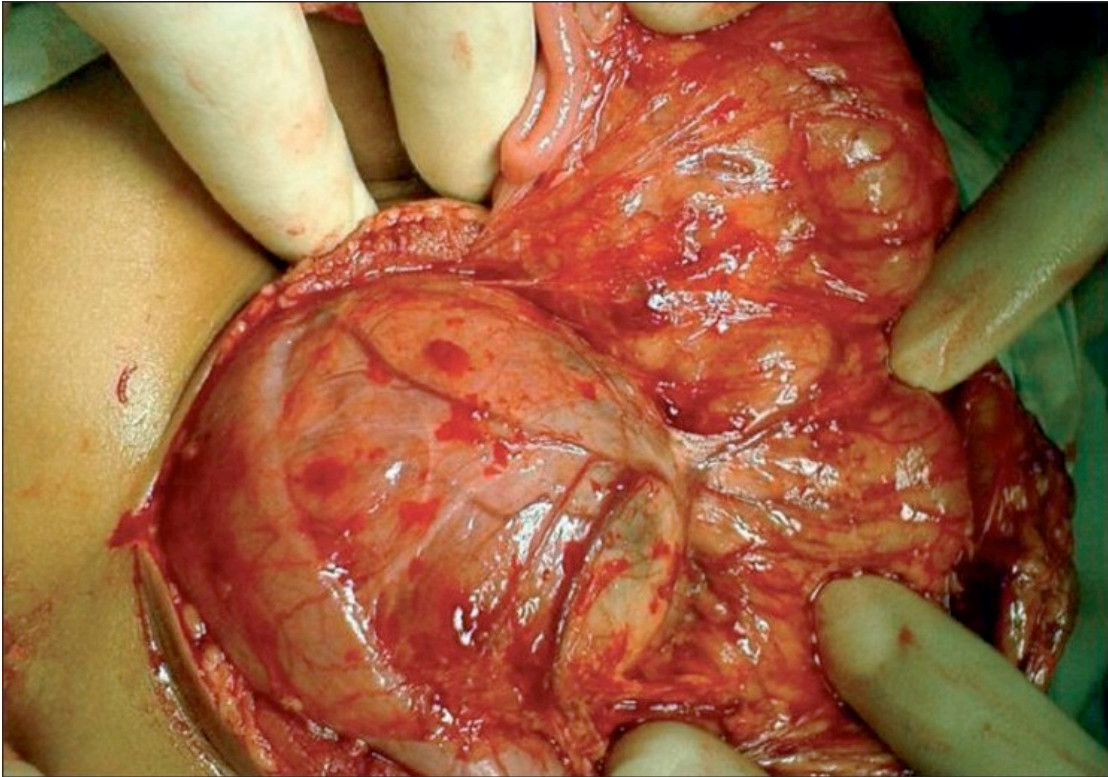


Fig. 75. This is an example of an edematous and swollen pancreas in a case of pancreatic divisum. Pancreatic Divisum is the commonest congenital anomaly of the pancreas with an overall incidence of 4% to 11% and is a common cause of acute pancreatitis in children. (afjrpaedsurg.org) (Wiki)

If the compression is severe enough the pancreas may rupture completely. Injuries from falls on to the handlebars of a bicycle or a misplaced seat belt placed across the abdomen are mechanisms that can produce this type of injury to the pancreas. Such a transection of the pancreas in a child, as is true in adults, occurs at the neck of the gland in line with the superior mesenteric vein. In one retrospective study conducted over a period of 14 years at a pediatric trauma center, only 26 cases of pancreatic injuries were identified, but 11 of the 26 cases were from falls onto the handlebars of bicycles.

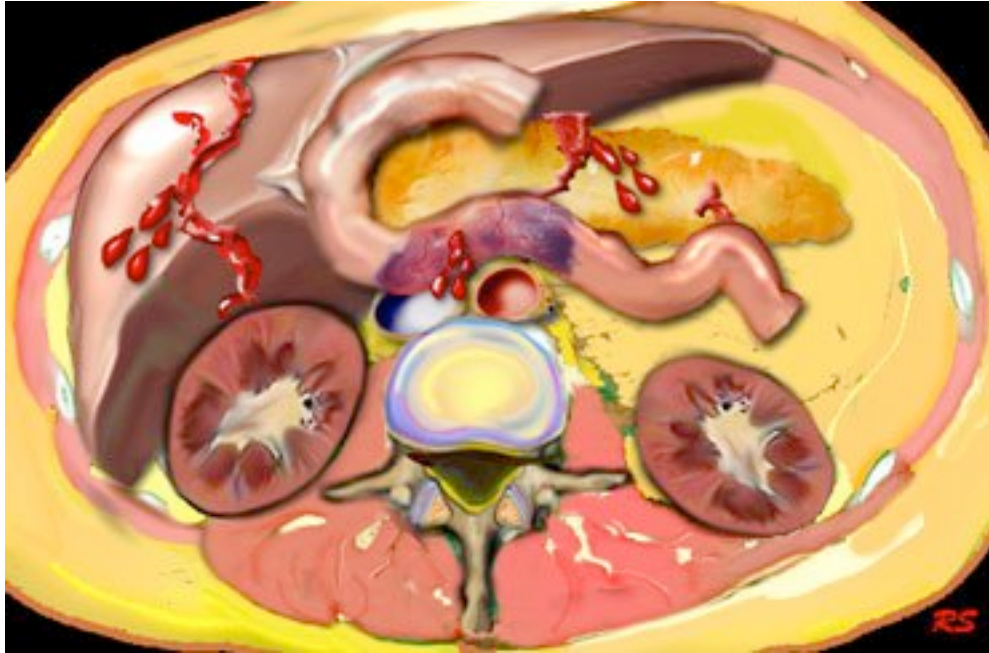


Fig. 76. Common abdominal injuries in abused children are liver laceration, duodenal hematoma and pancreatic laceration.

Visceral injury is seen at autopsy of young infants, but is rarely documented radiologically in living victims less than 1 year of age.

It is estimated that 2 to 10% of all abdominal injury results from child abuse. The mean age of these children is about 2 years. It is more common in boys than girls. The mortality rate is 50% due to 'patients and doctors delay'. Typically, these children are brought to the hospital days after the injury, when a perforation already has resulted in peritonitis and sepsis. The history given by the abusers usually does not correlate with the symptoms, which makes these cases very difficult to evaluate for the clinician.

The most common non-accidental abdominal injuries are: visceral perforation or hematoma; liver-and pancreatic laceration; and adrenal bleeding. The most common abdominal accidental injuries in adults, which are laceration or subcapsular bleeding of the spleen and the kidney, are unusual in abused children. (Diagnostic Imaging in Child Abuse, Non Accidental Trauma, Simon Robben, Radiology Department of the Maastricht University Hospital in the Netherlands) (Wiki)



Fig. 77. The above is a CT scan showing a case of pancreatic laceration in child abuse. (Diagnostic Imagine in Child Abuse, Non Accidental Trauma, Simon Robben, Radiology Department of the Maastricht University Hospital in the Netherlands) (Wiki)

The other mechanism that can produce either a severe contusion or a transection of the pancreas is a deceleration injury, when the pancreas is stretched over the lumbar vertebrae, typically L2 and L3. Such injuries can occur in falls from a height on to the back or in motor vehicular accidents.

In 60 to 80% of accidental blunt force trauma to the abdomen in which there is a pancreatic transection there is associated injury to other abdominal organs. The two most frequently affected organs are the liver and spleen. Also, in more than 10% of such cases, there is an associated injury to a hollow viscus. Pancreatic rupture carries a mortality rate close to 20%. The prognosis for pancreatic injury is typically good.

Conservative non-operative management of pancreatic injuries without ductal rupture is associated with a low morbidity.

Preoperative diagnosis of pancreatic trauma in a child is difficult. This is especially true in which the pancreatic injury is isolated, such as falling on to the handlebar of a bicycle. Often the child presents with nonspecific symptoms of moderate epigastric or back pain

and mild tenderness in the epigastrium. In many cases there are no, or very minor, symptoms appearing following an isolated injury. It is not uncommon for moderated symptoms not to appear until 24 hours after a complete pain-free period following the incident (Fig. 76). It is important to always keep in mind when evaluating abdominal trauma in a child, even minor blunt force trauma to the abdomen is capable of causing serious injury to the abdominal organs, including pancreatic rupture.

Perioperative diagnosis of pancreatic trauma in the pediatric age group using

serum amylase: As in adults, using serum amylase as an indicator of pancreatic trauma is fraught with difficulty. In one study, most children did not show a raised amylase until many days after the injury. Also, as in adults, elevation of serum amylase is not a specific sign of pancreatic injury. Ruszinko *et al.* published a study in 2005 in which enzyme levels increased only in 60 to 70% of children with blunt abdominal trauma, and such elevation occurred in even a smaller portion of children with penetrating injury. Elevated serum amylase occurred only in 33% of blunt abdominal trauma without pancreatic injury. It was their view that continuous monitoring of serum amylase was most effective. If there is constant elevation, or continuous high values, than pancreatic injury is probable.

In the child, the CT scan is the most useful imaging technique for detecting parenchymal lesions in the early phase, however, others suggest ultrasound to evaluate the pancreas and pseudocysts in both the initial and following phases (Figs. 78 to 81).



Fig. 78. The above ultrasound image shows a pancreatic rupture with hemoperitoneum. (ultrasoundcases.info) (Wiki)

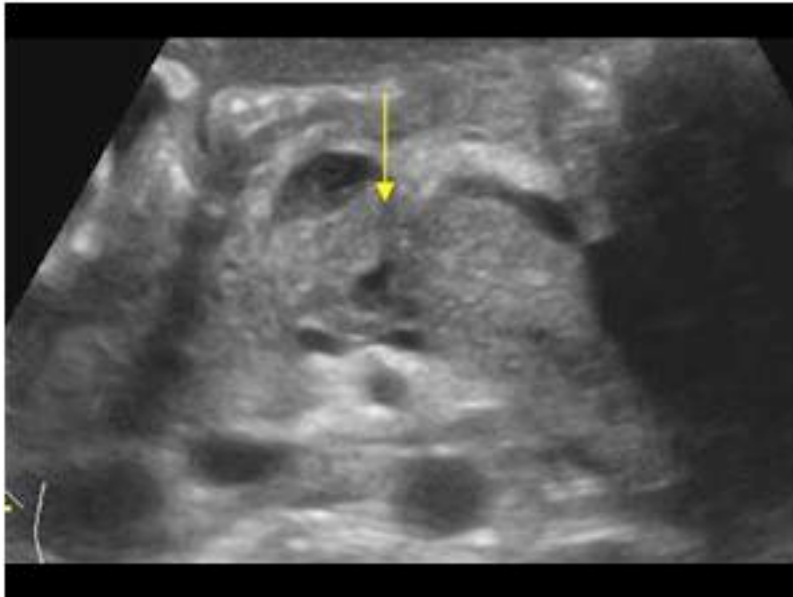


Fig. 79. This is an ultrasound image showing a pancreatic rupture. (ultrasoundcases.info) (Wiki)

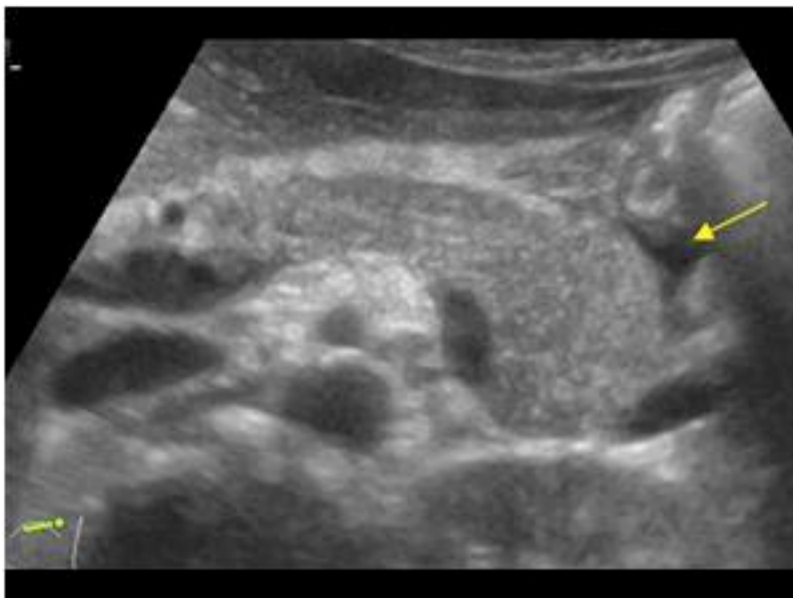


Fig. 80. This is an ultrasound image showing an effusion around the tail of the pancreas. (ultrasoundcases.info) (Wiki)



Fig. 81. This is an ultrasound image showing hemoperitoneum with blood in the lower abdomen, transverse. (ultrasoundcases.info) (Wiki).

There are two other associations with traumatic pancreatitis in children, although uncommon, which need to be mentioned. Both of these associated conditions involve lytic bone lesions. In some cases of pediatric traumatic pancreatitis, lytic bone lesions, due to medullary fat necrosis, develop 2 to 10 weeks after the injury. It appears that the release of pancreatic enzymes into the circulation following the traumatic pancreatitis event breaks down the medullary fat in the bone, producing the lytic lesions (Fig. 82).



Fig. 82. The above image shows multiple lytic bone lesions involving long bones. (Wiki)

The lower extremities, especially the small bones in the feet, are most often involved, which is often associated with pain and swelling. Spontaneous resolution occurs typically within a year. Unfortunately, on occasion, these lesions have been misinterpreted as bone lesions of leukemia and metastatic neuroblastoma.

The other association in pediatric traumatic pancreatitis are bone lesions occurring in conjunction with polyarthritis and cutaneous nodules. It appears the first such association was a fatal case reported by Ponfick in 1872.

Mullin *et al.* reviewed the literature in 1968, in which they described 23 patients with pancreatic disease and arthritis or skin lesions resembling erythema nodosum, or both, some of which had bone lesions as well. Fourteen of these cases were carcinoma of the pancreas, which were associated with subcutaneous fat necrosis or joint involvement (12 with arthritis) or both. Of the 23 patients 2 had traumatic pancreatitis.

In 1975, Moore discussed in the *Journal of Rheumatology*, the various syndromes resulting from dissemination of pancreatic enzymes. In the same journal issue, Gibson and Tannenbaum and their colleagues discussed the association of bone lesions, polyarthritis and skin lesions with pancreatic disease. Their belief was the disseminated fat necrosis that affected viscera and bone distant to the pancreas was the result of excessive quantities of lipase being released into the bloodstream due to obstruction of pancreatic outflow by tumors, calculi or edema or because of hypersecretion of functional acinar cell carcinoma and their metastases.

There is no specific treatment for this condition. It appears to be self-limited, with resolution of the pancreatitis, followed by complete healing of bone and joint lesions. The bone lesions typically regress almost completely within a few months to a year.

D. Kidneys: Normally, the kidneys are paired retroperitoneal structures, lying on the psoas muscles on either side of the vertebral column in the paravertebral gutter (Figs. 18, p 21 & 19, p 22). The superior aspects of the kidney are somewhat protected by the lower ribs. The posterior surface and upper portion of the right kidney rest on the 12th rib; the left kidney typically rests on the 11th and 12th ribs thus, the right kidney is usually lower than the left (Fig. 20, p 22). The lower poles of both kidneys are inferior to

the 12th ribs. The anterior surface of the right kidney is in contact with the right adrenal gland, and is next to the right lobe of the liver and right hepatic flexure. The anterior surface of the left kidney is in contact with the left adrenal gland, and is next to the stomach, spleen, jejunum and the pancreas. Thus, due to its somewhat protected position by the surrounding ribs, muscles (psoas and quadratus lumborum), perinephric fat, and peritoneum, the kidneys are rarely damaged by frontal blunt force trauma, but may be injured by impact laterally or posteriorly in the region of the loin.

The frequency of renal injury depends much on the patient population under consideration. Traumatic injuries to the kidneys accounts for approximately 3% (1 to 5%) of all trauma admissions and as many as 10% of patients who sustain abdominal trauma. The kidney is the most common injured genitourinary organ, with the genitourinary system itself stated to be involved in approximately 10% of all traumatic injuries. In most trauma centers blunt force trauma is more common than penetrating trauma and in some trauma centers it occurs as much as nine times more frequently than penetrating injuries, accounting for up to 80 to 90% of all cases, with motor vehicular accidents being the most common cause; less common causes include: a direct blow to the flank or abdomen during an assault, a fight or sports activity (e.g., bicycling, horseback riding, and a fall from a height). Both kidneys are equal in frequency of being injured, penetrating or non-penetrating, although, there are reports in the literature the right is injured more frequently than the left.

Children and young adults are victims of blunt force traumatic injuries more often than the older person, with the incidence in males being approximately ten times as high as it is in females. These differences are because young males are more active and tend to engage in activities which have a greater chance to lead to renal injury.

Preexisting renal disease renders the involved kidney more susceptible to traumatic injury, even with slight trauma. Preexisting renal abnormalities predispose the kidney to an increased risk of not only injury, but a decreased potential for renal salvage following blunt abdominal trauma. Preexisting renal cysts are the most common predisposing anomaly and may undergo rupture, or bleeding, with or without, communication with the collecting system (Figs. 83, 84 & 85).

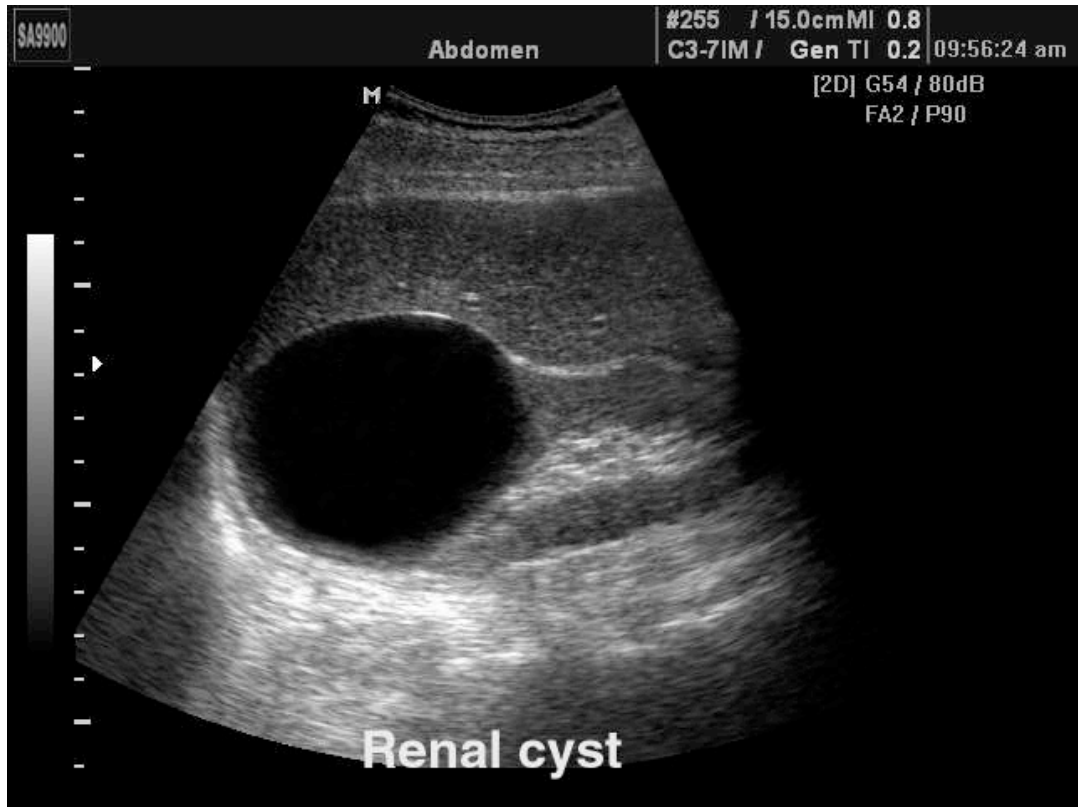


Fig. 83. This is an echogram of a renal cyst taken by ultrasound. (Wiki)

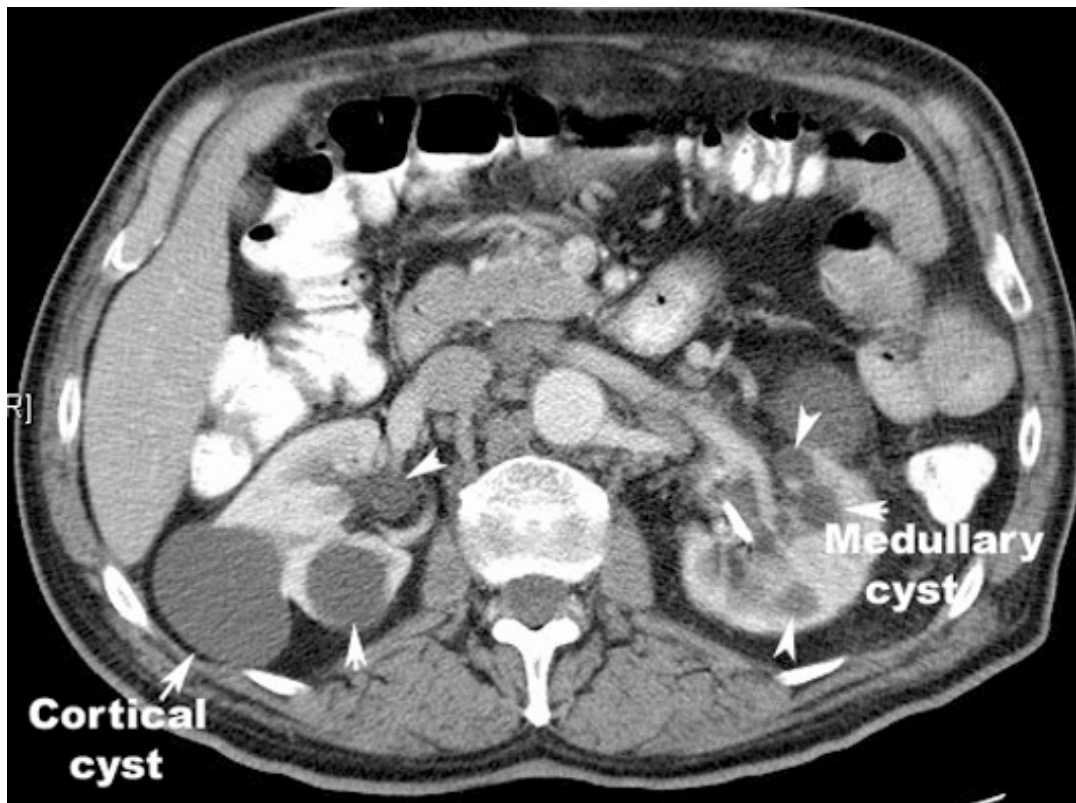


Fig. 84. The above image is a CT scan showing multiple renal cysts. (Wiki)



Fig. 85. This is a photograph of a kidney removed at the time of autopsy showing multiple renal cysts. Renal cysts have thin walls and are fluid filled. They can be multiple, as shown here, but they are never as numerous as in polycystic disease. They also do not predispose to chronic renal failure or to neoplasia. Such renal cysts become more common as a person ages. (Wiki)

One of the first reports of this phenomenon was by Bailey in 1924. Bailey reported on 26 cases of traumatic renal rupture, which were operated on. He observed 6 of the traumatically injured kidneys had preexisting severe disease. Establishing the existence of a preexisting renal disease in a traumatically injured kidney, has importance in determining the appropriate surgical approach. Not infrequently the renal disease, which predisposed to injury is bilateral. Harrison in 1940 was one of the first to stress knowing the condition of the uninjured kidney before removing the injured kidney. Although, the logic to Harrison's caution is irrefutable, there is another factor that needs to be kept in mind and that is the experimental observations made by Falcone. He experimented on eight dogs, in which he crushed one kidney. What he noted in the

uninjured kidney within a few days was extreme congestion, glomerular and interstitial hemorrhages, with oliguria, and the presence of albumin and white and red blood cells. As the days went forth, the quantity of urine increased, its color improved, but the albuminuria nearly always persisted, in some cases with cylindruria (presence of renal cylinders of pseudo-casts in the urine, typically in association with hyaline casts and proteinuria). Falcone went on to suggest, that one should continue without delay to remove the injured kidney.

Renal trauma management today first requires addressing concomitant injuries to the spleen, liver, bowel, or diaphragm, which often dictates the approach that is ultimately used to treat renal injuries, although nephrectomy or repair of renal injuries will eventually be needed as well. Today, renal management has three goals: (1) minimize hemorrhage; (2) maintain urinary flow without obstruction to preserve renal function; and (3) prevent extravasation of urine outside the urinary tract, thereby decreasing the risk of local and systemic infection. What is also important to remember is that 95 to 98% of isolated renal injuries are considered minor injuries and are managed non-surgically because they usually heal spontaneously and without complications.

Another issue to keep in mind, the same condition which predisposes a kidney to injury from what would be regarded as minimal trauma, may also cause **spontaneous rupture**. One of the first to address the relationship between diseases of the kidney and spontaneous rupture was Connell in 1916, in which he reported on 30 cases of spontaneous renal rupture.

Non-traumatic retroperitoneal hemorrhage due to spontaneous kidney rupture is an uncommon event, which was first described by Wunderlich in 1856, who pointed out that a perirenal hematoma may be due to spontaneous kidney rupture. Since his report approximately 200 cases have been reported. In most instances the spontaneous renal rupture is associated with underlying disease of the kidney. Renal tumors account for most cases, with clear cell carcinoma being most frequent, followed closely by angiomyolipomas and then vascular diseases (Figs. 86, 87 & 88). Infection, nephritis and blood dyscrasias were far less common as an etiology. Spontaneous rupture of the renal parenchyma during pregnancy has been reported, however, when this occurs it is typically associated with underlying renal disease.

