Pathophysiology

Dr. Yasser Mufid

Lecture 13

V. Disorders of cardiovascular system

XIV. Hypotension

The term "hypotension" is usually used only when blood pressure has fallen so far that enough blood can no longer reach the brain, causing dizziness and fainting. It is often defined as systolic/diastolic : 90/60 mmHg, but 100 mmHg may be more appropriate if the patient normally has hypertension.

Causes of hypotension

•Emotional stress, fear, insecurity or pain (the most common causes of fainting)
• Dehydration, which reduces blood volume
• The body's reaction to heat, which is to shunt blood into the vessels of the skin, leading to
dehydration
• Blood donation
• Internal bleeding, such as a perforated stomach ulcer
• Blood loss from trauma, such as a road accident or deep cut
• Pregnancy
• Medications for high blood pressure
• Diuretics, which produce fluid loss
• Medications for depression
• Medications for certain heart conditions
• Allergic reaction to certain drugs or chemicals
• Some forms of infection, such as toxic shock syndrome
• Heart disease, which can hamper the pumping action of the heart muscle
• Some nervous system disorders, such as Parkinson's disease

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Normal BP results from a balance between the peripheral vascular resistance and the cardiac output (CO), with total blood volume affecting both. Cardiac output is a product of the stroke volume (SV) and the heart rate (HR): CO = SV x HR
Hypotension results when either the stroke volume or the heart rate is decreased. In addition, blood volume provides the "substrate" that the resistance vessels "push" against in order to regulate BP. ***Thus, even maximal vasoconstriction will be ineffective if volume status is inadequate.*** This key point resurfaces in managing many hypotensive patients.
The peripheral vascular resistance (PVR) is regulated by a variety of mechanisms. Only a small proportion of the blood volume is involved in perfusing tissues at any given time. Most of the total blood volume is contained in the venous system. The veins serve as blood reservoirs that are mobilized by the neuroendocrine system in time of need. Certain organs, such as the heart and brain, are autoregulated. Their perfusion is influenced by metabolic factors and not by the neuroendocrine system. Thus, blood flow is preserved and can actually be enhanced in early volume loss.
Adrenergic receptors are located in organs based on their function in the "fight or flight" response to stress. Non-essential organs in acute stress events (such as the gastrointestinal tract) have high concentrations of vasoconstrictive alpha-1 (A1) receptors, while those essential to survival in acute stress (the heart, lung, and skeletal muscles) have high concentrations of vasodilatory beta-2 (B2) receptors. Cardiac beta-1(B1) receptors produce increased chronotrope and inotropy with consequent increased oxygen demand. Dopaminergic receptors are primarily located in the splanchnic and the renal beds. These receptors are stimulated by mediator release from nerve endings (norepinephrine) and the endocrine system (epinephrine). Mediator release is stimulated by the vasomotor centers located in the medulla and hypothalamus. Inhibitory outputs from cardiac, renal, and blood vessel baroreceptors affect these centers. Pathological drops in blood pressure cause decreased outputs to be sent from the baroreceptors, disinhibiting the vasomotor centers. Sympathetic nervous system output or tone is thus augmented; "vagal tone" is conversely decreased.
In low pressure states, like hypovolemia, there is less baroreceptor stimulation which leads to ADH release. The release of ADH leads to: 1) An increase in water absorption in the distal renal tubules and then an increase in vascular blood

volume; and 2) Peripheral vasoconstriction. Other mediators that increase adrenergic tone include carbon dioxide and hydrogen ions.
The kidney plays a role in the regulation of blood pressure through the following mechanisms:

- Glomerular filtration rate (GFR) decreases in hypotension which decreases sodium transit time in the tubules and increases its absorption. In turn, this increases the absorption of water.
- Increased water absorption mediated by ADH in the distal tubule.

Syndromes associated with hypotension

A. **Orthostatic hypotension**: also called **postural hypotension**, is a common form of low blood pressure. It occurs after a change in body position, typically when a person stands up from either a seated or lying position. It is usually transient and represents a delay in the normal compensatory ability of the autonomic nervous system.
B. **Neuro-cardiogenic syncope** (vasovagal syncope) enhancement of parasympathetic nervous system (vagal) tone and withdrawal of sympathetic nervous system tone leading to a cardio inhibitory response, characterized by a drop in heart rate (negative chronotropic effect) and in contractility (negative inotropic effect) leading to a decrease in cardiac output that is significant enough to result in a loss of consciousness. On the other end of the spectrum is the vasodepressor response, caused by a drop in blood pressure (to as low as 80/20) without much change in heart rate. This phenomenon occurs due to vasodilation, probably as a result of withdrawal of sympathetic nervous system tone.
C. **Postprandial hypotension**, A decline in blood pressure that occurs 30 to 75 minutes after eating substantial meals. When a great deal of blood is diverted to the intestines (a kind of "splanchnic blood pooling") to facilitate digestion and absorption, the body must increase cardiac output and peripheral vasoconstriction to maintain enough blood pressure to perfuse vital organs, such as the brain. Postprandial hypotension is believed caused by the autonomic nervous system not compensating appropriately, because of aging or a specific disorder.

