MINISTRY OF HEALTH OF UKRAINE
ODESSA NATIONAL MEDICAL UNIVERSITY

Faculty : International

Department of Surgery №3

CONFIRMED by

Acting vice-rector for scientific and pedagogical work
_____ Svitlana KOTIUZHYNSKA

September 1, 2022

RECOMMENDATIONS
to students independed study

Faculty : International , Year **5**

Discipline **"Surgery with pediatric surgery"**

Recomendations are approved at the meeting of the Department of Surgery No. 3
Minutes No. 1 dated August 28, 2022.

The head of the department, professor

Volodimir BONDAR

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Theme: "ACQUIRED HEART DEFECTS. ".

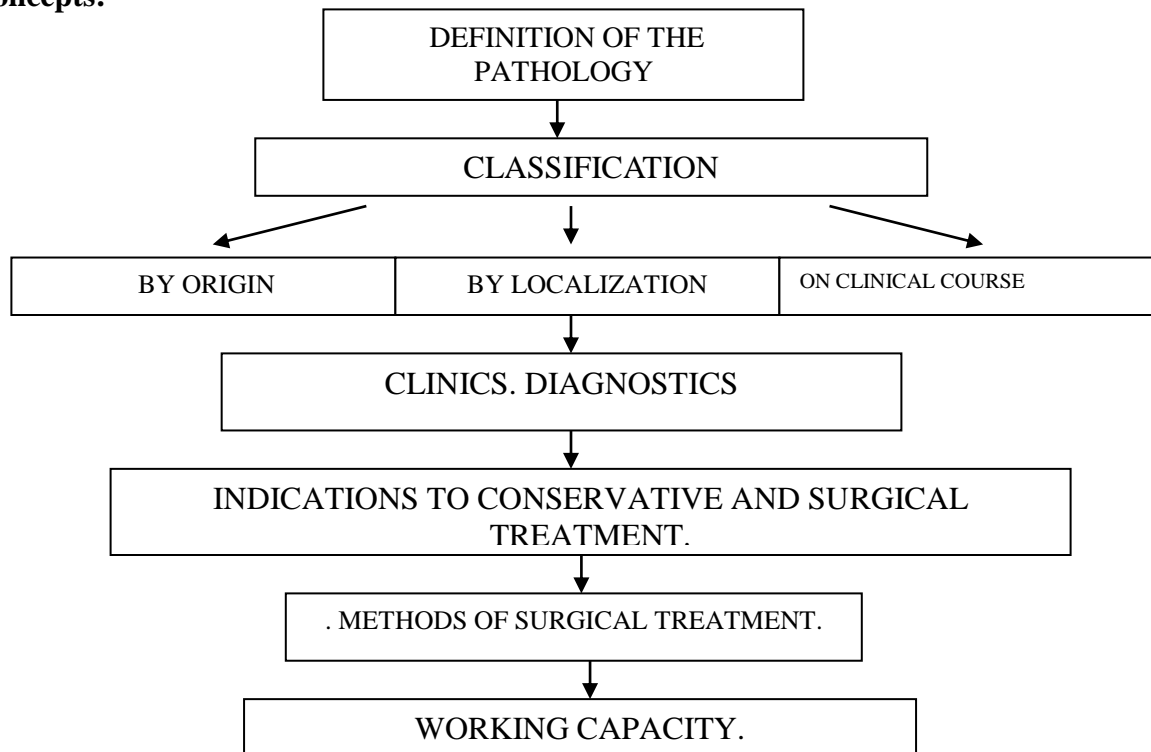**Goal :** to acquaint with aetiology and pathogenesis of acquired heart diseases

- to know variants of acquired heart diseases and developments of haemodynamic infringements.
- to know the basic attributes of these diseases.
- to know additional methods of diagnostics.
- to know opportunities of operative defects correction , indications to them and terms of performance of
operation.

Basic concepts:

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Equipment: Notebook, multimedia

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Plan.

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1. **Theoretical questions:**

Surgery for valvular heart disease
The number of operations for valvular heart disease has not risen as dramatically as those for coronary artery disease. The function of the heart valves is to maintain the forward flow of blood through the chambers of the heart. Disease may affect a valve by making the orifice smaller (stenosis) or by allowing back-flow or leakage through the valve (regurgitation or incompetence). Both stenosis and incompetence may coexist.
A stenotic valve produces a pressure load on the cardiac chamber immediately proximal to it. This chamber responds by becoming hypertrophied. Back-pressure on other chambers and vessels may follow. An incompetent valve produces a volume load, which has effects both upstream and downstream from the valve. The proximal heart chamber will enlarge predominantly by dilatation but also to some extent by hypertrophy.
Aetiology of heart valve disease.
Rheumatic fever
In the develop rheumatic fever has largely disappeared. However, in developing countries its incidence is still high. The pathology of rheumatic fever is an immunemediated acute inflammatory reaction that affects predominantly the heart valves (although the epicardium and myocardium may also be involved). It is due to cross-reaction between surface antigens of group A β-haemolytic streptococci and certain cardiac proteins.
In the acute phase a valvulitis occurs that is followed by further haemodynamic trauma, resulting in progressive valve failure with fibrosis and calcification. The initial valvulitis may be diagnosed by detecting the presence of a murmur. Stenosis results when there is fusion of the valve leaflets and regurgitation occurs when there is retraction and shortening of the scar tissue. Congenital valve abnormalities Several valve abnormalities may occur as part of the spectrum of congenital heart disease, e.g. Fallot's tetralogy but detailed discussion of the various congenital abnormalities is beyond the scope of this book.
The aortic valve is normally tricuspid but in 1–2% of individuals it has only two cusps. A functional disturbance presents in middle adult life related to long-term turbulent flow associated with the bicuspid valve. The disturbance is related to progressive calcification of the valve, which leads to stenosis and incompetence.
Degenerative valve disease Degenerative valve disease is caused by progressive 'wear and tear' of the valvular apparatus. It is being increasingly recognized as a feature of the elderly population. A syndrome described as the mitral floppy valve syndrome is a particular form of degenerative valve disease. Not only is there cystic change within the mitral valve leaflets but the chordal apparatus becomes increasingly elongated. It
leads to mitral regurgitation. Pathophysiology of heart valve abnormalities

Aortic valve
Aortic stenosis
A narrowed aortic orifice results in increased pressure in the left ventricle. Because the valve is stenosed, the pressure in the ventricle will be higher than the pressure in the root of the aorta during systole, i.e. a gradient exists across the valve. Because the ventricle has to work harder to overcome the obstruction, it hypertrophies. As the size of the ventricle increases, there is a corresponding increase in myocardial oxygen requirements. When supply exceeds demand the patient experiences angina. Syncopal attacks may also occur, particularly on exertion. Syncope occurs because the left ventricle is unable to increase its output in response to exercise-induced systemic vasodilatation. Left ventricular hypertrophy is a compensatory mechanism.
However, there is a limit to the amount of compensation that can be achieved and as the stenosis becomes tighter the compensatory mechanisms begin to fail, resulting in progressive ventricular dilatation and heart failure.
Operation is performed to improve prognosis and to relieve symptoms, particularly if the aortic valve gradient is in excess of 60 mmHg (8 kPa).

Aortic regurgitation
When the aortic valve is incompetent blood floods back into the ventricle during diastole. Thus the ventricle has to deal with an increased volume load, which it does by dilatation. Aortic regurgitation is usually better tolerated than aortic stenosis but will progressively lead to left ventricular failure.

Mitral valve

Mitral stenosis

A pressure load is placed on the left atrium. This is transmitted to the pulmonary circulation, which responds by progressive pulmonary hypertension. The left atrium is not usually dilated. As pulmonary arterial pressure increases there is an increasing load on the right ventricle.

In due course the tricuspid valve, right atrium and liver suffer the effects of back-pressure (congestive cardiac failure). In the early stages of mitral stenosis acute pulmonary oedema may occur, often as paroxysmal nocturnal dyspnoea. This will occur when left atrial pressure exceeds oncotic pressure in the pulmonary circulation. In some cases this may be precipitated by the development of an atrial arrhythmia, characteristically atrial fibrillation. In long-established cases, pulmonary oedema occurs less frequently. Surgery is performed for symptomatic deterioration deterioration, particularly New York Heart Association (NHYA) class III or IV. A valve area of less than 1 cm is a further indication for operation. The left atrium may contain thrombus, which makes systemic embolization particularly likely, especially if atrial fibrillation has developed.

This constitutes a further reason for operation.

Mitral regurgitation

A volume load is placed on the left atrium and is also associated with dilatation of the left ventricle. When this occurs slowly, large increases in cardiac chamber size may occur before any increase in pulmonary artery pressure develops. In the situation where regurgitation develops suddenly due to rupture of a papillary muscle or chorda (e.g. after myocardial infarction), the left atrial volume is small, with the result that pulmonary vascular pressure rises abruptly, causing pulmonary oedema and severe hypoxia. In the acute situation surgery may be life-saving. The case is more complex for the patient with chronic mitral regurgitation. Valve replacement in this situation will not affect the damaged and dilated ventricle, which will still continue to require a high filling pressure. Overall, the timing of surgery should be judged on the basis of left ventricular function as well as symptoms. Tricuspid valve The majority of patients with tricuspid valve disease have

tricuspid regurgitation secondary to right ventricular failure (e.g. caused by mitral valve disease) with long-term dilatation of the tricuspid valve annulus. The clinical signs are those of congestive cardiac failure: prominent jugular venous pulsations, liver congestion, ascites and peripheral oedema. In a small number of cases the pathological process is rheumatic fever or carcinoid syndrome. Operations for these conditions are uncommon in the UK and are frequently performed only in association with other valve replacements.

Investigations of patients with heart valve disease

As has been stressed earlier in this chapter, the history and clinical examination of the patient are of great importance. While the symptoms and physical signs of mitral valve disease frequently mirror the severity of the valvular defect, the symptoms of aortic valve disease may present at a later state when the point of optimal surgical intervention has been passed.
1. ECG: defines the cardiac rhythm and gives an indication of heart hypertrophy.
2. Chest radiography: demonstrates cardiac size and heart chamber size (in some cases) and provides evidence of pulmonary oedema.
3. Echocardiography (transthoracic and transoesophageal): used increasingly to provide evidence of chamber enlargement, imaging of the heart valve and calcification.
4. Doppler techniques: provide a measure of valve gradients and of regurgitation.
These assessments negate in many circumstances the need for cardiac catheterization. In certain situations, cardiac catheterization may be required. It allows imaging of the coronary arteries, which is important in elderly patients, particularly with the symptoms of angina. It also allows direct measurement of transvalvular gradients and visualization and quantification of the degree of valvular regurgitation.

Surgery for heart valve disease

The vast majority of operations are performed with cardiopulmonary bypass and cardioplegia. The heart chambers are opened, allowing the surgeon to see the valves. Minimally invasive techniques are gaining in popularity with technological advances but are not currently standard practice.

Valve replacement vs. valve conservation Artificial heart valves are non-physiological. Therefore, if possible, the patient's diseased valve should be remodelled. This is usually only possible with the mitral and sometimes the tricuspid valve. The criteria for valve repair are as follows:

• valve damage is minimal;
• valve leaflets are mobile; and
• there is no calcification.

Artificial heart valves

There are three main groups of artificial heart valves.

Prosthetic valves

Prosthetic valves are the most popular choice for the majority of patients. These valves are either of the balland-cage variety or use a tilting disc occluder or bileaflet mechanism. The initial use of metal alloys and plastics is now being superseded by pyrolytic carbon. A sewing ring made of Dacron allows suture of the valve to the native valve annulus. Patients with prosthetic valves implanted are at risk from thromboembolism, endocarditis (which may also produce septic emboli) and valve 'wear-out'. Because of the risk of thromboembolism, lifelong anticoagulation is required in patients with prosthetic heart valves. Warfarin is commenced within 48 h of operation. The currently recommended range for the international normalized ratio (INR) has been arbitrarily set at 3–4.5. Any interventional procedures on patients with prosthetic heart valves, including dentistry, should be performed with antibiotic cover to prevent valve endocarditis occurring . bioprosthetic valves Animal tissue is mounted on a synthetic frame or stent. A common tissue used is porcine aortic valve, which is treated with glutaraldehyde to reduce antigenicity. For patients with bioprosthetic valves, anticoagulation is carried on for 3 months until the valve has become epithelialized.

Biological valves

These contain only biological tissue. Cadaveric tissue is used to create a homograft valve. In the hands of enthusiastic proponents they give excellent haemodynamic performance and are relatively free from thromboembolic risk. Anticoagulation is not required for biological valves.

2. Questions for self-control:
1. Aetiology of heart valve disease
2. Classification.
3. Aortic stenosis. Staging. Pathogenesis.
4. Aortic regurgitation. Staging. Pathogenesis.
5. Mitral stenosis. Staging. Pathogenesis.
6. Mitral regurgitation. Staging. Pathogenesis.
7. Investigations of patients with heart valve disease.
8. Surgery for heart valve disease.
9. Artificial heart valves.
10. Prosthetic valves.
11. Biological valves.

3. Case scenarios:

1. A 40-year-old man complains of dyspnoea, dry cough, and general weakness. On objective inspection acrocyanosis is being defined; on palpation a "cat's purr" symptom is being detected in the projection of the heart apex. Auscultation reveals a strengthening of 1st sound; the sound of mitral valve opening is audible on the apex. Establish initial diagnosis.
    A. Aorta valve insufficiency
    B. Mitral insufficiency
    C. Aorta valve stenosis
    D. Mitral stenosis
    E. Endocarditis

2. In a patient during operation due to mitral stenosis an expressed fibrous changes in shutters and calcification of mitral valve has not been revealed. What type of operation is indicated for the patient?
    A. Closed mitral commissurotomy
    B. Prosthetic repair of the valve
    C. Aortocoronary shunt
    D. Endocardiac electrocardiostimulation
    E. Partial removal of the valve

3. A 52-year-old woman complains of dyspnoea and palpitation during physical exertion. Auscultation reveals the 1st sound weakened; the 3rd sound and systolic bruit is audible on the apex of the heart. The X-ray research reveals a rounding of arch on the left contour of the heart on the anterior-posterior projection. Establish initial diagnosis.
    A. Aortal insufficiency
    B. Mitral stenosis
    C. Mitral insufficiency
    D. Aortal stenosis
    E. Endocarditis

4. A 52- year-old man complains of dyspnoea, palpitation, irregularities in the heart, dizziness and periodic faints. Systolic arterial pressure is increased; a rough systolic bruit spreading to carotids is audible in a projection of the aortal valve. Establish initial diagnosis.
    A. Mitral insufficiency
    B. Aorta valve stenosis
    C. Mitral stenosis
    D. Aorta valve insufficiency
    E. Ischemic heart illness

5. During operation for aortal stenosis the shutter of the aortal valve is changed insignificantly .What type of operation is indicated for the patient?
    A. Partial removal of the valve
    B. Prosthetic repair of the valve
    C. Aortocoronary shunt
    D. Endocardial electrostimulation
    E. Division of adherent shutters along commissures

6. A 68-year-old man complains of a retrosternal pain which is temporarily arrested after validolum intake. Auscultation reveals weakened heart sounds; any bruits are not audible. Establish initial diagnosis.
    A. Mitral stenosis
    B. Aorta valve stenosis
    C. Ischemic heart illness
    D. Mitral insufficiency
    E. Endocarditis

7. During operation for ischemic heart illness a segmental narrowing of the coronary arteries was being revealed .What type of operation was indicated for the patient?
    A. Division of adherent shutters along commissures
    B. Endocardial electrostimulation
    C. Partial removal of the valve
    D. Aortocoronary shunt
    E. Endocardial electrostimulation

8. In a 45-year-old man a general weakness, hypotension, cyanosis of the upper trunk, vein dilatation on the neck developed after blunt trauma of the thorax with fracture of the breastbone. Pleural puncture failed reveal any contents. P: 120/min, is rhythmical and of poor volume. What diagnosis the most likely?
A.Heart tamponade

B.Thromboembolism of the lung artery
C.Concussion of the heart
D.Myocardial infarction
E.Clotted haemopericardium

9. A 54-year-old man was admitted to the hospital with expressed acrosyanosis, dilated cervical veins, enlarged liver and ascites. Boundary lines of the heart are expanded. Heart sounds are not audible; the apical jerk is not being defined. BP: 100/50mmHg. The X-ray of the thorax reveals the shadow of the heart like a trapeze. Which pathology can explain the specified symptoms in the patient?
A. Heart tamponade
B. Exudate pleurisy.
C. The combined heart defect.
D. Acute cardiac insufficiency.
E. Hiatus hernia.