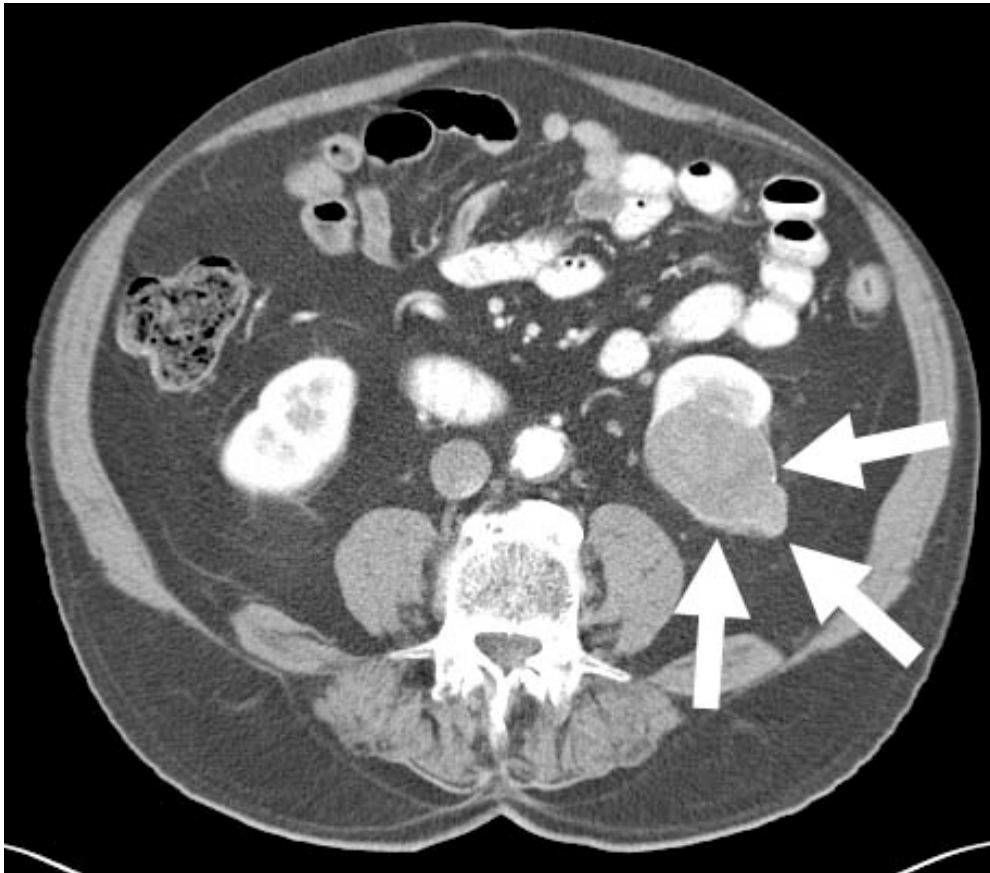


Fig. 86. This is a CT scan of the abdomen of a 67-year-old man with a renal cell carcinoma showing a lobulated and heterogeneously enhancing mass arising from the lower aspect of the left kidney (arrows). (radiology.casereports.net) (Wiki)

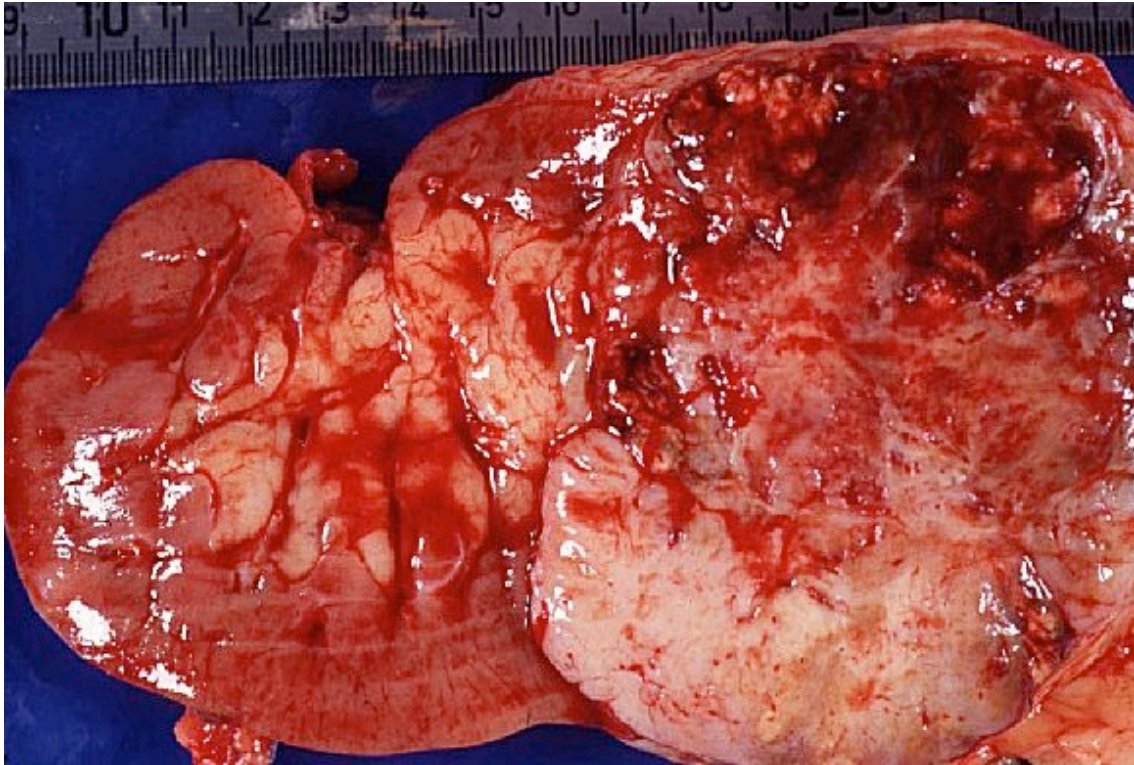


Fig. 87. This is a picture of a kidney, which has been cut in half parallel to its longitudinal dimension, showing a yellow, hemorrhagic mass, consistent with renal cell carcinoma. (pathguy.com) (Wiki)

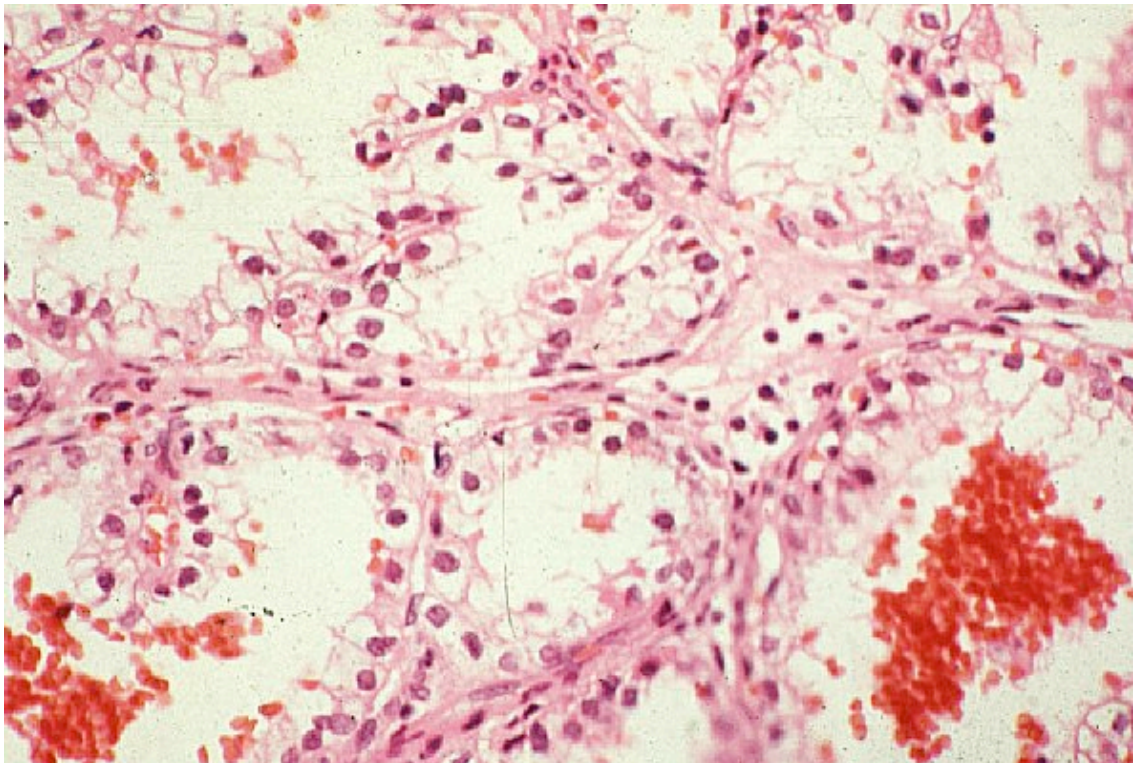


Fig. 88. The above image is a photomicrograph of a renal cell carcinoma in which the cells have a clear cytoplasm with easily determined cellular borders. Some of the clear staining is due to lipid, which gives the tumor a yellow color. Foci of acute hemorrhage are noted, which is due to the rich vascularity of these tumors. It is this vascularity, which accounts for their propensity to bleed into themselves and to rupture. (pathguy.com) (Wiki)

One of the sources of spontaneous renal rupture are renal cysts, which have been discussed above (Figs. 83, 84 & 85). Renal cysts occur in approximately 50% of the adult population over 50. They can develop complications such as obstruction, infection, rupture or hemorrhage, confined either to the cyst or causing subcapsular or peri-renal hemorrhage.

Although it has been reported that spontaneous rupture of a normal kidney does not occur, McDougal and colleagues reported of four cases in 1975 and Valtonen in 2008 reported on another case.

The etiology of traumatic injuries to the kidneys are fundamentally of two types: **penetrating**, which include gunshot (Figs. 89, 90 & 91) and stab wounds (Fig. 92) and **non-penetrating**, which include blunt force trauma, either directly over the kidneys, such as can occur by a pedestrian being struck on the anterior abdominal wall, or a kick delivered to the victim lying supine with the tip of the shoe impacting directly below the slightly raised rib cage, between the rib margin and hip, or the blow can be delivered posteriorly or laterally (Figs. 93, 94 & 95). Impacts to the flank can compress the kidney against the lumbar vertebrae.

Another source of injury to the kidneys are iatrogenic procedures, such as endourologic procedures, extracorporeal shock wave lithotripsy, renal biopsy, percutaneous renal procedures and diagnostic peritoneal lavage.

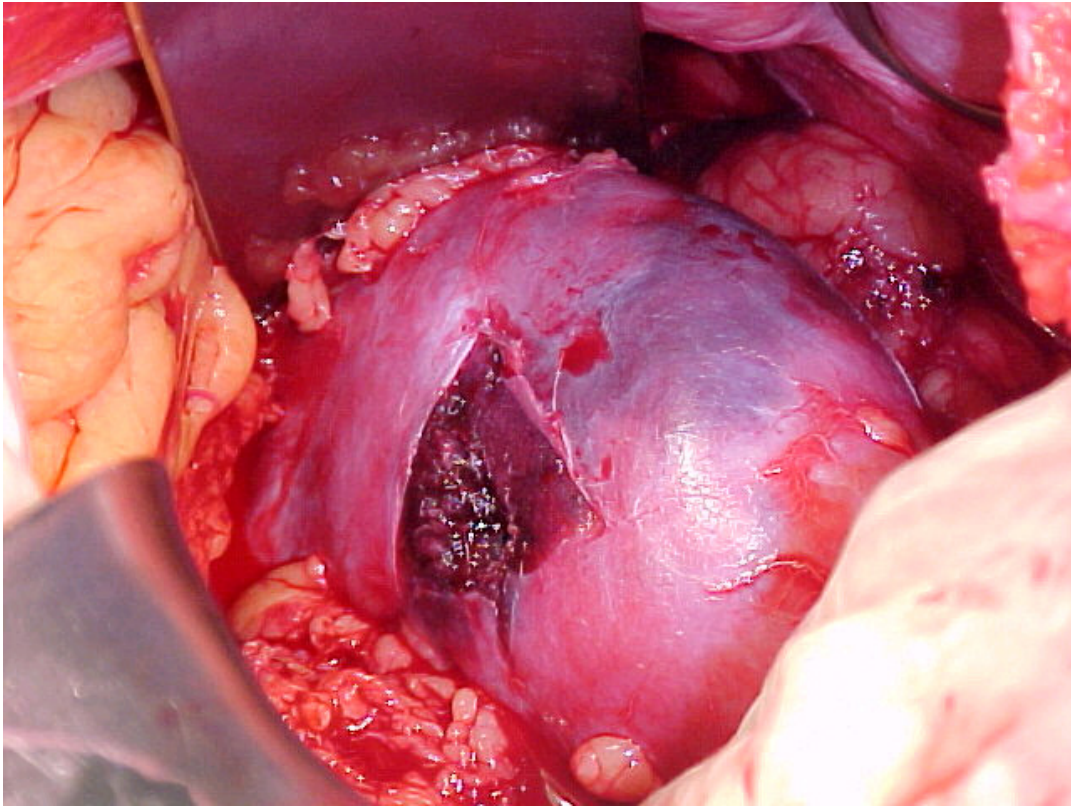


Fig. 89. This image shows an entrance gunshot wound to the right kidney. (Trauma.org) (Wiki)

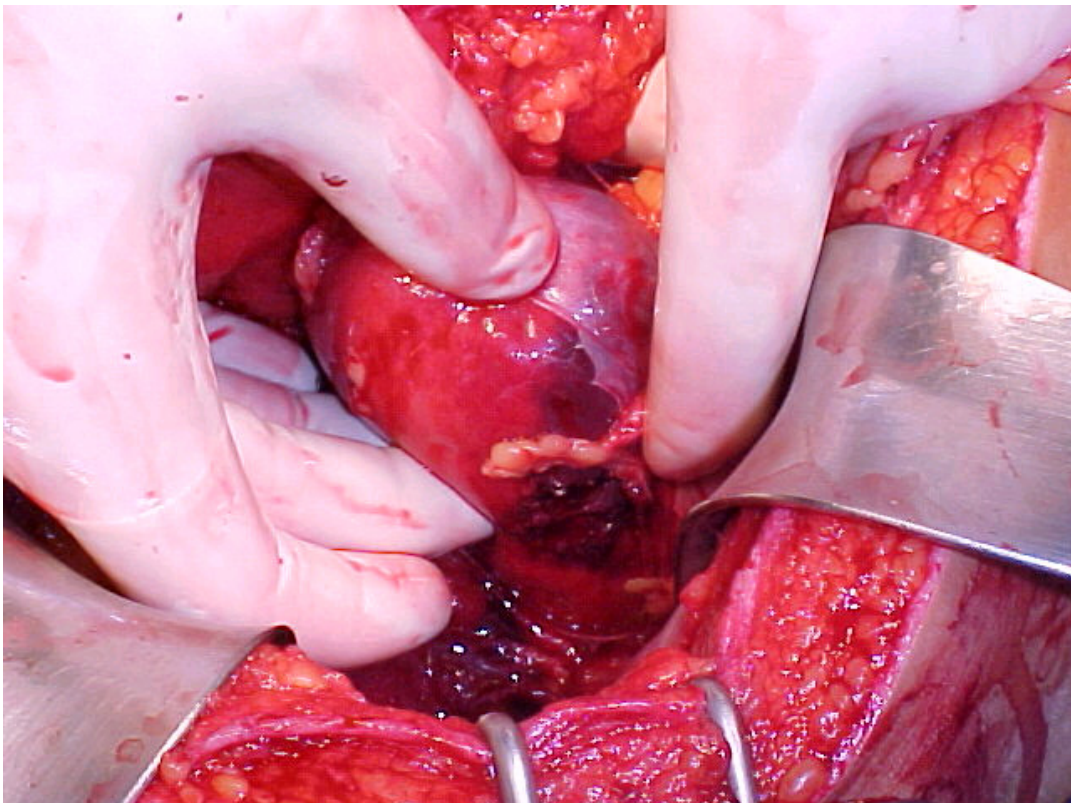


Fig. 90. The above image is the exit wound of the gunshot wound of entrance shown in Fig. 89. (Trauma.org) (Wiki)

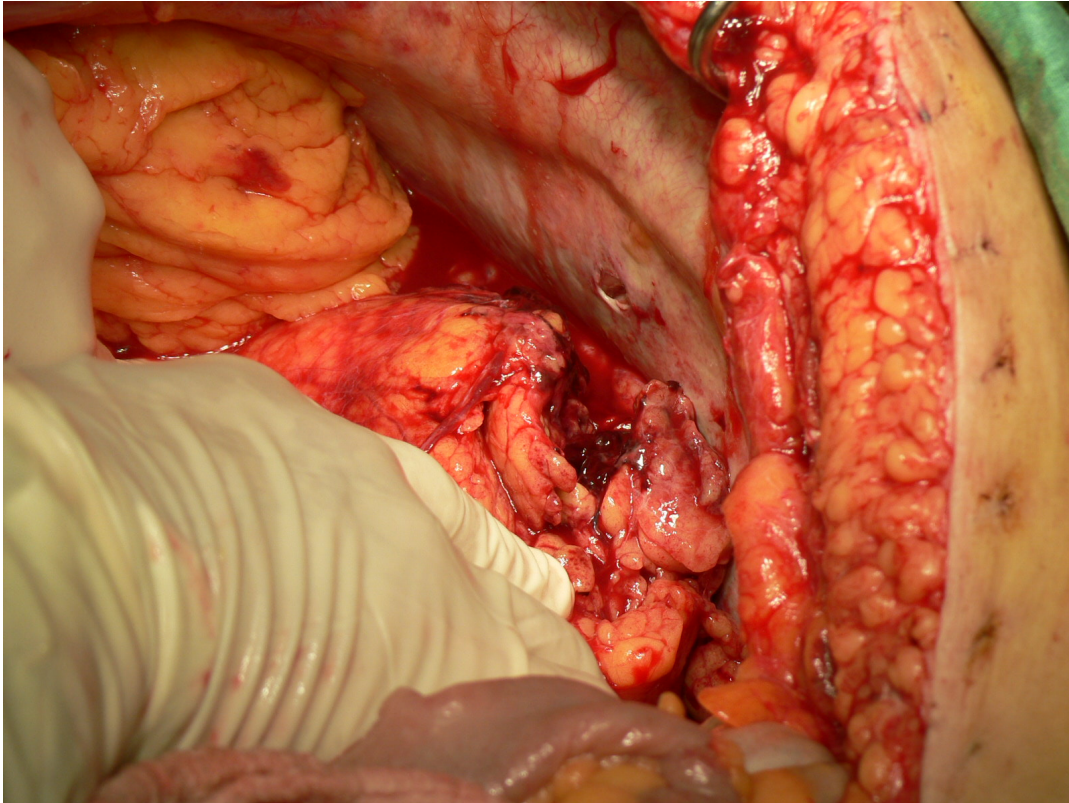


Fig. 91. This is a laceration of the left kidney produced by the wound tract of a missile. (Trauma.org) (Wiki)



Fig. 92. This is photograph showing two stab wounds of the kidney.
(malmignatte.tumblr.com) (Wiki)

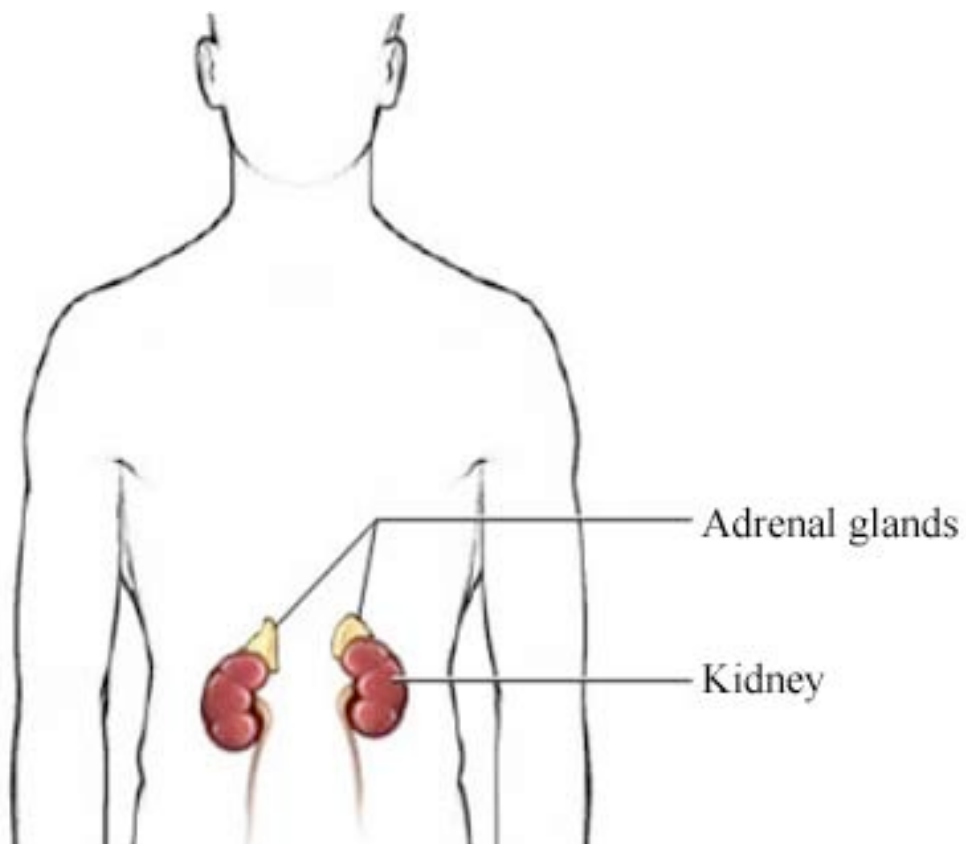


Fig. 93. This is a drawing showing the location of the kidneys in the region of the loin.
(Wiki)



Fig. 94. This is a contusion (bruise) in the region of the left loin, which overlies the left kidney. (Wiki)

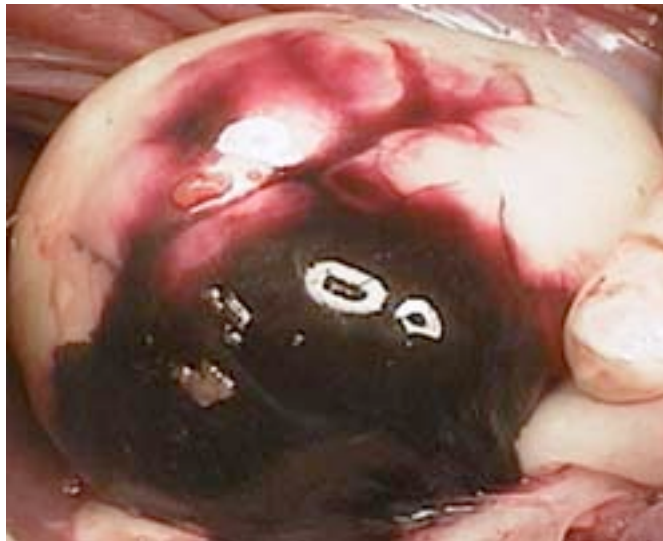


Fig. 95. This is a contusion of the kidney. (Wiki)

Biophysics of Renal Injury: Küster published a paper in 1896 in which he established the underlying mechanisms of non-penetrating traumatic injuries to the kidney. He noted that non diseased cadaver kidneys or kidneys removed at the time of autopsy could withstand a great deal of impact force before rupturing. However, if he took the same kidneys and filled the pelvis and vessels with fluid, tying them off, the kidney would readily rupture with moderate impact. He was able to reproduce both the location, as well as the character of the traumatic injuries, which were seen clinically and at the autopsy table. The ruptures, according to Küster, were usually transverse and occurred in the interlobular lines of the embryonic kidneys. It was from these experiments that Küster determined the blunt force traumatic injuries were best explained because of explosive hydraulic effects of sudden compression of a structure, the fluid content of which could not be evacuated quickly enough through its anatomical channels.

Henry Morris published a paper in 1901 in which he contended the ribs also play a part in the production of traumatic injuries to the kidneys. He opined that when impact force is applied to the back or laterally, that force is directly transmitted to the kidneys through the impact of the ribs. Likewise, when the impact force is to the front of the abdomen it is indirectly transmitted, by impact of the ribs, to the kidneys. In either case the kidney is driven against the vertebral column. It is difficult however to apply Morris's hypothesis to rupture of the kidneys through falling on ones feet. Perhaps, Küster's hydraulic pressure theory is more applicable, the kinetic energy of which enters the trunk through the lower extremities.

Non-penetrating Injuries to the Kidneys: The major anatomical lesions to the kidneys due to blunt force trauma are:

1. Perinephric injuries: Blunt force trauma applied to the kidneys, whether anterior, laterally or posteriorly, can injure the perinephric fat regardless whether the kidney itself is damaged. The only evidence of such an injury may be the presence of foci of hemorrhage into the perinephric fat and psoas muscles (Figs. 96 & 97).

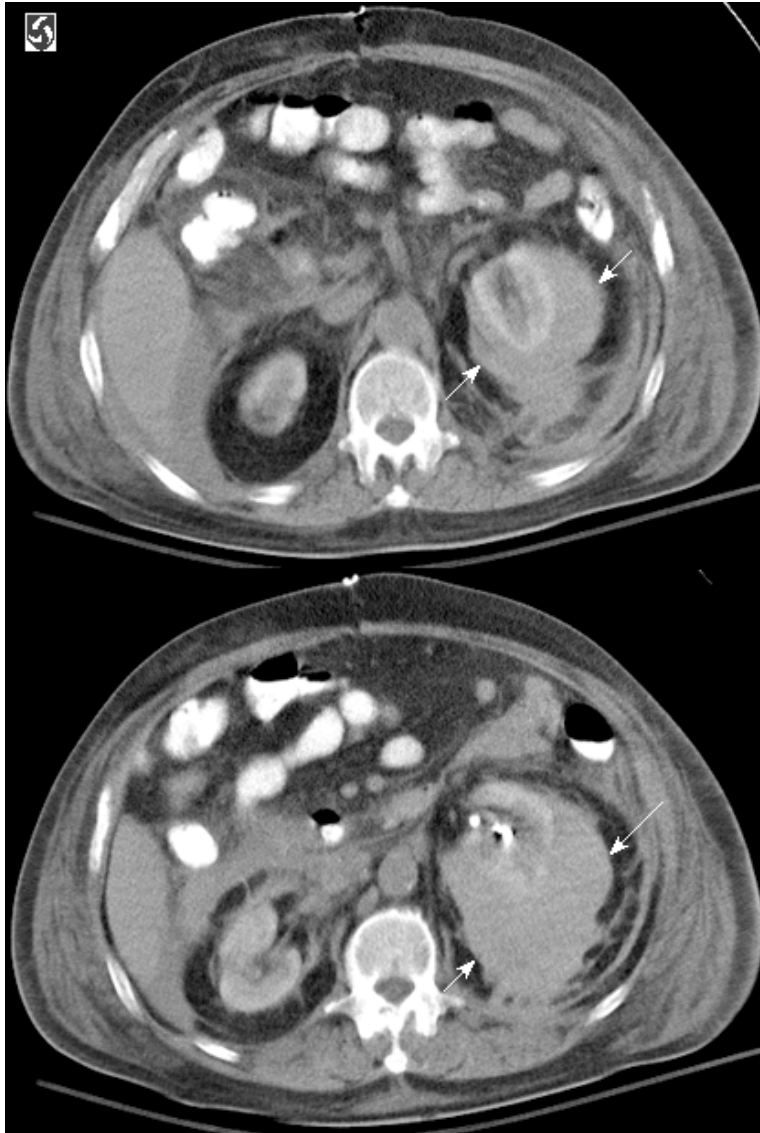


Fig. 96. These images are contrast-enhanced abdominal CT scans demonstrating a large (8 x 7 cm) left perinephric fluid collection (arrows) arising from the lower pole and extending 13 cm inferiorly. The left kidney is elevated anteriorly. The density of the mass is similar to that of the liver, which is consistent with a hematoma. The adjacent Gerota's fascia is significantly thickened and shows blood tracking into the anterior pararenal spaces on both sides, into the peritoneal cavity around the liver, and into the paracolic gutters. Diffuse edema is present and is most pronounced within the pelvis. The etiology of perinephric hematoma can be either spontaneous (such as a malignant or benign tumor [Fig. 97], vascular disease, arteriovenous malformation, hemorrhagic cyst, infection, or infarction), or traumatic (such as blunt or penetrating trauma, percutaneous nephrotomy, biopsy, surgery, or anticoagulation therapy). Patients with perinephric hematoma generally present with acute flank pain, and sometimes with hematuria. (brighamrad.harvard.edu) (Wiki)

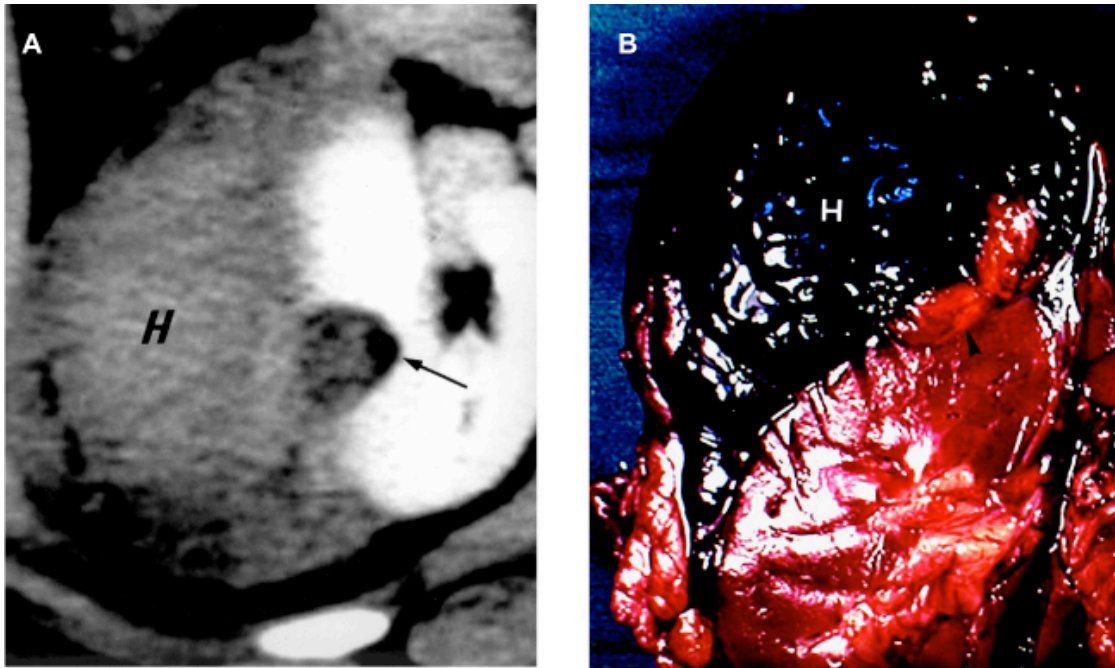


Figure 1 - Small ruptured angiomyolipoma associated with spontaneous renal hemorrhage. A) - Enhanced helical CT scan shows a large perirenal hematoma (H). A heterogeneous exophytic mass can be seen at the lateral portion of the right kidney (arrow). A small amount of fatty tissue can be seen at the periphery of the lesion (arrowhead). B) - Gross specimen shows a small angiomyolipoma (arrowhead) causing a large hematoma (H).

Fig. 97. The left image is an enhanced helical CT scan showing a perirenal hematoma (H). A heterogenous exophytic mass can be see at the lateral portion of the right kidney (arrow). The B image shows a small angiomyolipoma (arrowhead), which is the source of the hematoma (H). (International braz j urol, vol.29 no.3 Rio de Janeiro May/June 2003) (Wiki)

In addition, due to the disruption of perinephric blood vessels, foci of fat necrosis may develop. As the foci of fat necrosis evolve, the foci of hemorrhage are likely to be absorbed, being represented microscopically by hemosiderin laden macrophages. This is why it is very important in such cases, perinephric fat and muscle be taken for microscopic examination. Overtime, the foci of fat necrosis will be absorbed, and may be represented by fluid-filled spaces. At the periphery of these resolving areas of fat necrosis will be a zone of fibroblast. Within the inner most layer of these proliferating fibroblast you may fine isotropic crystals, some of which are free and others are within multinucleated foreign body giant cells. Scattered throughout are mononuclear cells and lymphocytes. Again, this is another reason why it is important to take samples of

perinephric tissue for microscopic examination. In some cases, the entire traumatized area will be surrounded by a thick fibrous capsule. The smaller lesions typically absorb completely. However, the larger lesions may persist for months and some may progressively increase in size due to continued inflammatory reaction, such that they may be misdiagnosed as a tumor.

