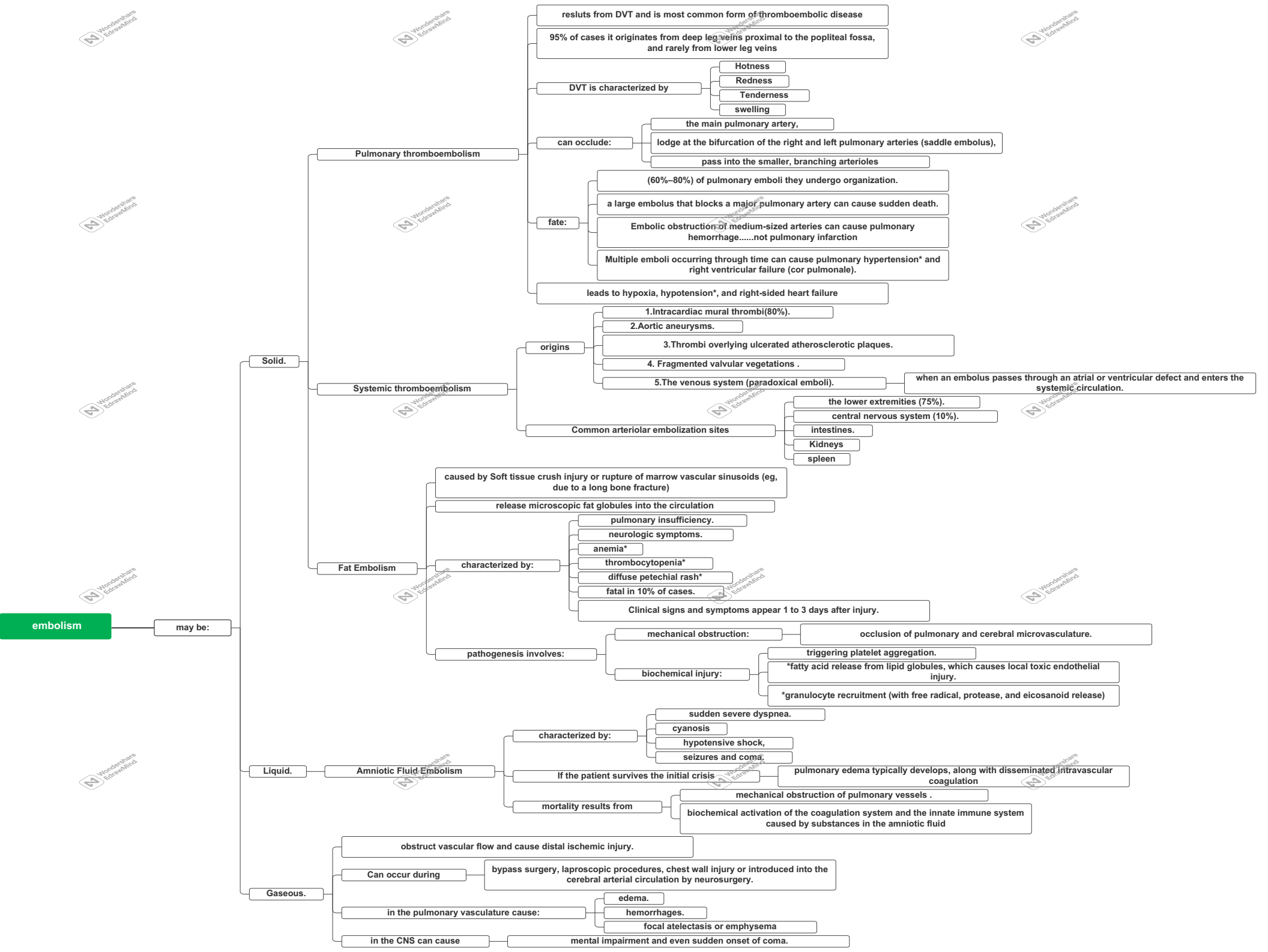