4. Individual tasks for applicants for higher education on the topics:
1. Modern approaches to the treatment of acquired heart defects.
2. Prevention of acquired heart disease.

5. List of recommended literature:

Basic

1. SABISTON: TEXTBOOK OF SURGERY: THE BIOLOGICAL BASIS OF MODERN SURGICAL PRACTICE, TWENTY FIRST EDITION Copyright © 2020
2. Gozie Offiah, Arnold Hill//RCSI Handbook of Clinical Surgery for Finals. 4th ed. 2020
3. Цигикало О. В. Clinical Anatomy and Operative Surgery=Клінічна анатомія і оперативна хірургія.. Підручник для ВМНЗ IV р.а.: Рекомендовано МОЗ: 2020/ 528 с.
4. Березницький Я. С. (за ред.) General Surgery=Загальна хірургія. — 2-ге вид. Підручник для ВМНЗ III—IV р.а.: Рекомендовано ДУ"Центр.метод.каб.з вищої мед.освіти МОЗ України": 2020/ 328 с.
5. Christian de Virgilio, Areg Grigorian//Surgery: A Case Based Clinical Review. 2nd Ed. 2020
6. JANE C. ROTHROCK// Alexander's Care of the Patient in Surgery. 16th Ed. 2019
7. RUTHERFORD'S VASCULAR SURGERY AND ENDOVASCULAR THERAPY, 9th ed. Volume 1, Volume 2. Copyright © 2019 by Elsevier, Inc.
8. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 11th Ed. Copyright © 2019 by Elsevier Inc.
9. SEIDEL'S GUIDE TO PHYSICAL EXAMINATION: AN INTERPROFESSIONAL APPROACH. Copyright © 2019 by Elsevier, Inc.

Additional.

10. Hamilton Bailey's Physical Signs: Demonstrations of Physical Signs in Clinical Surgery, 19th Edition Paperback – Import, by John S.P Lumley (Author), Anil K. D'Cruz (Author), Jamal J. Hoballah (Author), Carol E.H. Scott-Connor (Author) 25 Feb 2016
11. Schwartz's Principles Of Surgery With DVD Hardcover – 2014 by F. Charles Brunicardi (Author), Dana K. Andersen (Author), Timothy R. Billiar (Author), David L. Dunn (Author), John G. Hunter (Author), & 2 More

6. Electronic informative resources

1. http://moz.gov.ua – Міністерство охорони здоров'я України
2. www.ama-assn.org – Американська медична асоціація / American Medical Association
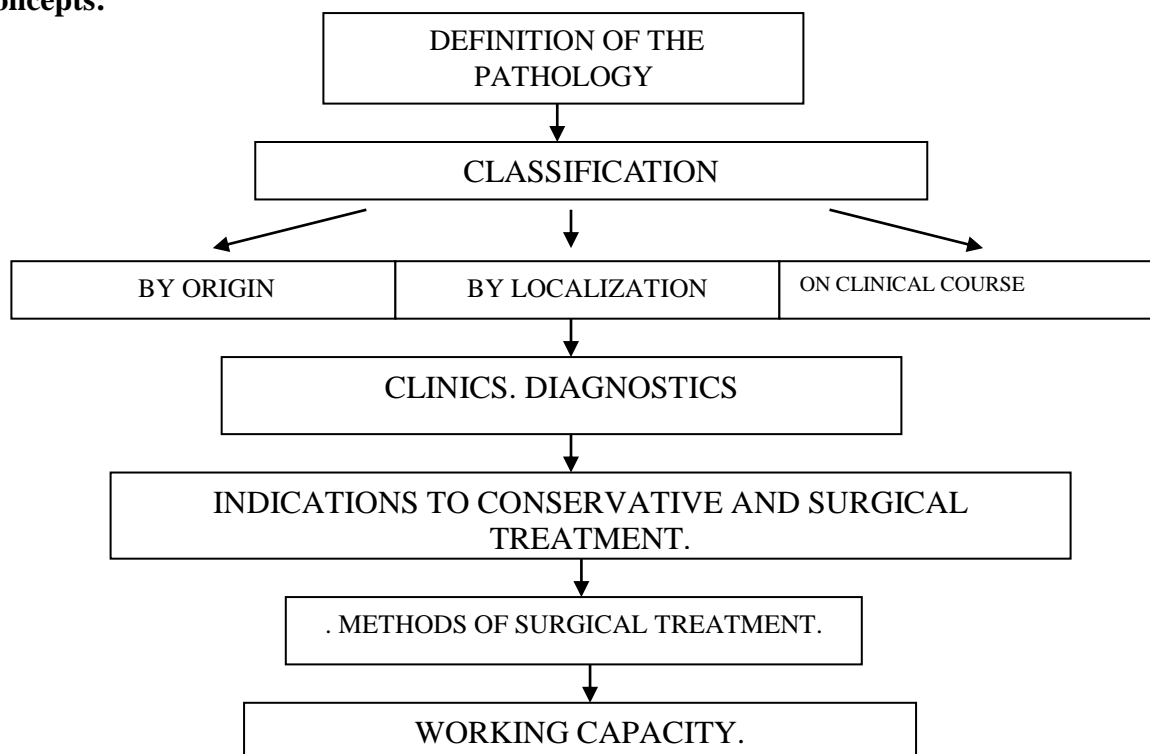3. www.who.int – Всесвітня організація охорони здоров'я

4. www.dec.gov.ua/mtd/home/ - Державний експертний центр МОЗ України
5. http://bma.org.uk – Британська медична асоціація
6. www.gmc-uk.org - General Medical Council *(GMC)*
7. www.bundesaerztekammer.de – Німецька медична асоціація
8. http://medforum.in.ua/partners- Асоціація хірургів України
9. http://endoscopy.com.ua/ - Асоціація ендоскопічних хірургів України
10. http://thoracic-surgery.com.ua/ - Асоціація торакальних хірургів України
11. https://youcontrol.com.ua/ - Асоціація судинних хірургів України

Theme: " ISCHEMIC HEART DISEASE".

Goal : acquaint with aetiology and pathogenesis of ischemic heart disease.
- know variants of ischemic heart disease and developments of haemodynamic infringements.
- know the basic attributes of these diseases.
- know additional methods of diagnostics.
- know opportunities of operative defects correction , indications to them and terms of performance of operation.

Basic concepts:

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Equipment: Notebook, multimedia

Plan.

2. **Theoretical questions:**

Coronary artery disease

Ischaemic heart disease is the most common cause of death in the western world, claiming more than 3000 deaths per million each year in the UK. It results from atheromatous narrowing of the coronary arteries and may present as sudden death or as acute myocardial infarction, but more commonly presents as angina pectoris. This is typically provoked by exertion and relieved by rest. Physical examination is frequently normal, although the stigmata of hypercholesterolaemia or diabetes may be present. The auscultation of a heart murmur or carotid bruit will require further investigation.

Investigations
• Resting and exercise ECGs will demonstrate evidence of exercise-induced ischaemia, previous infarction or arrhythmias. This test provides objective evidence so that medical therapy may be instituted, monitored and the need for further intervention assessed.
• Coronary angiography is performed when it is felt that patients need revascularization, whether this is by angioplasty or coronary bypass surgery. Indications for surgery
Medical therapy is recommended when the ischaemia is prevented by anti-ischaemic drugs that are well tolerated. With one- or two-vessel disease not involving the left anterior descending artery, medical therapy (or angioplasty) is recommended first, with coronary artery bypass grafting (CABG) being reserved for refractory ischaemia. With limited coronary artery disease refractory to medical therapy, angioplasty should be considered before recommending surgery, unless there is a compelling reason such as left main artery disease. If angioplasty is high risk or the lesions are technically unsuitable, CABG is the correct way to proceed.
Coronary artery surgery may benefit the patient by providing symptomatic relief and in many cases is of prognostic benefit. Data accumulated from three large, prospective, randomized trials have shown that surgery is better than medical therapy for improving survival in left main stem or triple-vessel disease (i.e. stenosis in each of the three main arteries: right, left anterior descending and circumflex), double-vessel disease involving the left anterior descending artery, and chronic ischaemia
leading to left ventricular dysfunction.

Surgical procedure
1 Following the institution of cardiopulmonary bypass and application of the aortic cross-clamp, the heart is arrested in diastole and protected from ischaemic damage by the administration of cardioplegia. Usually 1 L is infused in the first instance, followed by further infusions at further time intervals thereafter. Topical cooling in the form of ice is applied to the myocardial surface, and the patient is also systemically cooled by the heat exchanger on the bypass machine.
2 The graft of choice is the internal mammary (internal thoracic) artery. It is anastomosed (joined with sutures) most commonly to the left anterior descending artery. In this situation it confers greater protection from subsequent cardiac events (angina, myocardial infarction and sudden death). Its patency rates are superior to long saphenous vein (95% and 85% patency at 5 and 10 years, respectively, can be expected).
3 Reversed long saphenous vein is also used extensively as a conduit. However, vein grafts are prone to occlusion at a rate of 10–20% in the first year, with an occlusion rate of 2–3% per year thereafter. Treatment with low-dose aspirin following the operation enhances patency.
4 An arteriotomy is made distal to the coronary artery stenosis. The reversed saphenous vein is then anastomosed using a fine polypropylene suture. All distal anastomoses are performed in this fashion; if the internal mammary artery is being used, it is anastomosed last. When all distal anastomoses have been performed, the aortic cross-clamp is removed, allowing the heart to reperfuse and beat. As the patient rewarms, the proximal anastomoses to the aorta are performed.
5 Following completion of all anastomoses and warming of the patient, ventilation is recommenced and, providing the patient is in stable rhythm, cardiopulmonary bypass is discontinued. Following removal of venous and arterial cannulae, protamine is given to reverse the anticoagulant effects of heparin.

Outcome
The hospital mortality following CABG is 1–4% and this is likely to be a reflection of operating on older patients and those with impaired left ventricular function. Although the majority of patients obtain relief from angina, recurrent angina is most likely to occur within the first year following surgery. This is usually due to graft failure as a result of poor anastomotic technique, or an inadequate distal vessel. Only 50% of vein grafts are patent at 10 years. The internal mammary artery has a superior patency rate. Approximately 10% of patients will have a second operation within the ensuing 10 years. Reoperation is associated with an increased operative mortality and is reserved for patients with severe symptoms refractory to maximal medical therapy.

Complications of myocardial infarction
Most complications of acute myocardial infarction occur in the period immediately after the infarction. The patient is frequently in cardiogenic shock. The aetiology of the circulatory collapse must be ascertained before a management plan can be formulated. As a general rule the earlier the occurrence of the complication, the higher the overall mortality. The surgically correctable complications of myocardial
infarction are:
• VSD;
• mitral regurgitation;

- left ventricular aneurysm;
- ventricular arrhythmias;
- ruptured ventricle.

The patient is normally profoundly unwell and is admitted directly to the ICU. Full invasive monitoring, including the insertion of a Swan–Ganz catheter (which may demonstrate an interventricular shunt), will be necessary. Cardiogenic shock is treated by inotropic drugs, vasodilators and diuretics. Shock refractory to these therapies will frequently require a mechanical device to assist the failing heart. Such a device is known as an intra-aortic balloon pump, which is positioned in the descending aorta via the femoral artery and provides synchronized alterations in left ventricular afterload to improve cardiac output. If arrhythmias are the cause of heart failure they should be treated by the use of antiarrhythmics, pacing or direct current (DC) cardioversion.

An ECG will demonstrate the presence and extent of an infarct, and associated arrhythmias. Echocardiography will demonstrate mitral regurgitation, VSD, left ventricular aneurysm, left ventricular dysfunction and a ruptured left ventricle.

If the patient is stable, then angiography can proceed so that revascularization may be performed in association with any other corrective procedure. This is often not possible as the risks of catheterization maybe prohibitive. Transplantation should be considered for some patients with poor left ventricular function and heart failure refractory to medical or surgical therapy.

Ischaemic heart disease at a glance

Definition

Ischaemic heart disease: a common disorder caused by acute or chronic interruption of the blood supply to the myocardium, usually due to atherosclerosis of the coronary arteries, i.e. coronary artery disease

Epidemiology
- M > F before age 65
- Increasing risk with increasing age up to 80 years
- Most common cause of death in the western world

Aetiology
- Atherosclerosis and thrombosis
- Thromboemboli
- Arteritis (e.g. periarteritis nodosa)
- Coronary artery spasm
- Extension of aortic dissecting aneurysm
- Syphilitic aortitis

Risk factors
- Cigarette smoking
- Hypertension
- Hyperlipidaemia
- Type A personality
- Obesity

Pathology
- Reduction in coronary blood flow is critical when lumen is decreased by 90%
- Angina pectoris results when the supply of oxygen to the heart muscle is unable to meet the increased demands for oxygen, e.g. during exercise, cold, after a meal
- Thrombotic occlusion of the narrowed lumen precipitates acute ischaemia
- Heart muscle in the territory of the occluded vessel dies (myocardial infarction). May be subendocardial or transmural

Clinical features
Angina pectoris
- Central chest pain on exertion, especially in cold weather, lasts 1–15 min
- Radiates to neck, jaw, arms
- Relieved by glyceryl trinitrate (GTN)
- Usually no signs

Myocardial infarction
- Severe central chest pain for > 30 min duration
- Radiates to neck, jaw, arms
- Not relieved by GTN
- Signs of cardiogenic shock
- Arrhythmias

Investigations
Angina pectoris
- Full blood count for anaemia
- Thyroid function tests
- Chest X-ray: heart size
- ECG: ST-segment changes
- Exercise ECG
- Coronary angiography

Myocardial infarction
- ECG: Q waves, ST-segment and T-wave changes
- Cardiac enzymes: LDH/CPK, CPK-MB
- Chest X-ray
- Full blood count, urea and electrolytes

Management
Angina pectoris
- Lose weight
- Avoid precipitating factors (e.g. cold)
- GTN (sublingual)
- Calcium channel blockers
- β-Blockers, nitrates, aspirin
- Treat hypertension and hyperlipidaemias
- Coronary angioplasty or CABG

Indications for CABG
- Left main stem disease
- Triple-vessel disease
- Two-vessel disease involving left anterior descending
- Chronic ischaemia + left ventricular dysfunction

Myocardial infarction
- Bedrest, oxygen, analgesia
- Thrombolysis
- Aspirin
- Treat heart failure (diuretics)
- Treat arrhythmias

Complications of myocardial infarction
- Arrhythmias
- Cardiogenic shock
- Myocardial rupture
- Papillary muscle rupture causing mitral incompetence
- Ventricular aneurysm
- Pericarditis
- Mural thrombosis and peripheral embolism

3. **Case scenarios:**

1. A 40-year-old man complains of dyspnoea, dry cough, and general weakness. On objective inspection acrocyanosis is being defined; on palpation a "cat's purr" symptom is being detected in the projection of the heart apex. Auscultation reveals a strengthening of 1st sound; the sound of mitral valve opening is audible on the apex. Establish initial diagnosis.
 A. Aorta valve insufficiency
 B. Mitral insufficiency
 C. Aorta valve stenosis
 D. Mitral stenosis
 E. Endocarditis

2. In a patient during operation due to mitral stenosis an expressed fibrous changes in shutters and calcification of mitral valve has not been revealed. What type of operation is indicated for the patient?
 A. Closed mitral commissurotomy
 B. Prosthetic repair of the valve
 C. Aortocoronary shunt
 D. Endocardiac electrocardiostimulation
 E. Partial removal of the valve

3. A 52-year-old woman complains of dyspnoea and palpitation during physical exertion. Auscultation reveals the 1st sound weakened; the 3rd sound and systolic bruit is audible on the apex of the heart. The X-ray research reveals a rounding of arch on the left contour of the heart on the anterior-posterior projection. Establish initial diagnosis.
 A. Aortal insufficiency
 B. Mitral stenosis
 C. Mitral insufficiency
 D. Aortal stenosis
 E. Endocarditis

4. A 52- year-old man complains of dyspnoea, palpitation, irregularities in the heart, dizziness and periodic faints. Systolic arterial pressure is increased; a rough systolic bruit spreading to carotids is audible in a projection of the aortal valve. Establish initial diagnosis.
 A. Mitral insufficiency
 B. Aorta valve stenosis
 C. Mitral stenosis
 D. Aorta valve insufficiency
 E. Ischemic heart illness

5. During operation for aortal stenosis the shutter of the aortal valve is changed insignificantly .What type of operation is indicated for the patient?
 A. Partial removal of the valve
 B. Prosthetic repair of the valve
 C. Aortocoronary shunt
 D. Endocardial electrostimulation
 E. Division of adherent shutters along commissures

6. A 68-year-old man complains of a retrosternal pain which is temporarily arrested after validolum intake. Auscultation reveals weakened heart sounds; any bruits are not audible. Establish initial diagnosis.
 A. Mitral stenosis
 B. Aorta valve stenosis
 C. <u>Ischemic heart illness</u>
 D. Mitral insufficiency
 E. Endocarditis

7. During operation for ischemic heart illness a segmental narrowing of the coronary arteries was being revealed .What type of operation was indicated for the patient?
 A. Division of adherent shutters along commissures
 B. Endocardial electrostimulation
 C. Partial removal of the valve
 D. <u>Aortocoronary shunt</u>
 E. Endocardial electrostimulation

8. In a 45-year-old man a general weakness, hypotension, cyanosis of the upper trunk, vein dilatation on the neck developed after blunt trauma of the thorax with fracture of the breastbone. Pleural puncture failed reveal any contents. P: 120/min, is rhythmical and of poor volume. What diagnosis the most likely?
   A. <u>Heart tamponade</u>
   B. Thromboembolism of the lung artery
   C. Concussion of the heart
   D. Myocardial infarction
   E. Clotted haemopericardium

9. A 54-year-old man was admitted to the hospital with expressed acrosyanosis, dilated cervical veins, enlarged liver and ascites. Boundary lines of the heart are expanded. Heart sounds are not audible; the apical jerk is not being defined. BP: 100/50mmHg. The X-ray of the thorax reveals the shadow of the heart like a trapeze. Which pathology can explain the specified symptoms in the patient?
   A. <u>Heart tamponade</u>
   B. Exudate pleurisy.
   C. The combined heart defect.
   D. Acute cardiac insufficiency.
   E. Hiatus hernia.