Laceration of the perinephric fat accompanied by hemorrhage occurs more commonly than **contusions** of the perinephric fat described in the previous paragraph (Fig. 98). These lacerations come about through the displacement of the kidney within its perinephric fat capsule. Such displacement leads to tearing of not only small vessels, but those of considerable size as well. Thus, the following hemorrhage may be quite small and insignificant or quite large forming a hematoma (Fig. 99). If the displacement was such that small vessels, which extend from the perinephric fat through the capsule are torn you may also see hemorrhage on the external surface of the capsule, within the capsule, or directly beneath it. These foci of hemorrhage may form a cystic hematoma, which through the process of absorption may form a cystic hygroma, either perirenal or subcapsular. Some of the larger perirenal or pararenal hematomas may become calcified, cystic, or infected. The larger hematomas tend to undergo central liquefaction forming a cyst encapsulated by a fibrous capsule. Ultimately, the contents of these larger cyst are filled with clear fluid. The lining of such cyst is not epithelial, but is formed by irregular masses of granulation tissue with both extracellular hemosiderin and macrophages laden with hemosiderin.

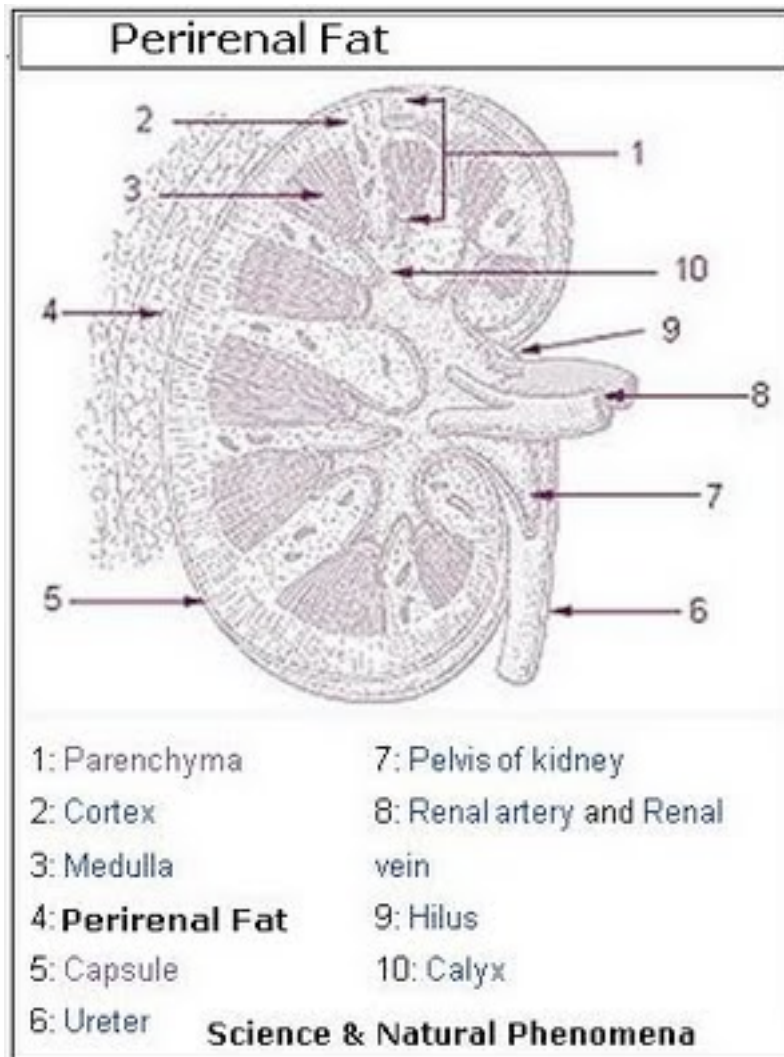


Fig. 98. The above illustration shows the various anatomical components of the kidney including the perirenal (perinephric) fat (4). (Korean medical library engine) (Wiki)

Renal trauma grading scale

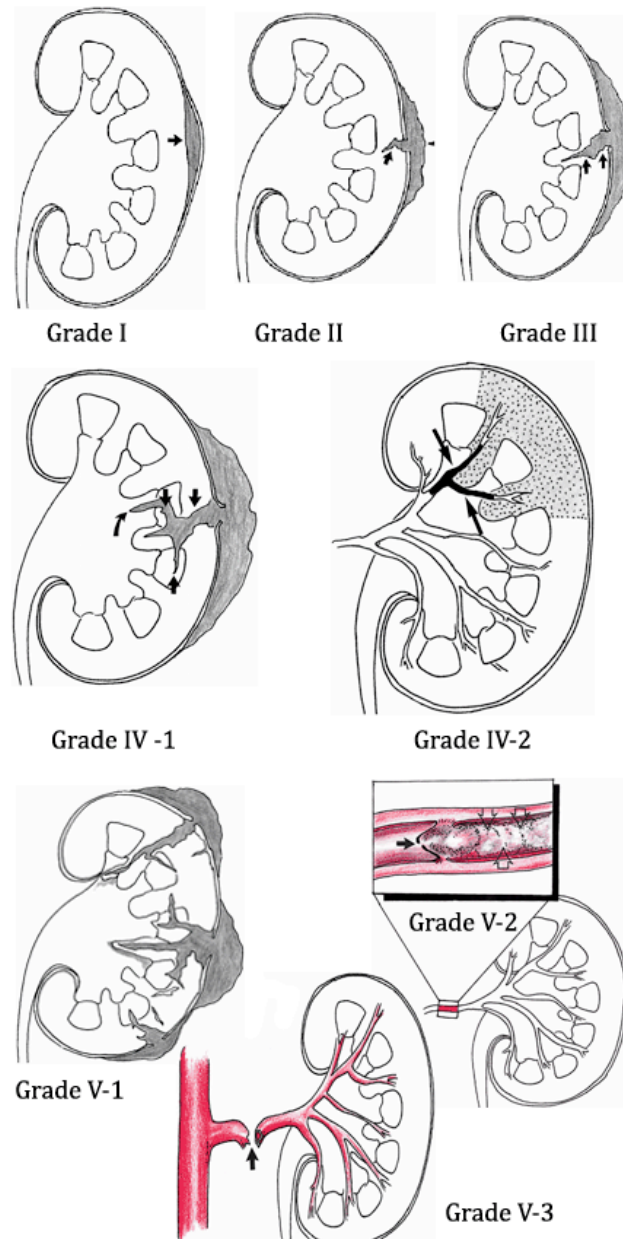


Fig. 99. This is an illustration of the various grades in severity of injury to the kidney, extending from Grade 1, which is a subcapsular hematoma to a Grade 5, which includes a completely shattered kidney. (Wiki)

Approximately 82% of injuries may be classified as grade 1 and include parenchymal contusions and isolated subcapsular hematomas. Grade 2 injuries include superficial cortical lacerations less than 1 cm in depth and non-expanding perirenal hematomas. Grade 3 injuries include lacerations greater than 1 cm in depth without extension into the collecting system or evidence of urinary extravasation. Deep lacerations that involve the collecting system, traumatic thrombosis of a segmental renal arterial branch, and injuries to the main renal artery not associated with renal devascularization are all

grade 4 injuries. Grade 5 injuries, the most severe, include shattering of the kidney into multiple fragments and devascularizing injuries to the renal pedicle. They also include avulsion of the renal artery, as well as thrombosis of an intact renal artery, usually due to a shearing injury to the intima. In one large urologic series, minor parenchymal lacerations (grade 2) accounted for 6% and major lacerations (grades 3 & 4) accounted for 7% of injuries. Vascular injuries (grades 4 & 5) accounted for only 5.5% of cases. (radiographics.rsna.org) (Wiki)

2. Injuries of the Renal Pedicle: Blunt force injuries to the renal pedicle occur infrequently. Renal pedicle injuries account for up to 5% of all renal trauma and are often associated with injuries to other organs. If the injury is isolated, there may be no hematoma, or other signs of injury, and hematuria may also be absent (Fig. 96). Blunt force trauma to the renal pedicle may cause kinking, contusion, laceration, renal artery thrombosis, renal vein disruption, or renal pedicle avulsion (Figs. 99, 100, 101 & 102). The most common form of vascular pedicle injury is renal artery occlusion (Fig. 99). This is due to the stretching of the renal artery, which produces an intimal tear, or dissection, followed by platelet aggregation and subsequent vascular occlusion that progresses distally.

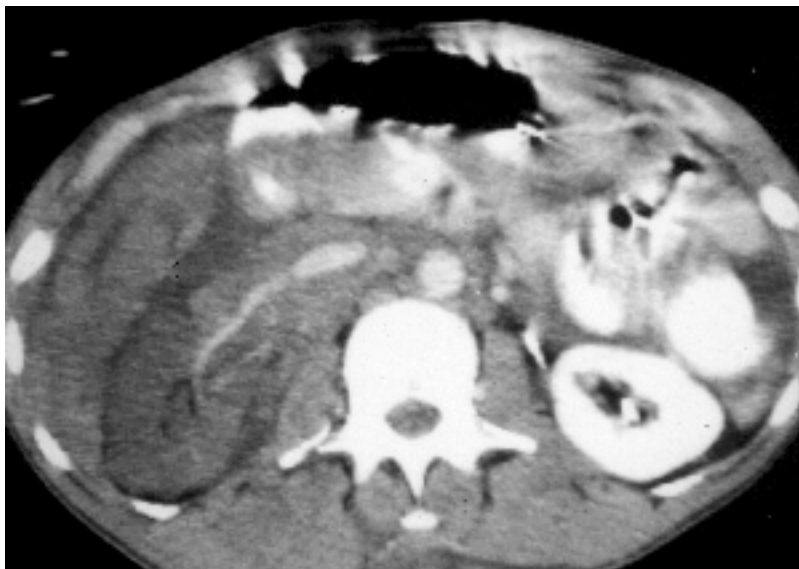


Fig. 100. This is a CT image showing no enhancement of the right kidney in a patient who was involved in a motor vehicular accident. Avulsion of the right renal artery was the cause. A hematoma can be seen in the renal hilum. There is reflux of contrast into the right renal vein from the inferior vena cava. (med.wayne.edu) (Wiki)



Fig. 101. This is an image of a traumatic renal arterial and venous injury in a 23-year-old man with a vascular pedicle injury after a motorcycle collision. This nephrographic phase CT scan shows near total absence of enhancement of the left kidney. Left renal artery (arrow) ends abruptly. There was also left perinephric hematoma, as well as hemoperitoneum from an associate splenic injury, which is not shown. (ajronline.org) (Wiki)

Most patients are injured in motor vehicular accidents. An important component of motor vehicular accidents is the degree of deceleration. Rapid deceleration may cause renal artery thrombosis, renal vein disruption, or renal pedicle avulsion (Fig. 99). The seatbelt, steering wheel, or air bag may compress the renal artery between the abdominal wall and the spine causing a thrombosis. Such a thrombosis of the main renal artery, or vein, results in a devascularization of the kidney (Figs. 99 & 103). What is essential with renal pedicle injuries is the injury be quickly recognized and managed appropriately if preservation of renal function is to be preserved.



Fig. 102. This is a picture of a reno-vascular pedicle injury with renal pelvic ureter junction disruption. (Picture provided by Mr. J C Campbell, Derriford Hospital, Plymouth, United Kingdom) (Wiki)

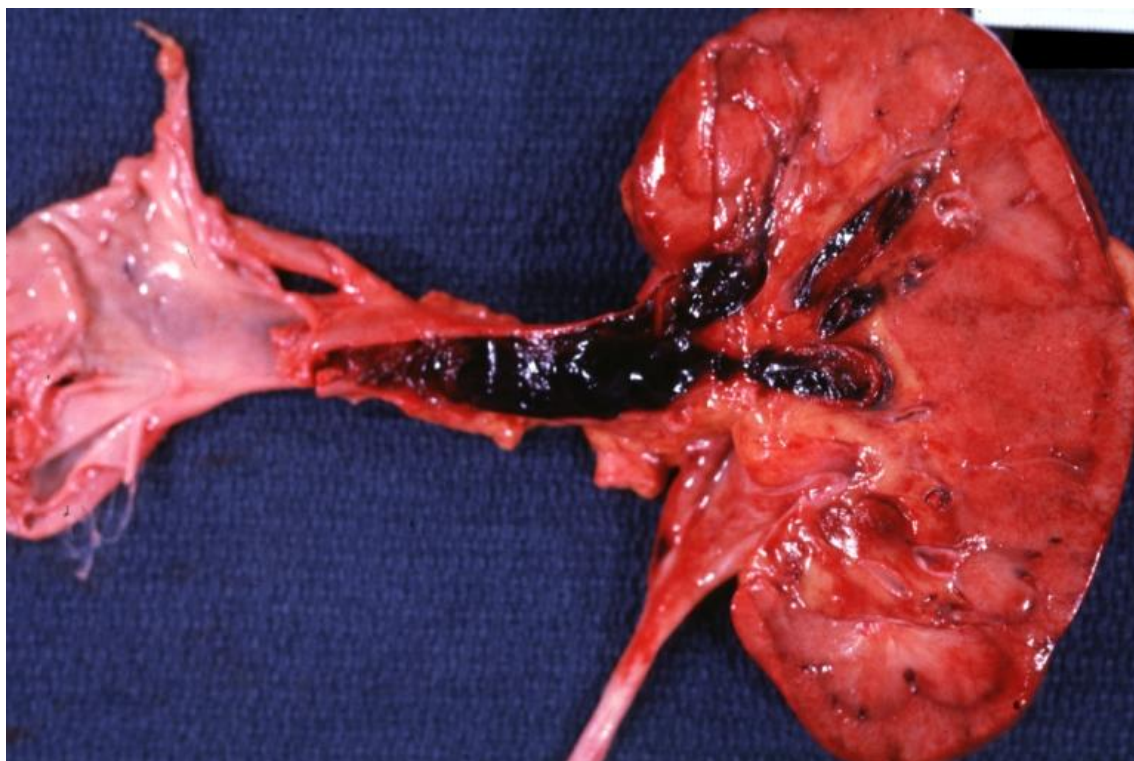


Fig. 103. This image shows a thrombosis completely occluding the renal vein. (Wiki)

Typically, the anatomic location of the kidneys is sufficiently elastic that even with momentary displacement due to blunt force impact, the kidney returns to its original position. However, should the impact result in either a movable or permanent dislocation serious complications can result. One of these complications is ureteral obstruction, which can lead to chronic or intermittent hydronephrosis. This can then lead to the development of pyelonephritis. Another complication of a movable or displaced kidney is the interference with blood flow either through the renal artery or vein (Figs. 104 & 105). If the interference is complete, the kidney will be infarcted. However, if the interference is incomplete, this may lead to renal atrophy (Fig. 106).

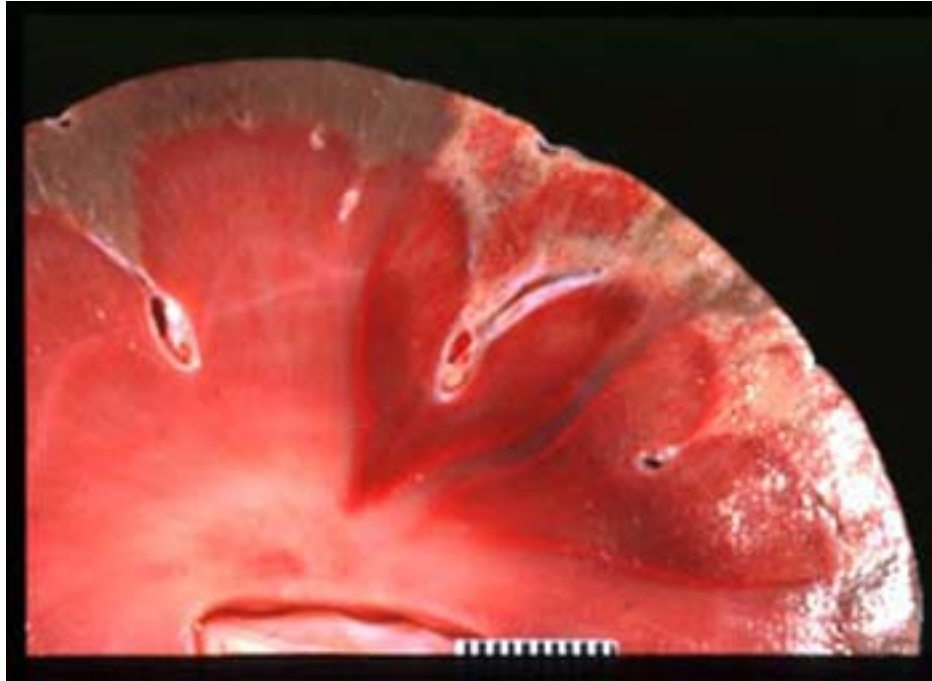


Fig. 104. This is a large infarct, which resulted from thrombosis of a major vessel, most likely the interlobular artery. (kosvi.com) (Wiki)



Fig. 105. This image depicts several triangular infarcts involving the cortex and the outer medulla, which is consistent with an occlusion of an arcuate artery. (kosvi.com) (Wiki)

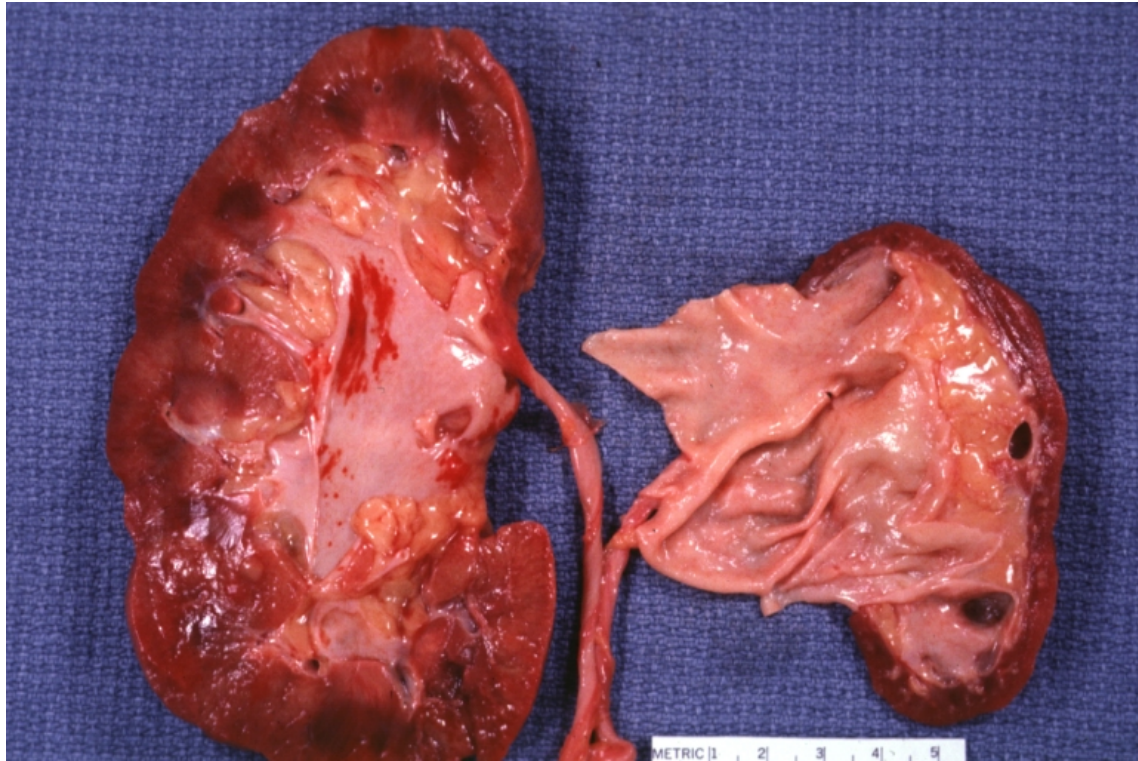


Fig. 106. This image shows a normal sized kidney on the left with a markedly atrophic kidney of the right. The atrophy was due to a severe compromise in blood supply. (Wiki)

If the renal pedicle is driven into the lower thoracic or lumbar vertebral column, the renal artery or vein may be contused, which in turn may lead to a thrombosis of either vessel. Again, as indicated above, this can result in a renal infarction involving much of the kidney. If the impact force is especially severe, the renal artery and or vein can be lacerated resulting in a fatal retroperitoneal hemorrhage with a possible intraperitoneal component. Occasionally, the renal artery will not be completely torn, but will sustain a partial tear leading to a false aneurysm of the renal artery (Figs. 107 & 108).

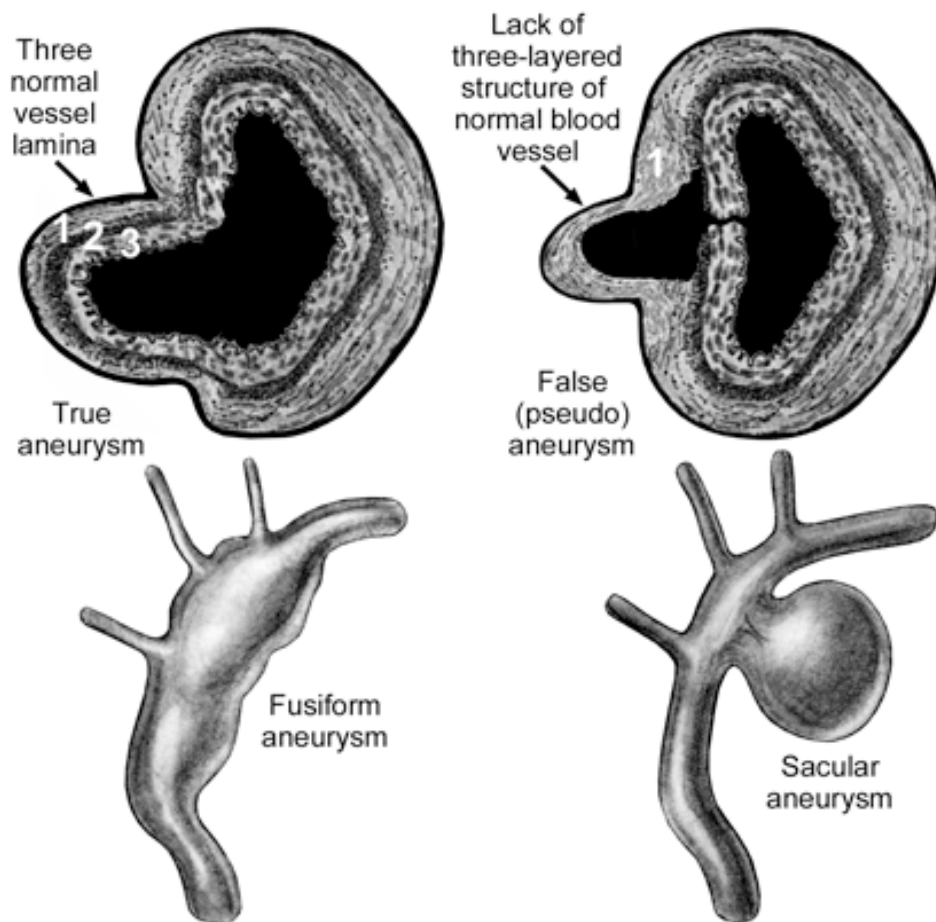


Fig. 107. This is an illustration depicting the types of aneurysms which can involve the renal artery. True aneurysms most often result from blunt force trauma with damage to the internal elastic lamina. The aneurysm wall, although abnormal, contains elements of the three normal vessel lamina. False aneurysms typically result from penetrating trauma. They project from the side of the vessel and lack the three-layered structure of a normal blood vessel. (msdlatinamerica.com) (Wiki)

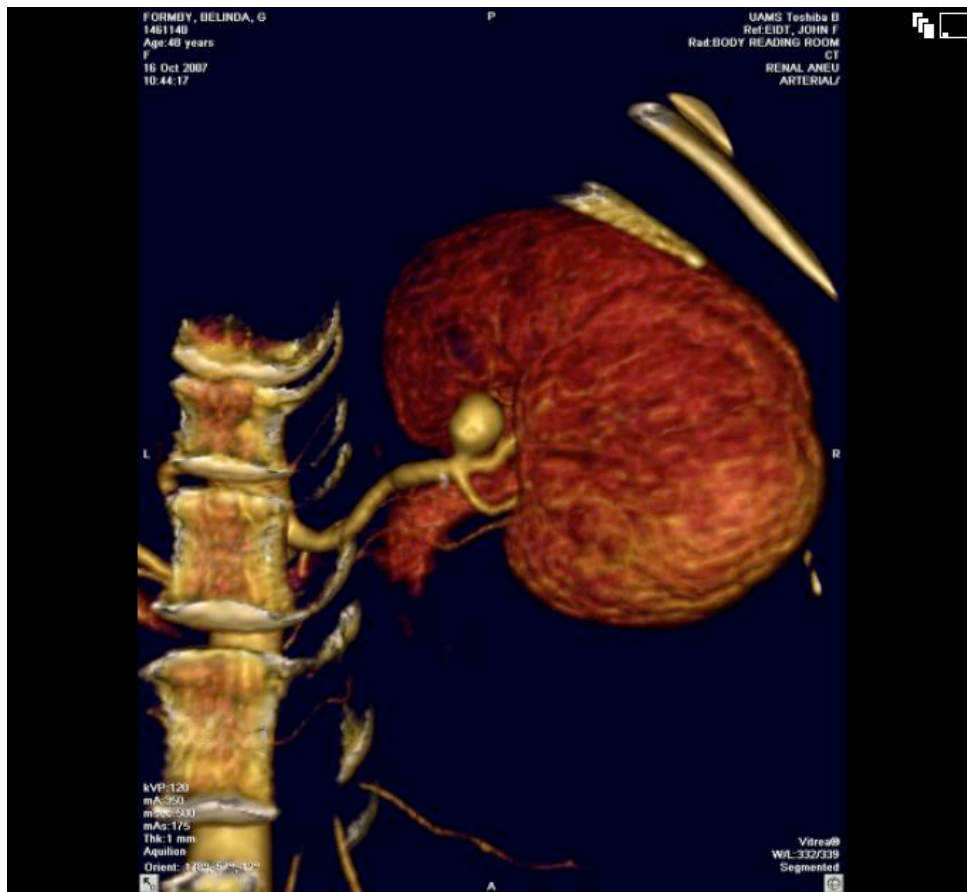


Fig. 108. This image is of a false aneurysm arising from the renal artery. (korean medical library engine) (Wiki)

The first published report of a rupture of a false aneurysm of the renal artery was by Rouppe in 1770. His case involved a sailor who fell onto his right flank. The ruptured renal artery false aneurysm was identified at autopsy. It is important to remember there are other causes of false aneurysms besides trauma, which include anastomotic iatrogenic during endovascular procedures, spontaneous, dissection, Mycotic and Kawaski disease. Remember, a false aneurysm does not include all layers of the artery (Fig. 107), are typically saccular in appearance (Figs. 107 & 108), and can be extra-parenchymal or intraparenchymal. A true aneurysm includes all layers of the artery, usually the result of inherited disorders (Fibromuscular dysplasia and Ehlers-Denlos syndrome), can be fusiform or saccular in appearance (Fig. 107), and 90% of these are

extraparenchymal. It has also been recently recognized that one of the complications to renal transplantation is either a false or true aneurysm.

Another complication of blunt force traumatic dislocation of the kidney is rupture of the extrarenal portion of the pelvis, or the upper portion of the ureter. However, this is believed not likely to occur unless these structures have been weakened by preexisting disease. Conditions such as hydronephrosis (Figs 109 & 110) and pyelonephritis (Figs. 111, 112, 113 & 114) can predispose the renal pelvis to spontaneous rupture, or to rupture with minimal trauma.