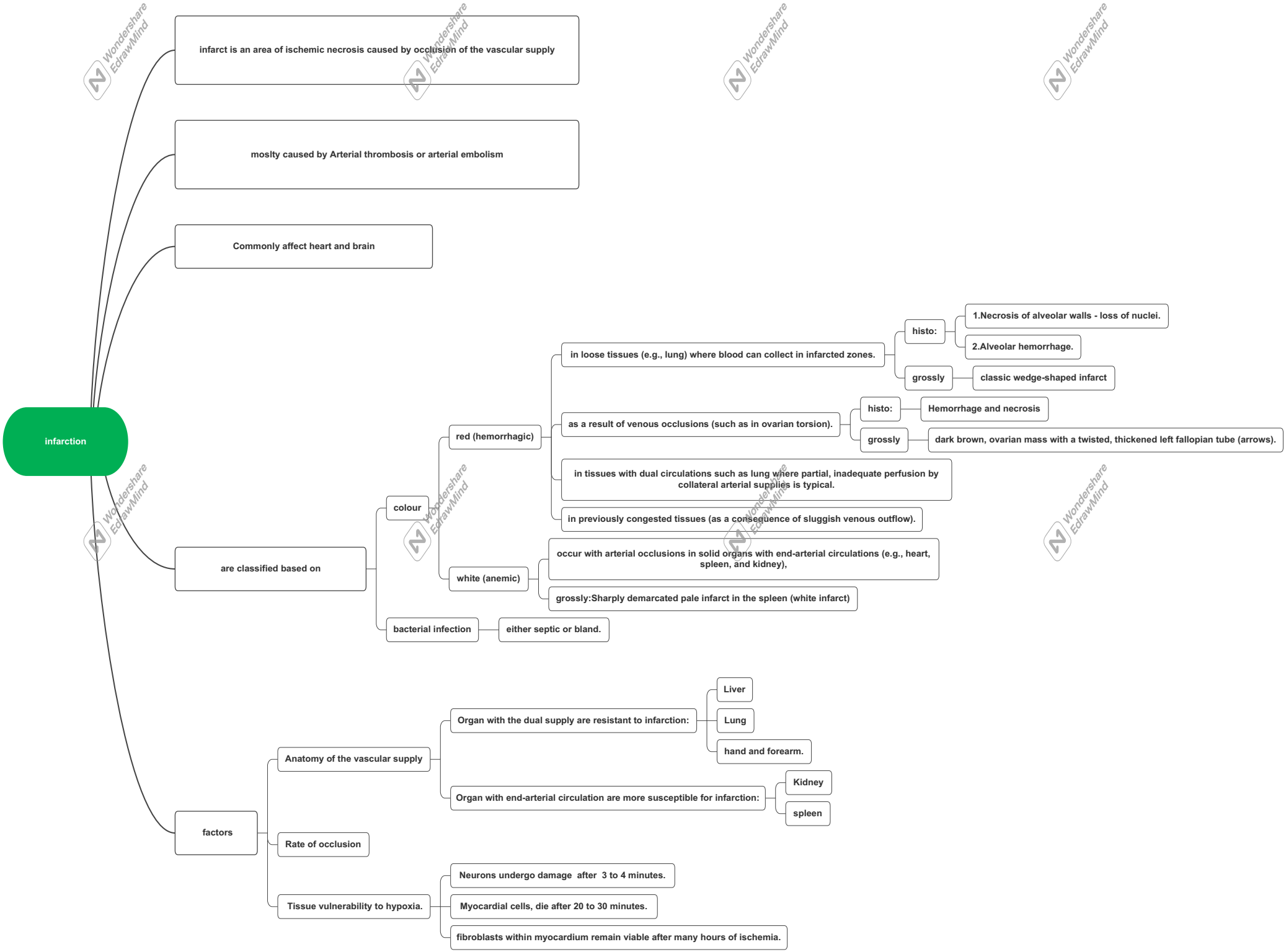

embolism
may be:
Solid.