10. Which of the following statements is true concerning aortocoronary bypass grafting?
   A. <u>It is indicated for crescendo (preinfarction) angina</u>
   B. It is indicated for congestive heart failure
   C. It is not indicated for chronic disabling angina
   D. It is associated with a 10% operative mortality
   E. It is only indicated if significant triple vessel disease is documented angiographically

 The answer is **a**. (Schwartz, 7/e, pp 865–867.) Coronary artery bypass surgery was developed in the late 1960s and is now being regularly performed. Indications for surgery include chronic disabling angina and crescendo (or preinfarction) angina. Cardiac catheterization with selective coronary angiography defines the extent of disease, which generally is localized to the proximal segments of the vessels. Operative mortality is about 2%, and relief of angina is obtained in most affected patients. Patients with left main coronary artery disease as well as those with triple vessel disease and ventricular dysfunction have an increased longevity fol- lowing successful bypass. Data regarding extension of life in other groups is conflicting. Coronary artery bypass is not indicated for congestive heart failure unless this condition is ischemic in origin and angiography identifies disease amenable to surgical revascularization.

4. Individual tasks for applicants for higher education on the topics:
1. Treatment of patients with coronary artery disease
2. Tactics in the treatment of patients with myocardial infarction

5. List of recommended literature:

Basic

10. SABISTON: TEXTBOOK OF SURGERY: THE BIOLOGICAL BASIS OF MODERN SURGICAL PRACTICE, TWENTY FIRST EDITION Copyright © 2020
11. Gozie Offiah, Arnold Hill//RCSI Handbook of Clinical Surgery for Finals. 4th ed. 2020
12. Цигикало О. В.   Clinical Anatomy and Operative Surgery=Клінічна анатомія і оперативна хірургія.. Підручник для ВМНЗ IV р.а.: Рекомендовано МОЗ: 2020/ 528 с.
13. Березницький Я. С. (за ред.)   General Surgery=Загальна хірургія. — 2-ге вид.   Підручник для ВМНЗ III—IV р.а.: Рекомендовано ДУ“Центр.метод.каб.з вищої мед.освіти МОЗ України”: 2020/ 328 с.
14. Christian de Virgilio, Areg Grigorian//Surgery: A Case Based Clinical Review. 2nd Ed. 2020
15. JANE C. ROTHROCK// Alexander's Care of the Patient in Surgery. 16th Ed. 2019
16. RUTHERFORD'S VASCULAR SURGERY AND ENDOVASCULAR THERAPY, 9th ed. Volume 1, Volume 2. Copyright © 2019 by Elsevier, Inc.
17. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 11th Ed. Copyright © 2019 by Elsevier Inc.
18. SEIDEL'S GUIDE TO PHYSICAL EXAMINATION: AN INTERPROFESSIONAL APPROACH. Copyright © 2019 by Elsevier, Inc.

Additional.

10. Hamilton Bailey's Physical Signs: Demonstrations of Physical Signs in Clinical Surgery, 19th Edition Paperback – Import, by John S.P Lumley (Author), Anil K. D'Cruz (Author), Jamal J. Hoballah (Author), Carol E.H. Scott-Connor (Author) 25 Feb 2016
11. Schwartz's Principles Of Surgery With DVD Hardcover – 2014 by F. Charles Brunicardi (Author), Dana K. Andersen (Author), Timothy R. Billiar (Author), David L. Dunn (Author), John G. Hunter (Author), & 2 More

6. Electronic informative resources
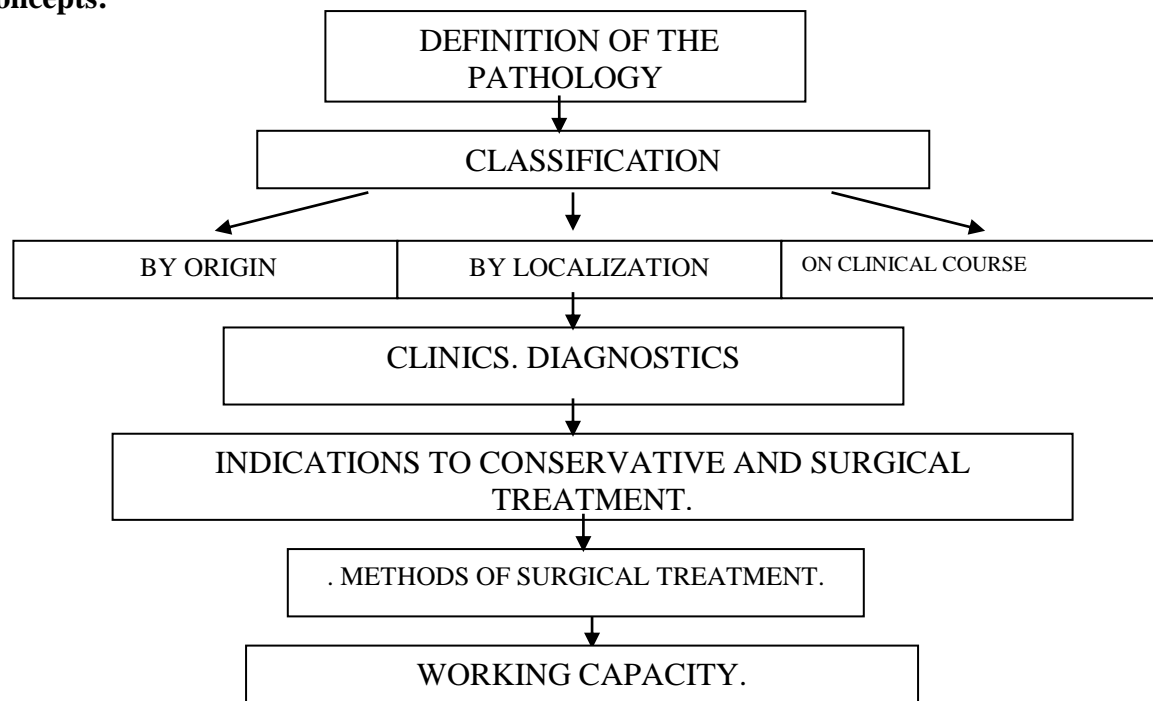
1. http://moz.gov.ua – Міністерство охорони здоров'я України
2. www.ama-assn.org – Американська медична асоціація / American Medical Association
3. www.who.int – Всесвітня організація охорони здоров'я
4. www.dec.gov.ua/mtd/home/ - Державний експертний центр МОЗ України
5. http://bma.org.uk – Британська медична асоціація
6. www.gmc-uk.org - General Medical Council (GMC)
7. www.bundesaerztekammer.de – Німецька медична асоціація
8. http://medforum.in.ua/partners- Асоціація хірургів України
9. http://endoscopy.com.ua/ - Асоціація ендоскопічних хірургів України
10. http://thoracic-surgery.com.ua/ - Асоціація торакальних хірургів України
11. https://youcontrol.com.ua/ - Асоціація судинних хірургів України

Theme: «Pericardium diseases ".

Goal : Applicants for higher education should know the anatomical structure and physiology of the cardiovascular system; survey methodology, indications and methods of surgical treatment of acquired heart disease and pericarditis. Be able analyze the basic pathophysiological processes and pathological changes analyze additional methods; conduct a differential diagnosis between different diseases.

Basic concepts:

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│      DEFINITION OF THE          │
│         PATHOLOGY               │
└─────────────────────────────────┘
                │
                ▼
┌─────────────────────────────────┐
│         CLASSIFICATION          │
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│ BY ORIGIN │ BY LOCALIZATION │ ON CLINICAL COURSE  │
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│      CLINICS. DIAGNOSTICS       │
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│ INDICATIONS TO CONSERVATIVE AND SURGICAL      │
│            TREATMENT.                          │
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│ . METHODS OF SURGICAL TREATMENT.     │
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│        WORKING CAPACITY.             │
└─────────────────────────────────────┘
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Equipment: Notebook, multimedia

Plan.

3. **Theoretical questions:**

NORMAL PHYSIOLOGY
Although there are no significant consequences of the congenital absence or surgical resection of the pericardium as long as the defect does not lead to cardiac herniation, the pericardium has some function in the normal patient. The pericardium anchors the heart and prevents torsion and acute distension.
The pericardium contributes to the diastolic coupling of the ventricles along the Starling curve.
Mechanoreceptors in the pericardium may also regulate blood pressure and heart rate. The pericardium stretches up to 20% with small changes in pressure but becomes abruptly stiff and resistant with larger volumes.
Compliance depends on the rate of fluid accumulation, and the hemodynamic response is also partially dependent on intravascular volume status.
The normal pericardial pressure is less than atmospheric pressure and is the same as the intrapleural pressure.
With inspiration, right-sided venous returns and preload increases. Blood pools in the lungs and decreases left-sided venous return and aortic blood flow. The arterial pressure normally decreases less than 10 mm Hg with inspiration.. The awave is the normal atrial contraction. The c wave reflects the bulging of the atrioventricular valve into the atrium during isovolumic ventricular systole. The v wave represents passive atrial filling from the vena cava. The x descent occurs with systolic collapse during ventricular systole and atrial relaxation.
The y descent reflects diastolic collapse with opening of the atrioventricular valve and passive ventricular filling.
Inspiration decreases intrathoracic pressure and leads to a lower x descent compared to the y descent.
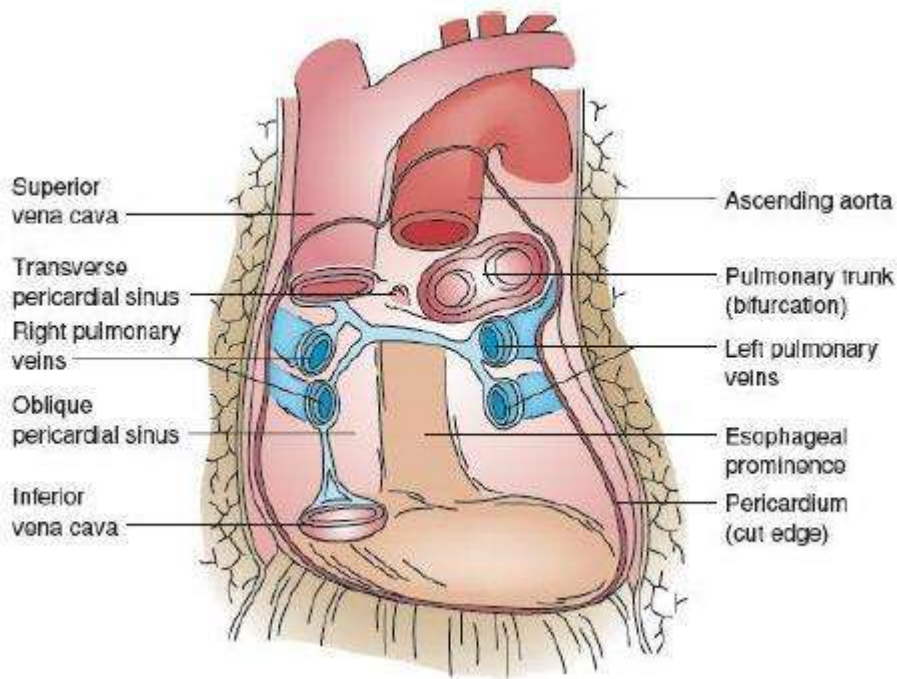
Figure 86-1. This drawing illustrates the pericardial attachments of the great vessels and pulmonary veins. The oblique sinus forms a blind cul-de-sac behind the left atrium, and the transverse sinus is a space between the aorta and pulmonary artery superiorly and the left and right atrium inferiorly.

DIAGNOSTIC STUDIES
While the electrocardiogram is nonspecific, it can be helpful in suggesting the diagnosis. In acute pericarditis the EKG classically shows diffuse ST elevations, and a low-voltage QRS is seen with a large pericardial effusion. The chest radiograph in a patient with a pericardial effusion shows an enlarged cardiac silhouette described as a water bottle. Pericardial calcification can be seen in chronic constrictive pericarditis secondary to tuberculosis.
 The echocardiogram is the most useful noninvasive test in evaluating pericardial disease. The echocardiogram can identify an effusion, pericardial thickening, or masses.
By using Doppler and assessing changes in chamber size, echocardiogram is useful in assessing hemodynamics and can help differentiate tamponade, constriction, and restriction. It can also be used to help guide procedures like pericardiocentesis. Computed tomography and magnetic resonance imaging can identify pericardial masses and pericardial thickening or calcification.9 Cardiac catheterization provides pressure tracings that help to distinguish cardiac tamponade, constriction, and restriction. Endomyocardial biopsy can also be useful in diagnosing restrictive cardiomyopathy.

ACUTE PERICARDITIS

Acute pericarditis is an inflammatory process that has involved the pericardium for less than 2 weeks. Patients often present with a 3- to 7-day prodrome of low-grade fevers, malaise, and muscle aches. Acute pericarditis occurs in 5% of those who present to the emergency department with nonischemic chest pain10 and in 1% of those with ST elevation.11 It is important to distinguish pericarditis from the chest pain of an acute myocardial infarction. Acute pericarditis usually causes sharp, pleuritic pain that can last several days. The pain most commonly radiates to the trapezius ridge and is improved by leaning forward. Patients can present with shortness of breath, a nonproductive cough, and clear lung fields. The differential diagnosis also includes aortic dissection and pneumothorax. On physical examination, a friction rub may be heard and is sometimes intermittent. The classic friction rub has three components during systole, early diastolic filling, and atrial contraction.

Table 86-1 Causes of Pericarditis

Idiopathic
Infectious
Neoplastic
Hemopericardium
Blunt and penetrating trauma
Postcardiac surgery
Aortic dissection
Autoimmune and inflammatory
Connective tissue disorders
Postmyocardial infarction
Medication induced
Miscellaneous
Radiation induced
Hypothyroidism
Uremic

T

he electrocardiogram is important in the diagnosis and classically shows diffuse ST elevations without Q waves or T-wave inversion. PR depression can also be seen. There is a four-stage progression in the changes seen on EKG with diffuse ST elevations followed by normalization of ST segments with flattening of T waves. The EKG then evolves with T-wave inversions prior to normalization of the EKG.

Pericarditis and associated myocarditis can cause elevations in creatinine kinase and troponin The minimal workup should include an EKG, complete blood count, cultures, chemistry profile, and antibody titres for collagen diseases.

Idiopathic and Viral Pericarditis

Idiopathic causes of pericarditis are the second most common after neoplastic disease. The majority are likely viral although routine testing is not usually performed. A virus is only identified in 15% to 20% of cases with the most common being Coxsackievirus, echovirus, adenovirus, influenza, and cytomegalovirus. Patients present with chest pain, malaise, and fever and often have an elevated erythrocyte sedimentation rate. The episode is self-limited in 70% to 90% of cases.10 Initial treatment is with nonsteroidal anti-inflammatory drugs (NSAIDs).

There is a 15% to 30% relapse rate at which point specific causes such as autoimmune disorders should be investigated. A repeat course of NSAIDs, colchicine, or steroids is generally successful. Pericardiectomy is recommended if the patient unresponsive to medical treatment or constriction develops.