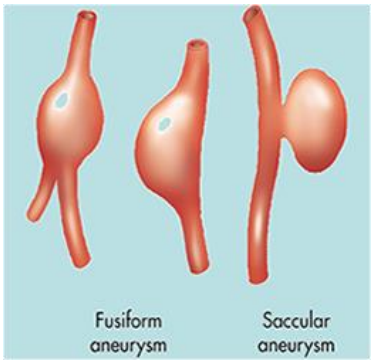
XV. Aneurysm

A localized widening (dilatation) of an artery, a vein, or the heart. At the point of an aneurysm, there is typically a bulge. The wall of the blood vessel or organ is weakened and may rupture. Aneurysms may be classified by type, morphology, or location.

Morphological types of aneurysm

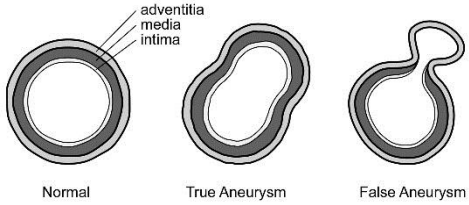
Aneurysms can also be classified by their macroscopic shape and size.

A. Saccular aneurysms are spherical in shape and involve only a portion of the vessel wall; they vary in size from 5 to 20 cm (8 in) in diameter, and are often filled, either partially or fully, by a thrombus.
B. Fusiform aneurysms ("spindle-shaped" aneurysms) are variable in both their diameter and length; their diameters can extend up to 20 cm (8 in). They often involve large portions of the ascending and transverse aortic arch, the abdominal aorta, or less frequently the iliac arteries.



Aneurysms could be either True or false aneurysms

A true aneurysm is one that involves all three layers of the wall of an artery (intima, media and adventitia). True aneurysms include atherosclerotic, syphilitic, and congenital aneurysms, as well as ventricular aneurysms that follow transmural myocardial infarctions (aneurysms that involve all layers of the attenuated wall of the heart are also considered true aneurysms).



A false aneurysm, or pseudo-aneurysm, is a collection of blood leaking completely out of an artery or vein, but confined next to the vessel by the surrounding tissue. This blood-filled cavity will eventually either thrombus (clot) enough to seal the leak, or rupture out of the surrounding tissue.

Pseudoaneurysms can be caused by trauma that punctures the artery, such as knife and bullet wounds, as a result of percutaneous surgical procedures such as coronary angiography or arterial grafting, or use of an artery for injection.

Types according to Location

Aneurysms can also be classified by their location:

The **heart**, including coronary artery aneurysms, ventricular aneurysms, aneurysm of sinus of Valsalva, and aneurysms following cardiac surgery.

The **aorta**, namely aortic aneurysms including thoracic aortic aneurysms and abdominal aortic aneurysms.

The **brain**, including cerebral aneurysms, berry aneurysms, and Charcot-Bouchard aneurysms.

The **legs**, including the popliteal arteries.

The **kidney**, including renal artery aneurysm and intraparechymal aneurysms.

Capillaries, specifically capillary aneurysms.

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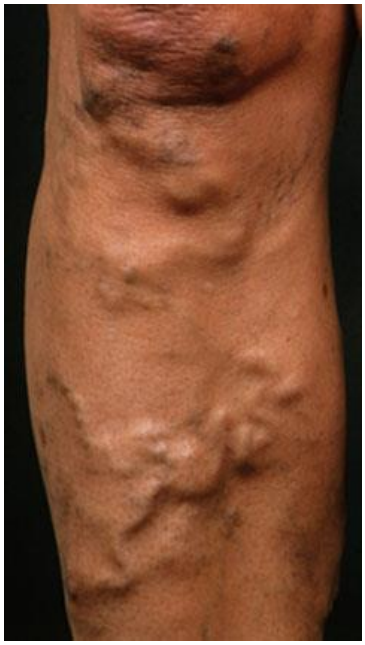
1. Most aneurysms are caused by degenerative disease affecting the vessel as in atherosclerosis
2. Structural weakness & Hemodynamic forces leading to damage and loss of intima resulting in reduction in the elastin and collagen content of the media
3. Loss of the Collagen; tensile strength of the adventitia of the vessel.
4. Absence of the elastin; recoil capacity of the media.
5. Rare causes