Fig. 109. This image is that of a kidney that has undergone extensive dilation due to hydronephrosis. Note the extensive atrophy and thinning of the renal cortex. Hydronephrosis refers to distention and dilation of the renal pelvis and calyces, usually caused by obstruction of the free flow of urine from the kidney. Untreated, it leads to progressive atrophy of the kidney. Hydroureteronephrosis refers to distention of both the ureter and the renal pelvis and calyces. (Wiki)

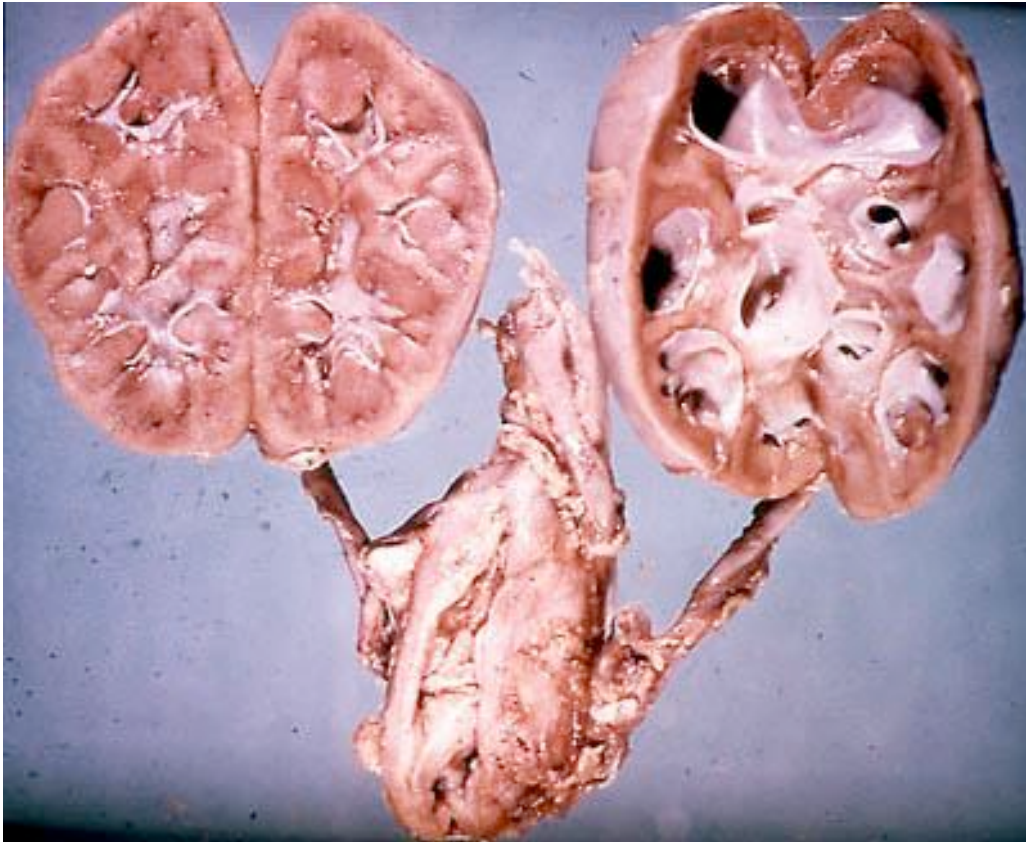


Fig. 110. This is another example of hydronephrosis. The kidney of the left side of the image is normal. The kidney on the right shows an opaque and thickened pelvic mucosa, suggesting chronic obstruction and possible infection. The dilatation involved only one kidney indicating the obstruction was unilateral (e.g., calculus, ureteral stricture, or unilateral obstruction of ureteric orifice). (cueflash.com) (Wiki)

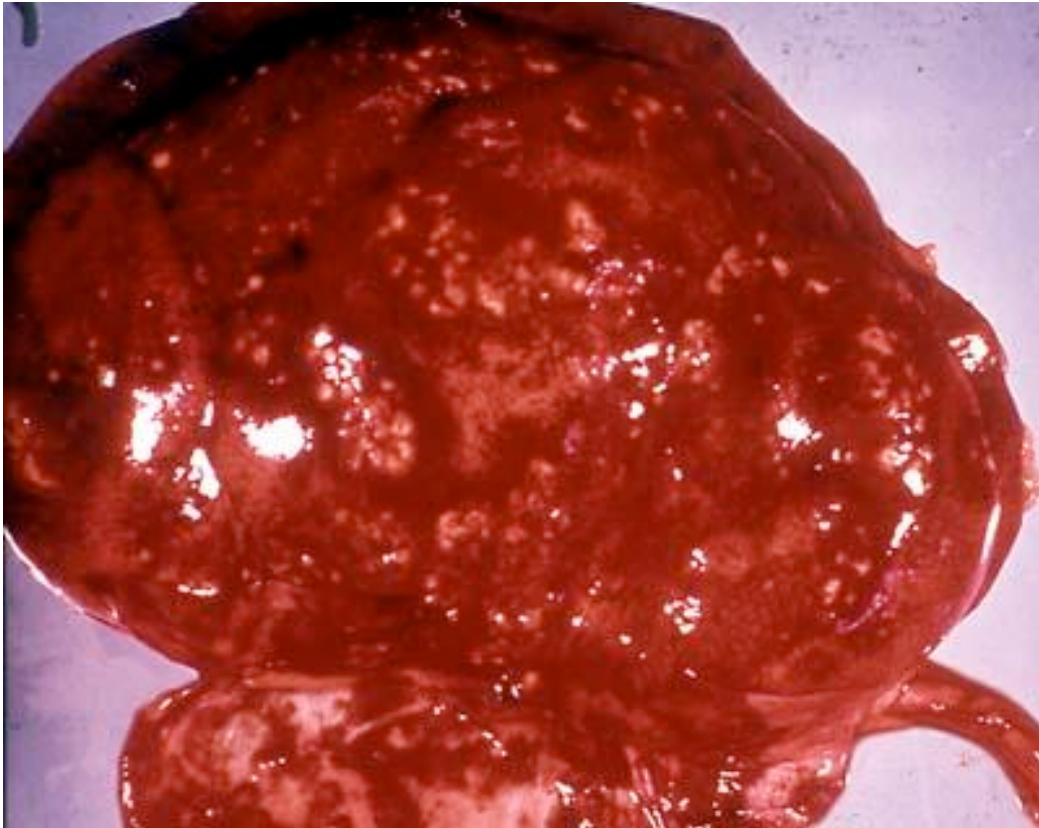


Fig. 111. This is an image of acute pyelonephritis. Note the intense hyperemia and multiple abscesses. (cueflash.com) (Wiki)

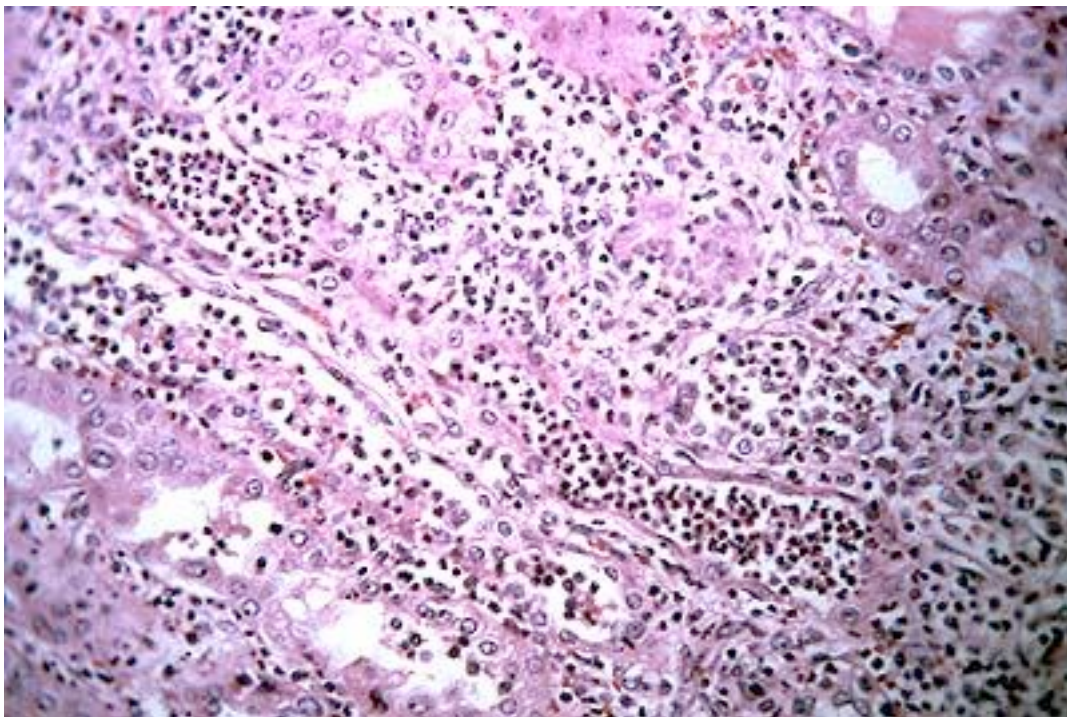


Fig. 112. The above image is a photomicrograph of acute pyelonephritis. Note the tubules are filled with acute inflammatory cells with surrounding congestion. (cueflash.com) (Wiki)

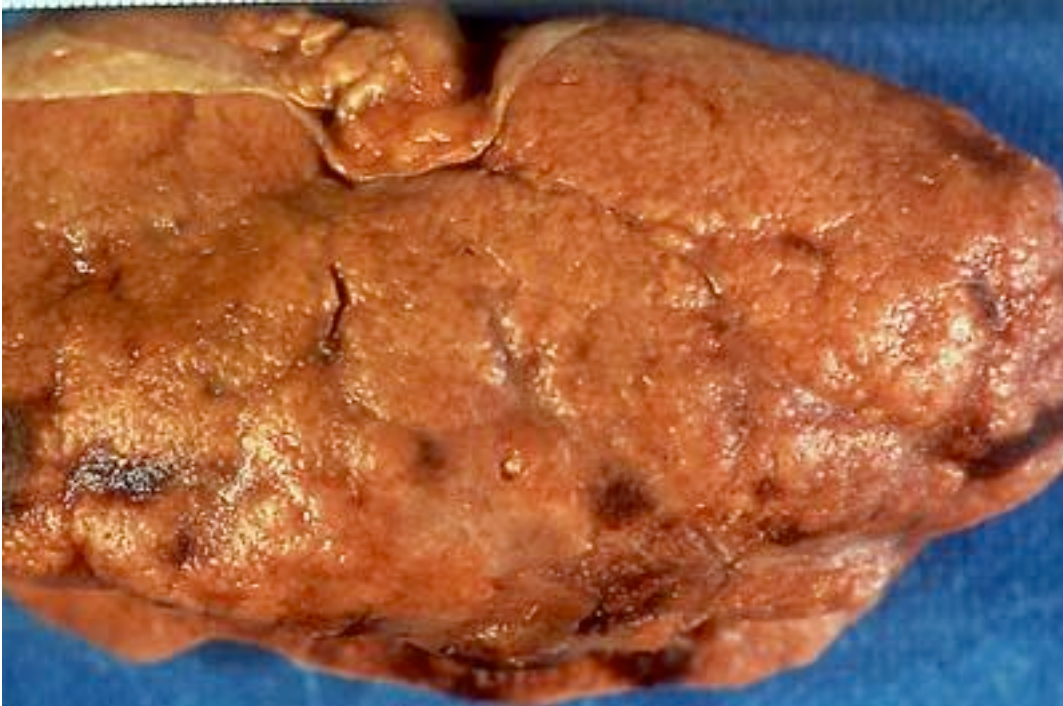


Fig. 113. This image is of a case of chronic pyelonephritis. Note the irregular scars on the surface, as well as the contraction of the kidney. (cueflash.com) (Wiki)

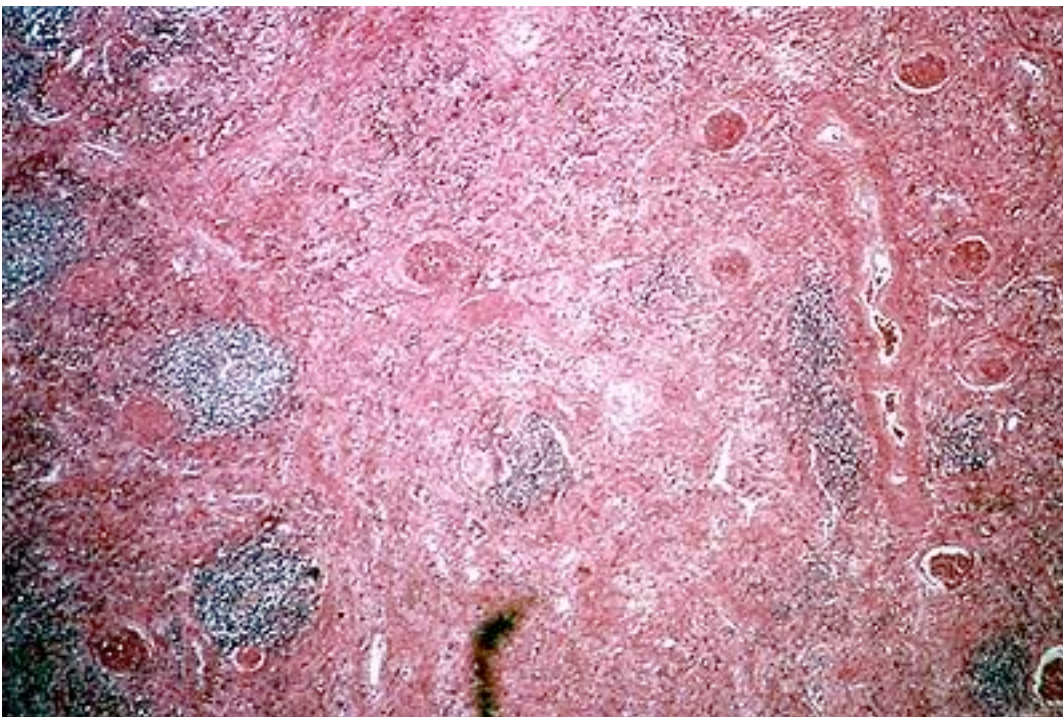


Fig. 114. The above image is a photomicrograph of chronic pyelonephritis. Note the diffuse fibrosis, few glomeruli with periglomerular fibrosis, and numerous lymphoid follicles. (cueflash.com) (Wiki)

It appears that hydronephrosis is associated with a sixfold reduction in the tissues forming the renal pelvis, as well as the immediate adjacent parenchyma. It is also accompanied by a ninefold reduction in the energy absorptive capacity of the immediate adjacent parenchyma as compared with the renal pelvis. Other factors which play a role in hydronephrosis contributing to rupture of the renal pelvis even with minimal trauma are internal pressure, the law of mass variation (the acceleration of tissues induced by the kinetic energy released by the impact force will increase the mass effect of the contained urine within the hydronephrotic pelvis), and the energy absorptive capacity of the surrounding anatomic structures.

Such ruptures must be immediately recognized and treated appropriately, for not being recognized and treated will lead to infection with the development of perinephric cellulitis, or abscess formation (Fig. 115).

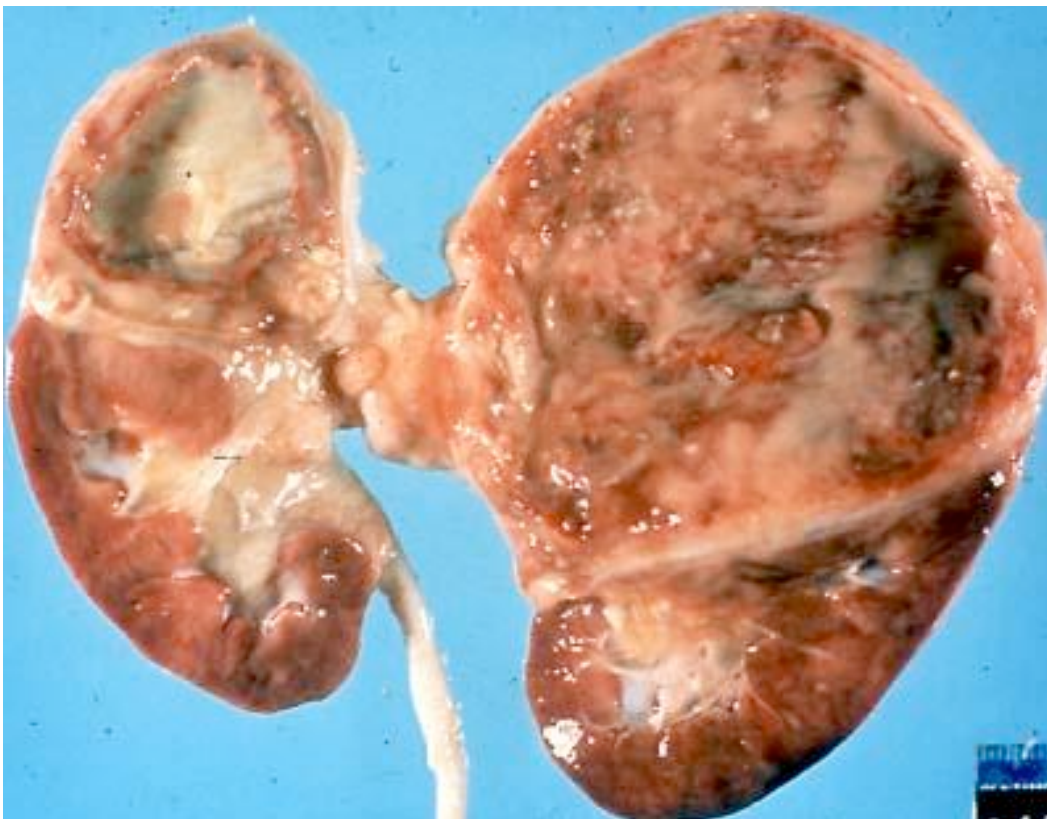


Fig. 115. The above image shows the cut surface of a kidney with a single, large abscess. (cueflash.com) (Wiki)

It is also important to remember trauma itself can cause the development of hydronephrosis. One of the first to report on this was Sutter, who published a series of cases in 1905, in which approximately 14% of kidneys damaged by blunt force trauma, if the victim survived, developed hydronephrosis.

Lastly, renal pedicle injuries are typically associated with other severe abdominal injuries, which according to Strum and colleagues results in a mortality of 50%.

3. Injuries of the Kidneys: One of the initial series of cases involving traumatic injuries of the kidneys was by Delbet in 1901. This was followed by Watson in 1903, Suter in 1905, Lardennois in 1908, and Connell in 1911. Connell series of 147 cases brought the total number of cases reported by 1911 to 875.

There are fundamentally four types of blunt force traumatic injuries which can involve the kidneys: **contusions, lacerations, avulsion, and partial or total fragmentation (pulpfaction)**. These injuries may be brought about through: (1) direct trauma, such as kicks; falls on to objects, such as furniture, rocks, etc.; and motor vehicular accidents either due to crushing or deceleration. (2) indirect trauma, such as stressful horseback riding; falling from a height and landing on one's feet. (3) and those due to strenuous muscular action, such as heavy weight lifting; sudden movement to catch an object or dodge a blow; and springing backward.

In 45 out of the 770 cases reviewed by Lardennois, the cause of the rupture was a shock wave or blow to the anterior abdominal wall, and in only 3 of these cases were other abdominal organs injured.

What became apparent from these early series of cases is the force or blow need not be very severe to cause serious injury, even complete rupture, of the kidney. For example, Gargam reported in 1881, a case of rupture of the kidney occurring from an individual striking against a table. In another case cited by Voit, a women, while dancing with her husband, was held very tightly about the waist, felt a sever pain in her right side. It subsequently was discovered her right kidney had ruptured.

a. Contusions: This is the most common blunt force traumatic injury to the kidney, accounting for 75 to 85% of cases. It may be focal or diffuse. Typically, focal

contusions occur on the posterior surface or near the lower pole (Fig. 95, p 103). It is not uncommon for there to be multiple focal contusions on the posterior surface; their configuration is consistent with them having been produced by the impact against the ribs. Such contusions are consistent with Morris's hypothesis previously discussed (p 104). These contusions may be represented by an area of subcapsular hemorrhage (Figs. 99 & 116) to a far more serious lesion in which there is underlying cortical infarction (Figs. 104 & 105).

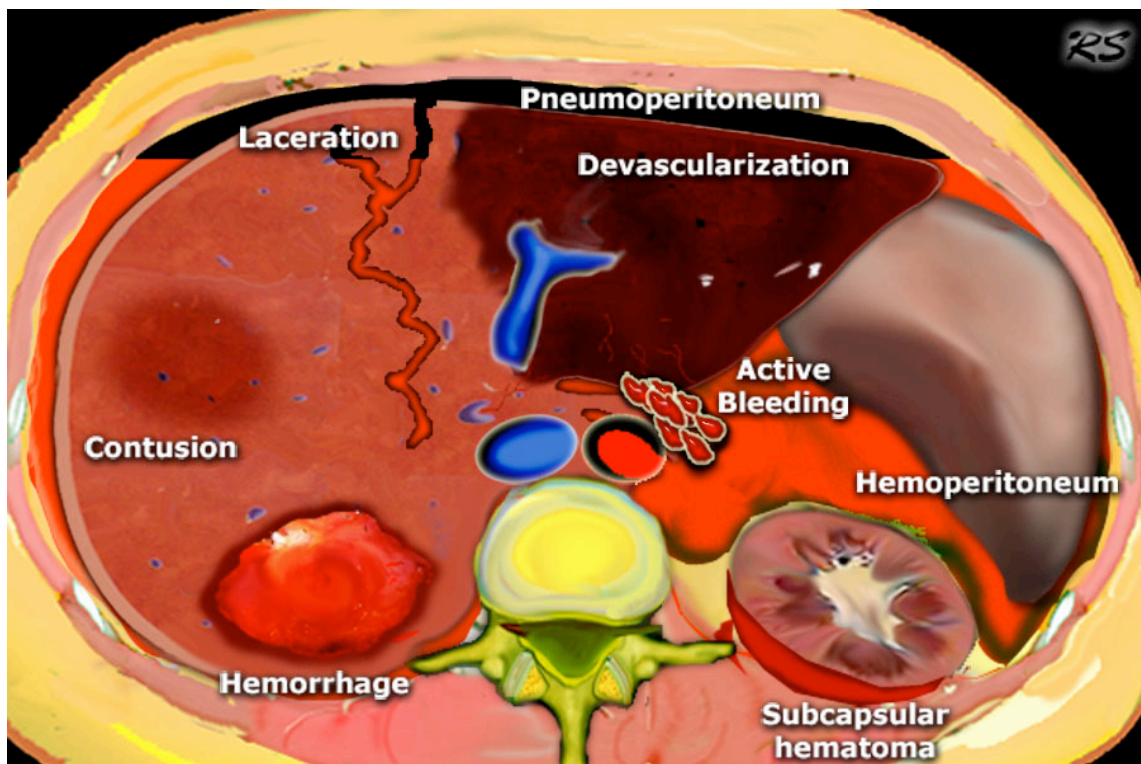


Fig. 116. In this illustration a number of abdominal injuries are demonstrated, one of which is a subcapsular hematoma. (radiologyassistant.nl) (Wiki)

b. Lacerations: The location of lacerations are much like contusions in that they tend to occur on the posterior surface and usually nearer the lower pole than the upper. Unlike contusions, they also have a propensity to occur along the lateral surface. Typically, lacerations are due to blunt impact to the posterior surface of the trunk in the region of the loin (Fig. 94, p 103), or along the lateral surface of the lower rib cage. There are five types of lacerations: **capsular, subcapsular, transcapsular,**

transrenal, and central.

(1). **capsular lacerations** are not uncommonly associated with contusions and like contusions involve only the capsule with no underlying cortical involvement (Fig. 95, p 103).

(2). **subcapsular lacerations** occur beneath an intact capsule and do not extend to the pelvis as does a **transcapsular laceration**. The resulting hemorrhage is confined to the kidney. Typically, it is not associated with gross hematuria. Also, it is not uncommon for this injury in-of-itself not to be recognized clinically, however, the contusion of the overlying muscle is often recognized (Figs. 99, p 109 & 117).

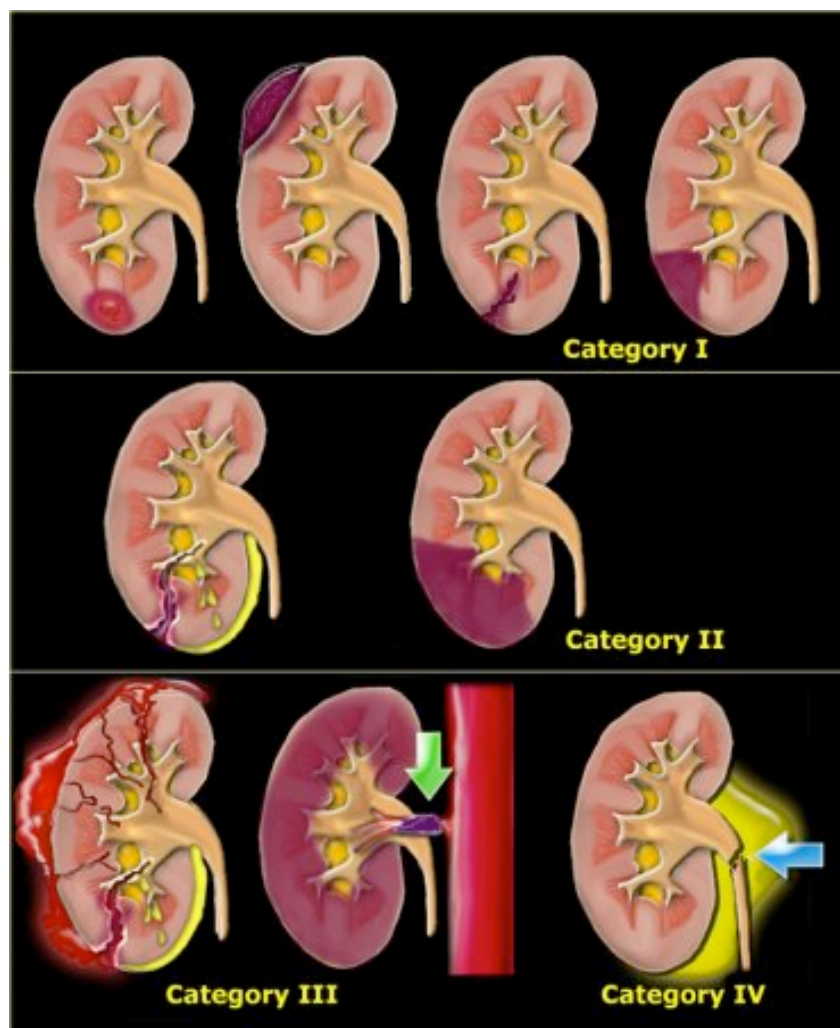


Fig. 117. The above figure shows the four categories of renal injuries as given by Michael Federle:

1. Minor injury: renal contusion; intrarenal and subcapsular hematoma; minor laceration with limited perinephric hematoma without extension to the collecting system or medulla; and small subsegmental infarct.
 2. Major injury: major laceration into medulla or collecting system; and segmental infarct.
 3. Catastrophic injury: maceration of the kidney; and total devascularization due to arterial occlusion.
 4. Rupture of collecting system.
- (radiologyassistant.nl) (Wiki)

(3). transcapsular lacerations are those in which the laceration involves both the capsule and underlying cortex (Fig. 99, p 109). Such lacerations are associated with significant hemorrhage, which leads to a hematoma that can involve the adjacent perinephric region (Figs. 99, p 109 & 117). If such a lesion becomes infected it can lead to an abscess. What is important to remember is such lacerations can cause an infarction of the adjacent cortex (Figs. 104 & 105, p 114). *Remember, the parenchyma of the kidney has a segmental arterial supply with blunt injuries tending to disrupt the parenchyma along the planes between segmental vessels, whereas penetrating injuries cross segmental vessels.*

It is important to keep in mind segmental infarctions can occur without associated lacerations. Such infarctions are caused by thrombosis, dissection, or laceration of an accessory capsular artery, or intrarenal segmental branch. These segmental infarctions are often multifocal and are usually associated with other renal injuries.

(4). transrenal lacerations extend from the capsule to the renal pelvis. It is not uncommon for these lacerations to extend across the lateral margin dividing the kidney into two separate parts, and upper or lower, or one pole may be completely avulsed from the rest of the kidney. Clinically, the patients present with gross hematuria. This is associated with blood and urine extravasation into the retroperitoneal space and pelvis. Although, it is difficult to comprehend, some of these cases have been associated with spontaneous healing without surgical intervention (Figs. 99, p 109 & 117).

(5). central laceration is a laceration, which originates in the calyx but does not extend through the cortex. The bleeding from such an injury is substantial, forming a hematoma either in the renal pelvis with possible extension into the ureter, or into the peripelvic fat. If inappropriately treated, the intrapelvic and or intraureteral coagulated blood can lead to hydronephrosis. If the laceration involves a large segmental renal artery or vein a segmental infarction can arise.

There are two possible complications of traumatically induced renal infarctions (Figs. 104 & 105, p 114). It is not uncommon for what initially appears to be a small laceration, which originally has a small area of infarction, to be accompanied by a large area of ischemic necrosis, the area of which exceeds what the lacerated vessel should have caused. The other oddity is the continued enlargement of the original area of ischemic infarction over the space of several weeks following the original injury. The progressive enlargement appears to evolve in intervals, such that microscopic examination shows the oldest area of infarction to show evidence of healing, whereas the most peripheral zone shows an infarction, which is far more acute. In some of these cases retrograde thrombosis of the originally injured vessel provides the explanation for these sequential areas of infarction, however, in some cases there is no retrograde thrombosis or explanation.

c. Avulsion of the ureteropelvic junction: These injuries occur because of sheering stress at the renal pelvis. The ureter has two fixation points, the proximal ureteropelvic junction and the distal ureterovesical junction. At this point we will address only the ureteropelvic junction. With sudden deceleration and the resulting hyperextension, the ureter is tensed across the transverse process, resulting in a laceration or avulsion of its proximal point of fixation (Figs. 99, p 109, 117 & 118).