Acquired Immunodeficiency Syndrome

A pericardial effusion develops in up to 20% of patients with HIV and is usually a poor prognostic sign. This may be partly due to a generalized capillary leak syndrome as well as increased cytokine expression seen in the more advanced stages of HIV. Other contributing factors include tubercular and mycobacterial infections, lymphoma, Kaposi's, or congestive heart failure. The majority are idiopathic and do not require further therapy if asymptomatic. Symptomatic effusions are drained.

Tuberculous Pericarditis

Tuberculous pericarditis occurs in 1% to 8% of patients. In immunocompromised patients infection by Mycobacterium avium or Mycobacterium intracellulare can lead to pericarditis. The incidence of tuberculous pericarditis has decreased although it continues to be a significant issue in immunocompromised patients, particularly HIV patients in Africa. Patients present with fever, night sweats, cough, dyspnea, and weight loss. While infection usually results from hematogenous spread, it can also extend directly from lymph nodes or through lymphatics. Pericardial changes occur in four stages including fibrinous, effusive, fibrous, and constrictive fibrous stages. Making the diagnosis from pericardial fluid alone is rare. Pericardial biopsy with acid fast staining provides the diagnosis 80% to 90% of the time. Treatment includes multidrug antitubercular therapy and pericardiocentesis. Steroids have not been shown to be beneficial for mortality or progression to constriction but leads to a faster resolution of symptoms and reaccumulation.16 If patients present with late constriction, pericardiectomy may be required.

Purulent Pericarditis

Bacteria can be introduced into the pericardial space by direct injury, pneumonia, extension from head and neck infections, or hematogenous and lymphatic spread. Patients present with chest pain, fever, and leukocytosis. The most common organisms in adults are staphylococcus, pneumococcus, and streptococcus. Purulent pericarditis can evolve rapidly into tamponade and can be confused with septic shock. Fungal infections are less common, but immunocompromised patients and drug addicts are at greater risk. Pericardial fluid in purulent pericarditis has low glucose, high protein, and elevated LDH and neutrophils. An air–fluid level may be seen on chest radiograph with gas-producing organisms.

Treatment includes antibiotics and drainage. Surgical drainage may be needed for thick fluid or refractory effusions. The prognosis for purulent pericarditis is poor with a survival of 30%.

Uremic Pericarditis

Fifty percent of patients with untreated renal disease develop pericarditis. The incidence is decreased to approximately 20% in patients on hemodialysis. The exact cause is unknown but is not directly related to the BUN or creatinine. Other possible etiologies include hypercalcemia, viral infection, and autoimmune disease. Chest pain is usually less common, and the large effusions accumulate gradually.

Treatment includes more intensive dialysis, NSAIDs, and steroids. The fluid is generally bloody and care should be taken in heparinizing these patients for dialysis. If patients develop tamponade or the effusion is unresponsive, pericardiocentesis or surgical drainage is performed.

Vasculitis, Connective Tissue Disease, and Drugs

Pericarditis can result from a variety of vasculitic and connective tissue diseases including rheumatoid arthritis, Wegener granulomatosis, rheumatic fever, lupus, scleroderma, Reiter syndrome, Behcet disease, dermatomyositis, polyarteritis nodosa, dermatomyositis, sarcoidosis, and amyloidosis.

Treatment includes management of the underlying disease process and NSAIDs. Pericardiocentesis is performed if necessary. Several medications can also lead to pericarditis including warfarin, hydralazine, isoniazid, procainamide, phenytoin, dantroline, cromolyn, and methysergide.

Dressler and Postpericardiotomy Syndrome

Pericarditis can occur following acute myocardial infarction in 3% to 5%. Pericarditis can develop early in the first 1 to 3 days particularly with transmural infarction. Forty percent of large Q-wave infarctions cause pericardial inflammation although the incidence is decreasing with the use of thrombolytics and early revascularization. Patients are usually asymptomatic and are found to have a pericardial rub on examination although they can present with pleuritic chest pain. Treatment is usually with NSAIDs. Steroids can prevent conversion of infarcted myocardium to scar leading to greater thinning and risk of rupture.

Patients can also present with pericarditis 1 week to a few months after a myocardial infarction with a friction rub, typical EKG changes, and chest pain although the incidence has decreased with increased revascularization. Pericardial inflammation is thought to be from an autoimmune reaction to myocardial cells. Similar symptoms can also occur after cardiac surgery, blunt trauma, and pacemaker placement.

Patients can develop a pericardial effusion and even tamponade. Treatment includes NSAIDs and colchicine for 2 to 3 weeks and occasionally steroids. The syndrome is usually self-limited.

Radiation-Induced Pericarditis

Pericarditis can result from mediastinal and thoracic radiation for cancer including lymphoma and breast carcinoma and is related to the dosage delivered. With modern techniques, the incidence is 2% but can be as high as 20% if the entire pericardium is treated. Patients may develop chest pain and fever. Symptoms are usually self-limited, and tamponade is rare. Delayed symptoms can develop years later and can result in pericardial constriction. Due to the history of malignancy, the etiology can be confused with a malignant effusion. If pericardial constriction develops, the treatment is pericardiectomy although the mortality is higher than for other etiologies.

PERICARDIAL EFFUSION AND TAMPONADE

Any of the etiologies mentioned above for pericarditis can also lead to a pericardial effusion. Other causes include blunt or penetrating trauma, retrograde bleeding from an aortic dissection, and a transudative effusion from congestive heart failure. Effusions due to a bacterial or fungal infection, HIV, or malignancy have a higher incidence of progressing to tamponade. Twenty percent of large symptomatic effusions of unknown cause are due to an undiagnosed cancer.

Normally there is only a small reserve volume before pericardial fluid causes significant cardiac compression and prevents adequate cardiac filling. The hemodynamic significance of a pericardial effusion depends on the volume and the rate of accumulation. The compensatory adrenergic response to a pericardial effusion leads to tachycardia and increased contractility, and patients on beta blockers are less likely to compensate. Tamponade usually occurs

when filling pressures reach 15 to 20 mm Hg although tamponade can occur at lower pressures in conditions where blood volume is reduced including dialysis, with diuretic therapy, and bleeding. Cardiac pressures become elevated and are most closely equalized during inspiration. Tamponade generally affects right heart filling first which then leads to underfilling of the left heart.

Patients may present with dyspnea, tachycardia, and diaphoresis. The three classic signs of Beck triad are hypotension, jugular venous distension, and muffled heart sounds although each may be absent in patients with tamponade. Symptoms can be confused with right heart failure and pulmonary embolus.

The jugular waveform changes characteristically with loss of the y descent.24 Since the total heart volume is fixed in tamponade, blood only enters the heart when blood leaves. The y descent represents opening of the tricuspid valve and is lost since no blood is ejected from the heart. Pulsus paradoxus is also noted with a fall in the systolic blood pressure of greater than 10 mm Hg with inspiration. Pulsus paradoxus is absent in left ventricular dysfunction, atrial septal defect, positive-pressure ventilation, aortic insufficiency, and regional tamponade. The EKG shows variation in the morphology of every other QRS complex, or electrical alternans, due to swinging of the heart in the pericardial effusion (Fig.86-3). Chest radiograph shows an enlarged, rounded cardiac silhouette, and the pericardial fat pad sign is seen when the pericardial fat is separated from the heart by the effusion. Echocardiography is the most useful test in diagnosing pericardial effusion and tamponade.

Signs of tamponade include early diastolic right ventricular and right atrial collapse (Fig. 86-4) and distension of the cava that does not diminish with inspiration.

 Doppler is useful in evaluating blood flow and shows exaggerated respiratory variation with an increase with inspiration on the right and decrease on the left side of the heart. Echocardiography is also useful in identifying localized atrial compression which can lead to tamponade with the other changes described above.
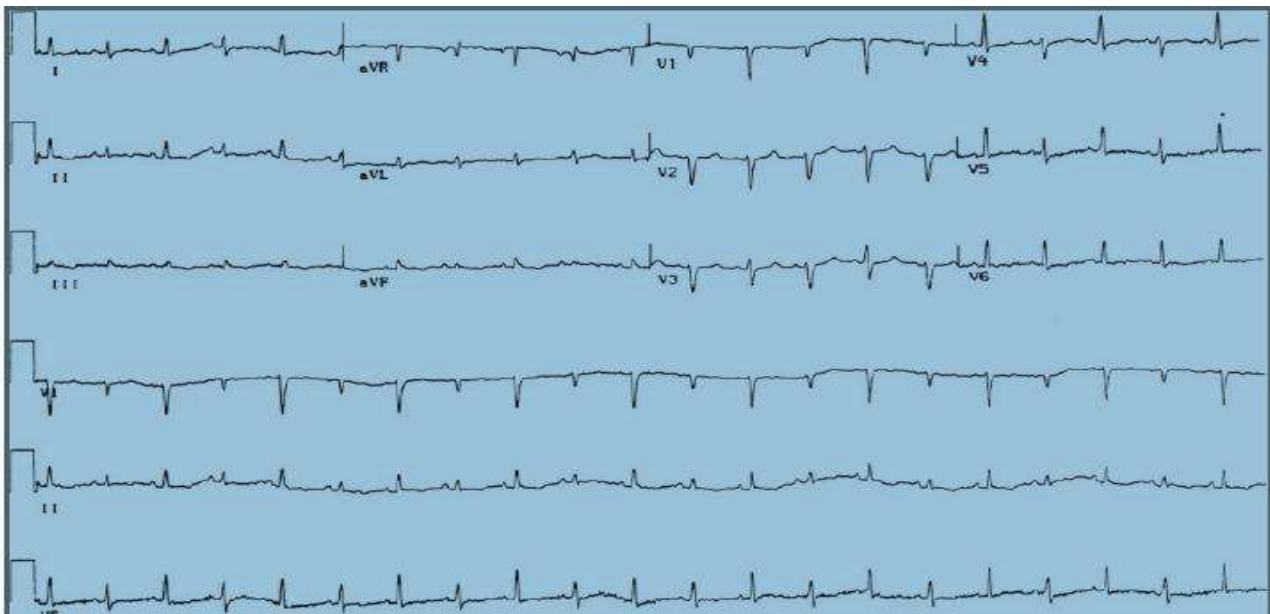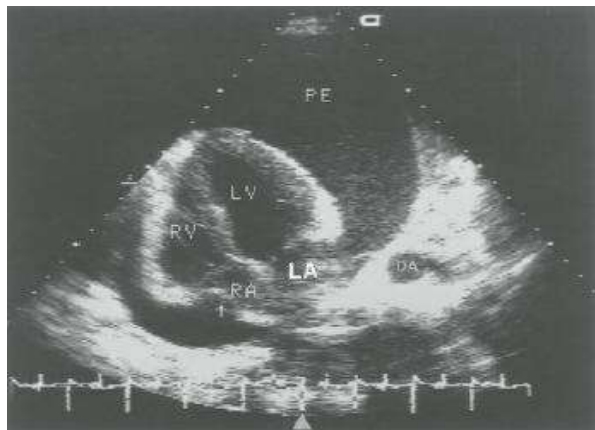

Figure 86-3. The 12-lead EKG shows electrical alternans with variation in the morphology of every other QRS complex in a patient with a large pericardial effusion.

If there are significant hemodynamic changes, emergent pericardiocentesis should be performed. Intravenous fluid and inotropes can temporize the hypotension but should not delay pericardiocentesis. If the effusion is loculated or contains blood clots, open drainage may be necessary. There should be a higher suspicion for tamponade in situations involving bleeding, bacterial or tuberculous pericarditis, or an acute or increasing moderate-to-large effusion.

Postoperative Cardiac Tamponade

Postoperative tamponade should always be in the differential diagnosis with low cardiac output after cardiac surgery. Patients may have rising filling pressures, hypotension, and decreased urine and cardiac output.

Mediastinal chest tube output may increase or abruptly stop. Hypovolemia during the postoperative period can limit the increase in filling pressures. Pulsus paradoxus may also be masked by positive pressure ventilation.

On chest radiograph, the cardiac silhouette is enlarged. In the postoperative cardiac patient, the epicardial pacing wires may move farther from the pericardium with increasing tamponade on chest films. Tamponade can result from relatively small amounts of blood with localized atrial compression from blood clots. There should be a low threshold for reexploration which also provides a definitive diagnosis. Patients can also present with delayed tamponade after being started on anticoagulants.

PERICARDIAL CONSTRICTION

Constriction develops when the heart becomes constrained by a fibrotic pericardium. The process can progress over years, and the most common causes in the developed world are radiation treatment, postsurgical changes, and idiopathic. Tuberculosis was the most common cause before antitubercular drug therapy. Other causes include amyloidosis, scleroderma, sarcoidosis, hemochromatosis, and malignant disease. Patients develop signs and symptoms of right heart failure due to obstruction of the right ventricular outflow tract including hepatomegaly, ascites, and peripheral edema. Patients can also have dyspnea, pleural effusions, fatigue, and weight loss. On physical examination, a pericardial knock, or a loud third heart sound, can be heard which corresponds to the abrupt cessation of diastolic filling.

Kussmaul sign is increased jugular venous distension with inspiration. Atrial fibrillation or flutter is present in up to 25% of patients, and tricuspid regurgitation is also common.

Pericardial calcification is pathognomonic but is only seen in 40% of cases. Thickened pericardium can be seen on CT or MRI (Fig. 86-5) although the pericardium was normal in thickness in 18% of patients in one series. The normal pericardial thickness is 2 mm on CT and 4 mm on MRI.

Echocardiography shows thickened pericardium and a septal bounce due to the abrupt displacement of the septum during early diastole. The ventricles are small with good function. Ventricular filling halts abruptly in diastole due to the limits of the stiff, fibrotic pericardium. There are signs of systemic venous congestion with a distended inferior vena cava. In contrast to tamponade, there is no decrease in early diastolic filling but a sudden decrease in late diastole. When the myocardium is involved by fibrosis, ventricular dysfunction is present which is associated with a poorer response to. On right heart catheterization, the y descent is deeper and more rapid than normal. The right atrial waveform is M- or W-shaped. The right ventricular tracing is described as the square root sign with an early diastolic decrease with a rapid increase and plateau. Pulmonary artery and right ventricular pressures can be moderately elevated, but more severe pulmonary hypertension suggests a different disease process. In addition, hypovolemia can mask these findings, and patients may need to be volume-challenged to see the diagnostic changes.

Distinguishing constrictive pericarditis from restrictive cardiomyopathy can be difficult but is important since the treatment is significantly different. In restrictive cardiomyopathy, there is no pericardial thickening. Decreased systolic function, mitral regurgitation, and left heart pressures greater than right are more common in restrictive cardiomyopathy. Early diastolic filling is slower. In addition, left and right ventricular pressures move in the same direction on inspiration rather than opposite directions.

Constrictive pericarditis is generally progressive although it can be transient after cardiac surgery and should be observed for several months. Diuretics with fluid and salt restriction help initially, but pericardiectomy is the only definitive treatment. Tachycardia is a compensatory response, and beta and calcium channel blockers should be avoided.
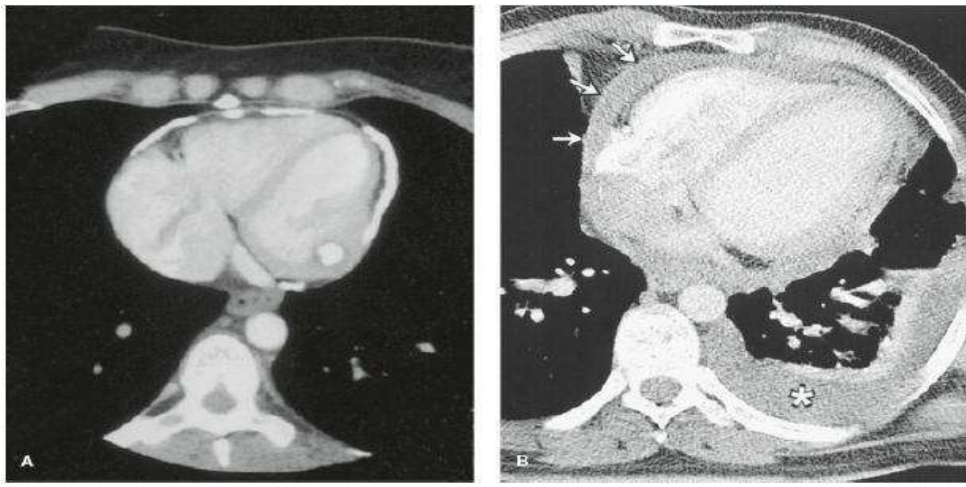
Figure 86-5. A: Chest CT shows extensive pericardial calcification. Constrictive pericarditis from a severely calcified pericardium. B: Chest CT shows thickening of the pericardium in a patient with constrictive pericarditis.

