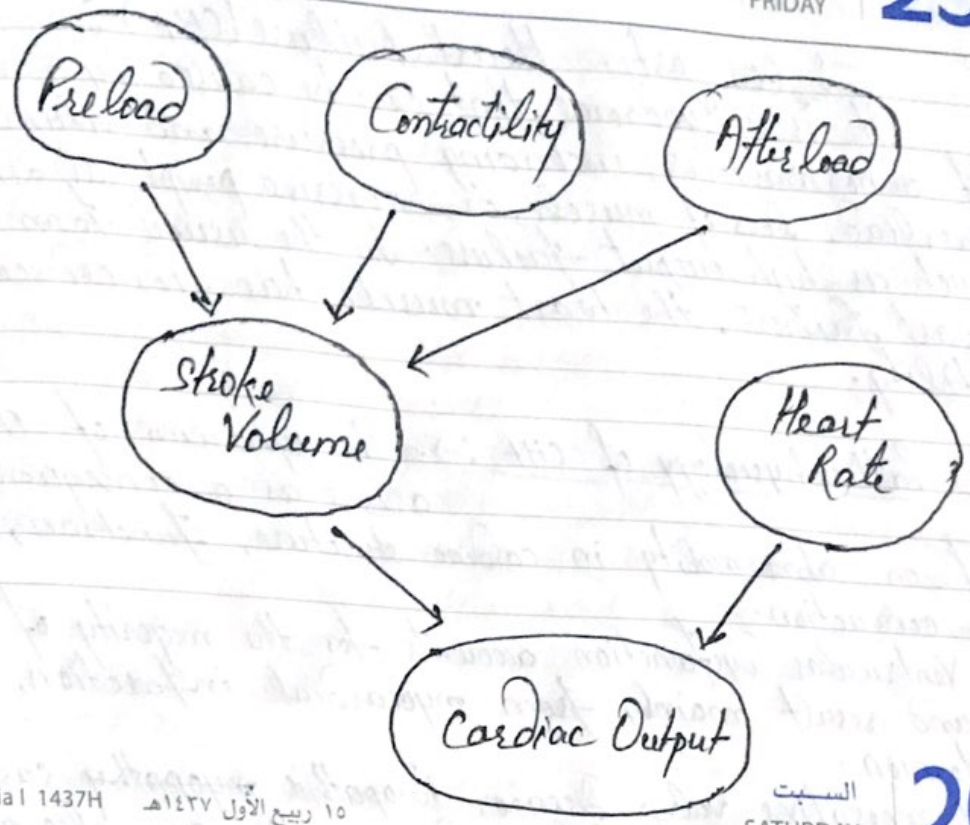
7 :- Congestive Heart failure (CHF) :-

It is a syndrome that can be caused by a variety
8 of abnormalities, including pressure and volume
overload, loss of muscle, or excessive peripheral demands
9 such as high output failure. In the usual form of
heart failure, the heart muscle has reduced contra-
10 ctility.

11 Pathophysiology of CHF :- = The syndrome of CHF arises as a consequence
12 of an abnormality in cardiac structure, function, & rhythm
or conduction.
13 = Ventricular dysfunction accounts for the majority of cases
and result mainly from myocardial infarction, hyper-
14 tension.
= Degenerative valve disease, idiopathic ~~myopathy~~ cardio-
15 myopathy, and alcoholic cardiomyopathy are also major
causes of heart failure.
16 = CHF indicates not only inability of the heart to main-
tain adequate oxygen delivery, it is also a systemic
17 response attempting to compensate for the inadequacy.
= The determinants of cardiac output include heart rate and
18 stroke volume. The stroke volume is further determined by
the preload (the volume that enters the left ventricle), contracti-
Notes lity and afterload (the impedence of the flow from ملاحظات
the left ventricle). These variables are important in under-
the pathophysiologic consequences of heart failure and
the potential treatment.

