Vascular surgery

Vascular tree can be devided into three main components
:
1..VEINS:which drain blood toward the heart ,they carry deoxygenated blood except the pulminary veins which carry bright blood from the lungs to left artium.
Venous side of low pressure and some times reach negative value e.g, neck vein .the veins consider as capacitant vesseles.
2.ATERIESthey distibute the blood from the heart to all body components theycarry oxygenated blood except the pulminary artery.
3..MICRO CICULATION;small sized vesselles less than 0.1 mm diameter responsible for distibution and collection of blood including the vascular tree themselves.
The arteries can be divided into 2 main types from functional point of view ,transporting arteries (elastic arteries)in which the intema and media composed of mutiple layers of elastin ,second type is disributive arteries (muscular arteries)those contain only single layer of elastin the thiird layer of the vessel called adventitia .
Some capillaries controlled by pre-cappilary sphincter while some lack such sphincter and considered as arerio-venous shunt.
because of thier ability for huge dilatation to take more bloodthe viens are called capacitant vesselstheir intima composed of only single layer of endothelium,small and medium sized vesseles semilunar valves that permit only unidirectional flow.

AUTOREGULATION:

It is the ability of vascular bed to provide constant blood flow regardless oerfusion pressure.for skeletal muscles 20-40mm hg ,,50-60mmhg for the brain is the lower limits for auto regulation.
ATHEROGENESIS: IT IS THE FORMATIONOF ATHEROMATOUSPLAQUE LEADING TO THROMBUS AND VASCULAR INJURY.
Three categories of vascular injuries can be recognized here:
a..type 1functional alteration of endo thelial cells without morphological changes.that might occur due to flow disturbance lipid accumulated within macrophages and(FOAM CELLS)are the earliest sign of atherosclerosis.
b..type 2the foam cells may rupture and release thier toxic products lead to proliferation and migration of smooth muscle cells causing fibro-intimallesion lead to formation of (after atime) smooth plaque.
c..type 3 the smooth plaque will undergo distruption and fissuring reaching to the media this will increase the platelets adhesion and extensive polifiration of smooth muscle cells ,this is called mural thrombus which involve the whole wall of the vessel.
Periphral arterial blood flow follows the physical principles of fluid dynamics i.ediameter and pressure.

CRITICAL STENOSIS:

Is defined as the dgree of stenosis sufficient to produce significant drop in the pressure .this pressure gradiant result in energy loss and do not become evident untill cross sectional area of the vesseles is reduced by more than 75%of the original lumen.the length of stenosis is less important than the diameter of stenosis (l) (r4).

COLLATERAL CIRCULATION:group of pre-existing pethways that enlarges as stenosis develops in the main arterial supply.collateral pathway may povide flow distal to the occlusion which is sufficient to preserve the viability BUT it is never effecient as patent artery, because the collateral resistant always exceeds the major artery.

Periphral vascular disease

Causes:

1.. atgerosclerosis.
2.. embolic phenomena.
3..traumatic injuries to the vesseles.

Atheroma tend to occurs in certain locations like proximal internal carotid artery ,infra renal aorta,common and external iliac arteries.
Atheroma also tends to form at branching points.
RISK FACTORS;

1.smoking 2.D.M 3.hypertension 4.hyperlipidaemia.

CLINICAL PRESENTATIONS::

A; claudication:-pain in major muscles distal to the stenosis during walking .the 10 years prognosis is good only 10%progress to sver ischemia and may ended by limb loss.

B; Ischemic rest pain :-when sever compromize to blood supply occur during even rest i.e the blood not sufficient to keep the limb viable at rest.this mean that rest pain regards as limb threating condition,85%loss thier limb in the next 5 years if revascularisation not achieved.

C; Ulceration &Gangrene; this most sever condition in which circulation is not enough to mantain tissue viability leading to tissue deathand dry gangrene which can complicated by infection cuasing wet gangrene.

Evaluation of patient

1; history.
2; physical examination.
3;Non invasive tests doppler u.s , duplex .
4; arteriography.
5;Magnatic resonant angiography.

Treatment

Medical treatment

A,stop smoking.
B,excesize programs to induce collateral circulation.
C, anti-coagulant.
D, ainti-platelets.

Interventional treatment

This is done by catheterisation of the vessel and expanding new techniques developedthrough (PTA)per cutaneous trans luminal angioplasty and baloon dikatation of the vessel can be achieved or placement of stents or mesh can be done and we can also remove the clots from the vesseles

Surgical treatment;include diffirent types of surgery to mantain blood supply to the organs;-

1;end arterectomy mean removal of the atheromotous material with the intema and part of the media.
2;resection of the stenosed part and end to end anastomosis.
3; resection and interposition graft from the same patient ,we used commonly saphnous vien grafts.
4;use of synthetic grafts like gortex , dacron.
5;AMPUTATION of gangrenous limb.

Acute arterial embolism

ONEof the most common emergencies of the vascular surgery department.
It occurs due to translocation and dislodgment of material within the arterial blood stream to more distal sites, resulting in interruption of blood supply.
It occurs commonly at bifurcation of the arteries especially the lower limbs like iliac and femoral vesseles.
Some times small micro-emboli cause more distal injury (blue toe syndrome).
The commonest cause of acute arterial emboli is cardiac origin due to atrial fibrilation,ventricular source from M.I .,ventricular aneurysm.
Rheumatic heart disease is another cause.
The next cause after cardiac is proximal arterial pathology either atherosclerotic plaque separation oraneurysmal area that become source for such thrombosis.

Clinical evaluation:

In acute embolism the symptom are of sudden onset.....

The cardinal signs of ischemia descriped as ps;
Pain ,pulseless, pallor ,parasethesia ,paralysis,poikilothermia.

Rapid assesment to detect the level of occlusion by physical examination,compare with the other side ,check the pulse level ,color changes.
Nerves are the most vulnarable to ischemia motor and sensory loss mandate urgent intervention.
Doppler study to asses the level of obstruction and collateral state.

Treatment:-

This condition is emergency surgical condition .after correction of hydration by I.V fluidsto protect thy kidneys from toxic material (myoglubinuria)that cause acute tubular necrosis anr acute renal faliure,bollus dose of heparin 5000i.u followed by 1000i.u per hour till the time of surgery.
Time factor for surgery is important (golden hours)are 6-8 hours before serious irreverssible tissue damage (nerve and muscle).
Surgical procedure including incision on the groin and open the femoral artery ,passing embolectomy catheter toremove the clot from the artery.
Some times bypass graft surgery needed in cases of adequate thrombo-embolectomy.
The thrombogenic agents are materials can dissolve the clot completely or partialy may be used to dissolve the rest emboli which are small or un accessable for surgery e.e streptokinase or uro kinase..