PERICARDIOCENTESIS

Pericardiocentesis can be diagnostic and therapeutic. Ideally, it should be performed in the catheterization laboratory so the hemodynamic response can be monitored. Normal pericardial fluid is an ultrafiltrate with an LDH 2.4 times serum and protein 0.6 times.28 Uremic fluid is bloody. Rheumatic effusions are high in protein and leukocytes and low in glucose. Cholesterol crystals are seen with myxedema, tuberculosis, and rheumatoid arthritis. Generally, a 16- to 22-gauge needle is inserted to the left of the xiphoid. The needle is angled at a 45- degree angle toward the left shoulder (Fig. 86-7). An EKG lead may also be attached to the needle to monitor for an injury current, although echocardiography is more commonly used for guidance. There is a 97% success rate with a complication rate of 4.7% (Table 86-4).29 If the effusion is anterior, a parasternal approach is used 1 to 2 cm to the left of the sternum in the fourth or fifth intercostal space.The removal of even a small amount of fluid can lead to significant hemodynamic improvement. For longer-term drainage a catheter can be placed over a guidewire. Pericardiocentesis may not be effective for purulent or bloody effusions that may require open drainage
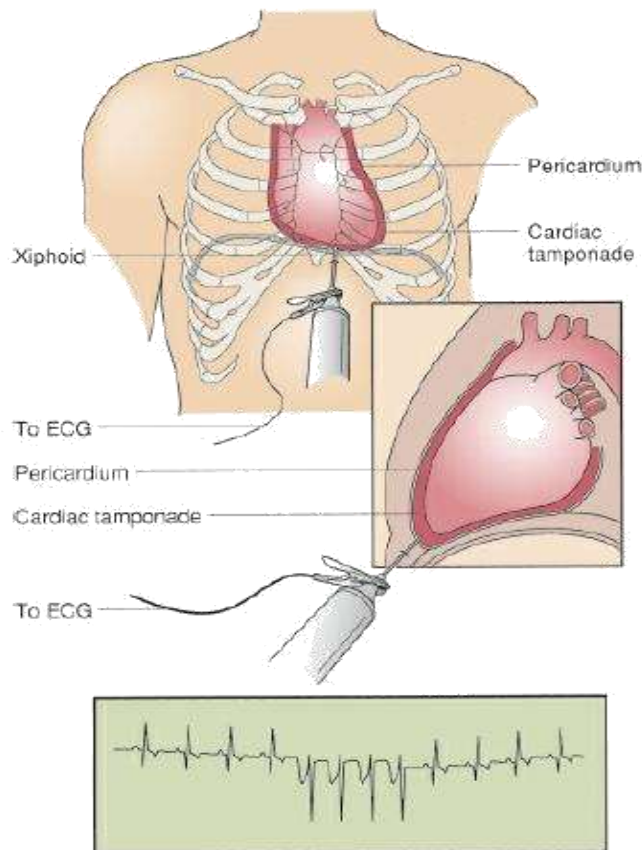
Figure 86-7. Pericardiocentesis is performed by inserting a needle to the left of the xiphoid and toward the left shoulder at a 45-degree angle. An EKG lead may be attached to the needle to monitor for Q waves indicating contact with the epicardium.

Table 86-4 Potential Complications From Pericardiocentesis

Injury to surrounding organs
Liver laceration
Lung (pneumothorax)
Coronary arteries
Myocardium
Arrhythmias
Pulmonary edema from acute left ventricular failure
Recurrence

PERICARDIAL BIOPSY AND SURGICAL DRAINAGE

Pericardial biopsy is useful when pericardiocentesis is nondiagnostic, which is not uncommon in neoplastic and tubercular effusions. This can be approached through a subxiphoid incision, anterior thoracotomy, or thoracoscopically (Fig. 86-8). Open drainage may be useful if the fluid is viscous, including bloody or purulent effusions, or in patients that have recurrent effusions. Loculated effusions may also require open drainage. The subxiphoid approach is better for purulent effusions to prevent contamination of the pleural space. Chronic or malignant effusions can be drained through a pericardial window into the left pleura. Preinduction pericardiocentesis or intravenous fluids may be necessary for patients to tolerate general anesthesia.

Xiphoid

Skin incision

Pericardium

Diaphragm
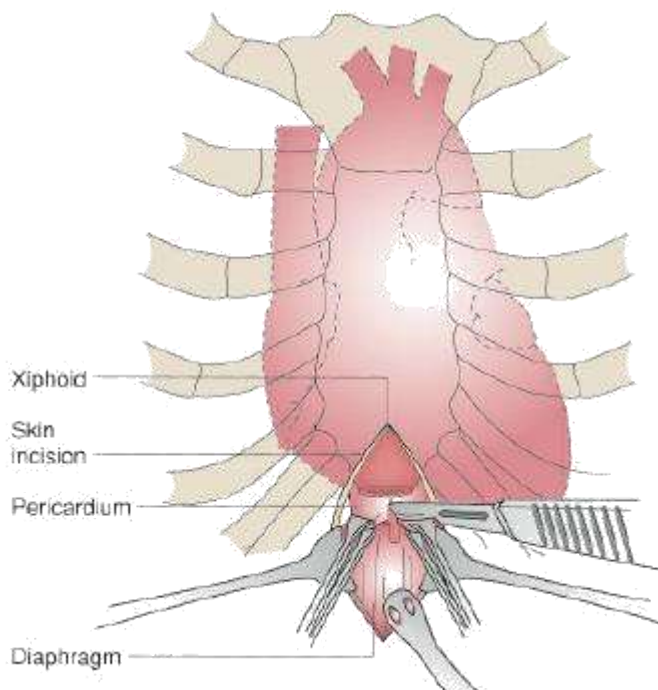
PERICARDIECTOMY

Pericardiectomy is most commonly performed for constrictive pericarditis. It can be performed through a median sternotomy or a left anterior thoracotomy in the fifth intercostal space. Anterior thoracotomy allows removal of the pericardium over the left ventricle with minimal manipulation. Femoral cannulation is used if bypass is necessary. Median sternotomy is the most common approach (Fig. 86-9).

Cardiopulmonary bypass increases the risk of bleeding and is used when significant cardiac manipulation is needed or the dissection is difficult. The pericardium over the left ventricle is resected first to avoid pulmonary edema from the right ventricle ejecting against a constricted left ventricle. The pericardium is removed from

phrenic nerve to phrenic nerve. Some patients have an immediate improvement in hemodynamics and symptoms while others do not improve until weeks or months later.

A delayed or incomplete response is thought to be due to an incomplete resection of the visceral pericardium or myocardial atrophy and fibrosis. Left ventricular function returned to normal in 40% of patients. Perioperative mortality has been reported between 5% and 15% and is due to low cardiac output, sepsis, bleeding, and renal or respiratory failure. Seventy percent of mortality is due to low cardiac output. Mortality is directly related to the patient's preoperative status35 and is 1% for New York Heart

Association class I to II, 10% for class III, and 46% for class IV.36 Five-year survival is 84%, and 99% of late survivors were class I or II. Patients with constriction due to radiotherapy have a higher mortality which may be due to radiation-induced myocardial injury. Other poor prognostic factors are renal failure, advanced age, pulmonary hypertension, hyponatremia, and reduced cardiac output.
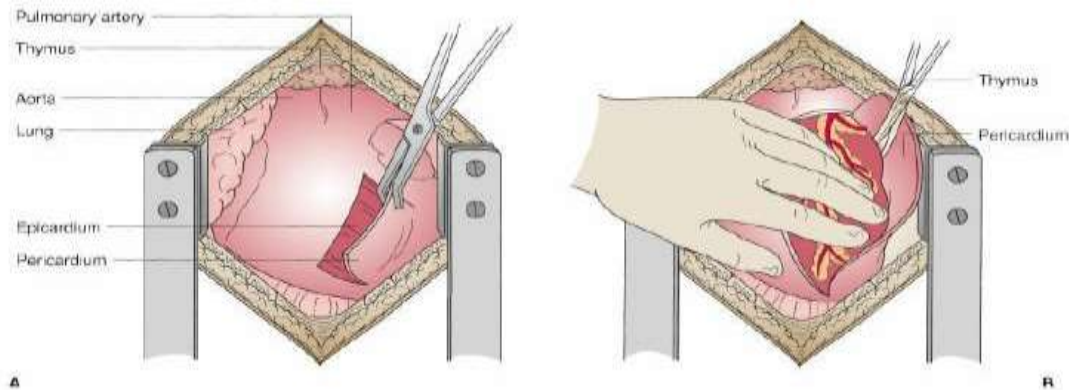

Figure 86-9. Pericardiectomy is most commonly approached through a median sternotomy. A: The pericardium is incised, and the fibrous pericardium dissected from the left ventricle. B: The heart is retracted to the right so that the pericardium can be resected from phrenic nerve to phrenic nerve.

2. Questions for self-control:
18. Aetiology of pericarditis.
19. Classification of pericarditis
20. Pathogenesis
21. Investigations
22. Surgery of pericarditis
23. Outcome

3. Case scenarios:

1.   A 63-year-old man is seen because of facial swelling and cyanosis, especially when he bends over. There are large, dilated subcutaneous veins on his upper chest. His jugular veins are prominent even while he is upright. Which of the following conditions is the most likely cause of these findings?
a. Histoplasmosis (sclerosing mediastinitis)
b. Substernal thyroid
c. Thoracic aortic aneurysm
d. Constrictive pericarditis
e. Bronchogenic carcinoma

Explanation
The answer is **E**. (Mulholland, p 1423.) Superior vena cava obstruction (SVC syndrome) is almost always due to malignancy (90% of cases) and in 3 out of 4 cases, results from invasion of the vena cava by bronchogenic carcinoma. Lymphomas are the second most common cause of the SVC syndrome. Fibrosing mediastinitis as a complication of histoplasmosis or ingestion of methysergide may occur, but is rare. Rarely, a substernal thyroid or thoracic aortic aneurysm may be responsible for the obstruction. SVC syndrome can be caused iatrogenically secondary to indwelling catheters. Although constrictive pericarditis may decrease venous return to the heart, it does not produce obstruction of the superior vena cava. Whatever the cause of the superior vena cava syndrome,

the resultant increased venous pressure produces edema of the upper body, cyanosis, dilated subcutaneous collateral vessels in the chest, and headache. Cervical lymphadenopathy may also be present as a result of either stasis or metastatic involvement. Initial management of superior vena cava syndrome consists of diuresis, and for malignancies, the treatment consists of radiation and chemotherapy if applicable. Occasionally, surgical intervention or thrombolysis may be indicated for severe life- threatening complications.

2. The basic clinical sign of exudative pericarditis is:

        A. Right-ventricle heart failure
        B. Left-ventricle heart failure
        C. Intoxication
        D. Respiratory failure
        E. Acrocyanosis

3. A 67-year-old man presents with an anterior myocardial infarction (MI) and receives thrombolytic therapy. Three days later, he develops chest pain that is exacerbated by lying down, and his physical findings are normal except for a friction rub. His ECG shows evolving changes from the anterior infarction but new PR-segment depression and 1-mm ST-segment elevation in all the limb leads. Which of the following is the most likely diagnosis?

    A. reinfarction
    B. pulmonary embolus
    C. viral infection
    D. post-MI pericariditis
    E. dissecting aneurysm

Explanation
 (D) Pericarditis secondary to transmural infarction is very common and most cases appear within 4 days. The most common manifestation of pericarditis is a friction rub along the left sternal border. It is evanescent, lasting only a few days. The pain is usually perceived by the patient to be different than that of the infarct. It is worsened by inspiration, swallowing, coughing, or lying down. It frequently is associated with a low-grade fever. *(Fuster, p. 1980,1995-1996)*

      A 23-year-old man develops sharp left-sided chest pain, fever, and a friction rub heard at the lower left sternal border, unaffected by respiration. The pain is also aggravated by lying down and relieved by sitting up. He is otherwise well with no other symptoms and the remaining physical examination is normal. Which of the following is the most likely cause for his symptoms?

    (A) rheumatic fever
    (B) tuberculosis (TB)
    (C) herpes simplex virus
    (D) MI
    (E) coxsackie virus

Explanation

(E) Pericarditis in clinical practice is commonly idiopathic and frequently assumed to be of possible viral origin. Coxsackieviruses are a common cause, but herpes viruses are not. Although TB, rheumatic fever, and MI can cause pericarditis, they are unlikely in this case.
    (Fuster, p. 1979)

5. A 47-year-old man is found to have edema, ascites, and hepatosplenomegaly. The examination of his neck veins reveals elevated venous pressure with a deep y descent. Heart size on x-ray is normal. Which of the following etiologies is not a possible explanation for this syndrome?

(A) rheumatic fever
(B) TB
(C) unknown cause
(D) previous acute pericarditis
(E) neoplastic involvement of the pericardium

Explanation

(A) Commonly, no cause is found for constric-tive pericarditis. Some patients do give a history of previous acute pericarditis. TB is now an uncommon cause. Cancer can cause constriction but is uncommon. Rheumatic fever does not cause pericarditis. *(Fuster, pp. 1989-1991)*

6.   4 weeks after myocardial infarction a 56-year-old patient developed acute heart pain, pronounced dyspnea. Objectively: the patient's condition is extremely grave, there is marked cyanosis of face, swelling and throbbing of neck veins, peripheral pulse is absent, the carotid artery pulse is rhythmic, 130 bpm, AP is 60/20 mm Hg. Auscultation of heart reveals extremely muffled sounds, percussion reveals heart border extension in both directions. What is the optimal treatment tactis for this patient?

A Pericardiocentesis and immediate thoracotomy
B Oxygen inhalation
C Puncture of the pleural cavity on the left
D Conservative treatment, infusion of adrenomimetics
E Pleural cavity drainage

7.   A week ago a 65-year-old patient suffered an acute myocardial infarction, his general condition deteriorated: he complains of dyspnea at rest, pronounced weakness. Objectively: edema of the lower extremities, ascites is present. Heart borders are extended, paradoxical pulse is 2 cm displaced from the apex beat to the left. What is the most likely diagnosis?

A Acute cardiac aneurysm
B Recurrent myocardial infarction
C Acute pericarditis
D Cardiosclerotic aneurysm
E Pulmonary embolism

4. Individual tasks for applicants for higher education on the topics:
1. Treatment of patients with Purulent Pericarditis
2. Tactics in the treatment of patients with fibrotic pericardium

5. List of recommended literature:

Basic

1.   SABISTON: TEXTBOOK OF SURGERY: THE BIOLOGICAL BASIS OF MODERN SURGICAL PRACTICE, TWENTY FIRST EDITION Copyright © 2020
2. Gozie Offiah, Arnold Hill//RCSI Handbook of Clinical Surgery for Finals. 4th ed. 2020
3. Цигикало О. В.   Clinical Anatomy and Operative Surgery=Клінічна анатомія і оперативна хірургія.. Підручник для ВМНЗ IV р.а.: Рекомендовано МОЗ: 2020/ 528 с.
4. Березницький Я. С. (за ред.)   General Surgery=Загальна хірургія. — 2-ге вид. Підручник для ВМНЗ III—IV р.а.: Рекомендовано ДУ"Центр.метод.каб.з вищої мед.освіти МОЗ України": 2020/ 328 с.
5. Christian de Virgilio, Areg Grigorian//Surgery: A Case Based Clinical Review. 2nd Ed. 2020
6. JANE C. ROTHROCK// Alexander's Care of the Patient in Surgery. 16th Ed. 2019
7. RUTHERFORD'S VASCULAR SURGERY AND ENDOVASCULAR THERAPY, 9th ed. Volume 1, Volume 2. Copyright © 2019 by Elsevier, Inc.

i.
8. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 11th Ed. Copyright © 2019 by Elsevier Inc.
9. SEIDEL'S GUIDE TO PHYSICAL EXAMINATION: AN INTERPROFESSIONAL APPROACH. Copyright © 2019 by Elsevier, Inc.

Additional.

