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Modern Surgery - Chapter 9. Thrombosis and Embolism

John Chalmers Da Costa
Jefferson Medical College

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IX. THROMBOSIS AND EMBOLISM.

**Thrombosis** is the ante-mortem coagulation of blood in the heart or in a vessel, the coagulum remaining at its point of origin and plugging up the vessel partially or completely. The process is known as thrombosis; the clot is called the thrombus. This process is an essential part in the arrest of hemorrhage; it occurs in phlebitis and arteritis, and affords a frequent basis for embolism. Thrombi may form in the veins, in the arteries, and in the heart. Clotting is due to destruction of white blood-cells, fibrin ferment being set free, causing the union of calcium and fibrinogen and thus forming fibrin. Thrombosis is more common in the veins than in the arteries, the slow blood-current and the existence of valves favoring the deposit, though not causing it. A thrombus forms gradually, being deposited layer by layer; hence it is stratified or laminated. Fig. 56 shows a thrombus in a vein. All thrombi are either septic or aseptic, and they are also spoken of as fibrinous, red, hemostatic, leukocytic, etc.

**Causes of Thrombosis.**—The essential cause of all intravascular thrombi is damage to the endothelial coat. Any condition which causes the blood to contain an excess of fibrin-forming elements favors thrombosis, in the sense that a slight injury of the vascular endothelium will be followed by clot formation. Among conditions favoring thrombosis we must note particularly slowing of circulation, however caused. A special predisposing condition is the retarded circulation in tuberculosis, influenza, and fevers, the blood clotting behind the vein-valves after the endothelium has been damaged by toxins. Among other favoring states are inflammations; wounds; fractures; the pressure of a bandage or of a splint; varicose veins; ligation of a vessel; injuries of a vessel; foreign bodies in a vessel; atheroma in arteries; sutures in a vessel; certain diseases, such as gout, typhoid fever, pregnancy, and septic processes; phlebitis or arteritis arising in the vessel or from extension of surrounding inflammation; and entrance of specific organisms.

It has been asserted that so long as the endothelium of a vessel is uninjured a clot does not form. Slowing of the blood-current in aseptic conditions, it is now taught, will not cause thrombosis. One of the functions of the endothelial coat is to keep the blood fluid by preventing corpuscular disintegration. A thrombus can form only when fibrin ferment is set free, and fibrin ferment can be set free only when white corpuscles disintegrate. When moving blood coagulates, the third corpuscles first settle out, and then the leukocytes. This is known as the white or "ante-mortem" thrombus—the clot of moving blood. Thrombi from moving blood are rarely pure white; they contain some red corpuscles, forming mixed thrombi. The red thrombus plugs vessels which are cut across or ligated; it also occurs in septic processes and is formed after death. A thrombus soon undergoes a change. An aseptic clot usually "organizes"—that is, the clot is absorbed and replaced by fibrous tissue. The walls of the injured vessel become filled with leukocytes, leukocytes invade the clot,
the vascular endothelium proliferates, and the young cells follow the colonies of leukocytes into the thrombus. The thrombus is gradually removed by leukocytes and replaced by fibroblasts, the new tissue is vascularized and becomes granulation tissue, the granulation tissue is converted into fibrous tissue, and the fibrous tissue contracts. In some instances a thrombus is implanted on the wall of the vessel, and the tube is not permanently occluded. Such a condition may be obtained by the application of a lateral ligature about a small tear in a large vein. In most instances, after the formation of an intravascular thrombus, the vessel is converted into a narrow cord of fibrous tissue. A thrombus may degenerate and break down (fatty degeneration), giving rise to emboli or undergoing calcification. A calcified thrombus in a vein is known as a phlebolith. An infected thrombus may undergo liquefaction, infective emboli being set free (Fig. 57).