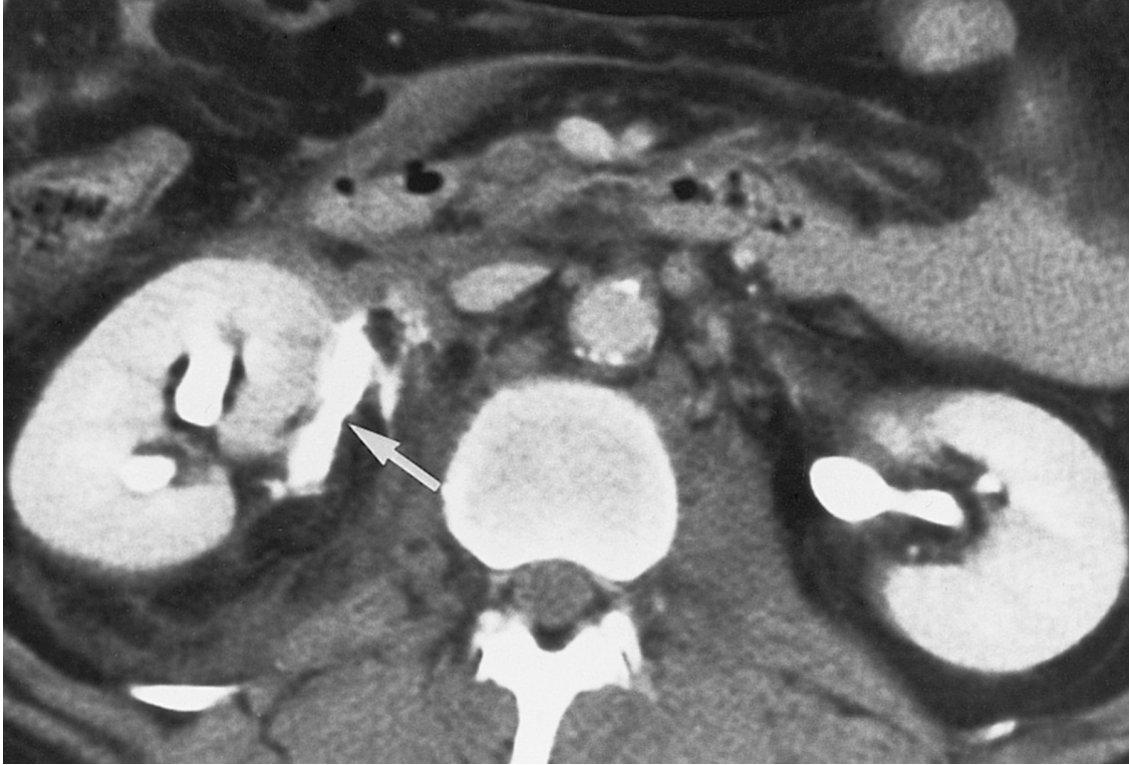


Fig. 118. This is a helical CT scan in a 49-year-old man who sustained blunt trauma causing avulsion of the ureteropelvic junction (category IV) injury. The above scan shows medial contrast material extravasation (arrow). (radiographics.rsna.org) (Wiki)

d. Partial or complete fragmentation of the kidney: This occurs when the kidney is fractured into multiple fragments. Such fragmentations are associated with one or more devitalized areas, injuries to the collecting system, severe hemorrhage, and active arterial bleeding. However, there are some instances in which such fragmented kidneys are associated with little bleeding, which is believed to be due to early thrombosis. Also, there are some cases in which the lacerations causing the fragmentation, run parallel to intervascular tissue planes and thus, do not tear any of the major arteries or veins, resulting in a completely amputated upper and lower renal pole that still have an intact blood supply (Figs. 99, p 109 & 119).

In those rare cases in which the fragmented kidney is not treated, typically the infarcted segments undergo organization, so much so, that all that remains is pigmented fibrous tissue with scattered areas of atrophic renal casts.

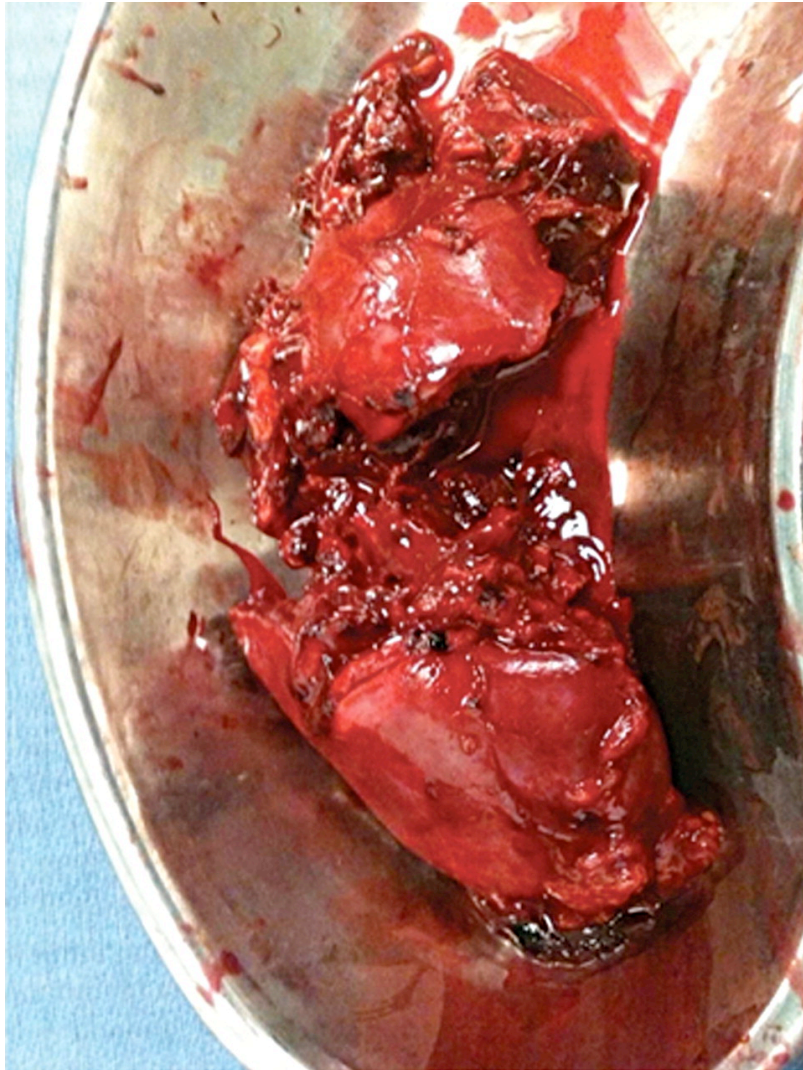


Fig. 119. This is an example of a fragmented kidney, which occurred in a 22-year-old male who presented to the emergency department four hours following an injury sustained during football practice. (casereports.bmj.com) (Wiki)

The most accepted classification system for traumatic renal injuries is that of the AAST (American Association for the Surgery of Trauma) grading system, which is based on findings at the time of surgery. An illustration of this grading system is seen in Fig. 99, page 109, along with a brief description. The AAST renal grading system is as follows: Grade I: Contusions, which are the most common type of renal injury, accounting for 75 to 85% of cases and include hematuria (either microscopic or gross), normal urologic findings, and includes non-expanding subcapsular hematomas with no associated laceration (Figs 95, p 103, 99, p 109 & 117).

Grade II. Includes non-expanding perinephric hematomas confined to the retroperitoneal space and superficial cortical lacerations measuring less than 1 cm in depth, with sparing of the collecting system (Figs 99, p 109 & 117).

Grade III. Includes renal lacerations deeper than 1 cm in depth that extend to the medulla, without involvement of the collecting system (Figs. 99, p 109 & 117).

Grade IV. Includes renal lacerations extending through the kidney into the collecting system, injuries involving the main renal artery or vein with contained hemorrhage, and segmental infarctions without associated lacerations (Figs. 99, p 109, 104 & 105, p 114 & 117).

Grade V. Includes shattered (fragmented) kidney (Figs 99, p 109 & 119), partial tears or complete laceration (avulsion) of the ureteropelvic junction (Figs. 99, p 109, 117 & 118), and thrombosis of the main renal artery or vein with devascularization of the kidney (Figs. 99, p 109, 103, p 113, & 117).

Penetrating Trauma to the Kidneys: Penetrating trauma accounts for approximately 10% of all renal injuries and is most commonly caused by gunshot or stab wounds (Figs. 91, p 100, 92, p 101 & 120) except for the few iatrogenic injuries resulting from renal biopsy or other medical procedures.

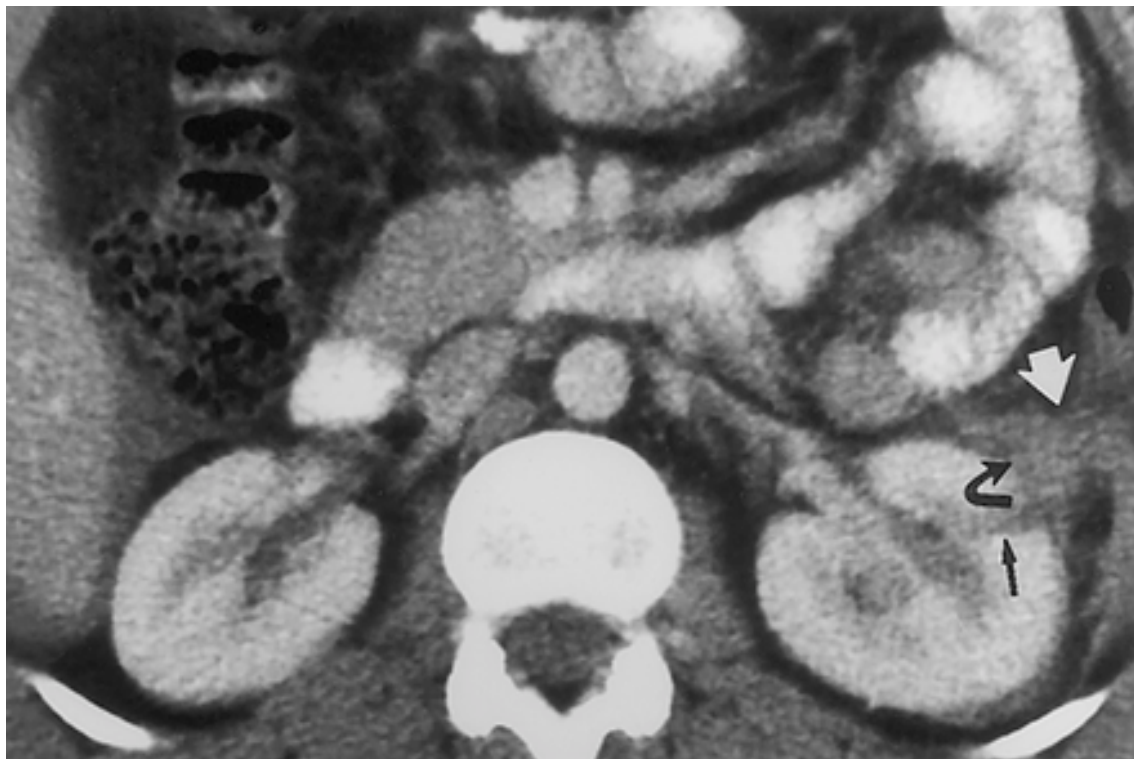


Fig. 120. The above is a transverse CT image showing a laceration (straight black arrow) in the upper pole of the left kidney. There is an associated hemorrhage in the left perirenal (curved arrow) and anterior pararenal (white arrow) spaces. The patient was treated conservatively, without surgery, and recovered satisfactorily. (radiology.rsna.org) (Wiki)

The mechanism of injury consist of direct damage to the parenchyma, excretory system, or vascular structures and violation of the peritoneum. Penetrating injuries are often associated with non-sterile conditions hence, there is a high incidence of infection and possibly the establishment of a urinary fistula. The resulting infection is typically, pararenal rather than intrarenal thus, it can lead to a pararenal abscess or peritonitis. If the penetrating wound involved the renal pelvis, then a urinary fistula may develop. Regarding gunshot wounds, the extent of renal damage is not determined by the size of the entrance wound, but by the velocity of the missile. Low-velocity missiles cause a small “permanent cavity” type of injury, much like a stab wound, with the kinetic energy transferred to the tissue next to the wound path being much less than the extensive damage induced by the wound produced by a high-velocity missile (Figs. 89 & 90, p 99). However, there is an exception to this statement; the destruction caused by a close range shotgun blast (low-velocity) is analogous to that produced by the destruction caused by a high-velocity missile (Fig. 121). The cavitation produced by a high-velocity missile may cause delayed necrosis of the adjacent non-penetrated tissue. In essence there are two mechanisms of injury from a missile, the “permanent cavity” produced by the missile passing through the renal parenchyma and the stretching of the surrounding tissue (temporary cavity) produced by the released kinetic energy. The temporary cavity created by the kinetic energy of a high-velocity missile is about 30 to 40 times larger than the size of the permanent cavity. *Remember, the kinetic energy, hence the tissue destruction, is determined by the square of the velocity of the missile, not its mass.* Also, a secondary effect of both the permanent and temporary cavities is the induction of a wide zone of tissue destruction due to infarction.

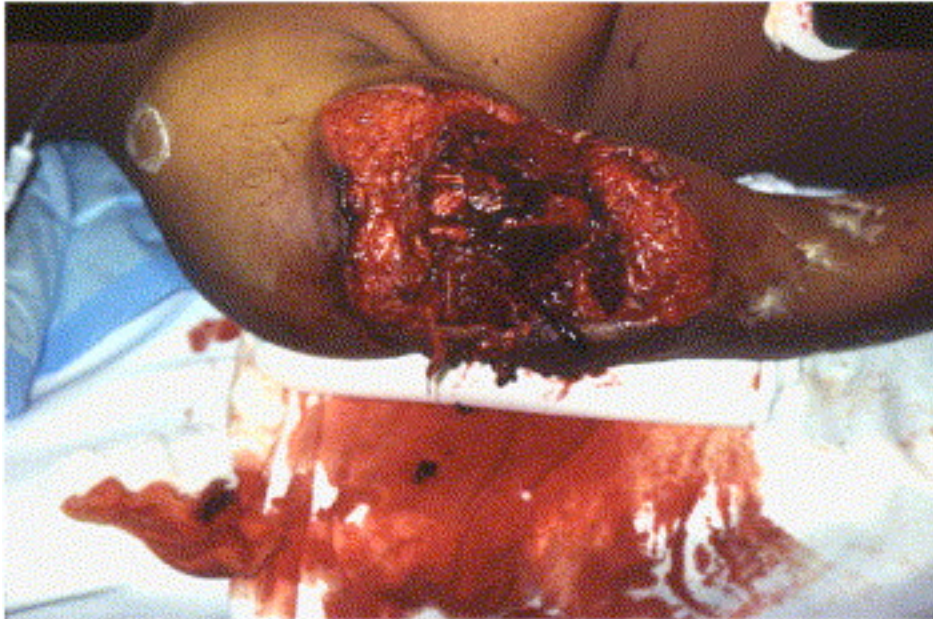


Fig. 121. This is an example of a close range shotgun wound, which gives one the impression of tissue injury due to a high-velocity missile. (sciencedirect.com) (Wiki)

Pediatric Injuries: In the pediatric age group blunt force trauma to the abdomen involves renal injury in 10 to 20% of cases. Renal trauma accounts for 1.6% of total injuries with 90% of these injuries being due to blunt force trauma. If you include the posterior abdomen and retroperitoneum in the definition of blunt abdominal trauma, then the kidney is not an uncommonly injured solid organ in pediatric blunt abdominal trauma (Fig. 125).

The increased risk of the child's kidney to blunt force trauma is due to several factors. It is proportionately larger within the abdomen, and is less protected due to a thinner perirenal fat cushion, underdeveloped flank muscles, and less ossification of the overlying ribs. Those children whose kidneys have persistent fetal lobulations have an increased propensity to disruption following blunt force trauma (Fig. 122). Also, the right and left pediatric kidneys differ in their susceptibility to certain types of injury. *The right kidney is better protected by the overlying right lobe of the liver, however, it has a larger bare area and is more mobile. Thus, the right kidney will escape minor crash forces, but will sustain injury following a severe compressive force. The left kidney is not*

covered by the left lobe of the liver, is less mobile due to its additional ligamentous attachments (Splenorenal and phrenocolic) and has a small bare area. Thus, it is more prone to minor injuries.

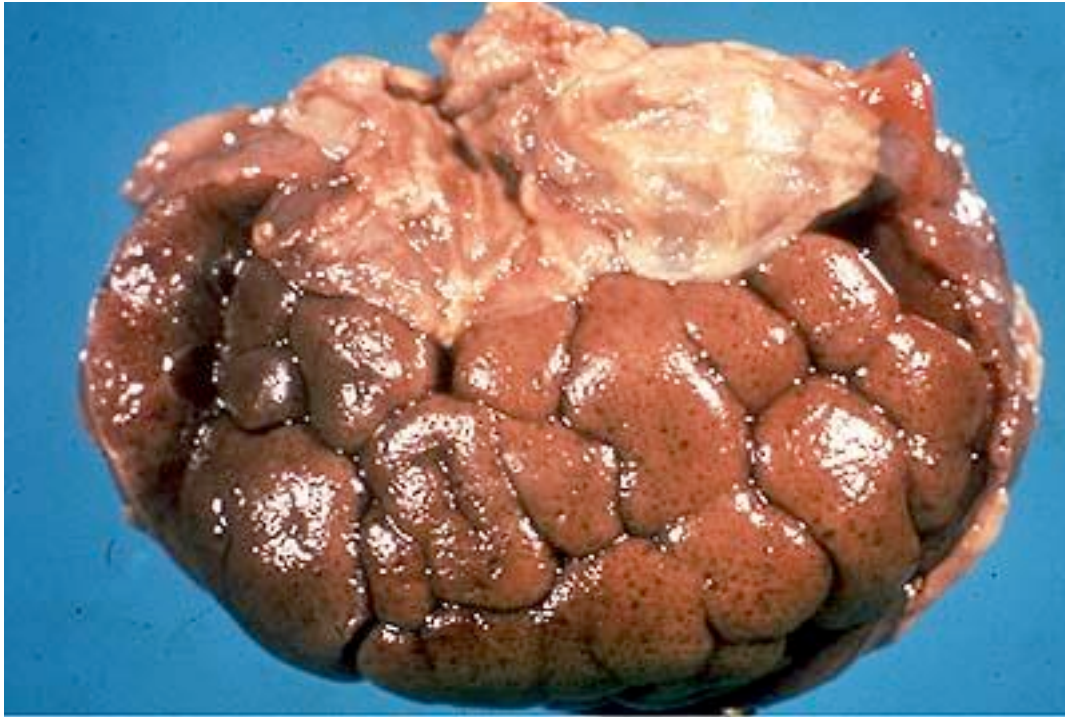


Fig. 122. This image is of a newborn kidney showing normal fetal lobulation. (cueflash.com) (Wiki)

The presence of a preexisting renal abnormality, as in the adult, predisposes it to severe injury even following minor trauma; if the preexisting abnormality includes a dilated renal pelvis, the site of bleeding is typically the arcuate artery. Ectopic renal location can make the kidney more susceptible to injury. If the kidney overlies the spine as in cross-fused ectopia or horseshoe kidney, it is susceptible to the same types of injury as the pancreas (Figs. 123 & 124).

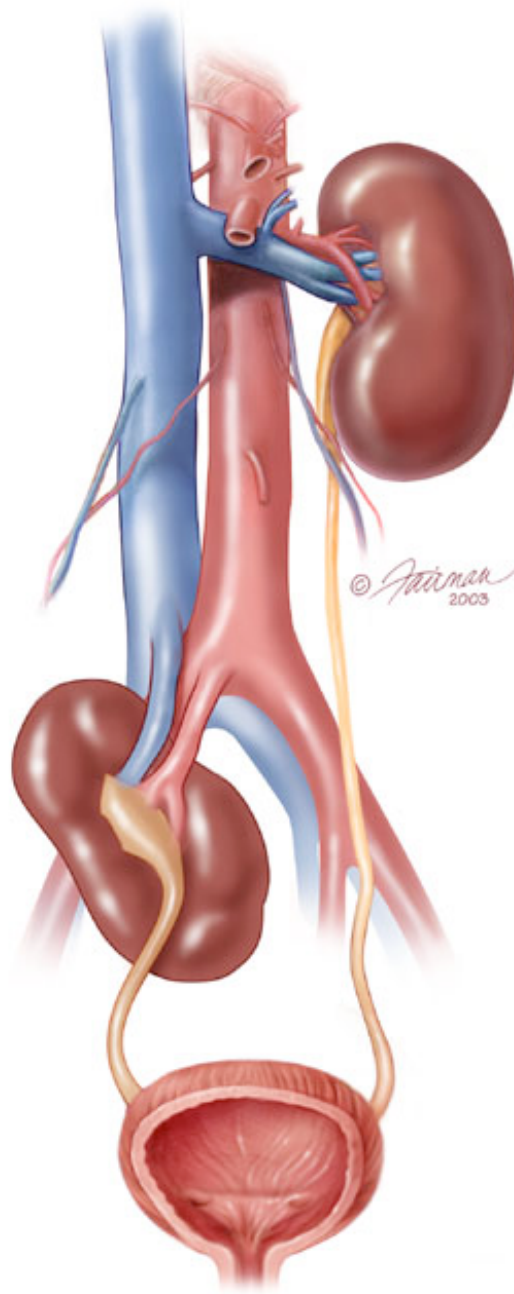


Fig. 123. This is an illustration of an ectopic kidney being retained in the pelvis. (Korean medical library engine) (Wiki)

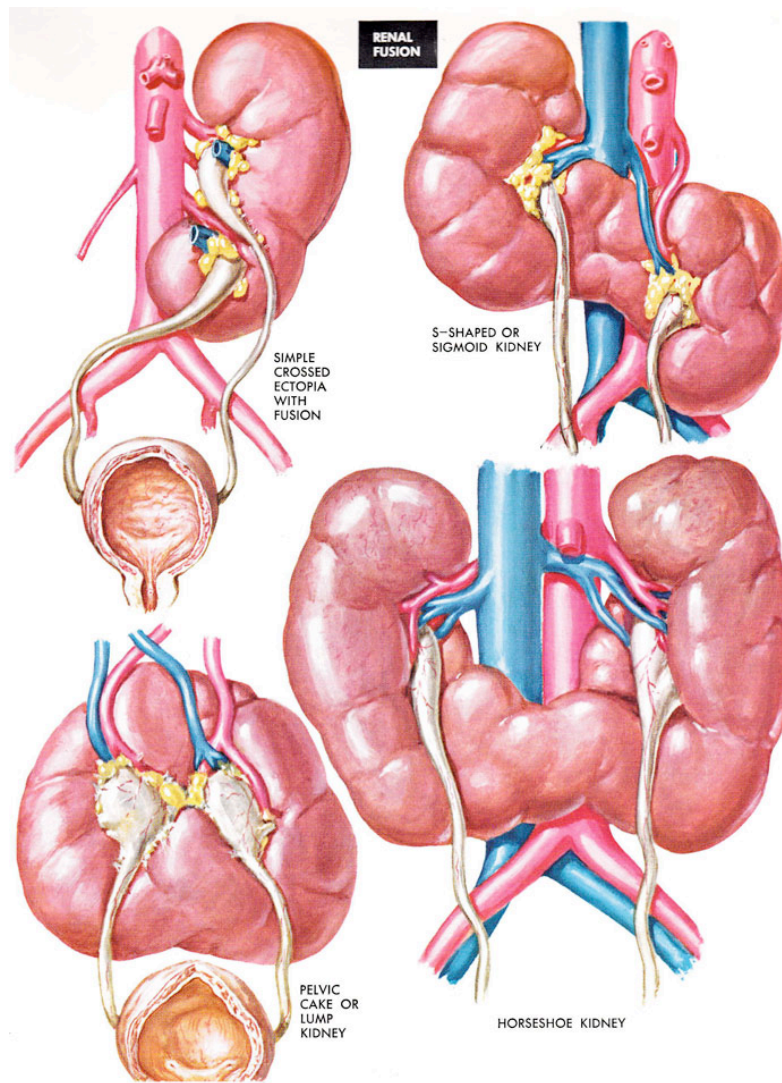


Fig. 124. This is an illustration showing a simple crossed-fused ectopia, a sigmoid kidney, pelvic case of lump kidney and a horseshoe kidney. (Korean medical library engine) (Wiki)

The most common cause of pediatric renal blunt trauma is a motor vehicular accident. As in the adult, most renal injuries are minor, consisting of simple contusions or subcapsular lacerations with an intact overlying capsule (Figs. 99, p 109 & 116, p 124). Occasionally, the capsule may be lacerated, but even in these cases the bleeding is contained within the perirenal fascia. Only rarely does serious, life threatening injury occur, such as renal avulsion or complete fragmentation (Figs. 99, p 109, 117, p 125, 118, p 128 & 119, p 129).

Non-accidental trauma (child abuse) rarely results in direct renal injury. In child abuse, functional renal abnormalities are more commonly due to a metabolic imbalance from major trauma, such as shock or myoglobinuria due to muscle breakdown. Renal traumatic injury, as in the liver, is rarely caused by a fractured rib. However, direct blows to the kidney can result in parenchymal lacerations and discontinuity of the collecting system and hemorrhage, which may extensively involve the retroperitoneum. As in the adult, the primary aim of clinical management is to prevent mortality, conserve the kidney, and reduce immediate and long-term morbidity. In the pediatric age group, clinical management in a hemodynamically stable child is conservative, with monitoring and follow-up imaging as warranted from the grade of the injury as determined by the initial CT (Fig. 125).

Figure 1. Kidney Injury.

Image from the abdominal CT scan of a 12-year-old boy who presented with gross hematuria after falling down multiple stairs at school. Note the ruptured left kidney (thin arrows) with surrounding hematoma (thick arrow). The pediatric surgeons who admitted this child successfully managed this injury non-operatively in a pediatric intensive care unit.



Fig. 125. The above image as the caption indicates is an abdominal CT scan of a 12-year-old boy who ruptured the left kidney after falling down the stairs at school. The kidneys have been reported to be the most commonly injured solid organ in pediatric blunt abdominal trauma, if the retroperitoneum and posterior abdomen are included as already stated. However, more recent studies suggest that kidney injuries are less common than other solid organ injuries. The most common etiology of renal injury is the motor vehicle collision. In a 1994 study of pediatric blunt abdominal trauma, 28 % of 1754 patients were found to have a renal injury. Kidney injuries are most commonly associated with deceleration mechanism or blunt trauma to the trunk. The isolated injury seen in this image is uncommon, as renal injuries are usually associated with other injuries. (ebmedicine.net) (Wiki)

Incidental genitourinary anomalies in renal trauma do not seem to have an impact on recovery from the injury itself. *Since blood pressure is not a reliable clinical indicator in children, serial hemoglobin and hematocrit levels should be monitored to keep track of continued blood loss.* Most renal injuries do not require surgical intervention.

Cortical scars may be the result of the healing process in severe contusions, however, they rarely cause significant functional abnormalities. Occasionally, traumatic injury to the renal pelvis may result in a persisting urinoma (a cystic collection of extravasated urine) (Fig. 126).



Fig. 126. The above image is a CT scan of a kidney which shows a septated fluid collection in the posterior-inferior aspect of the left perinephric space (urinoma) with leakage of contrast material around the inferior pole of the left kidney (peripelvic extravasation). (Indian Journal of Cancer. Urinoma: a cystic collection. 2011; 48 (4): 516-517) (Wiki)

E. Ureter: Trauma to the ureter is rare, accounting for less than 1% of all urologic traumatic injuries. However, undiagnosed ureteral injuries can result in significant morbidity and mortality. The rarity of ureteral trauma is due to the fact it is well protected in the retroperitoneal space by the bony pelvis, psoas muscles and vertebrae. However, it has two vulnerable fixation areas, which are fixation points that have been previously discussed. One is at the ureterovesical junction. As previously pointed out, with sudden deceleration and its accompanying hyperextension, the ureter is tensed across the transverse process, resulting in laceration or avulsion of its proximal point of fixation (Figs. 117, p 125 & 118, p 128). Also, the left ureteropelvic junction is posterior to the ligament of Treitz, pancreas and duodenal-jejunal junction. The inferior mesenteric artery and gonadal vessels cross in front of the left ureter at the inferior pole of the left kidney. The right ureteropelvic junction lies posterior to the duodenum and just lateral to the inferior vena cava, with the right colic and ileocolic vessels crossing in front. Hence, due to these anatomical relationships, isolated injuries to the ureter are uncommon. Typically, ureteral injuries are associated with significant collateral damage. Historically, the first reported case of trauma to the ureter was by Alfred Poland in 1868. This case involved avulsion of the right ureter below the renal pelvis. The victim had been pinned between a platform and a railway carriage. In 1884, Henry Morris reported on 13 cases of rupture of the ureter and 3 cases of penetrating wounds. In 1901, Morris reported on 8 additional cases of injuries to the ureter. In 1904, Morris described the first ureteral procedure, a ureterectomy, on a 30-year-old male who “fell from his van” catching one of the wheels across his right loin. Blauel, in 1906 reported 12 cases of injuries to the ureter. In 1981, Kirchner reported the first bilateral ureteral injury and repair secondary to a single low-velocity penetrating missile.

Traumatic injuries to the ureter are the result of **non-penetrating blunt force trauma** or **penetrating trauma**. When all penetrating and non-penetrating genitourinary traumatic injuries are evaluated, the ureter was damaged in less than 4% and 1% of cases

respectively. Approximately, 10 to 20% of ureteral injuries are caused by blunt trauma, and within this category, motor vehicular accidents predominate. Those traumatic injuries due to **non-penetrating trauma** are most commonly **iatrogenic**. The iatrogenic causes are typically associated with abdominopelvic surgery, or ureteroscopy. Abdominal hysterectomy at one point was the most common cause of iatrogenic injury. Approximately 52 to 82% of surgical ureteral injuries occur during gynecologic procedures, with hysterectomy accounting for most of these cases. Ureteral injury occurs in 1.3 to 2.2% of abdominal hysterectomies, however, it occurs in only 1.3% and 0.3% of laparoscopy and vaginal hysterectomies respectively. In iatrogenic traumatic injuries, it is the distal ureter, which is most commonly injured, typically around the infundibulopelvic ligament, or where the ureter passes inferior to the uterine artery. After gynecologic procedures, colorectal surgery is the next most common cause of iatrogenic ureteral injury, with the left ureter being more commonly involved than the right.