1. Hamilton Bailey's Physical Signs: Demonstrations of Physical Signs in Clinical Surgery, 19th Edition Paperback – Import, by John S.P Lumley (Author), Anil K. D'Cruz (Author), Jamal J. Hoballah (Author), Carol E.H. Scott-Connor (Author) 25 Feb 2016
2. Schwartz's Principles Of Surgery With DVD Hardcover – 2014 by F. Charles Brunicardi (Author), Dana K. Andersen (Author), Timothy R. Billiar (Author), David L. Dunn (Author), John G. Hunter (Author), & 2 More
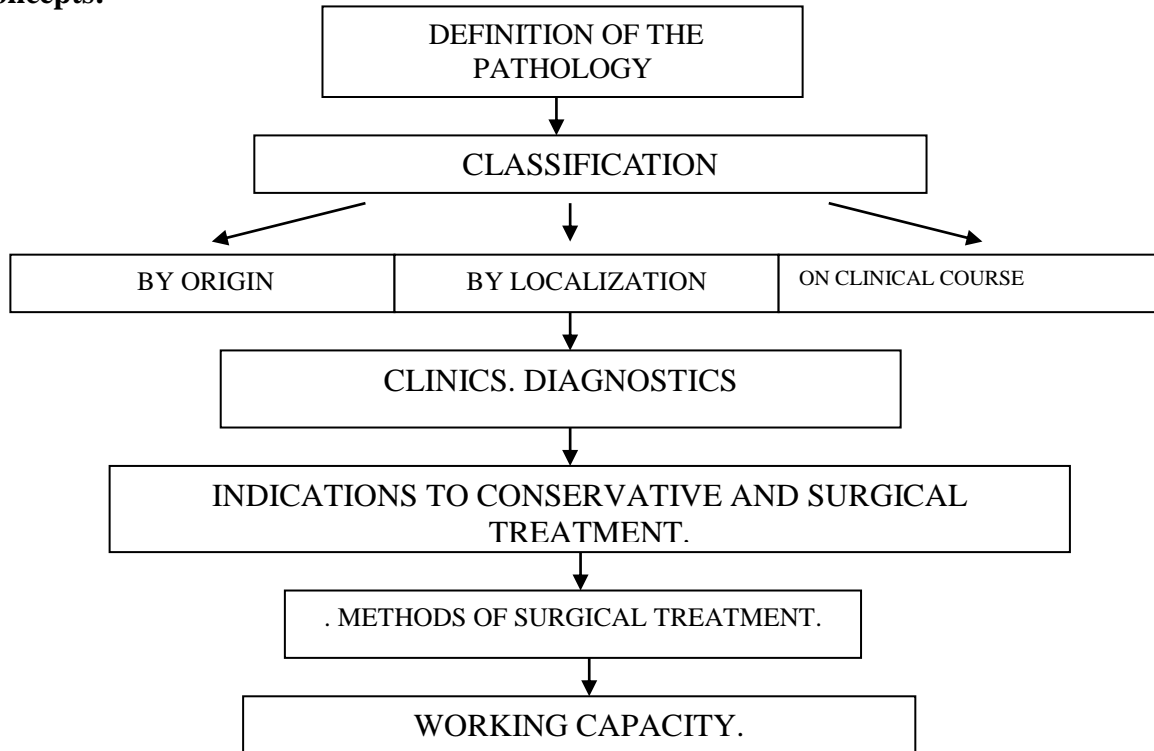
6. Electronic informative resources

1. http://moz.gov.ua – Міністерство охорони здоров'я України
2. www.ama-assn.org – Американська медична асоціація / American Medical Association
3. www.who.int – Всесвітня організація охорони здоров'я
4. www.dec.gov.ua/mtd/home/ - Державний експертний центр МОЗ України
5. http://bma.org.uk – Британська медична асоціація
6. www.gmc-uk.org - General Medical Council (GMC)
7. www.bundesaerztekammer.de – Німецька медична асоціація
8. http://medforum.in.ua/partners- Асоціація хірургів України
9. http://endoscopy.com.ua/ - Асоціація ендоскопічних хірургів України
10. http://thoracic-surgery.com.ua/ - Асоціація торакальних хірургів України
11. https://youcontrol.com.ua/ - Асоціація судинних хірургів України

Goal : Know the clinical anatomy and physiology of the parathyroid glands.
- know the classification structure, of diseases of the parathyroid glands.
- learn the clinical features and radiological signs parathyroid disease.
- know the structure, possible complications of diseases of the parathyroid glands.
- know the causes, types, clinical and radiological characteristics and methods of surgical treatment of diseases of the parathyroid glands.

Basic concepts:

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┌─────────────────────────────┐
│   DEFINITION OF THE          │
│   PATHOLOGY                  │
└─────────────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│      CLASSIFICATION          │
└─────────────────────────────┘
     │         │          │
     ▼         ▼          ▼
┌──────────┬──────────────┬────────────────────┐
│ BY ORIGIN│ BY LOCALIZATION│ ON CLINICAL COURSE│
└──────────┴──────────────┴────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│   CLINICS. DIAGNOSTICS       │
└─────────────────────────────┘
              │
              ▼
┌─────────────────────────────────────────────┐
│ INDICATIONS TO CONSERVATIVE AND SURGICAL      │
│ TREATMENT.                                    │
└─────────────────────────────────────────────┘
              │
              ▼
┌─────────────────────────────────────────────┐
│ . METHODS OF SURGICAL TREATMENT.              │
└─────────────────────────────────────────────┘
              │
              ▼
┌─────────────────────────────┐
│   WORKING CAPACITY.          │
└─────────────────────────────┘
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Equipment: Notebook, multimedia

Plan.

4. **Theoretical questions:**

Embryology. The superior parathyroid glands (IV) are derived from the fourth pharyngeal pouch, whereas the inferior glands (III) are from the more cephalad third pharyngeal pouch. The explanation for this apparent paradox is that of the common origin of the inferior glands and the thymus. From the fifth week of gestation, the thymus gland (also derived from the third pouch) descends into the superior mediastinum, dragging the inferior parathyroids with it. In consequence, some surgeons refer to the inferior parathyroids as 'parathymic'.

Anatomy. Because of their complicated embryological derivation, the precise location of individual glands is variable, although it tends to be symmetrical. The superior glands are usually found adjacent to the thyroid, typically within a 1 cm radius, above the junction of the inferior thyroid artery and the recurrent laryngeal nerve. The inferior glands are usually located in a condensation of fascia between the lower pole of the thyroid and the thymus (the thyro-thymic ligament). However, they may lie in the superior mediastinum or within the carotid sheath. The typical parathyroid gland is only 1 mm × 3 mm × 5 mm and weighs approximately 30 mg; the total weight of parathyroid tissue is thus about 120 mg. The blood supply for both pairs of glands is usually from the inferior thyroid artery. Fewer than 5% of individuals have five or more glands, with the supernumerary ones usually located within the thymus.

The cellular structure of the parathyroid gland is:
1)abundant chief cells which manufacture and secrete parathyroid hormone
2)sparse oxophil cells whose function is poorly understood.

Physiology of the control of the level of serum calcium

On the cell surface of every parathyroid cell is a calcium-sensing receptor. The concentration of plasma calcium is tightly controlled between 2.2 and 2.6 mmol/L. A reduction in serum calcium stimulates the secretion of parathyroid hormone (PTH) and vice versa.

Parathyroid hormone

The main product of the parathyroid glands has three principal actions:

1)stimulation of the activity of osteoclasts in bone and thus mobilisation of calcium from bone into the bloodstream

2)enhancement of the absorption of calcium from the gut into the bloodstream, an action facilitated by vitamin D

3)increase of the reabsorption of calcium by the renal tubules, thereby reducing urinary calcium excretion.

All of these tend to increase serum calcium concentration, which then has negative feedback on the secretion of PTH.

Vitamin D

Vitamin D3 is produced in the skin from the action of ultraviolet light upon 7-dehydrocholesterol; it is also ingested in the diet. This inactive form undergoes a two-stage hydroxylation process in the liver and kidney to produce the active vitamin D: 1,25-dihydroxycholecalciferol. The second hydroxylation is stimulated by PTH. Active vitamin D3enhances intestinal calcium absorption by PTH and facilitates bone mineralisation.

Calcitonin

Calcitonin is secreted by the parafollicular or C cells of the thyroid. Its actions are the direct opposite of those of PTH and, in particular, it decreases osteoclastic activity. However, its biological importance in calcium homeostasis is uncertain. Serum calcium concentration is normal both in patients who have undergone total thyroidectomy (and who therefore have negligible serum levels of calcitonin) and in those with medullary thyroid carcinoma (high levels of calcitonin).

Hyperparathyroidism

In this condition, there is overactivity of one or more parathyroid glands with secretion of excessive amounts of PTH. Three subtypes are recognised, as follows.

1)Primary hyperparathyroidism. Without any demonstrable stimulation, the parathyroid gland(s) secrete inappropriately raised amounts of PTH. Serum calcium concentration is raised and negative feedback is abolished, so the level of PTH is inappropriately high for the level of calcium. The cause is most commonly adenomatous change in one parathyroid, but less frequently there may be hyperplasia of all four or a carcinoma of one.

2)Secondary hyperparathyroidism. This occurs in chronic kidney disease (failure of tubular reabsorption); intestinal malabsorption and vitamin D deficiency. There is a reduction in the plasma concentration of calcium, which causes hyperplasia of all four glands. Increased production of PTH is therefore appropriate and, if the cause of hypocalcaemia can be corrected, in most instances the parathyroids return to normal.

3)Tertiary hyperparathyroidism. For patients with chronic kidney disease induced secondary hyperparathyroidism, the situation becomes chronic owing to dialysis. If a patient were then to receive a renal transplant, the implanted kidney retains the ability to activate vitamin D, which, in the presence of continuing parathyroid overactivity, leads to hypercalcaemia.

Primary hyperparathyroidism

Epidemiology.Primary hyperparathyroidism is the commonest subtype to present to the surgeon. The availability of the multichannel autoanalyser, which provides serum calcium concentrations on blood samples sent for other tests, has resulted in an increasing recognition of this disorder. It can occur at any age but its peak incidence is over 60 years. Women are more commonly affected. It has been reported to occur in 1 in 1000 patients in hospital, but community prevalence is much less.

Aetiology and pathological features. The cause remains obscure.

Eighty per cent of patients have a solitary adenoma. The adenomatous gland is enlarged, and the chief cells are hypertrophied and numerous. The other glands are suppressed, small, their chief cells few, and their stroma contains an abundant amount of fat. In 5% of patients, adenomas are multiple. The remainder (15–20%) have multiple-gland hyperplasia. Patients with hyperplasia may suffer from the multiple endocrine neoplasia (MEN) syndrome.

Clinical features. The symptoms are those of complications of the disorder, often summarised as 'stones, bones, abdominal groans and psychic moans' but more formally listed as:

1) urinary tract stones – mainly renal colic
2) bone decalcification, which may cause bone pain or a pathological fracture
3) abdominal pain – often of obscure cause but occasionally consequent on the presence of a peptic ulcer or recurrent pancreatitis
4) psychological disturbances of altered mood – mainly depression which may remain unrecognised by the patient or the doctor until successful treatment alters the mental state for the better.

Occasionally a rise of serum calcium concentration above 3.5 mmol/L produces a syndrome of vomiting, dehydration, renal failure and coma. The event may be potentially lethal.

Increasingly (up to 80%) patients are without overt symptoms and the possibility of the condition is signalled by an abnormal result on the autoanalyser profile. Most patients admit to fatigue, poor memory and bone and joint pains.

Physical findings. Examination rarely reveals any abnormality. It is most unusual to find a lump in the neck. Features of the complications outlined above may be present.

Investigation. Diagnostic criteria are as follows:

1) Unequivocal hypercalcaemia – blood is taken without applying a tourniquet to the arm, because this may raise serum calcium concentration by provoking regional acidosis. At least three measurements are made on different occasions; since most calcium in serum is bound to albumin, results are adjusted to a standard albumin concentration of 40 g/L.
2) Simultaneous finding of detectable or raised levels of PTH in the blood – excluding other causes of hypercalcaemia.

Other causes of hypercalcaemia to be excluded in the diagnosis of primary hyperparathyroidism

Cause	Method of exclusion
Secondary carcinoma of bone (common sites: breast, bronchus, thyroid, kidney and prostate)	History Typical bone X-rays Bone scan
Multiple myeloma	Typical bone X-rays
	Plasma electrophoresis
	Bence–Jones proteinuria
Vitamin D intoxication	History of intake
Sarcoidosis	
Thyrotoxicosis	
Rare tumours (usually carcinoma of bronchus) which secrete PTH-related peptide	
Familial hypercalcaemic hypocalciuria – diminished renal calcium excretion	Family history Low renal calcium output

Management

Hypercalcaemia. Severe hypercalcaemia (above 3.5 mmol/L) requires rehydration and the administration of bisphosphonates, which inhibit osteoclastic bone resorption. Calcium concentrations are measured at least daily and often need to be followed more frequently. Moderate hypercalcaemia (3.0–3.5 mmol/L) is usually controlled by intravenous rehydration.

Surgical. Surgical removal offers the only cure for primary hyperparathyroidism and should be offered to all symptomatic patients and those asymptomatic patients whose calcium concentration exceeds 2.75 mmol/L. The high success rate and low morbidity of exploration of the neck have led towards a more liberal surgical policy for minimally symptomatic patients with a plasma calcium between 2.60 and 2.75 mmol/L.

The preoperative preparation is as for thyroidectomy. Some surgeons use frozen section and others intra-operative (quick) PTH assay.

Operation. The approach and exposure are the same as for thyroidectomy. If an adenoma is found, it is removed. The surgical treatment of multi-gland hyperplasia is more difficult. The conventional management is subtotal parathyroidectomy (removal of three and a half glands), leaving half a gland in the neck. However, there is a chance of recurrence, and neck re-exploration is then more difficult and treacherous. Some surgeons now autograft the remaining half gland into the forearm. The graft survival rate is high (more than 90%) and further surgery in the neck is avoided. A third option is to cryopreserve some parathyroid tissue and delay its transplantation until hypocalcaemia is documented; however, graft survival is much lower. The fourth option is to excise all parathyroid tissue and place the patient on lifelong calcium and vitamin D supplements.

Postoperative care. Serum calcium concentration is measured the day after surgery. After a successful procedure, the calcium level often falls below normal before it rises to the normal range. If the calcium level does not fall, or returns to its original level after a transient decrease, the surgeon has failed to remove the disordered tissue.

The patient is questioned for early signs of hypocalcaemia such as paraesthesiae of the hands and lips. Trousseau's and Chvostek's signs may be elicited. It may be necessary to give oral calcium and vitamin D until the suppressed parathyroids recover.

Complications
PERSISTENT OR RECURRENT HYPERCALCAEMIA is the consequence of failure to remove an adenoma or enough hyperplastic tissue. Re-exploration must be carried out, but only by an experienced surgeon because the complication rate is much higher than for a first operation.

HYPOPARATHYROIDISM. Transient hypocalcaemia is common even after successful removal of a diseased gland but recovery occurs within a week. Permanent hypocalcaemia follows removal of too much parathyroid tissue. If cryopreserved tissue is available, some of it is implanted as already described; otherwise, the patient is treated with calcium and vitamin D for life.

Parathyroid carcinoma
This condition is very rare. Usually there is invasion of local tissues and recurrence after excision. Metastases are uncommon. Death is often caused by hypercalcaemia.

Clinical features. There may be symptoms of metastatic disease in addition to the progressive effects of hyperparathyroidism.Physical findings are of a mass in the neck which may be palpable; this should alert the surgeon to the possibility of carcinoma.

Investigation and management. The plasma calcium concentration is typically very high and is accompanied by a high level of PTH. CT of the neck demonstrates the local anatomy, and a chest X-ray should be taken to look for secondary spread.The surgical strategy is en-bloc resection of the tumour. Adjuvant oncotherapy should be considered, and bisphosphonates may be required to control the hypercalcaemia.