14 Rabia I 1437H ١٤ ربيع الأول ١٤٣٧هـ

الجمعة FRIDAY **25**



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15 Rabia I 1437H ١٥ ربيع الأول ١٤٣٧هـ

السبت SATURDAY **26**

= The failing heart in CHF can be best evaluated with the above variables considered together.
= If cardiac output falls, either the heart rate or stroke volume must change in order to maintain perfusion.
= If stroke volume can not be maintained then the heart rate must increase to maintain cardiac output.

Notes ملاحظات

= The pathophysiology behind CHF includes not only a structural abnormality, it also includes the cardiovascular response to poor perfusion with the activation of the neurohumoral system.

24 الخميس THURSDAY 13 Rabia I 1437H

= Activation of the renin-angiotensin system attempts to increase preload by stimulating retention of salt & water, increasing vasoconstriction (and thus afterload) and augmenting cardiac contractility.

= Prolong activation of renin-angiotensin system results in loss of myocytes and maladaptive changes in the surviving myocytes and the extracellular matrix.

= The stressed myocardium undergoes remodeling and dilation in response to the insult. This process has also detrimental effects on the functioning of the lungs, kidneys, muscles, blood vessels, and probably other organs.

= The symptoms of heart failure can be related to either the reduction of cardiac output (fatigue, weakness) or to excess fluid retention (dyspnea, orthopnea and cardiac wheezing).

= Patients with previous evidence of heart disease, diabetes mellitus, hypertension or documented coronary artery disease are at increased risk for CHF, and one should always consider CHF in the differential diagnosis of acute respiratory failure in these patients.

Notes ملاحظات

Therapy for CHF :→ Understanding the pathophysiology of heart failure allows one to acheivement the goals of treatment, which are to relieve symptoms, avoid hospital admissions, and prolong life.

- Treatment for CHF consists of a combination of phasmacologic and nonphasmacologic therapies.
- The basic theory include termination of the renin-angiotensin system to prevent the long-term complications of the cascade.
- Treatment often focuses on a combination of afterload reduction with angiotensin-converting-Enzyme (ACE) inhibitors,
  - Reduction of catecholamines surges with β blockers.
  - Preload reduction with diuretics.

Newyork heart association (NYHA) classification system, which separates patients based on the limitations they experience in performing certain activities. An NYHA class I patients has almost no evidence of limitation in daily performance of activities, whereas an NYHA class IV patients experiences severe symptoms event at rest.

ملاحظات
Notes

22 الثلاثاء TUESDAY 11 Rabia I 1437H ١١ ربيع الأول ١٤٣٧هـ

New York Heart Association classification of CHF

stat / Degree	Symptoms and Activity - Limitations
I - None	No symptoms from ordinary activities
II - Mild	Comfortable at rest or during mild exertion
III - Moderate	Symptomatic with any activity
IV - Severe	Symptomatic at rest. Confined to bed or chair

Ischaemic heart disease :→ Ischaemic heart disease is a disease which is characterized by reduced blood supply to the heart. The coronary arteries supply blood to the heart muscle and no alternative blood supply exists, so a blockage in the coronary arteries reduces the supply of blood to heart muscle.

←: ANGINA :→

Angina also known as angina pectoris, is chest pain, or pressure, usually due to not enough blood flow to the heart muscle. Angina is usually due to obstruction or spasm of the arteries that supply blood to the heart muscle. The main mechanism of coronary artery obstruction is atherosclerosis as part of coronary artery disease.

There is a weak relationship between severity of pain and degree of oxygen deprivation in the heart muscle. In some cases angina can be quite severe. People with an average age of 62 years, who have moderate to severe degrees of angina (grading by class II, III and IV) have a 5-years survival rate of approximately 92%.

Notes ملاحظات

Angina results when there is an imbalance between the heart's oxygen demand and supply. This imbalance can result from an increase in demand (eg. during exercise) without a proportional increase in supply.