The ureters may be traumatically injured during vascular surgery, the incidence of which has been reported as 2 to 4%.

Ureteral injuries occurring during urologic procedures account for 42% of all iatrogenic injuries. Endoscopic procedures accounted for 79% of injuries, while open surgery accounted for 26%. Again, most of these injuries occurred in the distal ureter (87%). Other surgical procedures associated with injury to the ureters are spinal disc surgery, vaginal surgery for pelvic prolapse, and appendectomy.

Non-iatrogenic blunt force trauma accounts for approximately 4% of traumatic injuries. Such injuries involve deceleration or acceleration mechanisms, typically, causing a partial or total avulsion at the ureteropelvic or ureterovesical junctions (Figs. 127 & 128). These injuries can result from high-speed motor vehicular accidents, falls from a substantive height, or a direct blow to the L2-L3 vertebrae.

There is another type of non-iatrogenic blunt force traumatic lesion, which is referred to as a **straddle injury**. These are more commonly seen in children than adults (Fig. 129). These are injuries produced when the person falls, striking the urogenital area with the force of their body weight. Injury is caused by the compression of soft tissues against the bony margins of the pelvic outlet (Figs. 138 & 139).

The proximal ureter is injured in approximately 59.7% of non-iatrogenic trauma cases, while the mid and distal ureter are involved in 25.6% and 20.8% respectively.

Associated injuries are present in 90.4% of patients. Small and/or large bowel injuries are most commonly involved in conjunction with traumatic ureteral injuries in 96% of those cases with associated injuries.

In one study, non-iatrogenic traumatic injuries to the ureter involved males in 83.4% of cases with the average age being 23.2-years-old. These figures reflect the young male predominance in violent trauma.



Fig. 127. This image is a CT scan of a 75-year-old woman with known left ureterohydronephrosis who sustained blunt trauma in a fall from a height of 1 m. The arrow denotes the presence of lithiasis in the proximal left ureter. A left retroperitoneal fluid collection is also seen (*).

Preexisting renal abnormalities predispose the urogenital system to an increased risk of injury. These abnormalities include longstanding hydronephrosis (Figs. 109, p 118 & 110, p 119) secondary to ureteropelvic junction stenosis or renal stones as seen in this image: extrarenal pelvis; congenital anomalies, such as ectopic or horseshoe kidney (Fig. 124); rupture of a tumor, such as angiomyolipoma or renal cell carcinoma (Figs. 87 & 88, pages 97 & 98, Fig. 97, p 106); and transplanted kidney, which is superficial in location and, therefore, more prone to blunt trauma. (radiographics.rsna.org) (Wiki)

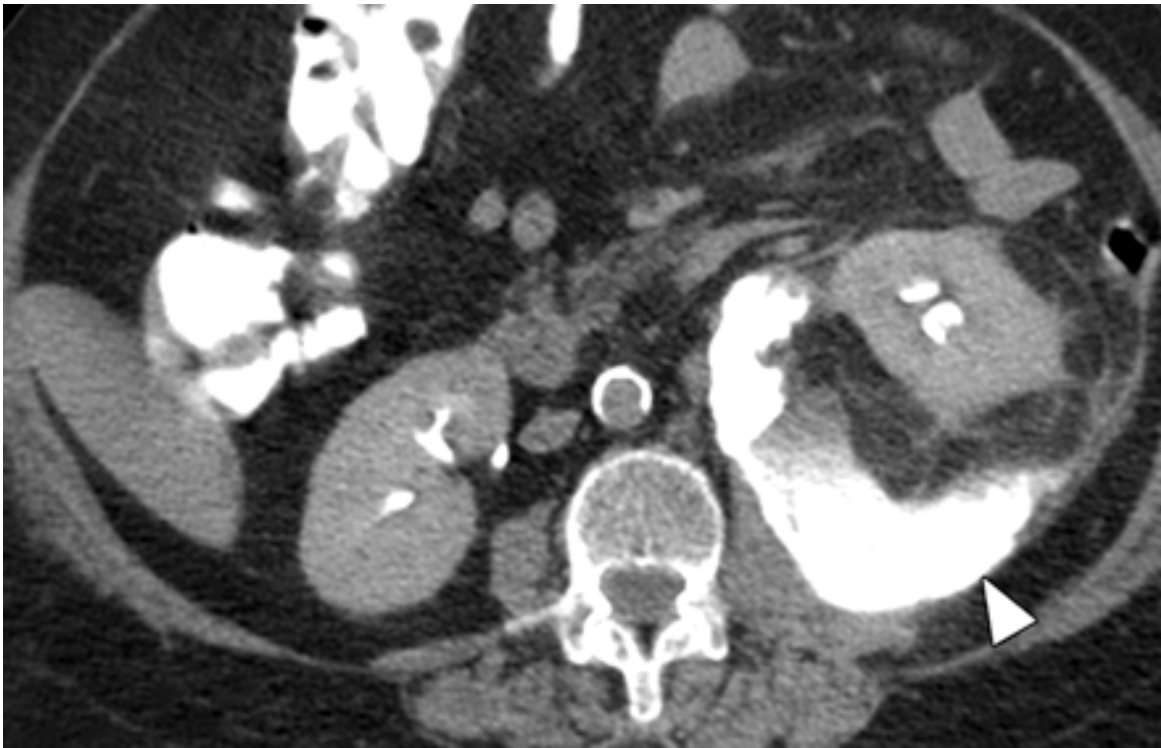


Fig. 128. This image is the delayed phase CT scan of the patient described in Fig. 127. It shows that the collection has filled with diluted intravenous contrast material (arrowhead). (radiographics.rsna.org) (Wiki)

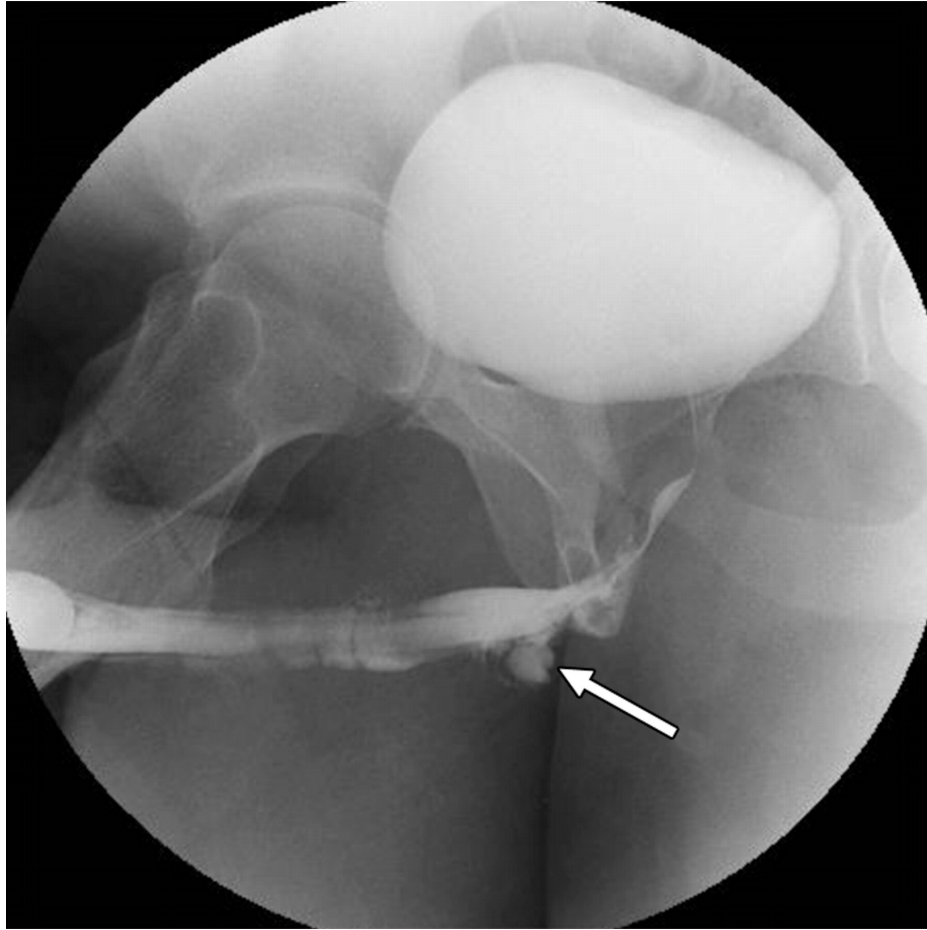


Fig. 129. This image is an example of a straddle injury. The voiding cystourethrogram shows partial urethral transection and extravasation at bulbar urethra (type 5 injury, arrow). (ajronline.org) (Wiki)

Penetrating trauma accounts for the majority of traumatic injuries to the ureter (61.1% \pm 45.7%). In one study, gunshot wounds accounted for 91% of external derived traumatic injuries (Fig. 130), with stab wounds accounting for 5% (Fig. 131).

The frequency of ureteral damage from gunshot wounds of the abdomen is due to two factors: **direct injury** due to the missile traversing (permanent cavity) the ureter leading to complete or partial transection; and **indirect injury** due to the blast effect (kinetic energy), which causes the formation of an instantaneous temporary cavity.

The **direct injury** is due to the permanent tract created by the missile physically passing through tissue as it produces its wound tract. This direct injury may be in the form of a contusion to a complete transection. The missile tract may also involve the

intramural blood supply of the ureter thus, leading to ureteral necrosis. It has been shown that disruption of the intramural blood supply can cause an area of ureteral necrosis that extends up to 2 cm above and below the area of partial, or complete transection.

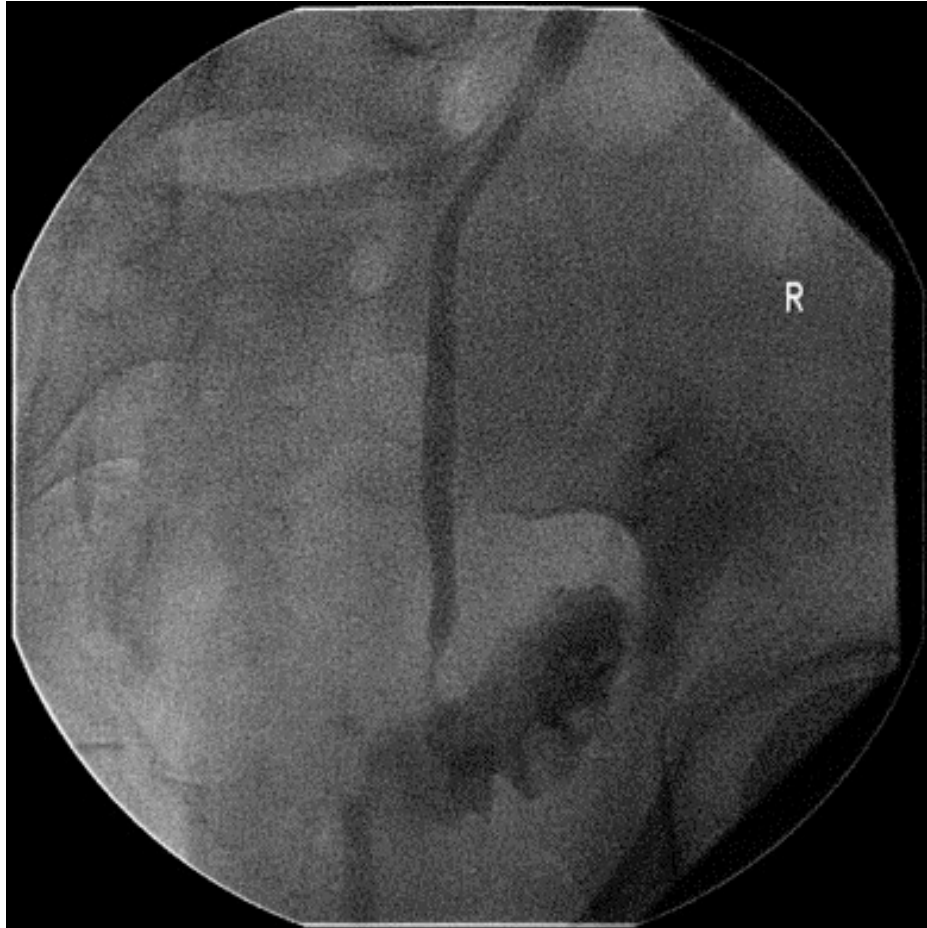


Fig. 130. This image is an antegrade nephrostogram at PCN showing extravasation from the distal ureter due to a gunshot wound. (sciencedirect.com) (Wiki)

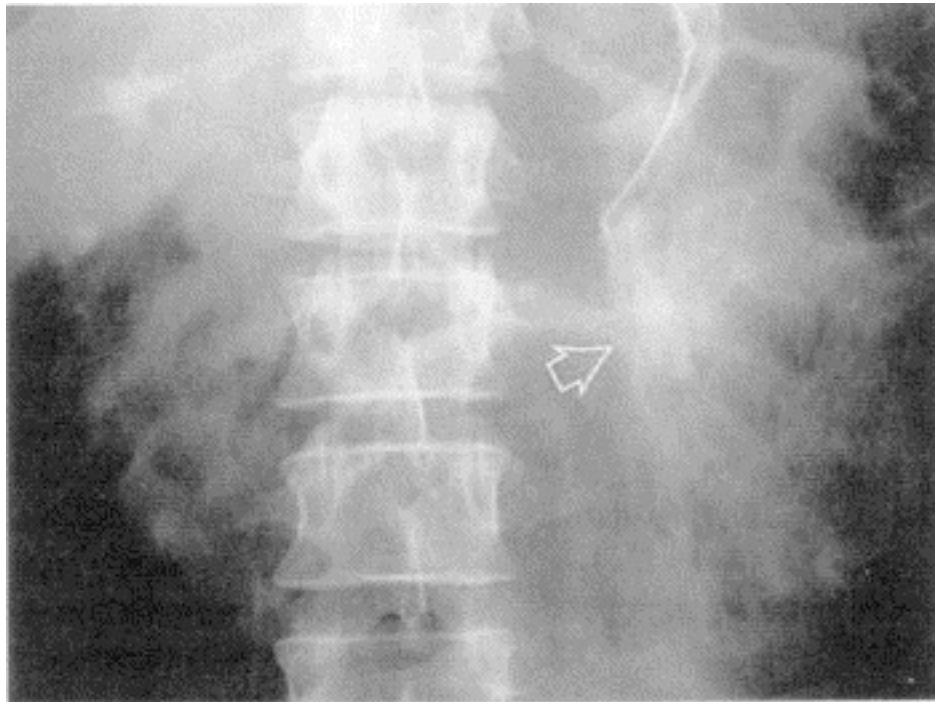


Fig. 131. This image is a one-shot IVP in a patient stabbed in the left abdomen showing contrast extravasation from an upper ureteral laceration. (sciencedirect.com) (Wiki)

The **indirect injury** is due to the release of kinetic energy as the missile traverses the tissues creating a blast effect, which appears as a temporary cavity. As previously pointed out, this temporary cavity is 30 to 40 times, depending on the velocity of the missile, greater than the size of the permanent cavity. This temporary cavity, especially from high velocity missiles can cause widespread tissue destruction. Even the temporary cavity formed by low velocity missiles can damage the small ureteral blood vessels leading to the formation of thrombosis and ischemia, which in turn leads to widespread tissue necrosis. *Remember, the size of the blast effect (temporary cavity) is determined by the square of the velocity of the missile. Thus, the higher the velocity the greater the size of the temporary cavity and tissue destruction.*

Penetrating ureteral traumatic injuries due to stab wounds are less frequent than those caused by gunshot wounds (Fig. 131). However, abdominal stab wounds due to long bladed knives, or stab wounds posterior to the mid-axillary line should raise concern of possible ureteral involvement (Fig. 132).

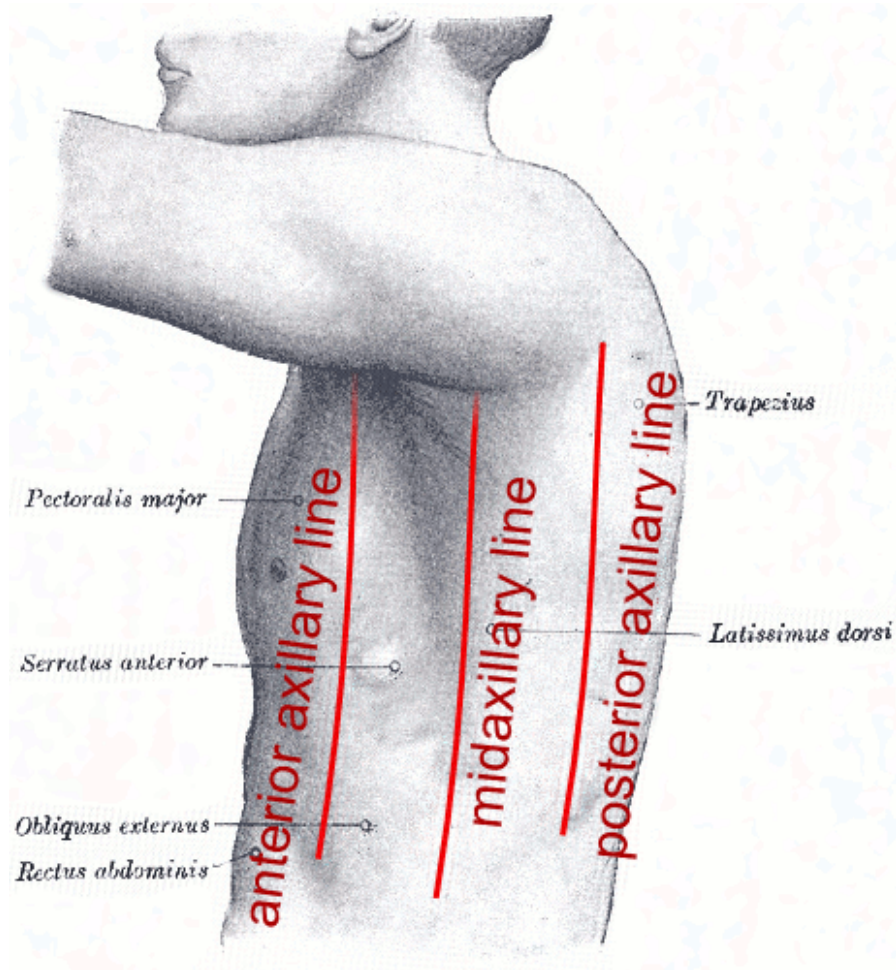


Fig. 132. The midaxillary line is a coronal line on the torso between the anterior axillary line and the posterior axillary line. (Wiki)

In many respects, the tissue damage caused by a stab wound is analogous to that of a low velocity missile minus the temporary cavity.

Pediatric Injuries: The most common ureteral injury in children is the ureteropelvic junction avulsion, which often occurs after severe deceleration (Fig. 133). Children's ureters are especially prone to injury at this location because of the hyperextensibility of their spines. This may occur due to the hyperflexion of the upper body over an improperly placed lap belt, which creates an axis of rotation near the level of the umbilicus during a motor vehicular accident. This results in the abdominal organs being subjected to compressive forces between the seatbelt and the spinal column, as well as increased luminal pressure and shearing forces. Such a mechanism of injury may also cause traumatic injury to the mid and distal portion of the ureter, as well as associated

injuries to the mesentery, small and large bowels, diaphragm, liver, spleen, pancreas and kidneys. A rare associated injury is a flexion-distraction vertebral column fracture, which occurs in the midlumbar region, typically L2, in the pediatric age group and in the thoracolumbar junction, T12-L2, in the population at large (Chance fracture) (Fig. 133).



Fig. 133. This is an image of a Chance fracture of T10 and fracture of T9 due to a seatbelt during a motor vehicular accident. (Wiki)

In the child, the head-to-body ratio is larger thus, providing a higher center of gravity and larger relative force about the rotation of axis. The relatively narrow immature pelvis and rib cage provide less protection for the abdominal organs and mesentery. Another mechanism of injury that can lead to ureteropelvic junction avulsion in a child is a substantive blow to the lower back. Such impact can cause hyperextension of the vertebral column, which in turn leads to tensing of the proximal ureter across the twelfth rib, as well as the transverse process of the upper lumbar vertebrae (Figs. 134, 135, 136 & 137).

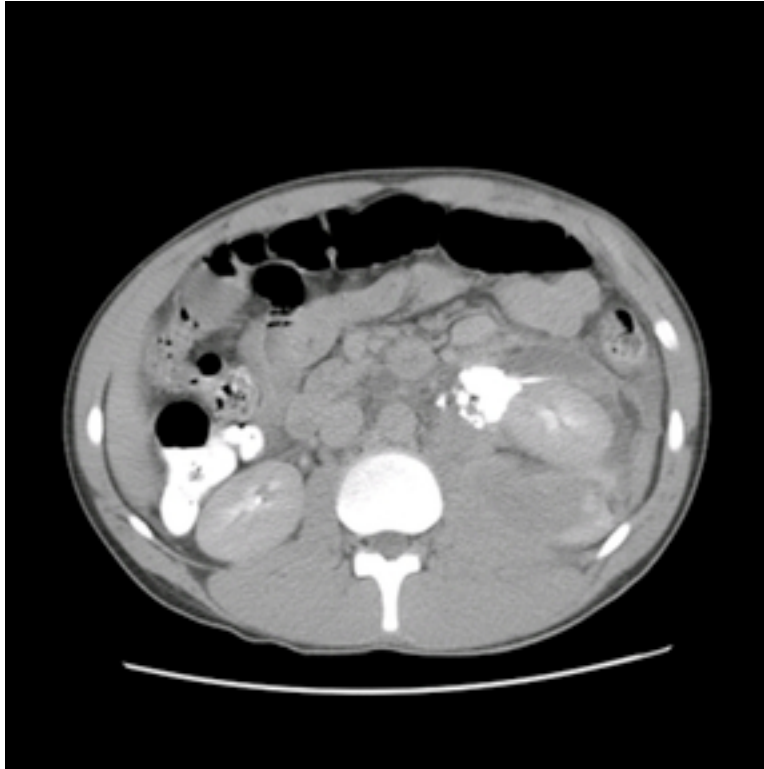


Fig. 134. This image is a delayed contrast CT image showing contrast extravasation into the medial aspect of the anterior perirenal space. The extravasation is from the proximal ureter. (ispub.com) (Wiki)

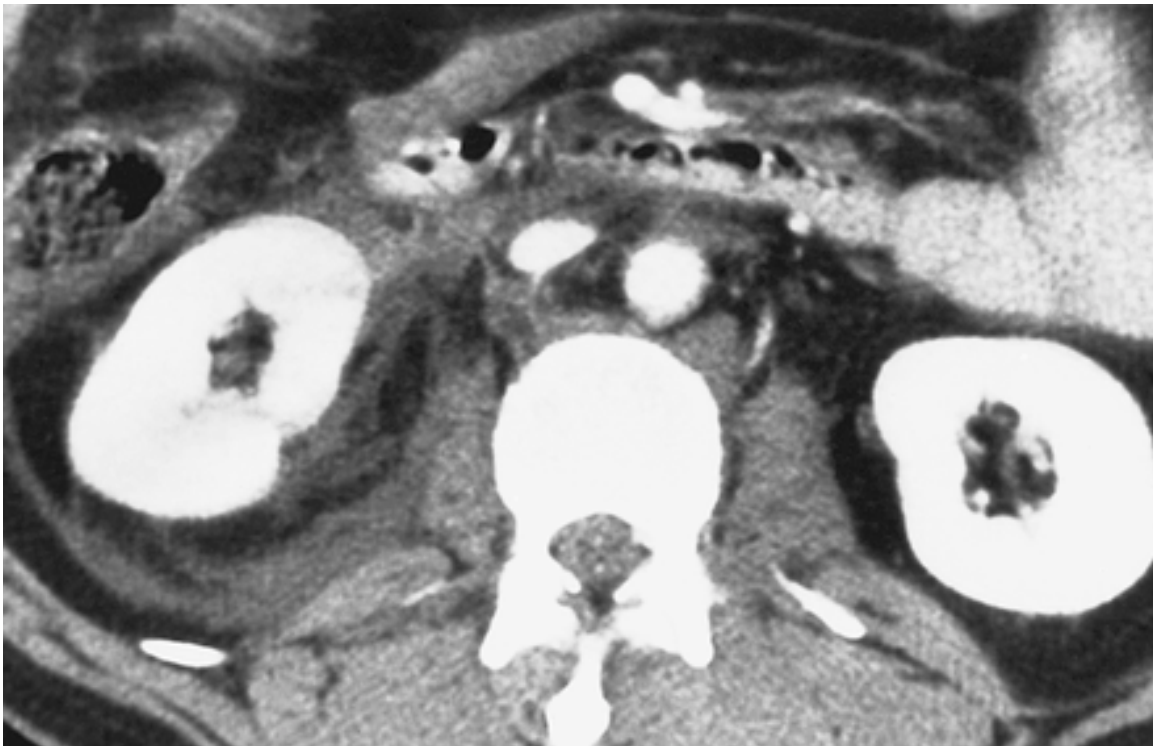


Fig. 135. The above image is a contrast-enhanced nephrographic-phase helical CT scan through the lower pole of the right kidney showing a perinephric fluid collection with no renal laceration in a victim who sustained blunt trauma causing an avulsion of the ureteropelvic junction (category IV) injury. No ureteral contrast material filling is noted. (radiographics.rsna.com) (Wiki)

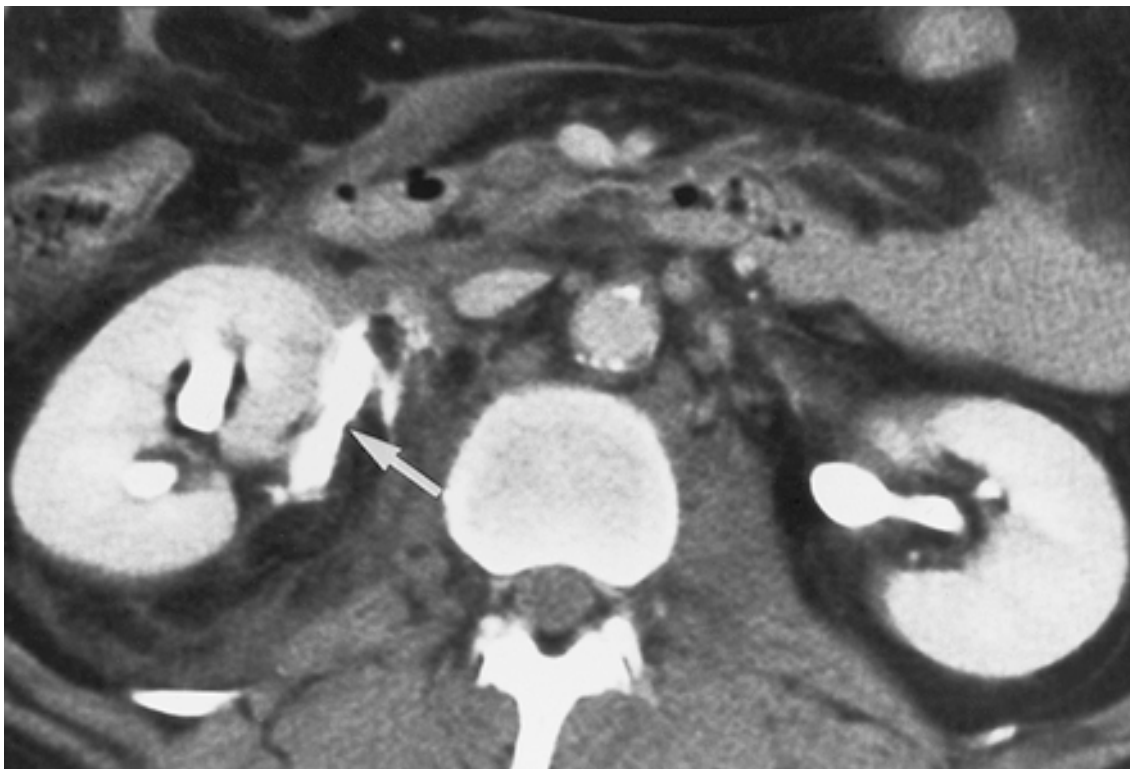


Fig. 136. This is the same case as Fig. 135. This image is a contrast-enhanced excretory-phase CT scan showing medial contrast material extravasation (arrow). (radiographics.rsna.com) (Wiki)



Fig. 137. This case is of a ureteropelvic junction laceration with preexisting obstruction (category IV) in a victim who had sustained blunt abdominal trauma. The image is an axial CT scan inferior to the lower pole of the left kidney, which shows contrast material extravasation at the point of a laceration of the ureteropelvic junction (straight arrow). A periureteral urinoma is also present. The enhanced ureter contains a filling defect (curved arrow), presumably secondary to a blood clot. (radiographics.rsna.com) (Wiki) Ureteropelvic junction injuries are classified into two groups: avulsion (complete transection, Figs. 135 & 136) and laceration (incomplete tear) this figure. The presence of contrast material in the ureter distal to the ureteropelvic junction helps differentiate laceration from avulsion. Ureteropelvic junction avulsion is usually treated surgically, whereas laceration may be treated conservatively or with stent replacement.

Blunt force trauma to the lower trunk, either anterior or posterior can cause partial or complete avulsion at the ureteropelvic junction, in the child, as well as the adult.

Another type of injury, which is especially common in children is compression of the soft tissues of the perineum against the bony margins of the pelvic outlet, the straddle injury. The straddle injury occurs when a child straddles an object as he or she falls, striking the urogenital area with the force of his or her body weight. These injuries occur most often during bicycle riding, falls, and playing on monkey bars. However, it is important

that a careful history and external examination at the time of autopsy be obtained in children with straddle injuries.