2. Questions for self-control:
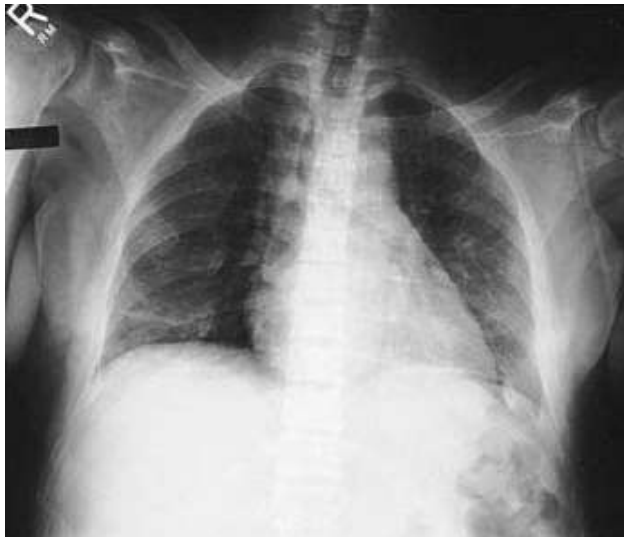Frontal survey on basic terminology.
Questions :
   24. Clinical anatomy and physiology of the parathyroid glands.
   25. Classification of benign tumours of the parathyroids.
   26. Pathogenesis
   27. Investigations
   28. Surgery of benign tumours of the parathyroids.
   29. Outcomes

   1. 3. **Case scenarios:**

   1. This 30-year-old woman presented with weakness, bone pain, an elevated parathormone level, and a serum calcium level of 15.2 mg/dL. Skeletal survey films were taken, including the hand films and chest x-ray shown. The most likely cause of these findings is

a. Sarcoidosis
b. Vitamin D intoxication
c. Paget's disease
d. Metastatic carcinoma
e. Primary hyperparathyroidism

2. A 52-year-old woman sees her physician with complaints of fatigue, headache, flank pain, hematuria, and abdominal pain. She undergoes a sestamibi scan that demonstrates persistent uptake in the right superior parathyroid gland at 2 hours. Which of the following laboratory values is most suggestive of her diagnosis?

a. Serum acid phosphatase above 120 IU/L
b. Serum alkaline phosphatase above 120 IU/L
c. Serum calcium above 11 mg/dL
d. Urinary calcium below 100 mg/day
e. Parathyroid hormone levels below 5 pmol/L

Explanation
The answer is c. (Brunicardi, pp 1376-1381.) Elevated parathyroid hormone (PTH) levels in conjunction with elevated calcium levels are diagnostic for hyperparathyroidism. Primary hyperparathyroidism is a common disease, affecting 100,000 individuals each year in the United States. Essential to the diagnosis of hyperparathyroidism is the finding of hypercalcemia. Though there are many causes of hypercalcemia, hyperparathyroidism is by far the most prevalent. The majority of patients with

primary hyperparathyroidism have a single parathyroid adenoma, which can be localized in 75% to 80% of patients with sestamibi scanning. Technetium 99m–labeled sestamibi is taken up by the parathyroid and thyroid glands. Hyperfunctioning parathyroid glands take up the sestamibi to a greater extent than normal glands, and therefore sestamibi scanning can be used to identify parathyroid adenomas. Patients with primary hyperparathyroidism have either normal or elevated urinary calcium. As the name suggests, patients with familial hypocalciuric hypercalcemia (FHH) have hypercalcemia. They also usually have elevated PTH, but urine calcium excretion is low (as opposed to normal to high as with a parathyroid adenoma). Surgery is not indicated in this relatively rare setting of hypercalcemia.

3. Which of the following patients with primary hyperparathyroidism should undergo parathyroidectomy?

        a. A 62-year-old asymptomatic woman
        b. A 54-year-old woman with fatigue and depression
        c. A 42-year-old woman with a history of kidney stones
        d. A 59-year-old woman with mildly elevated 24-hour urinary calcium excretion
        e. A 60-year-old woman with mildly decreased bone mineral density measured at the hip of less than 2 standard deviations below peak bone density

Explanation
The answer is c. (Brunicardi, p 1380.) Patients with symptomatic primary hyperparathyroidism as manifested by kidney stones, renal dysfunction, or osteoporosis should undergo parathyroidectomy.However, management of "asymptomatic" patients is controversial. Indications for surgical intervention for asymptomatic primary hyperparathyroidism include age less than 50 years, markedly elevated urine calcium excretion, kidney stones on radiography, decreased creatinine clearance, markedly elevated calcium or 1 episode of life-threatening hypercalcemia, and substantially decreased bone mass.

4. A 30-year-old woman presents with hypertension, weakness, bone pain, and a serum calcium level of 15.2 mg/dL. Hand films below show osteitis fibrosa cystica. Which of the following is the most likely cause of these findings?

        a. Sarcoidosis
        b. Vitamin D intoxication
        c. Paget disease
        d. Metastatic carcinoma
        e. Primary hyperparathyroidism

Explanation
The answer is e. (Brunicardi, pp 1377-1378.) Osteitis fibrosa cystica is a condition associated with hyperparathyroidism that is characterized by severe demineralization with subperiosteal bone resorption (most prominent in the middle phalanx of the second and third fingers), bone cysts, and tufting of the distal phalanges on hand films. These specific bone findings would not be present in sarcoidosis, Paget disease, or metastatic carcinoma. Vitamin D deficiency can lead to osteitis fibrosa cystica, but it would also be associated with hypocalcemia, not hypercalcemia.

5. A 35-year-old woman presents with a serum calcium level of 15.2 mg/dL and an elevated parathyroid hormone level. Following correction of the patient's hypercalcemia with hydration and furosemide, which of the following is the best therapeutic approach?

        a. Administration of steroids
        b. Radiation treatment to the neck
        c. Neck exploration and resection of all 4 parathyroid glands
        d. Neck exploration and resection of a parathyroid adenoma
        e. Avoidance of sunlight, vitamin D, and calcium-containing dairy products

Explanation

The answer is d. (Brunicardi, pp 1381-1383.) Treatment for primary hyperparathyroidism in this setting is resection of the diseased parathyroid glands after initial correction of the severe hypercalcemia. Parathyroidectomy without preoperative localization studies have a high success rate and low complication rate. Neck exploration will yield a single parathyroid adenoma in about 85% of cases. Two adenomas are found less often (approximately 5% of cases) and hyperplasia of all 4 glands occurs in about 10% to 15% of patients. If hyperplasia is found, treatment includes resection of 3½ glands. The remnant of the fourth gland can be identified with a metal clip in case reexploration becomes necessary. Alternatively, all 4 glands can be removed with autotransplantation of a small piece of parathyroid tissue into the forearm or sternocleidomastoid muscle.

6. A 52-year-old woman sees her physician with complaints of fatigue, headache, flank pain, hematuria, and abdominal pain. She undergoes a sestamibi scan that demonstrates persistent uptake in the right superior parathyroid gland at 2 hours. Which of the following laboratory values is most suggestive of her diagnosis?

    a. Serum acid phosphatase above 120 IU/L
    b. Serum alkaline phosphatase above 120 IU/L
    c. Serum calcium above 11 mg/dL
    d. Urinary calcium below 100 mg/day
    e. Parathyroid hormone levels below 5 pmol/L

Explanation
The answer is c. (Brunicardi, pp 1376-1381.) Elevated parathyroid hormone (PTH) levels in conjunction with elevated calcium levels are diagnostic for hyperparathyroidism. Primary hyperparathyroidism is a common disease, affecting 100,000 individuals each year in the United States. Essential to the diagnosis of hyperparathyroidism is the finding of hypercalcemia. Though there are many causes of hypercalcemia, hyperparathyroidism is by far the most prevalent. The majority of patients with primary hyperparathyroidism have a single parathyroid adenoma, which can be localized in 75% to 80% of patients with sestamibi scanning. Technetium 99m–labeled sestamibi is taken up by the parathyroid and thyroid glands. Hyperfunctioning parathyroid glands take up the sestamibi to a greater extent than normal glands, and therefore sestamibi scanning can be used to identify parathyroid adenomas. Patients with primary hyperparathyroidism have either normal or elevated urinary calcium. As the name suggests, patients with familial hypocalciuric hypercalcemia (FHH) have hypercalcemia. They also usually have elevated PTH, but urine calcium excretion is low (as opposed to normal to high as with a parathyroid adenoma). Surgery is not indicated in this relatively rare setting of hypercalcemia.

7. Which of the following patients with primary hyperparathyroidism should undergo parathyroidectomy?

    a. A 62-year-old asymptomatic woman
    b. A 54-year-old woman with fatigue and depression
    c. A 42-year-old woman with a history of kidney stones
    d. A 59-year-old woman with mildly elevated 24-hour urinary calcium excretion
    e. A 60-year-old woman with mildly decreased bone mineral density measured at the hip of less than 2 standard deviations below peak bone density

Explanation
The answer is c. (Brunicardi, p 1380.) Patients with symptomatic primary hyperparathyroidism as manifested by kidney stones, renal dysfunction, or osteoporosis should undergo parathyroidectomy. However, management of "asymptomatic" patients is controversial. Indications for surgical intervention for asymptomatic primary hyperparathyroidism include age less than 50 years, markedly elevated urine calcium excretion, kidney stones on radiography, decreased creatinine clearance, markedly elevated calcium or 1 episode of life-threatening hypercalcemia, and substantially decreased bone mass.

4. Individual tasks for applicants for higher education on the topics:
1. Treatment of patients with adenoma of the parathyroids.

5. List of recommended literature:

<p align="center">Basic</p>

1. SABISTON: TEXTBOOK OF SURGERY: THE BIOLOGICAL BASIS OF MODERN SURGICAL PRACTICE, TWENTY FIRST EDITION Copyright © 2020
2. Gozie Offiah, Arnold Hill//RCSI Handbook of Clinical Surgery for Finals. 4th ed. 2020
3. Цигикало О. В.   Clinical Anatomy and Operative Surgery=Клінічна анатомія і оперативна хірургія.. Підручник для ВМНЗ IV р.а.: Рекомендовано МОЗ: 2020/ 528 с.
4. Березницький Я. С. (за ред.)   General Surgery=Загальна хірургія. — 2-ге вид. Підручник для ВМНЗ III—IV р.а.: Рекомендовано ДУ"Центр.метод.каб.з вищої мед.освіти МОЗ України": 2020/ 328 с.
5. Christian de Virgilio, Areg Grigorian//Surgery: A Case Based Clinical Review. 2nd Ed. 2020
6. JANE C. ROTHROCK// Alexander's Care of the Patient in Surgery. 16th Ed. 2019
7. RUTHERFORD'S VASCULAR SURGERY AND ENDOVASCULAR THERAPY, 9th ed. Volume 1, Volume 2. Copyright © 2019 by Elsevier, Inc.
8. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 11th Ed. Copyright © 2019 by Elsevier Inc.
9. SEIDEL'S GUIDE TO PHYSICAL EXAMINATION: AN INTERPROFESSIONAL APPROACH. Copyright © 2019 by Elsevier, Inc.

Additional.

1. Hamilton Bailey's Physical Signs: Demonstrations of Physical Signs in Clinical Surgery, 19th Edition Paperback – Import, by John S.P Lumley (Author), Anil K. D'Cruz (Author), Jamal J. Hoballah (Author), Carol E.H. Scott-Connor (Author) 25 Feb 2016
2. Schwartz's Principles Of Surgery With DVD Hardcover – 2014 by F. Charles Brunicardi (Author), Dana K. Andersen (Author), Timothy R. Billiar (Author), David L. Dunn (Author), John G. Hunter (Author), & 2 More
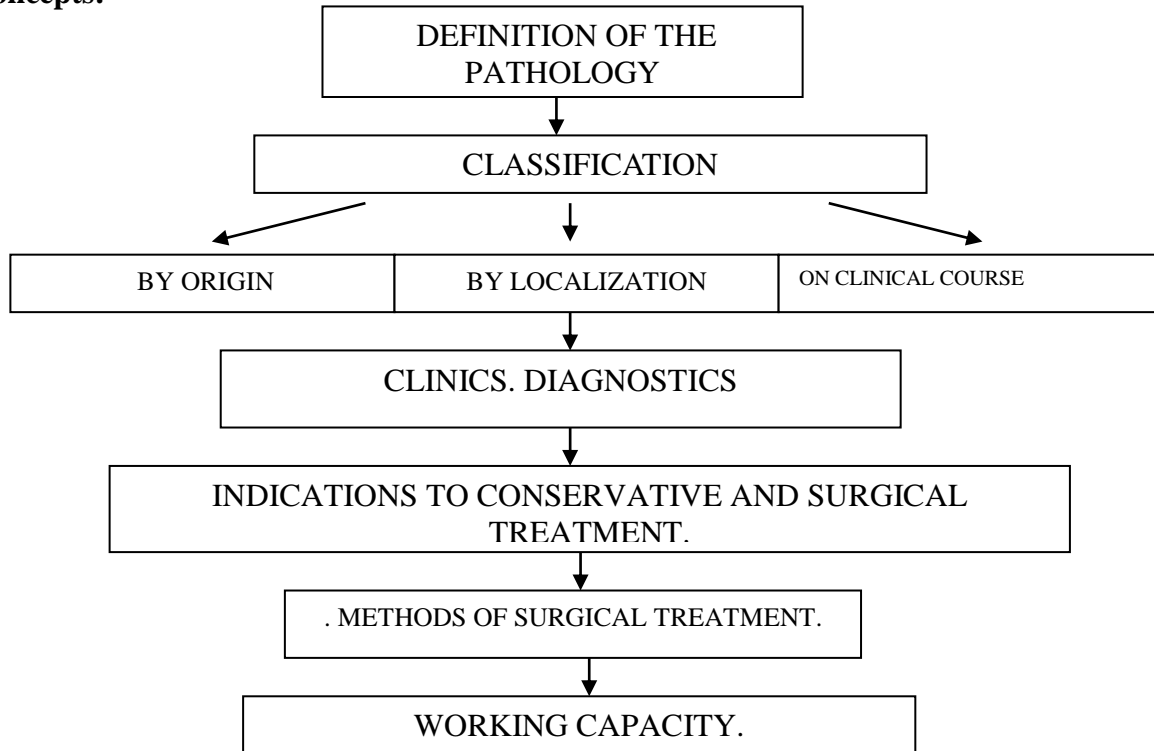
6. Electronic informative resources

1. http://moz.gov.ua – Міністерство охорони здоров'я України
2. www.ama-assn.org – Американська медична асоціація / American Medical Association
3. www.who.int – Всесвітня організація охорони здоров'я
4. www.dec.gov.ua/mtd/home/ - Державний експертний центр МОЗ України
5. http://bma.org.uk – Британська медична асоціація
6. www.gmc-uk.org - General Medical Council (GMC)
7. www.bundesaerztekammer.de – Німецька медична асоціація
8. http://medforum.in.ua/partners- Асоціація хірургів України
9. http://endoscopy.com.ua/ - Асоціація ендоскопічних хірургів України
10. http://thoracic-surgery.com.ua/ - Асоціація торакальних хірургів України
11. https://youcontrol.com.ua/ - Асоціація судинних хірургів України

Goal : Know the clinical anatomy and physiology of the adrenal glands.

- know the classification of diseases of adrenal glands.
- learn clinical and radiological signs of adrenal diseases.
- Know the possible complications of diseases of adrenal glands.
- know the causes, types, clinical and radiological characteristics and methods of surgical treatment of diseases of the adrenal glands.

Basic concepts:

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        ┌─────────────────────────────┐
        │  DEFINITION OF THE          │
        │  PATHOLOGY                  │
        └──────────────┬──────────────┘
                       ▼
        ┌─────────────────────────────┐
        │       CLASSIFICATION        │
        └──────────────┬──────────────┘
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  │  BY ORIGIN   │  BY LOCALIZATION │ ON CLINICAL COURSE │
  └──────────────┴────────┬─────────┴────────────────────┘
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        │     CLINICS. DIAGNOSTICS    │
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  │  INDICATIONS TO CONSERVATIVE AND SURGICAL      │
  │  TREATMENT.                                     │
  └──────────────────────┬────────────────────────┘
                         ▼
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      │ . METHODS OF SURGICAL TREATMENT.     │
      └──────────────────┬───────────────────┘
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        │     WORKING CAPACITY.       │
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Equipment: Notebook, multimedia

Plan.

5. **Theoretical questions:**

Adrenals

Embryology. The glands are derived from two components: the cortex and the medulla. The cortex is formed during the fifth week of life from proliferation of mesodermal cells. This is then invaded by ectodermal cells which have migrated from the neural crest (ectoderm) to form the adrenal medulla; these cells either differentiate into chromaffin cells containing granules of catecholamines (phaeochromocytes) or non-chromaffin sympathetic ganglion cells.

Neural crest cells are widely dispersed throughout the embryo but are usually replaced by lymphatic tissue shortly after birth. Ectopic nests of cells have been described in diverse sites including the urinary bladder, gonads and gastrointestinal tract. Persistence of these cells may give rise to extra-adrenal medullary neoplasms.

Anatomy. The adrenal glands are paired and similarly placed, bilaterally, above the kidneys; however, they are far from symmetrical. Each normal gland weighs approximately 5 g and is 5 cm long, 3 cm wide and

approximately 1 cm thick. The right gland is pyramidal while the left is semilunar. The blood supply arises from three arteries: branches from the aorta, renal and phrenic arteries.