Classification :→

(1) Stable angina :— Also known as 'effort angina' this refers to the classic type of angina related to myocardial ischemia (In some individuals, the ischemia is not always ~~alo~~ accompanied by pain, resulting in silent or ambulatory ischemia. In varient angina oxygen delivery decreases as a result of reversible coronary vasospasm. Variant angina is also called vasospastic or Prinzmetal's angina.

(2) Unstable angina :→ Unstable angina (this is a form of acute coronary syndrome) is defined as angina pectoris that changes or worsens. It has at least one of these three features.

(a) It occurs at rest (or within minimal exertion), usually lasting more than 10 minutes.

(b) It is severe and of new onset (i.e with in the prior 4-6 weeks)

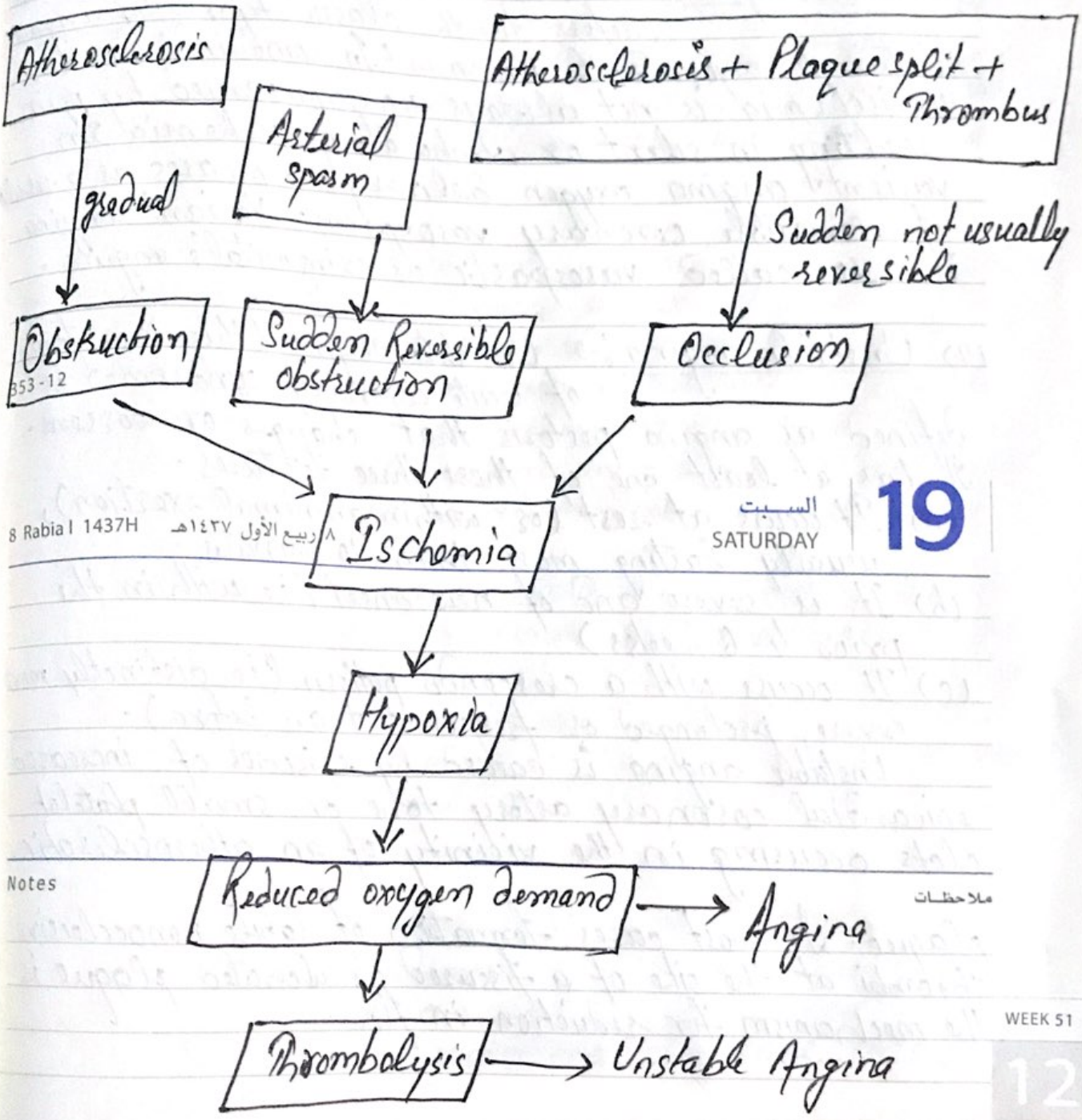
(c) It occurs with a crescendo pattern (i.e. distinctly more severe, prolonged or frequent than before).