It was taught for many years that when an artery is ligated a thrombus quickly forms and reaches to the first collateral branch. This view was formulated in preantiseptic days. It is now known that when aseptic ligation is performed the thrombus is small and rarely reaches the first collateral branch; and is often actually absent, vascular obliteration being obtained by proliferation of connective-tissue cells and of cells from the endothelial coat. If any infection takes place the clot will reach the first collateral branch. The old rule of surgery was as follows: If an artery is cut near a large branch, tie the branch as well as the artery, in order to permit of the formation of a lengthy clot. This rule is no longer followed unless infection exists or is anticipated.

A clot in a vein often extends a long distance. The author has seen in a post-mortem examination a venous thrombus reaching from the ankle to the vena cava. A spreading clot of this sort is known as a propagated thrombus.

**Symptoms**.—The symptoms are dependent on the seat of the obstruction. An organ or a part of an organ may exhibit functional aberration. The local signs in a vessel accessible to touch or sight are the presence of a clot; if it be in an artery, anemia and the absence of pulse below the clot; if it be a vein, swelling and edema below it. There is usually pain at the seat of trouble, and anesthesia below it. Moist gangrene may follow venous thrombosis, and dry gangrene, arterial thrombosis. Thrombosis of the mesenteric vein is followed by gangrene of the bowel. Thrombophlebitis is a spreading inflammation of a vein in which a septic thrombus forms. We see this condition sometimes in the lateral sinus of the brain as a result of suppuration in the middle ear; in any of the cerebral sinuses after infected compound fracture of the skull; and in the uterine veins in puerperal sepsis. Infective thrombophlebitis is an early step in pyemia. Thrombo-arteritis is a spreading inflammation of an artery in which a septic thrombus forms or in which a septic embolus lodges. It occasionally attacks an aneurysmal sac.

**Treatment**.—If a thrombus forms in a large vessel of a limb, raise the limb a few inches from the bed, keep it perfectly quiet to avoid detachment of fragments (emboli), apply a bandage lightly from the toes up, and place
warm bottles around the extremity. The great danger is the formation of emboli, hence movements and rough handling are to be avoided. Gangrene is another danger, hence it is wise to favor venous return and the development of the collateral circulation by warmth, elevation, and bandaging. In septic thrombophlebitis, if the vessel is accessible, tie it above and below the clot, open the vessel, remove the clot, irrigate, and pack the wound with iodoform gauze. The general treatment for a septic condition should be stimulant and supporting. Massage is unsafe in any condition of thrombosis, and is particularly dangerous in septic thrombosis. In thrombo-arteritis treat as in the thrombophlebitis. If gangrene follows thrombosis, treat as previously directed (page 140).

**Embolism** signifies vascular plugging by a foreign body (usually a blood-clot) which has been brought from a distance. The foreign body is called an embolus. Emboli may arise either in the venous or in the arterial system, but lodge only in an artery, in capillaries, or in the veins of the liver. The initial thrombus may form upon a diseased heart-valve or in a vein. It may be composed of fat, microorganisms, air, or a portion of a tumor. An embolus is arrested when it reaches a vessel whose diameter is less than its own. It is usually caught just above a bifurcation. When an embolus lodges, it at once partially or entirely obstructs the circulation, and increases in size by thrombosis. Fig. 58 shows an impacted embolus. A non-septic embolus usually "organizes," and, as described on page 155, is replaced ultimately by fibrous tissue. A soft embolus may disintegrate and permit the re-establishment of the circulation. An embolus may cause an aneurysm. A septic embolus breaks down, forms a metastatic abscess, and sends other emboli onward in the blood-stream.