Straddle injuries are typically unilateral and superficial. In both boys and girls they involve the anterior portion of the genitalia (Figs. 138 & 139). Most straddle injuries in girls involve the mons, clitoral hood, and the labia minora anterior or lateral to the hymen.

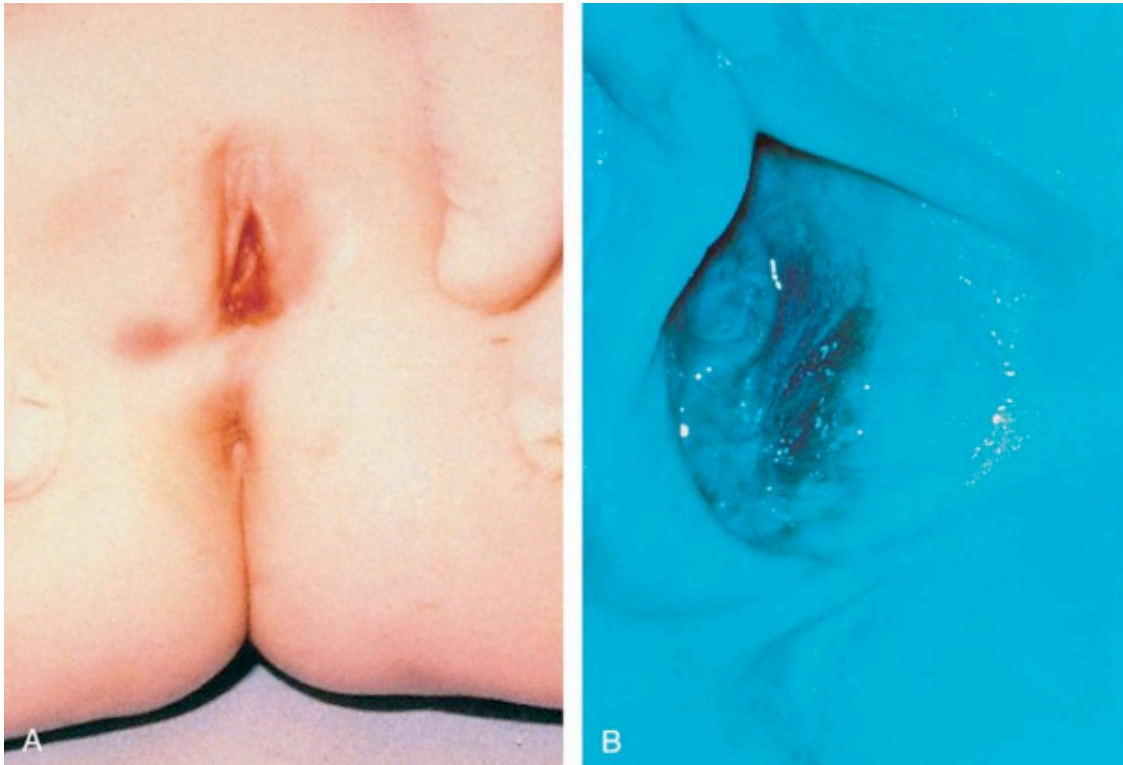


Fig. 138. Accidental trauma caused by straddle injury. A, This child with a straddle injury was brought in immediately for care because of slight bleeding. Superficial abrasions and contusions extend from the anterior portion of the labia minora to the posterior fourchette. Note the lesions are largely unilateral. B, Another child complained of dysuria and was noted to have a small amount of blood on her underwear following a straddle injury. The superficial laceration between the hymen and labia minora was barely visible in regular light, but was brought out by viewing it through a green filter. (health-7.com) (Wiki)



Fig. 139. This is another case of accidental trauma caused by a straddle injury. Note the unilateral swelling accompanied by a contusion with some extension to the opposite side superiorly. (accessemergencymedicine.com) (Wiki)

Straddle injuries to the hymen or posterior fourchette are less common and if present should raise concern for sexual abuse. For example, a laceration to the hymenal area that extends from 3 o'clock to 9 o'clock is consistent with a penetration injury and must be further explored.

Straddle injuries are classified as penetrating or non-penetrating. Non-penetrating injuries typically cause minor trauma to the external genitalia: superficial lacerations of the scrotum or penis in boys and lacerations or abrasions of the labia in girls. Perianal and testicular trauma rarely result from non-penetrating injuries. Penetrating injuries are more serious and extensive (e.g., vaginal-peritoneal perforation, rectal injury) and more often indicative of sexual assault.

A high index of suspicion for a possible ureteral injury should occur in either a child or adult who has sustained a fracture of the eleventh or twelfth ribs; fracture of the lumbar transverse process; fracture of the pelvis; and a penetrating injury of the abdomen, retroperitoneum or pelvis.

F. Adrenal Glands: Traumatic adrenal hemorrhage was first reported in 1863; few cases were reported in the literature until 1955, when Sevitt reported an autopsy series in which 28% of patients with significant abdominal trauma had evidence of adrenal traumatic injury. In a more recent study by Burks *et al.*, reported 2% of 1120 patients, who had abdominal CT assessment for blunt force trauma to the abdomen, had evidence of traumatic injury to the adrenal glands.

Isolated adrenal injuries are uncommon, and when they occur relatively benign and self limited. Typically, adrenal injuries are associated with more significant abdominal retroperitoneal and thoracic injuries. Liver injuries are the most common associated injury. The most commonly associated intrathoracic injuries are ipsilateral rib fractures, atelectasis, hemorrhage, pneumothorax and lung parenchymal contusions.

Intraabdominal associated injuries along with the liver are the spleen, stomach laceration, mesenteric contusions, and ruptured hemidiaphragm. In the retroperitoneal space the most common associated lesions are to the kidney, pancreas, and lumbar spine.

The most common traumatic injuries to the adrenal glands are acute hemorrhage (Figs. 140 & 141), hematoma (Fig. 142), and lacerations (Fig. 143).

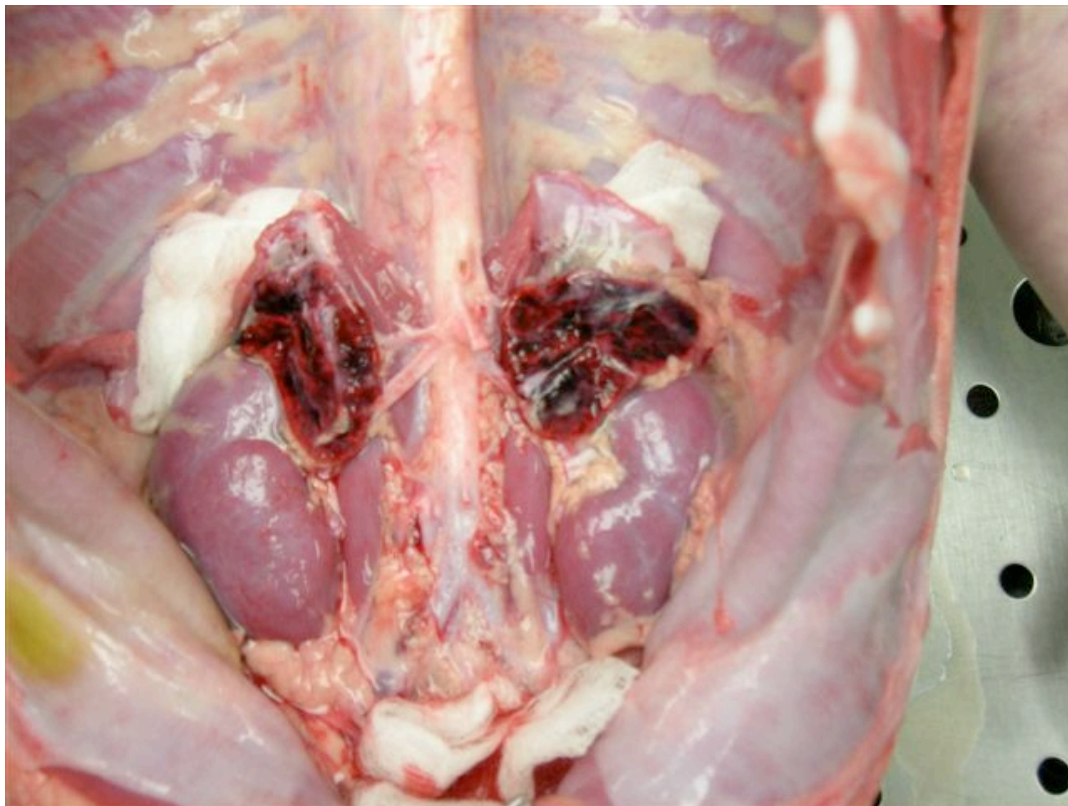


Fig. 140. This image is from an autopsy on a 10-month-old body who died from a fatal meningococemia. Note the hemorrhagic adrenal glands on the superior poles of the kidneys. (Courtesy of Dr. Eric L. Vey, Erie County Coroner's Office, Erie, PA) (pathologyoutlines.com) (Wiki)



Fig. 141. This image is from the same case as Fig. 140. Normal adrenal glands are on top and the hemorrhagic adrenal glands are on the bottom. (Courtesy of Dr. Eric L. Vey, Erie County Coroner's Office, Erie, PA) (pathologyoutlines.com) (Wiki)



Fig. 142. This image depicts a hematoma within an adrenal gland in a patient who died from Waterhouse-Friederichsen syndrome. (humpath.com) (Wiki)

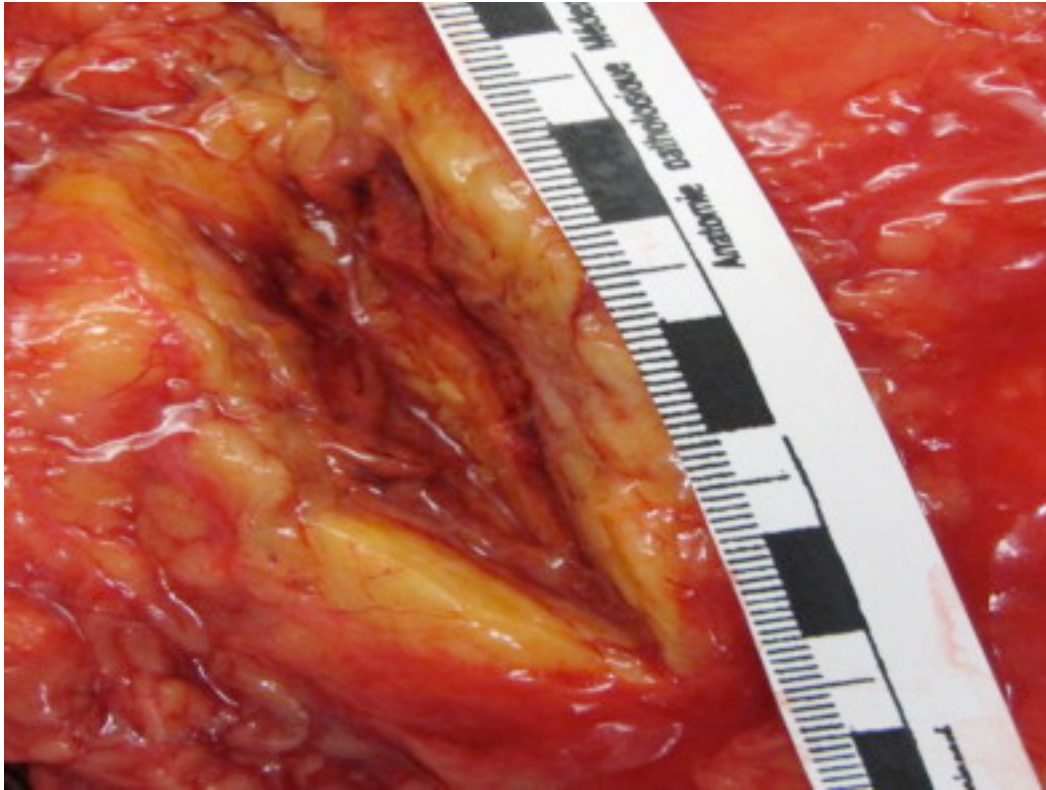


Fig. 143. This is an image of a laceration of an adrenal gland. (sciencedirect.com) (Wiki)

Traumatic injuries, most especially acute hemorrhage and hematomas, are most often right sided. In one study 77% of traumatic adrenal hemorrhage was right sided, 15% left sided, and 8% bilateral.

The most common periadrenal findings on CT scans include strand like hemorrhage (periadrenal stranding) infiltrating fat and/or a hematoma. Periadrenal stranding is identified in most cases of adrenal trauma (Figs. 144 & 145).

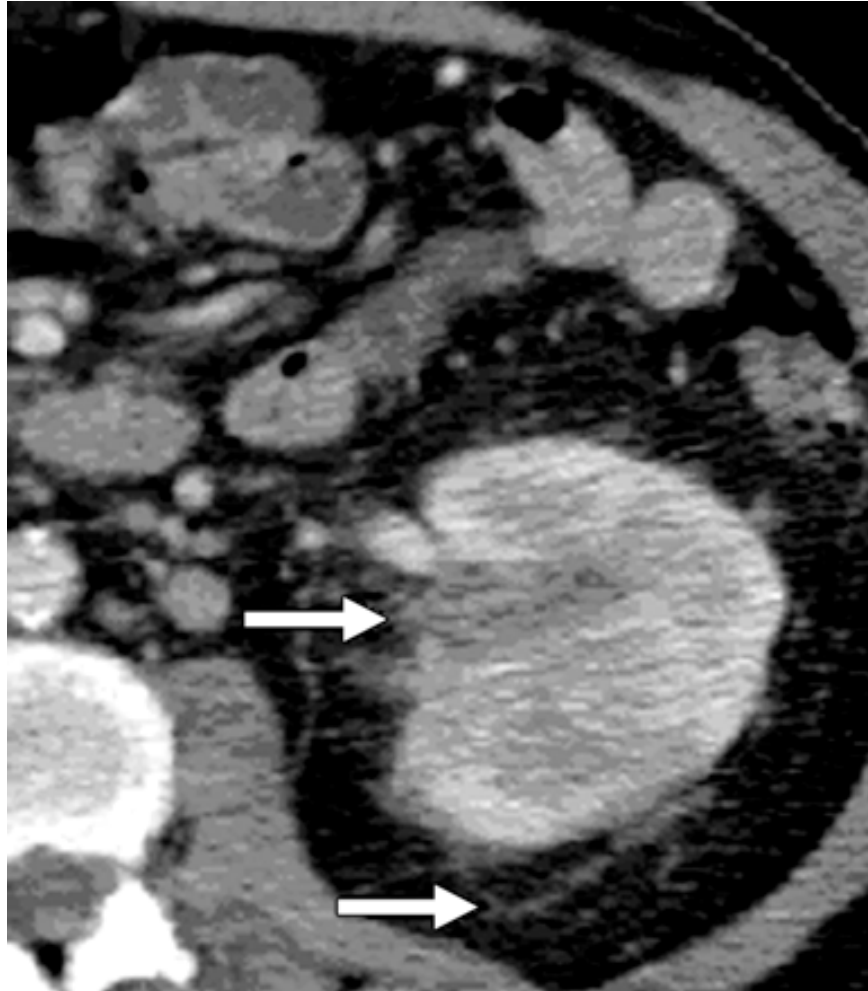


Fig. 144. This image is an axial contrast-enhanced CT image obtained during the late corticomedullary phase showing stranding in the renal hilum and perirenal fat. (radiographics.rsna.org) (Wiki)



Fig. 145. This CT image shows perinephric stranding and thickening of the posterior pararenal fascia (white arrows). Also seen are focal xanthomas (black arrow). (clinicalimagingscience.org) (Wiki)

Another common periadrenal abnormality is hemorrhage into the retroperitoneal space, especially the posterior pararenal space (Fig. 125, p 136). Less common periadrenal associated findings are hemorrhage into the anterior pararenal space, compression of the inferior vena cava by hematoma, acute hemorrhage into the psoas muscles, anterior thickening of Gerota's fascia and displacement of the kidney.

Mechanisms of injury: The mechanisms underlying traumatic injuries to the adrenal glands are varied. They include a crush injury of the right adrenal gland between the right lobe of the liver and the vertebral column, a crush injury of the left adrenal gland between the spleen and the vertebral column, an acute increase in venous pressure transmitted through the adrenal veins when the inferior vena cava is compressed,

thrombosis of the inferior vena cava with involvement of the adrenal veins, and shear injury to the small adrenal vessels due to rotational or deceleration forces.

Adrenal cyst and adenomas make the adrenal gland more susceptible to trauma.

Pathophysiological events: Pathologically, adrenal hemorrhages typically occur in the loosely textured adrenal medulla and juxta-medullary cortex due to the myriad of small vessels in the form of sinusoids and venules (Figs. 43, p 48, 44, p 49, 146 & 147).

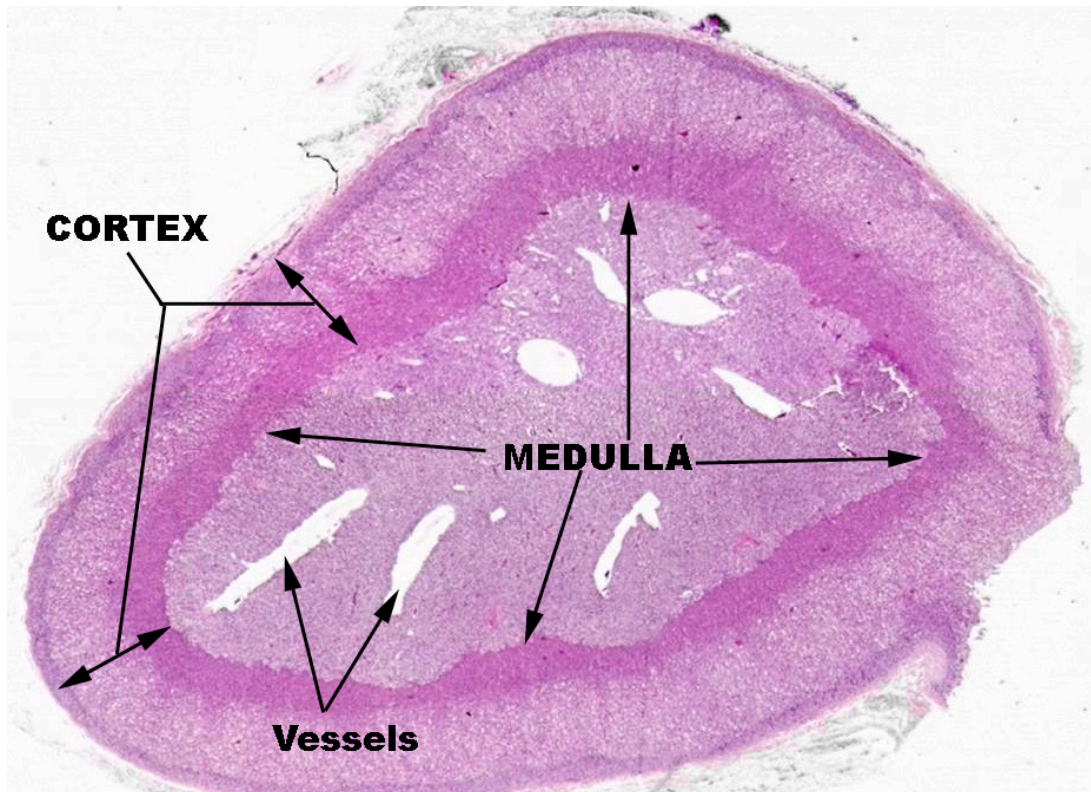


Fig. 146. This image shows the obvious distinction between the adrenal cortex and its medulla. Remember, they arise from different embryonic rudiments, have markedly different functions, and respond to different inputs, hence the anatomic differences should not be surprising.

The adrenal medulla is of neuroectodermal origin. The cortex arises from a condensation of embryonic mesoderm.

The name cortex, as the name implies (it means “rind” in Greek) surrounds the medulla. The medulla and cortex have a separate arterial blood supply, but share a common venous drainage. (vetmed.vt.edu) (Wiki)

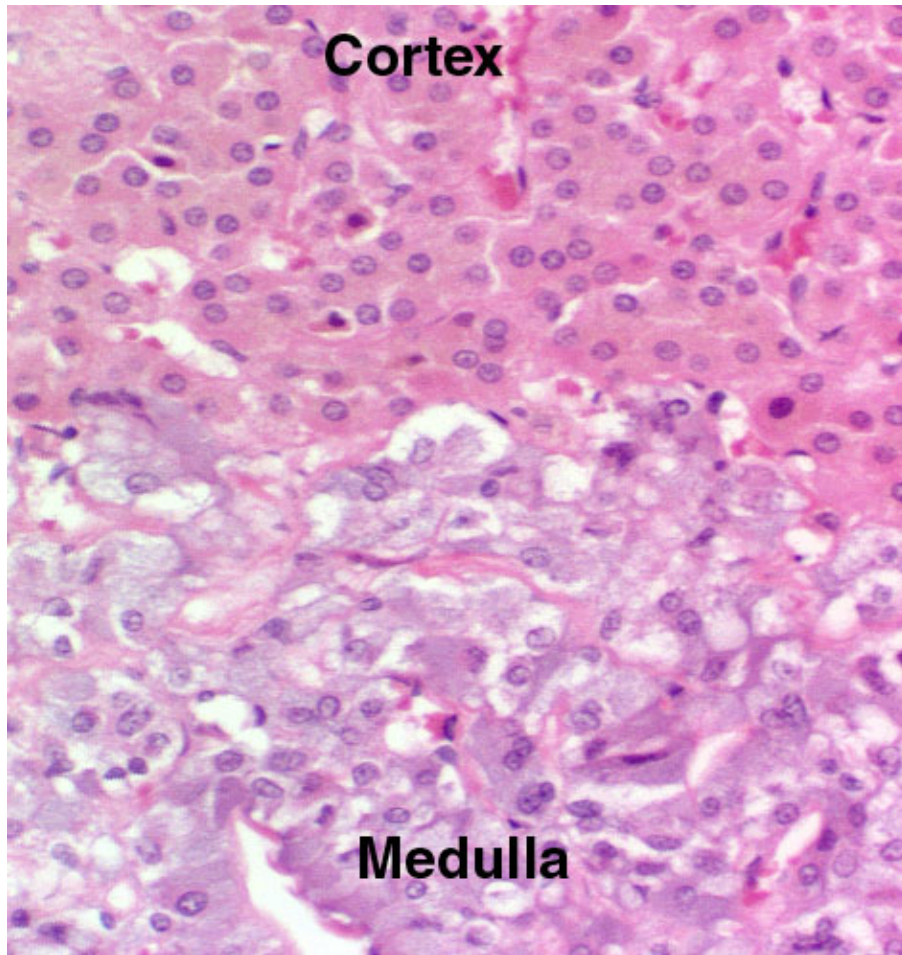


Fig. 147. This is a photomicrograph of Fig. 146. The corticomedullary junction is quite easy to see. The cells of the adrenal medulla are somewhat more basophilic than those of the cortex. (vetmed.vt.edu) (Wiki)

Sudden compression of the adrenal glands can easily rupture these vessels causing an acute hemorrhage. The right adrenal gland has greater susceptibility to this type of injury than the left due to it being directly opposed to the undersurface of the right lobe of the liver. Likewise, due to the fact the right adrenal vein is shorter than the left and drains directly into the inferior vena cava thus, acute pressure increase in the inferior vena cava is transmitted more directly to the right adrenal gland leading more readily to acute hemorrhage. The left adrenal vein drains into the left renal vein. Lastly, each adrenal gland is supplied by three or more suprarenal arteries, which pass through the adrenal capsule, ultimately dividing into a sinusoidal network within the cortex and

medulla. These sinusoidal vessels ultimately drain into a single central vein. All of these vessels are very susceptible to shear injury produced by rotational or deceleration forces.

Adrenal hemorrhage can also result from non-traumatic events, such as the stress caused by surgery, overwhelming stress, burns and hypotension (Fig. 148). Such hemorrhages are bilateral and may lead to life-threatening adrenal insufficiency.



Fig. 148. The above image shows bilateral adrenal hemorrhages due to shock. (Wiki)

The underlying mechanism for stressed induced adrenal hemorrhage is the increased secretion of adrenocorticotrophic hormone, which in turn leads to increased adrenal blood flow and a rise in adrenal venous pressure, which can be followed by venous hemorrhage. Non-traumatic adrenal hemorrhage is also associated with fulminant meningococemia (Waterhouse-Friderichsen syndrome) (Figs. 140, 141, 142 & 149),

pseudomonas infection, or infection with other gram-negative bacteria. Other stress related conditions associated with adrenal hemorrhage are pregnancy, cardiovascular disease, and the exogenous administration of adrenocorticotrophic hormone or steroids.



Fig. 149. This image shows acute hemorrhage in both adrenal glands, which caused acute adrenal insufficiency (Waterhouse-Friderichsen syndrome). (Yamagiku.co.jp) (Wiki)

Adrenal trauma, especially when it is unilateral and with minimal associated injuries, does not typically present a serious clinical problem. However, if the adrenal trauma is associated with major thoracic, abdominal or retroperitoneal organ injury, the prognosis is poor. Again, although bilateral post-traumatic adrenal hemorrhage is uncommon, when it occurs there is an increased risk of the development of acute renal insufficiency.

Pediatric injuries: Blunt force trauma as a cause of adrenal hemorrhage in the pediatric age group is rare. When it does occur it is typically unilateral, small, most often involving the right adrenal gland, and associated with ipsilateral intrathoracic, retroperitoneal, and intraabdominal injuries.

The underlying mechanisms are those of the adult. These include blunt force trauma to the abdomen or flank with compression of the right adrenal gland between the right lobe of the liver and the vertebral column, or compression of the left adrenal gland between the spleen and vertebral column, shearing of small vessels that perforate the adrenal capsule due to deceleration forces, and an acute rise of intraadrenal venous pressure due to compression of the inferior vena cava. The concepts of direct compression or shearing of small cortical adrenal vessels as mechanisms of injury is underscored by the high frequency of associated ipsilateral intraabdominal (61%) and intrathoracic (44%) injuries. Also, the concept that an acute rise in intraadrenal venous pressure due to compression of the inferior vena cava, as a mechanism, is supported by the more frequent occurrence of hemorrhage into the right adrenal gland (86%). *Again, the right adrenal vein drains directly into the inferior vena cava, whereas the left drains into the left renal vein.*

In the child, post-traumatic adrenal hemorrhage is typically made at the time of autopsy in up to 25% of severely injured patients. This is because specific clinical signs and symptoms are absent. Abdominal tenderness and hematuria are the most common indications for CT in children with adrenal hemorrhage. However, they are also the most common indications for CT in children studied after blunt force trauma who did not have adrenal hemorrhage, hence, there are no selective criteria for the detection of adrenal hemorrhage. Also, none of the children in one series had evidence of adrenocortical insufficiency. This is primarily due to the fact the injuries are unilateral in most children.

Neonates: Adrenal hemorrhage is more common in neonates than in older children, or adults, and is the most common adrenal mass in neonates. Such hemorrhage can occur prenatally due to a difficult labor or delivery, especially breech deliveries and especially in infants of diabetic mothers, or infants who are large for their gestational age. An accumulation of as much as 30 to 40 ml of blood may remain in the capsule, but with greater bleeding the capsule ruptures, producing perirenal hemorrhage. Typically, this hemorrhage is unilateral, usually the right side in 70% of cases, although, occasionally it is bilateral (Fig. 150).

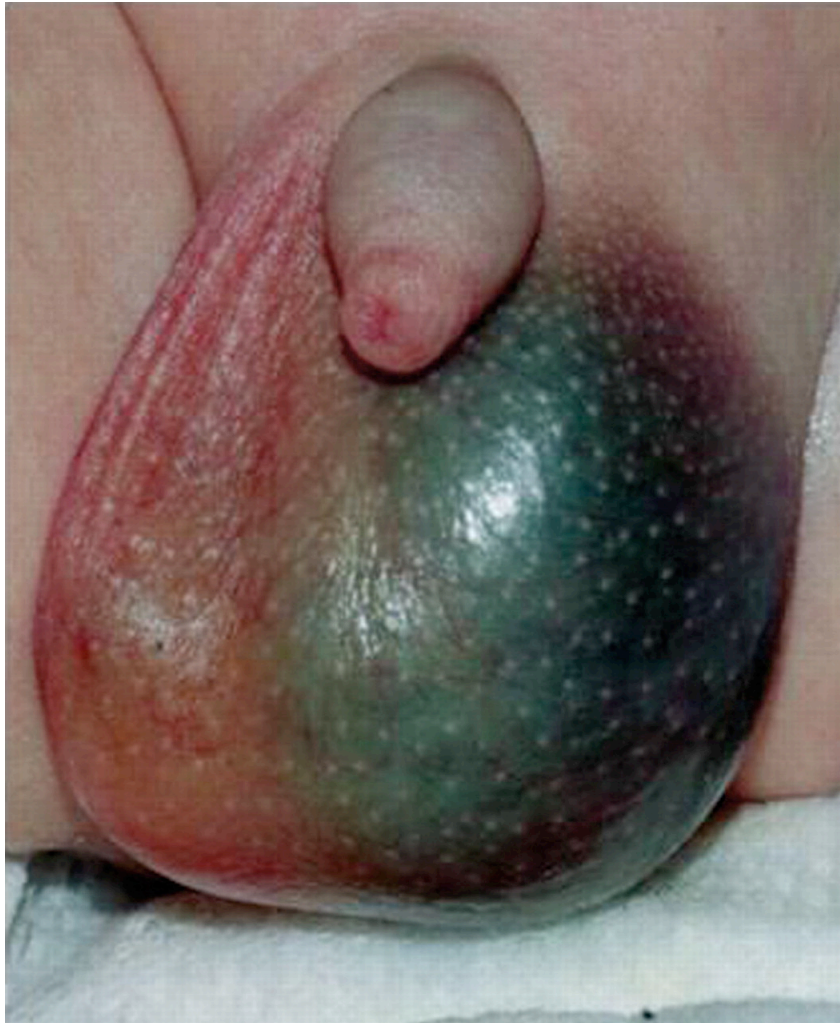


Fig. 150. The above image is from a case in which the neonate had an adrenal hemorrhage, which presented itself as a scrotal hematoma. In this case the hemorrhage is on the left. (fn.bmj.com) (Wiki)

Adrenal hemorrhage can also occur as the result of asphyxia, hypoxia, septicemia, or hemorrhagic disorders, such as disseminated intravascular coagulopathy and hypoprothrombinemia. This is believed to be due to the prominent dilation of the sinusoids and small vessels within the central part of the fetal zone of the adrenal glands during anoxia.