Unstable angina is caused by episodes of increased epicardial coronary artery tone or small platelet clots occuring in the vicinity of an atherosclerotic plaque. In most cases, formation of labile nonocclusive thrombi at the site of a fissured or ulcerated plaque is the mechanism for reduction in flow.

Notes ملاحظات

7 Rabia I 1437H ٧ ربيع الأول ١٤٣٧هـ

الجمعة FRIDAY **18**



8 Rabia I 1437H ٨ ربيع الأول ١٤٣٧هـ

السبت SATURDAY **19**

Notes ملاحظات

17 الخميس
THURSDAY
6 Rabia I 1437H

Major Risk factors :→

- Age (≥ 45 years for men, ≥ 55 years for women)
- Smoking
- Diabetes Mellitus
- Dyslipidemia
- Hypertension
- Kidney disease
- Obesity
- Physical inactivity
- Prolonged psychosocial stress

Treatment :→ The most specific medicine to treat angina is antianginal where nitroglycerin is a potent vasodilators that decreases myocardial oxygen demand by decreasing the heart's workload.

= Beta blockers
Calcium channel blockers] Reduce heart's workload.

The main goals of treatment in angina pectoris are relief of symptoms, slowing progression of the disease and reduction of future events, especially heart attacks and death.

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ملاحظات

β blocker (carvedilol, metoprolol, propranolol) have a large body of evidence in morbidity and mortality benefit and short acting nitroglycerine medications have been used for symptomatic relief of angina.

Myocardial Infarction

Myocardial infaction is the irreversible damage of myocardial tissue tissue caused by prolonged ischemia and hypoxia. This most commonly occurs when a coronary becomes occluded following the rupture of an atherosclerotic plaque, which then leads to the formation of blood clot.