a) Congenital: Marfan's syndrome, Berry aneurysms
b) Post-stenotic: Coarctation of the aorta, Cervical rib, Popliteal artery entrapment syndrome
c) Traumatic: Gunshot, stab wounds, arterial punctures
d) Inflammatory: takayasu's diseases, Behcet's disease
e) Mycotic: Bacterial endocarditis, syphilis
f) Pregnancy associated: Splenic, cerebral, aortic, renal, iliac & coronary

Risk factors

smoking, hypertension, hypercholesterolemia.

XVI. Varicose veins



a condition in which the superficial veins, especially of the legs, become tortuous, knotted, and swollen: caused by a defect in the venous valves or in the venous pump that normally moves the blood out of the legs when standing for long periods.

Causes

1. ***Primary***: Congenital abnormality, most common cause (weak mesenchymal tissue)

2. Secondary: Anything that raises intra-abdominal pressure or raises pressure in superficial/deep venous system:

a) Pregnancy
b) Abdominal/pelvic mass
c) Ascites
d) obesity
e) constipation
f) thrombosis of leg veins
g) spend long periods of time standing (barbers, for example)

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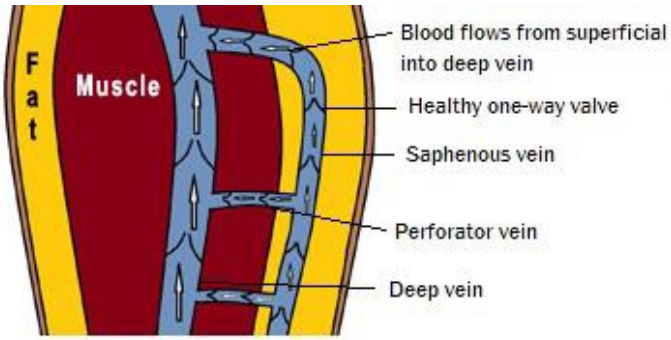
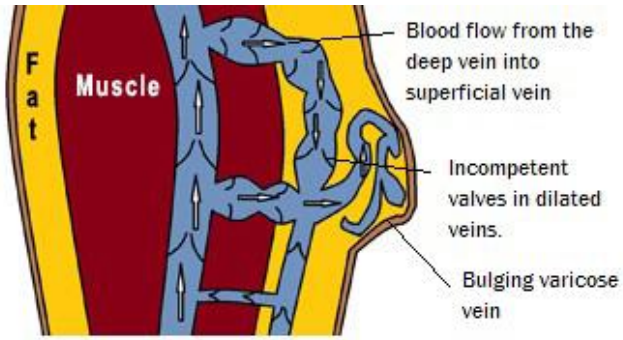
The two most widely accepted theories of primary varicose vein pathophysiology are (1) primary valvular incompetence and (2) primary (congenital) vein wall weakness. The primary valvular incompetence theory, postulates that varicose veins develop as the sequel of central valvular incompetence related to a paucity or atrophy of valves. This causes venous hypertension in the vein segment below, which in turn damages adjacent peripheral valves and causes propagation of varicose transformation in a central-to-peripheral direction.
The primary vein wall weakness theory states that varicose veins develop from a defect in vein wall integrity rather than from a problem with the valves

themselves. The components of a normal vein wall include collagen matrix that provides strength, elastic fibers that provide compliance, and three smooth muscle layers (circular media surrounded by longitudinal intimal and adventitial layers) that control venous tone.

Primary vein wall weakness can lead to valvular incompetence. In contradistinction to that of secondary varicose veins that result from post-thrombophlebitic valve leaflet damage, the mechanism of valve failure in primary varicose veins is abnormal collagen synthesis that leads to weakening and expansion of the valve annulus and in turn causes poor valve leaflet apposition and venous reflux despite relative absence of actual valve leaflet damage. As with other primary varicose veins, perforator reflux develops secondarily from a primary problem with vein wall integrity. Once established, perforator reflux contributes to superficial venous hypertension, especially in the setting of concomitant deep system venous reflux.



Blood Flow in Healthy Saphenous and Perforator Veins



Blood Flow in Dilated Sphenous and Perforator Veins