Adrenal hemorrhages may calcify in the neonate within a few days. These calcifications are often detected as an incidental finding in radiographic studies of the abdomen in

infants and children. Again, hemorrhage into the adrenal gland at or immediately after birth is probably the most common cause for these calcifications. Such calcifications rarely cause a functional disorder.

Adrenal hemorrhages, unless massive, are usually not fatal, because they are usually unilateral. However, adrenal insufficiency may result if the lesion is bilateral.

G. Abdominal Aortic Injuries: Blunt force traumatic injury to the abdominal aorta is an uncommon event. The infrequency with which blunt abdominal aortic injury occurs is likely due to the central protected position of the abdominal aorta. In contradistinction to the abdominal aorta, thoracic aortic injuries occur 20 times more frequently as seen in several autopsy series.

Abdominal aortic rupture is more commonly seen in cases of **penetrating abdominal trauma**, whereas the thoracic aorta is usually ruptured after **non-penetrating (blunt) trauma**. Blunt force traumatic injuries of the thoracic aorta are well known and account for about 95% of all injuries involving the aorta. Studies in the late 1930s and early 1940s reported an incidence of thoracic and abdominal aortic blunt trauma of less than 1% of all fatal abdominal trauma. In 1947, Strassman published a report on trauma to the aorta, as did Parmley *et al.* in 1954. When you combine these two studies, they reviewed a total of 8,710 autopsies, all done on trauma victims. Three hundred and forty-seven of these cases involved non-penetrating aortic trauma, of which only 16 cases involved the abdominal aorta (4.6%). Between 1958 and 1980, there were many reports on series of arterial injuries, but little on injuries to the abdominal aorta. In 1981, Lassonde *et al.* published a report on "Blunt Injury to the Abdominal Aorta." They reviewed up to that point all cases of traumatic injury to the abdominal aorta. In their review, males sustained injury to the abdominal aorta more frequently than females, with motor vehicular accidents being the most common cause in 20 out of 27 cases. Of these 27 patients, 10 sustained their injury due to the steering wheel, 5 struck some other structure within the vehicle, and 4 had a seatbelt injury; one patient was struck by a vehicle. The remaining 7 patients had sustained blunt force trauma to the abdomen from a variety of causes, i.e., falls, surfboarding, etc. In 1990, Frame *et al.* reported on penetrating injuries of the abdominal aorta through a review of 56 patients with such an injury. In their report the mechanism of injury was gunshot wound, 82%; shotgun

wound, 5%: and stab wound, 13%. The overall mortality was 73%, with gunshot wound, 78%; with shotgun wound, 67%; and 43% with stab wound. In 1992, Feczko *et al.* reported an incidence of blunt abdominal aortic trauma of 10 to 15%. This increase was due to the use of seatbelts (especially lap-type), with the impact from steering wheels, and most probably with the higher speed of motor vehicles (69% of abdominal aortic injuries are vehicle related).

Abdominal aortic injuries occur more commonly in the infrarenal portion (Fig. 151).

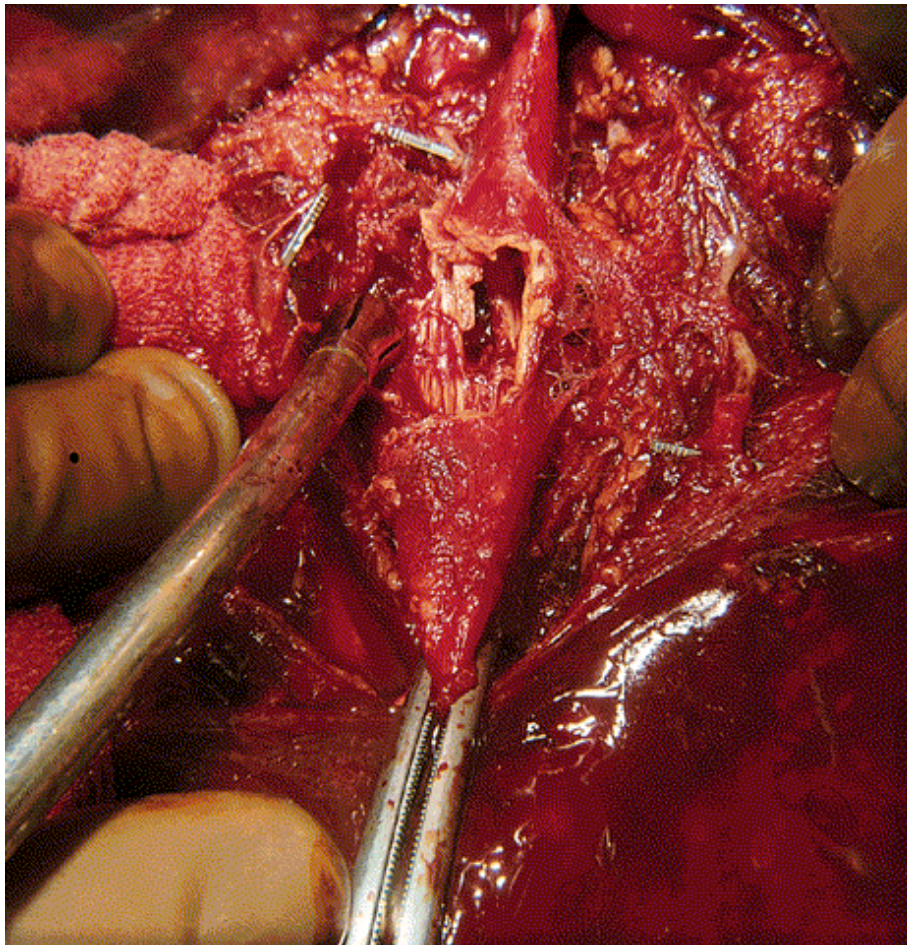


Fig. 151. This image is of a gunshot wound to the infrarenal portion of the abdominal aorta. (acssurgery.com) (Wiki)

Within this segment, it is the distal segment (Fig.151), that segment distal to the origin of the inferior mesenteric artery, which is most often involved. It is believed that the mid-abdominal location of the distal infrarenal segment makes it highly susceptible to

traumatic injuries and accounts for 98% of traumatic injuries to the abdominal aorta. Likewise, the paucity of traumatic injuries involving the suprarenal portion is due to the protection offered by the lower thoracic rib cage.

Although, from a pragmatic standpoint, **associated injuries** are most often not the immediate clinical concern, abdominal aortic injuries, as is true of abdominal vascular injuries, seldom occur alone. It appears multiple associated injuries are the rule rather than the exception. Often these associated injuries are severe, however, they typically do not effect the mortality rate. The most common associated injury was to the intestines.

The most common **clinical presentation** is acute, typically, severe hypotension, or presenting as an acute abdomen, immediately following the injury. However, some patients may have a delayed response in the presentation of their clinical symptoms, which may vary from a few weeks, up to a year. Typically, the clinical presentation of those with a delayed response was as an abdominal bruit, with or without an associated abdominal mass, claudication, persistent abdominal pain, and decreased pulses in the lower extremities.

Pathologically, the most common injury of blunt force trauma to the abdominal aorta is intimal disruption (Fig. 152). A common complication of intimal disruption, which is often seen, is secondary aortic thrombosis. Other injuries include false aneurysm, true aneurysm, intramural hematoma, fibrous stenosis, post-traumatic thrombosis, and distal embolization due to atherosclerotic debris (Figs 102, p 116 & 103, p 117). A common complication of intramural hematomas is varying degrees of aortic occlusion.



Fig. 152. This image shows intimal disruption manifested by intimal dissection. The dissection occurred just distal to the origin of the renal arteries (infrarenal). The patient was an unrestrained driver involved in a single vehicle rollover accident. (trauma.org) (Wiki)

A potential complication of aortic occlusion is the development of neurologic deficits ranging from sensory loss to paraplegia due to spinal artery syndrome. It is the position of some experts, the spinal artery syndrome secondary to abdominal aortic trauma is typically due to compromise of the Adamkiewicz artery, or associated vertebral fracture with direct spinal cord injury.

The Adamkiewicz artery is the largest anterior segmental medullary artery, which has a variable origin. In one study it was stated the artery originated on the left side of the aorta, between T8 and L1 in 75% of people. In another study, it arose from a left posterior intercostal artery, which branches from the aorta, and supplies the lower two -

thirds of the spinal cord through the anterior spinal artery. In some people it arises from a lumbar vessel.

The classic presentation, if the occlusion is total, is extensive bilateral tissue destruction that spares the posterior columns of the spinal cord. All spinal cord functions-motor, sensory, and autonomic-are lost below the level of the lesion, except for retained vibration and position sensation. In the case of traumatic injuries to the abdominal aorta, the neurologic deficiencies range from distal anaesthesia to paralysis of the lower extremities, due to aortic occlusion, distal embolization, anterior spinal artery syndrome due to compromise of the Adamkiewicz artery, or associated vertebral fracture with direct spinal cord injury.

Spinal cord ischemia with paraplegia occurs in 10% of traumatic cases reported in the literature.

Blute and Rey postulate that transitory lumbar arterial spasm can also affect spinal cord blood supply and thus, be a foundation for spinal cord injury. For example, an ischemic myelopathy has been reported in cocaine users, preceded sometimes by episodes of cord dysfunction resembling transient ischemic attacks, the foundation of which is cocaine induced arterial spasms.

Mechanism of injury: The mechanism of injury in non-penetrating blunt trauma to the abdominal aorta usually involves an impact force to the anterior abdominal wall, which crushes the abdominal aorta against the spine. Such direct impact force can cause a contusion of the aortic wall, intimal dissection (Fig. 152), intramural hematoma (Figs. 153, 154, & 155), thrombosis, pseudoaneurysm, or frank rupture of the aorta, either from the resultant pressure or from the associated thoracolumbar fracture. The aortic wall can also be stretched and compressed against a high-pressure column of blood. The elevated pressure above the injury can cause an intimal tear, pseudoaneurysm, thrombosis, or rupture.

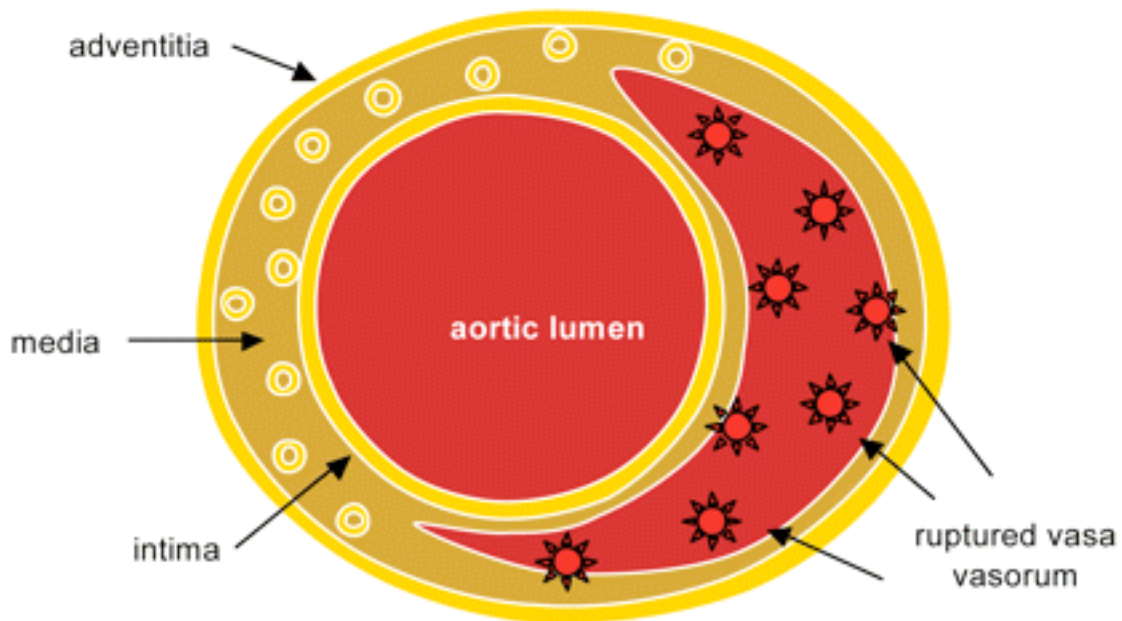


Fig. 153. This is an illustration which shows the pathogenesis of the aortic intramural hematoma. Hemorrhage resulting from rupture of the vasa vasorum weakens the media of the aortic wall and leads to enlargement of the aortic diameter. In an intramural hematoma, unlike an aortic dissection or penetrating ulcer, the intima remains intact. (radiographics.rsna.org) (Wiki)

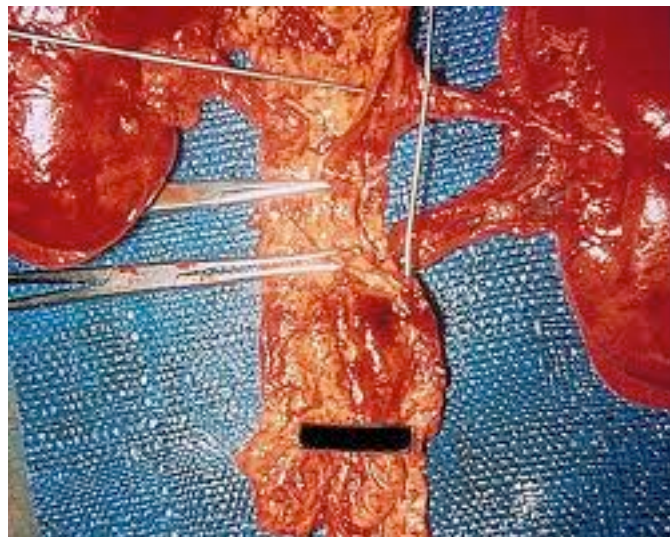


Fig. 154. This image shows the expansion of the infrarenal abdominal aorta due to an intramural hematoma. (cueflash.com) (Wiki)



Fig. 155. The above image is a T2-weighted transverse MRI of a descending aortic intramural hematoma. The descending aorta is dilated, and the wall is thickened. The signal from the wall is abnormally intense, consistent with an intramural hematoma. (chestjournal.chestpubs.org) (Wiki)

Another mechanism, which may affect the abdominal aorta, are deceleration forces. The abdominal aorta is a relatively fixed anatomic structure, whereas the mesenteric arteries are more mobile thus, their deceleration rate is different from the abdominal aorta. It is this difference in the rate of deceleration, which increases the likely hood of the mesenteric vessels being torn. According to Fox *et al.*, forces of more than 1,000 to 25,000 mm Hg are required to cause total rupture of the aorta.

Another possible mechanism is with substantive blunt force trauma to the abdomen, the structures within the abdomen can be driven into the pelvic cavity, stressing points of origin of the superior and inferior mesenteric arteries from the abdominal aorta.

Lastly, the kinetic energy transmitted through the anterior abdominal wall into the organs of the abdomen may in turn be transmitted through these organs into the abdominal aortic wall.

As is true of the kidney, preexisting disease of the abdominal aorta increases its susceptibility to traumatic injury. The most common preexisting condition is atherosclerosis. Another is a preexisting abdominal aortic aneurysm. Lastly, the structural integrity of the anterior abdominal wall also plays a role, especially the force of the anterior abdominal walls muscular contractions.

The mortality rate associated with traumatic injuries to the abdominal aorta varies between 24 to 30%.

Pediatric Injuries: Traumatic aortic rupture in the pediatric age group, which is the age group under 17 years, is extremely rare. Up to the published report of Katsoulis *et al.* in 2006, there were only 8 reported pediatric cases of operated abdominal aortic ruptures. The first reported pediatric operated abdominal aortic rupture was by Wilson in 1974. This case involved a 12-year-old boy who was horseback riding when the horse fell on him causing a rupture of the abdominal aorta posteriorly at the level of the bifurcation (infrarenal) associated with a comminuted fracture of the right iliac fossa, and a pulseless right leg due to a thrombus blocking the right common iliac artery.

Aortic trauma in this age group includes intimal disruption, with or without thrombosis, pseudoaneurysms, stenosis, and distal embolization by thrombotic debris. Although the frequency of traumatic aortic rupture differs in children and adults, multi-system trauma is common to both age groups. In the pediatric age group, the mechanism of injury, chest and abdominal film abnormalities, shock, abdominal pain, neurologic deficits, abnormal vascular examination, or any combination thereof should raise the suspicion of traumatic aortic rupture.

In the pediatric age group, complete aortic transection occurring during a motor vehicular accident is virtually always at the aortic isthmus. It is believed that due to the lack of atherosclerosis in the pediatric aorta, especially the abdominal aorta, the

integrity of the intima is complete thus, the intima and medial layers are believed to be more distensible and stronger, allowing it to withstand greater tension. As previously discussed, atherosclerosis of the aorta in the adult leads to loss of integrity of the intima, and thus, the intima becomes weaker. In addition, the atherosclerotic plaques cause a loss of elasticity and compliance of the aortic wall. All of these pathologic changes in the aorta, which are typically more severe in the abdominal aorta below the origin of the renal arteries, contribute to the more frequent traumatic rupture of the abdominal aorta in the adult.

In the pediatric age group, one of the mechanisms of injury of traumatic rupture of the abdominal aorta is **non-accidental trauma (child abuse)**. The first reported case may have been by Fox *et al.* in 1996, in which a 16-month-old child sustained an abdominal aortic transection below the origin of the inferior mesenteric artery, who presented with an ischemic sigmoid colon, lacerated inferior vena cava, and a fracture of the vertebral body of L3. It was suspected that these injuries were the result of child abuse.

It is noteworthy in 2009, Heck *et al.* reported a case involving a 16-month-old child with a traumatic rupture of the abdominal aorta, also infrarenal. However, this child's rupture was due to a motor vehicular accident in which the child was improperly secured in a five-point restraint car seat.

The second case was reported by Yumul *et al.* in 2002. This case was a 2½-year-old girl, who was reported to have been thrown against a couch by her mother's boyfriend. This child sustained a complete transection of the aorta approximately 3 cm below the renal arteries origin, which was tamponaded by a retroperitoneal hematoma.

Mechanism of Injury: The mechanism of injury to the abdominal aorta in the pediatric age group is the same as that in the adult. However, because of the anatomical differences between children and adults (e.g. incomplete bony ossification, short thoracic cage, and weakness of the abdominal musculature), even minor degrees of external force can cause significant internal injury. Thus, in all cases in which there is evidence of significant blunt force trauma you must assume the existence of underlying visceral and/or vascular injury.

H. Inferior Vena Cava Injuries: Traumatic injuries to the inferior vena cava are rare. In a retrospective study of 5059 patients with blunt trauma over a 10 year period conducted by Nett *et al.*, they found only 10 patients with injury to the inferior vena cava (Figs. 156 & 157).

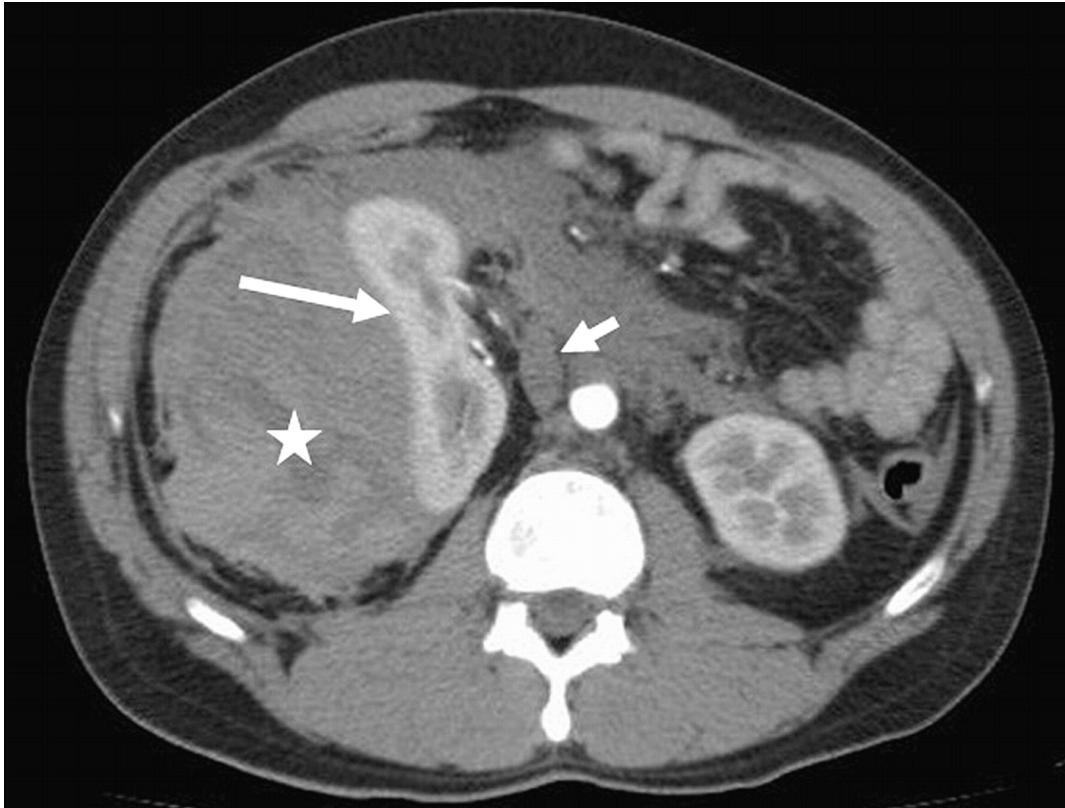


Fig. 156. A 35-year-old man was admitted to the hospital in hemorrhagic shock after a motor vehicular accident. The right adrenal gland was hemorrhagic. The above CT scan shows a retroperitoneal hematoma marked by the star which in turn shifted the right kidney (long arrow), which caused compression of the inferior vena cava (short arrow). (emj.bmj.com) (Wiki)

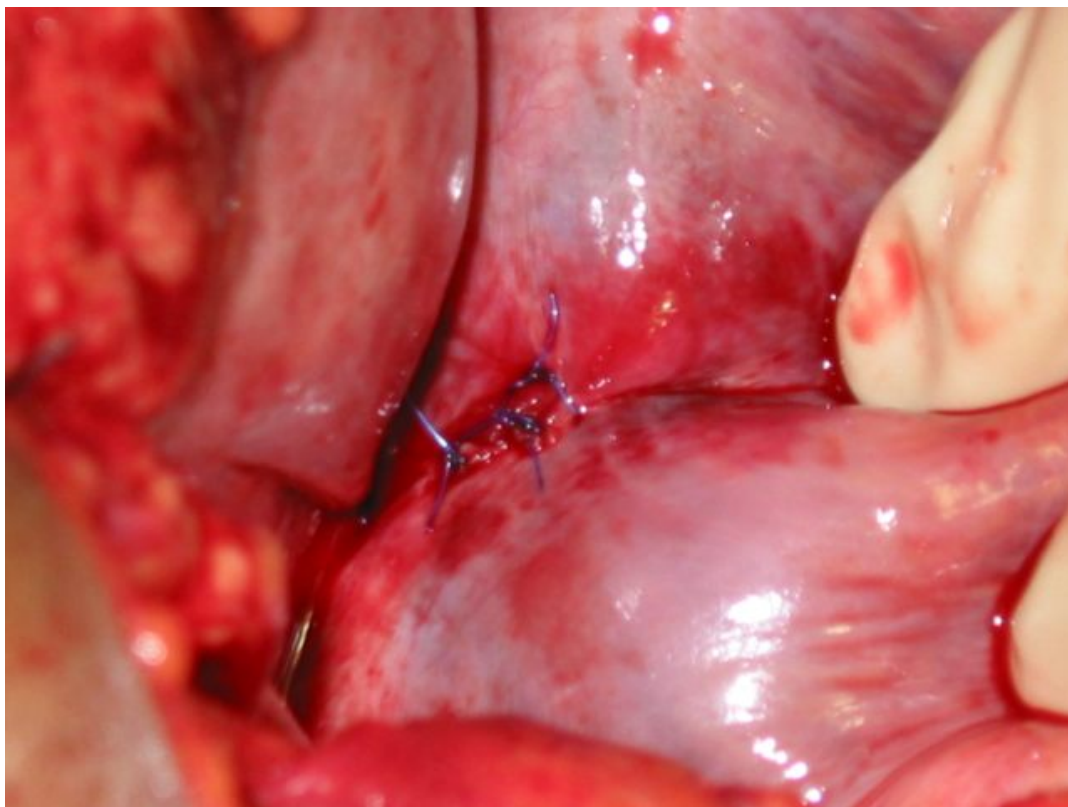


Fig. 157. The above image shows a laceration to the suprahepatic inferior vena cava at the right atrial junction. (trauma.org) (Wiki)

In a 2007 study by Kurguzov and Nadaria, they reported on 21 patients with injury to the inferior vena cava seen at the Central Clinical Hospital in Moscow, Russia. In their study 14 patients sustained stab wounds to the inferior vena cava, 5 had gunshot wounds and 2 had lacerations due to blunt force trauma. The infrarenal segment was involved in 17 of these patients, the renal segment in two, and the suprarenal segment in two. In 19 of these patients, the injuries to the inferior vena cava was associated with visceral injuries. The postoperative mortality rate was 38%. In 1978, Graham *et al.* reported on 301 patients with injury to the inferior vena cava treated over the previous 30 years. Again, penetrating injuries predominated, with gunshot injuries accounting for 228 patients. In their study the inferior vena cava was injured above the renal veins in 84 patients. Thirty-five patients died before treatment could begin. Those who went to surgery, the mortality was 30%.

Kudos *et al.* reported in 1984, their 14 year experience treating inferior vena caval injury. In their series, approximately 33% of patients with traumatic rupture or a tear of the inferior vena cava (Fig. 157) died before reaching the hospital. In the remaining 67%, the mortality was reported as high as 50%. In their review of the literature up to 1984, the mortality rate varied between 37 to 95%. In 1996, Degiannis *et al.* reported on a retrospective study of 74 patients with penetrating injuries of the abdominal inferior vena cava; the cause of injury was gunshot wound in 91% and stabbing in 9% (Figs. 158). Eighteen percent died before any caval repair could be carried out. The overall perioperative mortality was 39%.



FIGURE 27 (case 7).—Migratory intravascular foreign body. Lower portion of inferior vena cava showing wound of entry and superimposed metal fragment that had passed as embolus to branch of left pulmonary artery.

Fig. 158. The above image shows a penetrating injury to the distal inferior vena cava, immediately above the juncture of the common iliac veins. (history.amedd.army.mil) (Wiki)

If there is evidence of severe liver trauma, then one should suspect lacerations into the porta hepatis including the retrohepatic inferior vena cava.

Traumatic injuries to the inferior vena cava are typically associated with a large retroperitoneal hematoma.

There are cases of spontaneous rupture of the inferior vena cava, which is a rare clinical entity. It is believed the first reported case was by Nair *et al.* in 1986. In 2000, Mulkern *et al.* reported a case which involved a 71-year-old male patient who experienced a sudden onset of central abdominal pain, after moving some furniture at home 10 days before his admission. On the day of his admission the pain began to radiate down to his lower back, abdomen and groin. A review of his past medical history revealed he had undergone a right nephrectomy for tuberculosis approximately 35 years ago. It was postulated the cause of the tear in the inferior vena cava, was due to postoperative adhesions following the right nephrectomy, which because of the sharp movement of the lumbar spine during the moving of the furniture induced a small tear in the inferior vena cava. This small tear gradually enlarged, producing a large retroperitoneal hematoma over the ensuing 10 days. In Nair *et al.* case there was no previous history of surgery.

Pediatric Injuries: As in adults blunt traumatic injury to the inferior vena cava is rare in children, and when it does occur, it is associated with a high mortality rate. They have been reported as a complication of seatbelt injuries. For example, in 1999, De Cou *et al.* reported a seatbelt transection of the pararenal vena cava in a 5-year-old child, in a motor vehicular accident. She had also sustained a right renal vein transection, laceration of the right kidney, duodenal injury, and a fracture of L2. She not only survived her surgery, but was alive and well 10 months after the accident, with no sequelae of caval ligation and with normal right renal function.

The next chapter will cover the organs of the pelvis, which will include the urinary bladder, the rectum, the internal genital organs, and blood vessels and nerves.

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