Pulmonary thromboembolism
resluts from DVT and is most common form of thromboembolic disease
95% of cases it originates from deep leg veins proximal to the popliteal fossa, and rarely from lower leg veins
DVT is characterized by
Hotness
Redness
Tenderness
swelling
can occlude:
the main pulmonary artery,
lodge at the bifurcation of the right and left pulmonary arteries (saddle embolus),
pass into the smaller, branching arterioles
fate:
(60%–80%) of pulmonary emboli they undergo organization.
a large embolus that blocks a major pulmonary artery can cause sudden death.
Embolic obstruction of medium-sized arteries can cause pulmonary hemorrhage.....not pulmonary infarction
Multiple emboli occurring through time can cause pulmonary hypertension* and right ventricular failure (cor pulmonale).
leads to hypoxia, hypotension*, and right-sided heart failure
Systemic thromboembolism
origins
1.Intracardiac mural thrombi(80%).
2.Aortic aneurysms.
3.Thrombi overlying ulcerated atherosclerotic plaques.
4. Fragmented valvular vegetations .
5.The venous system (paradoxical emboli).
when an embolus passes through an atrial or ventricular defect and enters the systemic circulation.
Common arteriolar embolization sites
the lower extremities (75%).
central nervous system (10%).
intestines.
Kidneys
spleen
Fat Embolism
caused by Soft tissue crush injury or rupture of marrow vascular sinusoids (eg, due to a long bone fracture)
release microscopic fat globules into the circulation
characterized by:
pulmonary insufficiency.
neurologic symptoms.
anemia*
thrombocytopenia*
diffuse petechial rash*
fatal in 10% of cases.
Clinical signs and symptoms appear 1 to 3 days after injury.
pathogenesis involves:
mechanical obstruction:
occlusion of pulmonary and cerebral microvasculature.
biochemical injury:
triggering platelet aggregation.
*fatty acid release from lipid globules, which causes local toxic endothelial injury.
*granulocyte recruitment (with free radical, protease, and eicosanoid release)
Liquid.
Amniotic Fluid Embolism
characterized by:
sudden severe dyspnea.
cyanosis
hypotensive shock,
seizures and coma.
If the patient survives the initial crisis
pulmonary edema typically develops, along with disseminated intravascular coagulation
mortality results from
mechanical obstruction of pulmonary vessels .
biochemical activation of the coagulation system and the innate immune system caused by substances in the amniotic fluid
Gaseous.
obstruct vascular flow and cause distal ischemic injury.
Can occur during
bypass surgery, laproscopic procedures, chest wall injury or introduced into the cerebral arterial circulation by neurosurgery.
in the pulmonary vasculature cause:
edema.
hemorrhages.
focal atelectasis or emphysema
in the CNS can cause
mental impairment and even sudden onset of coma.


infarction
infarct is an area of ischemic necrosis caused by occlusion of the vascular supply
mosIty caused by Arterial thrombosis or arterial embolism
Commonly affect heart and brain
are classified based on
colour
red (hemorrhagic)
in loose tissues (e.g., lung) where blood can collect in infarcted zones.
histo:
1.Necrosis of alveolar walls - loss of nuclei.
2.Alveolar hemorrhage.
grossly
classic wedge-shaped infarct
as a result of venous occlusions (such as in ovarian torsion).
histo:
Hemorrhage and necrosis
grossly
dark brown, ovarian mass with a twisted, thickened left fallopian tube (arrows).
in tissues with dual circulations such as lung where partial, inadequate perfusion by collateral arterial supplies is typical.
in previously congested tissues (as a consequence of sluggish venous outflow).
white (anemic)
occur with arterial occlusions in solid organs with end-arterial circulations (e.g., heart, spleen, and kidney),
grossly:Sharply demarcated pale infarct in the spleen (white infarct)
bacterial infection
either septic or bland.
factors
Anatomy of the vascular supply
Organ with the dual supply are resistant to infarction:
Liver
Lung
hand and forearm.
Organ with end-arterial circulation are more susceptible for infarction:
Kidney
spleen
Rate of occlusion
Tissue vulnerability to hypoxia.
Neurons undergo damage after 3 to 4 minutes.
Myocardial cells, die after 20 to 30 minutes.
fibroblasts within myocardium remain viable after many hours of ischemia.