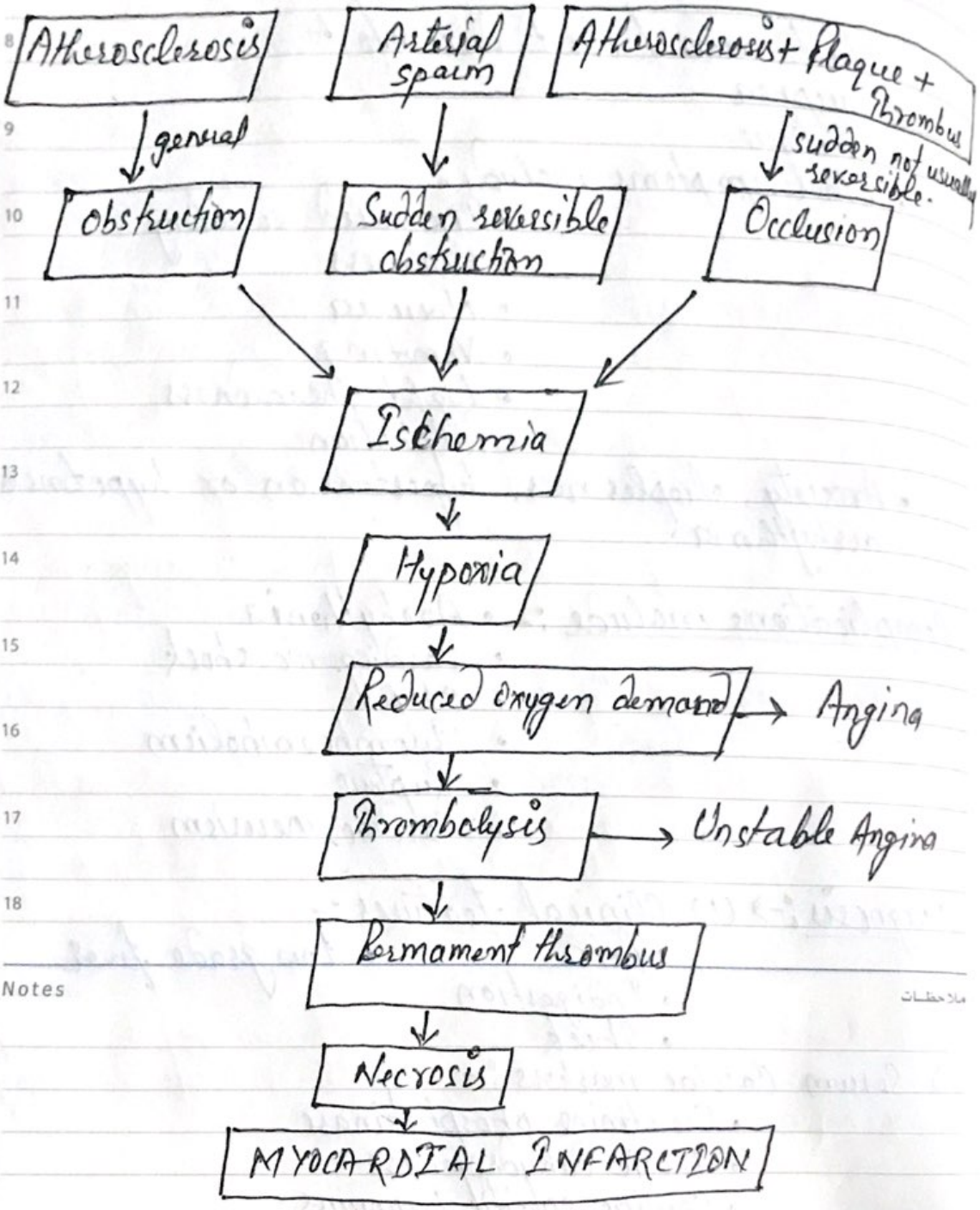
If a vessel becomes completely occluded, the myocardium normally supplied by that vessel will become ischemic and hypoxic.

Collateral blood flow is an important determinant of infarct size and whether or not the border zone becomes irreversibly damaged. Infarct tissue does not contribute to tension generation during systole, and therefore can alter ventricular systolic and diastolic function and disrupt electrical activity.

Ischemia can develop within 10 seconds and it is lasts longer ~~that~~ than 20 minutes, irreversible cell and tissue death occurs.

Notes ملاحظات

15 الثلاثاء TUESDAY 4 Rabia I 1437H



Notes

Sign and symptoms :→

- chest pain / chest discomfort
- Dyspnea
- fatigue
- Other symptoms include:
  - Increased sweating
  - Weakness
  - Nausea
  - Vomiting
  - Light-headedness
  - Palpitation
- Anxiety, sleeplessness, hypertension or hypotension, arrhythmia

Complications include :-
- Arrhythmia
- Cardiogenic shock
- CHF
- Thromboembolism
- Rupture
- Cardiac aneurism

Diagnosis :→ (1) Clinical features :-
- Pain
- Indigestion
- Shock
- Low grade fever

Notes ملاحظات

(2) Serum Cardiac markers :-
- Creatinine phosphokinase
- Lactic dehydrogenase
- Cardiac specific troponins.

(3) ECG changes:-
- ST segment elevation
- T wave inversion
- Appearance of wide deep Q waves.