An embolus is more serious than a thrombus: it causes sudden plugging, which makes dangerous anemia inevitable, and it will produce gangrene if the collateral circulation fails. Embolism of the mesenteric artery causes necrosis of the intestine. In organs with terminal arteries (spleen, kidney, brain, and lung) there is no collateral circulation and embolism causes *infarction*. For instance, if an embolus lodges in the lung it produces an area of anemia; the removal of all propulsion upon the venous blood causes it to flow back and stagnate, and vascular elements exude, forming a wedge-shaped area of red tissue, the embolus being the apex of the wedge. This is known as the *red infarction*, and is often seen in the lung (Fig. 59). The white infarction, seen in the brain and kidney, is not due to retrogression of venous blood,
but is due to anemia and resulting coagulation-necrosis. A septic embo-
lus causes septic thrombosis and a septic infarction, and a septic in-
farction is followed by suppuration and the production of a pyemic abscess.
If emboli arise from a thrombus in one of the veins of the pulmonary circu-
lation, they usually lodge in the lungs, and rarely, though occasionally, pass
through. Emboli formed in vessels of the systemic circulation lodge most
often in the lungs, brain, kidney, or spleen (Nancrede). Emboli passing
into the portal vein lodge in the liver and operations upon the rectum may be
followed by hepatic embolism and abscess of the liver.

Symptoms.—The symptoms depend upon the organ involved. They
are sudden in onset, and are due to loss of function, which may be permanent
or which may be followed by inflammation, softening, or gangrene. Embo-
lism of the cerebral arteries may cause aphasia, paralysia, or coma. Embo-
lism of the pulmonary artery may cause almost instant death. Embolism
of a large artery of a limb produces symptoms identical with thrombus,
except more sudden and decided. This condition is frequently followed by
gangrene. Embolism of the superior mesenteric artery produces symptoms
similar to those caused by acute intestinal obstruction, and results in gangrene
of a portion of the intestine.

Treatment.—The treatment of aseptic embolism depends upon the part
involved. In a limb, keep the part warm in order to stimulate the collateral
circulation, elevate the extremity several inches from the bed, apply a bandage
lightly from the periphery, and insist on perfect quiet. Massage is unsafe.
If gangrene ensues, await a line of demarcation and amputate. In septic
thrombo-arteritis in an accessible region it would be good surgery to act as
in septic thrombophlebitis. After an operation upon veins (as the operation
for varicocele, for varix of the leg, or for hemorrhoids), after any cutting
operation, and after the infliction of a fracture, avoid as much as possible,
and for some time, movements or handling, as fragments of thrombus may
be detached.

Fat-embolism was first described in 1884 by von Recklinghausen. It
is a process which leads to an accumulation in the capillaries of liquid fat after
injuries of adipose tissue, high tension having forced the fat into the open
mouths of veins. Some little fat may get into the blood by means of the
lymphatics. Fat-embolism occasionally arises during osteomyelitis, after
extensive bruises, crushes, or lacerations, and after amputations, fractures,
resecciones, or rupture of the liver.* This fluid fat accumulates especially
in the capillaries of the lungs and brain. It may plug systemic capillaries.
If the patient recovers, he does so because the fat has been forced through
the vessels; if he dies, the death results from mechanical hindrance to function
and nutrition. Normal blood contains a small amount of finely emulsified
fat (from 1 to 3 parts per 1000). In a number of physiological and patho-
logical conditions the circulating blood contains considerable free fat. It
may be found in a pregnant woman, a nursing baby, a fat individual, or in
any one during digestion. "It has been noted in the following conditions:
chronic alcoholism; diabetes mellitus; certain diseases of the liver, heart,
and pancreas; chronic nephritis; splenitis; tuberculosis; malarial fever,
typhus fever, Asiatic cholera; and poisoning by phosphorus and by carbon

*G. H. Makins, in Heath's Dictionary.
Fat-embolism

monoxid. Lipemia commonly occurs as the result of lacerated wounds of the blood-vessels situated in fatty tissue, and after fractures of the long bones involving injury of the fatty matter” (“Clinical Hematology,” by John C. DaCosta, Jr.). In lipemia fatty embolism may occur if the amount of fat becomes excessive or if vascular damage favors plugging.