Management :→

(1) Non-Pharmacological
- Counselling and education of patients
- Life style measures
- Smoking cessation
- Avoid alcohol intake
- Diet and nutrition
- Salt restriction

(2) Pharmacological
- Thrombolytic agents
- Anticoagulants
- Antiplatelet agents
- Antihypertensive agents
- Lipid lowering Drugs
- Vasodilators.

Notes ملاحظات

Notes

Causes :→ Atherosclerosis starts with damage or injury to the inner layer of an artery. The damage may be caused by:

29 Safar 1437H ٢٩ صفر ١٤٣٧هـ
الجمعة FRIDAY **11**

ATHEROSCLEROSIS

- Atherosclerosis also known as Arteriosclerotic Vascular Disease or ASVD.
- The condition in which an artery wall thickness as the result of a build-up of fatty materials such as cholesterol.
- This affecting arterial blood vessels, a chronic inflammatory response in the walls of arteries.
- This condition (atherosclerosis) is happen due to the accumulation of macrophage white blood cells and promoted by low-density lipoproteins without adequate removal of fats and cholesterol from the macrophages by functional high density lipoproteins.
- It is commonly reffered to as hardening of the arteries.

1 Rabia I 1437H ١ ربيع الأول ١٤٣٧هـ
السبت SATURDAY **12**

- It is caused by the formation of multiple plaques within the arteries.
- It can restrict blood flow. These plaques can also burst, causing a blood clot.
- Atherosclerosis is preventable and treatable condition.

Notes ملاحظات

Causes → Atherosclerosis starts with damage or injury to the inner layer of an artery. The damage may be caused by:

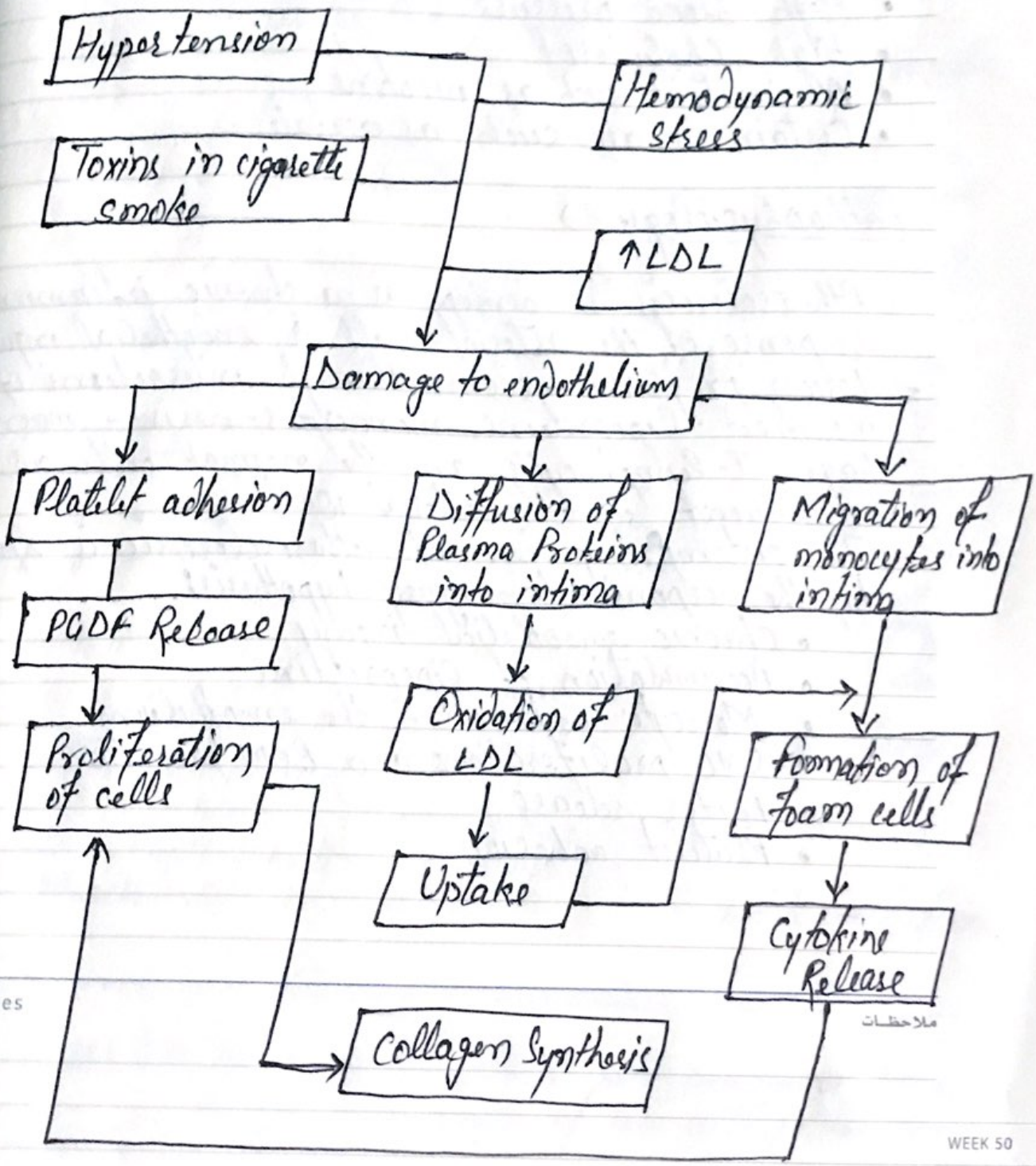
- High blood pressure
- High Cholesterol
- An irritant such as nicotine
- Certain disease such as diabetes.