Symptoms.—The symptoms are those of edema of the lungs and exhaustion, often with coma or delirium, and sometimes, in the beginning, are wrongly thought to be due to shock. There are restlessness, dyspnea, rapid pulse and respiration, normal or subnormal temperature, and pallor followed by cyanosis. The chest exhibits many coarse râles, but on percussion gives a clear note. If pulmonary edema becomes marked, the patient spits up a bloody froth. If life is prolonged a day or two, oil is found in the urine. Small amounts of oil may be found in the urine after serious injuries or operations when no symptoms of embolism exist. For instance, for two or three days after a fracture it is often present. Nevertheless, the presence of the oil is always a cause of anxiety, and is often a warning. It is maintained by Grouhè that the amount of fat in the urine is in inverse ratio to the amount in the blood; the greater the amount excreted in the urine, the less the amount retained in the blood. Hence, fat in the urine makes the surgeon anxious, and a sudden diminution of the amount in the urine is a sign of grave danger if there develops increasing difficulty in respiration (“Rev. de Chir.,” July, 1895). The inverse ratio said to be maintained between fat in the blood and fat in the urine, if it really exists, is similar to a finding of Lépine in diabetes, that is, if a diabetic is given diuretics, the sugar in the urine increases and the sugar in the blood decreases. These symptoms never occur until at least twelve hours after the accident, and rarely before the third day. The symptoms occur at a later period than those of shock, and at an earlier period than those of ordinary embolism of the lung. If some of the oil is forced through the vessels of the lung, it will lodge in other regions and produce other symptoms. Oil may appear in the urine as above stated. Urinary suppression may occur. Delirium may arise, there may be twitching, convulsions, or paralysis, or the patient may pass into coma. Severe cases of fat embolism are commonly fatal; milder cases are often recovered from. I have lost a case operated upon for carcinoma of the breast from this cause.

Treatment.—The treatment consists in the administration of stimulants, such as strychnin, alcohol, and carbonate of ammonium, the use of external heat; the employment of oxygen by inhalation; and the administration of diuretics and of nitroglycerin hypodermatically. Artificial respiration may tide a patient over a crisis. If an external wound exists, the drainage must
be free, and the damaged part should be thoroughly immobilized. In order to prevent fat-embolism after a severe injury insist on rest. Massage used early after some injuries is dangerous, as it may force fluid fat into the vessels. When a severe contusion causes the formation of a large cavity filled with blood, Groubé advises incision, to lessen the danger of fat-embolism.*

Air-embolism.—Air may enter a vein during a surgical operation or it may be injected accidentally while giving a hypodermatic injection, hypodermoclysis, or a saline infusion into a vein. It is very rarely that any symptoms follow. It was long thought that such an accident must be extremely dangerous. The experiments of my colleague, Professor Hare, indicate that quantities of air may be injected into the veins of a dog without apparent harm. The entry of a small amount of air into the veins of a human being will not be apt to induce dangerous symptoms, but it may be fatal. The more rapidly it is introduced and the greater the amount, the greater is the danger. The manner in which it can induce death is doubtful. Some maintain that it causes the blood in the right side of the heart to froth, and thus prevents normal action of the valves, the heart becoming unable to propel blood through the lungs. If a surgeon divides a large vein, air may be sucked in, and there is particular danger in such an accident if a vein at the root of the neck or a cerebral sinus is torn or incised, or if the damaged vessel lies in scar tissue and cannot collapse.

Symptoms.—There is a sucking sound and serious symptoms may or may not follow. Twice I have wounded the subclavian vein and have heard this sound, but no alarming symptoms developed, but no alarming symptoms developed. If serious symptoms are produced, they arise suddenly, and consist of extreme failure of circulation, gasping for air, convulsions, and possibly death.

Treatment.—Compress the vein with the finger and clamp it quickly. Suspend the anesthetic, lower the head, employ artificial respiration and inhalation of oxygen, and give strychnin hypodermically.