Pathophysiology :→

= Atherosclerosis is develops as a chronic inflammatory response of the arterial wall to endothelial injury.
= Lesion progression occurs through interactions of modified lipoproteins, monocytes-derived macrophages, T-lymphocytes, and the normal cellular constituents of the arterial wall.
= The contemporary view of atherosclerosis is expressed by the response-to-injury hypothesis:

- Chronic endothelial injury
- Accumulation of lipoproteins
- Monocyte adhesion to the endothelium
- SMC proliferations and ECM production
- factor release
- Platelet adhesion

Notes ملاحظات



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Symptoms :- Atherosclerosis symptoms depend on which artery are affected. for example -

- Atherosclerosis in heart artery, have symptoms similar to those of a heart attack, such as chest pain.
- Atherosclerosis in the arteries leading to brain, have symptoms such as sudden numbness or weakness in your arms or legs, difficulty speaking or slurred speech, or drooping muscles in the face.
- Atherosclerosis in the arteries of the arm and legs, produces decreased blood flow is called peripheral artery occlusive disease, have symptoms such as leg pain when walking.

Physiologic factors that increase risk :-

(1) Modifiable :-
- Diabetes or Impaired glucose tolerance (IGT)
- Dyslipoproteinemia
- Tobacco, smoking
- High blood pressure
- Obesity
- Physical inactivity
- High carbohydrate intake

(2) Non-Modifiable :->
- Age
- Heredity
- Genetic abnormalities.

Notes ملاحظات

Tests and diagnosis :→ Depending on the results of the physical exam, doctors may suggest one or more diagnostic tests, including.

- Blood tests
- Doppler ultrasound
- Ankle-brachial index
- Angiogram
- Electrocardiogram (ECG).

Treatments :→

= Life style changes, such as eating a healthy diet and exercise are often the first line of defense in treating atherosclerosis.

= Various drug can slow- or sometimes even reverse- the effects of atherosclerosis.

- Cholesterol medications.
- Anti-platelet medications
- Anti-coagulants
- Anti-hypertensive drugs

= Thrombolytic therapy

= Bypass Surgery

Notes ملاحظات