Physiology. The cortex secretes steroid hormones from three distinct zones. The outer zona glomerulosa secretes the mineralocorticoid aldosterone whose major actions are in the control of renal sodium and potassium excretion in conjunction with renin and angiotensin. The middle zona fasciculata secretes the glucocorticoid cortisol whose many physiological effects include hepatic gluconeogenesis, protein catabolism, some mineralocorticoid effects and regulation of the response to inflammation. Secretion of cortisol is regulated by adrenocorticotrophic hormone (ACTH) from the anterior pituitary. The inner zona reticularis secretes adrenal androgens and accounts for 15–20% of total androgen activity in males. The cells of the adrenal medulla are part of the amine precursor uptake and decarboxylation (APUD) system and secrete catecholamines noradrenaline (norepinephrine) and adrenaline (epinephrine).

Classification of disorders

Hyperplasia or neoplasia of the adrenal cortex produces characteristic syndromes which are dependent on the zone of origin:
1)zona glomerulosa – primary hyperaldosteronism
2)zona fasciculata – Cushing's syndrome
3)zona reticularis – virilism.
These entities can occur on their own or there may be a mixed picture which is suggestive of an adrenal carcinoma.

Primary hyperaldosteronism

Epidemiology and pathological features.This condition is relatively rare, occurring in less than 2% of patients with hypertension. It presents most commonly between the ages of 20 and 50 years and is more common in men. There are two subtypes:

1)idiopathic hyperaldosteronism – bilateral adrenal hyperplasia

2)Conn's syndrome – a single, usually small, canary-yellow tumour of the adrenal cortex, which is nearly always benign (98%).

Idiopathic hyperaldosteronism is three times less common than Conn's syndrome as a cause of primary hyperaldosteronism. It is important to distinguish between a single tumour, which is best treated surgically, and bilateral adrenal hyperplasia, which can be managed medically. Inappropriate autonomous oversecretion of aldosterone leads to sodium retention and potassium loss, causing hypertension and muscle weakness. High levels of aldosterone suppress the renin–angiotensin–aldosterone axis and, in consequence, the plasma renin concentration is low.

Clinical features. Symptoms are either vague or absent. Patients may complain of lethargy, muscle weakness and thirst. Clinical examination is normal, but there is hypertension.

Investigation. There are two stages of investigation: initially to confirm the diagnosis and then to localise a tumour if one is thought to be present.

 Localisation.CT scan may show a small (1 cm) tumour, but it must be remembered that there is a significant incidence of adrenal incidentalomas.

Scanning with radiolabelled cholesterol can distinguish a solitary functional adrenal tumour from bilateral increased uptake. The gold standard test is bilateral adrenal venous sampling to measure aldosterone

concentrations. However, this technique is difficult to undertake and carries a risk of infarction to one or both adrenals.

Management

Non-operative. If a tumour has been diagnosed, it is essential that serum potassium concentration be returned to normal before surgical treatment is considered; this can be achieved by the use of spironolactone, which blocks aldosterone receptors and reduces aldosterone secretion; 200–400 mg/day is used for 3–6 weeks before operation. Hyperplasia is not an indication for exploration and bilateral adrenalectomy because this renders the patient permanently dependent on steroids and restores the blood pressure to normal in only one-third of all patients. Spironolactone in doses of 200–400 mg/day controls the hypokalaemia, but other agents may be necessary to reduce blood pressure.

Operative. Adenomas are treated by unilateral adrenalectomy. This can be achieved laparoscopically. There is a high cure rate and virtually no morbidity or mortality. The hypokalaemia of primary aldosteronism is almost universally cured, but hypertension persists in 30% of patients and may recur in a further 20%, with, therefore, a long-term cure of hypertension of only 50%.

Cushing's syndrome

This condition is characterised by glucocorticoid excess. The underlying causes are
1)ACTH-dependent
a)Ectopic ACTH secretion – 15%
b)Cushing's 'disease' – 65%
2)ACTH-independent
a)Adrenocortical adenoma – 10%
b)Adrenocortical carcinoma – 10%
3)Iatrogenic steroid therapy – variable but should never be forgotten
ACTH, adrenocorticotrophic hormone.
Epidemiology. Cushing's syndrome affects patients between 20 and 40 years old and has a predilection for women; however, ectopic ACTH secretion has an equal sex incidence which reflects the chief cause, which is bronchogenic carcinoma. Adrenocortical carcinoma as a cause is very rare.
Pathophysiology. Primary ACTH-secreting tumours of the pituitary are described later in this chapter. Ectopic ACTH production stimulates both adrenal glands to produce excess cortisol. Adrenocortical adenomas and carcinomas secrete excess levels of cortisol with suppression of ACTH, and each accounts for about 10% of patients with Cushing's syndrome.

Clinical features. The effects of excess cortisol on the body are wide-ranging and produce characteristic clinical features. Typical symptoms are:
1)facial and truncal obesity
2)menstrual irregularity
3)hirsutism
4)muscle weakness
5)osteoporosis.
Clinical findings.The typical physical signs are

1.moon face
2.buffalo hump
3.central obesity ('lemon-on-sticks' appearance)
4.muscle wasting and weakness
5.striae
6.hirsutism
7.ecchymoses
8.hypertension.
Investigation. Biochemical and endocrinological

The following are used to confirm the presence of the disorder:

The 24-hour urinary free cortisol is elevated, and a low-dose dexamethasone suppression test depresses the hypothalamic–pituitary axis in normals but not in Cushing's syndrome.

Localisation of a tumour/Methods used are CT and selective venous sampling.

Management.

Medical. If an operation is indicated, steroid cover with hydrocortisone must be given both preoperatively and perioperatively and reduced to a maintenance dose of 20–30 mg/day of hydrocortisone usually within 10 days after operation. Patients with ectopic ACTH secretion are best treated by resecting the primary source, but all too often this is either not technically feasible or the patients have advanced metastatic disease and a poor prognosis. In such circumstances, drugs which block steroid synthesis, such as metyrapone and amino-glutethimide, are used.

Surgical. Adenomas are treated by unilateral adrenalectomy. Adrenal carcinomas are usually advanced, and metastases are common. Despite their poor prognosis (less than 50% survival at 2 years), surgery is the treatment of choice in an attempt to reduce the bulk of the tumour and palliate the symptoms of steroid excess. This can be aided in part by the use of mitotane , a mitochondrial poison, which, in the majority of patients, reduces the secretion of both cortisol and adrenal androgens.

Phaeochromocytoma

This is a catecholamine-secreting tumour of chromaffin cells in the adrenal medulla or in the paraganglionic tissues adjacent to the sympathetic chain at any level. It is a rare tumour with an incidence of 1 per million and which therefore accounts for less than 1% of cases of hypertension.

Pathological features.It has been called the '10% tumour' because 10% are bilateral, 10% are malignant and 10% are extra-adrenal. Extra-adrenal tumours are more likely to be malignant and can be found anywhere from the pelvis to the base of the skull. The most common extra-adrenal site is the organ of Zuckerkandl, which lies at the aortic bifurcation, but instances in the urinary bladder, the mediastinum and the neck have all been described.Malignancy is usually characterised by the presence of established metastases in liver, lymph nodes, bones and lungs.Phaeochromocytoma occurs in half of patients with MEN II and is also associated with other uncommon disorders such as von Hippel–Lindau disease (phaeochromocytoma, angioma, renal carcinoma), neurofibromatosis and tuberous sclerosis.

Clinical features. The typical symptoms are secondary to the effects of excessive α-adrenoreceptor stimulation. Sweating is very common in phaeochromocytoma and occurs in almost 90% of patients. Attacks are usually spontaneous, but sometimes they are precipitated by exercise, overeating, defecation or sexual intercourse. Patients describe them as consisting of:
1)paroxysmal headache
2)palpitations
3)profuse perspiration
4)sometimes precordial pain
5)a fearful feeling of impending death (angor animi).
These rarely last more than 15 minutes but tend to become more frequent over time.
Death from cardiovascular episodes and myocardial infarction has occurred during an attack. It must be remembered that invasive investigative procedures may precipitate an acute episode – an important consideration in efforts to localise the tumour.

Physical findings.Hypertension is the most common finding: over 50% of patients have persistent and sustained hypertension, but in the other 50% it is intermittent. Examination is otherwise normal but an abdominal tumour is occasionally found.

Investigation

Blood examination.The circulating plasma volume may be reduced by a combination of intense vasoconstriction and hypertension. A secondary rise in haematocrit is then evident.

Cardiac.There may be evidence of left ventricular hypertrophy on the ECG.

Hormonal.Twenty-four-hour measurement of the urinary metabolites of adrenaline and noradrenaline – nor- and metanephrines – is an effective screening test and is rarely normal in patients with symptomatic disease. If urinary metanephrines are high then plasma catecholamines are measured. Once an endocrine diagnosis has been made, abdominal CT will localise the tumour and whole-body radioisotope scan might be indicated for multiple tumours when metastases are suspected.

Management

Medical. Phaeochromocytomas are potentially lethal, and the major advance in their management has been the preoperative use of α- and β-adrenergic-blocking agents which have reduced operative mortality to 1%. Alpha-receptor blockade is mandatory before removal of the tumour and, when cardiac function is compromised, must be introduced gradually; phenoxybenzamine, a non-selective α-blocker, is the agent of choice. Phenoxybenzamine decreases the vasoconstriction, so that mild to severe postural hypotension develops, and the blood volume may need to be restored by increased oral fluid intake. The subjective symptoms are relieved, and the patient may appear to be drowsy as though sedated. Tachycardia may increase so that β-blockade becomes necessary but only after α-blockade is well established and the circulating blood volume has been restored.

Surgical. After the above preparation and precise localisation of the tumour, exploration and removal constitute the treatment of choice.

Multiple endocrine neoplasia

During embryonic life, the cells destined to form the endocrine organs arise from a single group in the neuroectoderm of the fetus. These cells, despite their widespread location in the body, have common histochemical features summarised by the acronym APUD (amine precursor uptake and decarboxylation). When such a tumour arises from one endocrine gland, it is likely that it is associated with tumour in other endocrine organs. The distribution of these tumours is not haphazard; certain associations are more common. Multiple endocrine neoplasia types I and II (MEN I, MEN II) are such autosomal dominant familial cancer syndromes.

MEN I

This is the more common type and involves the MEN I tumour suppressor gene on chromosome 11. The syndrome comprises neoplasia or hyperplasia of the parathyroids, the pancreatic islet cells, the pituitary and the thyroid, and rarely adrenal cortical tumours, carcinoids and lipomas. Not all tumours are present in any one patient.

The pattern of neoplasia in MEN I

Gland(s)	Abnormality	Frequency	Effect
Parathyroids	Hyperplasia	90%	Hyperparathyroidism
Pancreatic	Multiple	60–80%	Zollinger–Ellison syndrome or

Gland(s)	Abnormality	Frequency	Effect
islets	adenomas		insulinoma or glucagonoma or VIPoma
Pituitary chromophobes	Adenoma	50–70%	Prolactinoma or acromegaly
Thyroid	Adenoma	20%	Non-functional

Management

Parathyroid hyperplasia
There is a very high recurrence rate following subtotal parathyroidectomy, and some surgeons advocate total parathyroidectomy and lifelong maintenance with calcium and vitamin D supplementation.
Pancreatic islet cell hyperplasia
Pancreatic adenomas tend to be multiple and recur after partial pancreatectomy. Insulinomas are treated by either distal pancreatectomy or enucleation of tumours that are in the pancreatic head. Gastrinomas frequently present at a late stage and are successfully managed medically.
Pituitary adenomas
These can be treated medically (see above) or by excision if medical treatment is unsuccessful.
Thyroid adenoma
The main consideration is to exclude malignancy and for this reason the adenoma is best excised.
MEN II
This is an inherited cancer syndrome characterised by medullary thyroid cancer (MTC) and its precursor C-cell hyperplasia. There are three distinct subtypes:
1)familial – MTC is the sole feature
2)MEN IIa – MTC, phaeochromocytoma and parathyroid hyperplasia
3)MEN IIB – as with MEN IIA (although parathyroid involvement is rare) but with the additional developmental abnormalities of marfanoid features, mucosal neuromas and intestinal ganglioneuromas.
Genetics.The MEN II gene has been identified as the ret proto-oncogene on chromosome 10, which encodes the transcellular tyrosine kinase receptor. Mutations of the ret proto-oncogene have also been identified in patients with Hirschsprung's disease.
Management
 Adrenal tumours. These must be removed. Recurrence is likely and patients must be carefully followed up.
Parathyroid hyperplasia
The disease is usually mild, and surgical resection is limited to the enlarged gland(s).
Screening relatives
MEN II

The disease is usually mild, and surgical resection is limited to the enlarged gland(s).
Screening relatives

2. Questions for self-control:
frontal survey on basic terminology
 questions :
1. Aetiology of hyperaldosteronism.
2. Classification of hyperaldosteronism.
3. Pathogenesis. Presentation.
4. Investigations .
5. Cushing's syndrome. Pathogenesis.
6. Presentation. Investigations
7. Treatment.
8. Outcomes.

3. **Case scenarios:**

1. A 45-year-old woman presents with hypertension, development of facial hair, and a 7-cm suprarenal mass. Which of the following is the most likely diagnosis?

> a. Myelolipoma
> b. Cushing disease
> c. Adrenocortical carcinoma
> d. Pheochromocytoma
> e. Carcinoid

Explanation

The answer is **c.** (Brunicardi, pp 1397-1398.) The constellation of symptoms in this patient is typical of a functional adrenocortical tumor (androgens). Approximately 50% of adrenocortical tumors are functional and can secrete cortisol, androgens, estrogens, aldosterone, or multiple hormones. The single most important determinant of malignancy is the size of the tumor. Treatment consists of en bloc resection of the tumor and involved adjacent organs, such as the kidney or the tail of the pancreas. Symptoms related to hormone production can be minimized by complete resection despite the inability to cure advanced disease. Mitotane has been utilized as adjuvant therapy for unresectable or metastatic disease, but has not been proven to decrease mortality. Cushing disease refers to hypercortisolism due to a pituitary tumor and subsequent bilateral adrenal hyperplasia. Pheochromocytomas are characterized by hypertension and symptoms of excessive catecholamine production. Myelolipomas are benign adrenal lesions.

2. A 36-year-old woman presents with palpitations, anxiety, and hypertension. Workup reveals a pheochromocytoma. Which of the following is the best approach to optimizing the patient preoperatively?

> a. Fluid restriction 24 hours preoperatively to prevent intraoperative congestive heart failure
> b. Initiation of an **α**-blocker 24 hours prior to surgery
> c. Initiation of an **α**-blocker at 1 to 3 weeks prior to surgery
> d. Initiation of a **β**-blocker 1 to 3 weeks prior to surgery
> e. Escalating antihypertensive drug therapy with β-blockade followed by α-blockade starting at least 1 week prior to surgery

Explanation

. The answer is **c.** (Brunicardi, pp 1399-1400.) Patients with pheochromocytomas should be treated preoperatively with **α**-blockade using phenoxybenzamine 1 to 3 weeks before surgery. **β-**

Blockade may be necessary in addition to **α**-blockade for optimal blood pressure control, but should not be started in the absence of **α**-blockade because of the risk of cardiovascular collapse. With α-blockade, patients also require volume expansion.

3. A 34-year-old woman presents with hypertension, generalized weakness, and polyuria. Her electrolyte panel is significant for hypokalemia. Which of the following is the best initial test given her presentation and laboratory findings?

       a. Plasma renin activity and plasma aldosterone concentration
       b. Urine electrolytes
       c. Plasma cortisol level
       d. Overnight low-dose dexamethasone suppression test
       e. Twenty-four-hour urinary aldosterone level

Explanation

The answer is **a**. (Brunicardi, p 1392.) The biochemical diagnosis of hyperaldosteronism requires demonstration of elevated plasma aldosterone concentration (PAC) with suppressed plasma renin activity (PAR). A PAC: PAR ratio of 25 to 30:1 is strongly suggestive of the diagnosis. Hyperaldosteronism must be suspected in any hypertensive patient who presents with hypokalemia. Hypokalemia occurs spontaneously in up to 90% of patients with this disorder. Other individuals who should be evaluated for hyperaldosteronism include those with severe hypertension, hypertension refractory to medication, and young age at onset of hypertension. Plasma cortisol level and overnight low-dose dexamethasone suppression test are laboratory studies used in diagnosing Cushing syndrome. Neither urine electrolytes nor 24-hour urinary aldosterone level is beneficial in diagnosing hyperaldosteronism.

4. A 52-year-old woman presents with hypertension, obesity, and new skin striae. You are concerned about possible Cushing syndrome. Which of the following is the most common cause of Cushing syndrome?

       a. Adrenocortical hyperplasia
       b. Adrenocorticotropic hormone (ACTH)–producing pituitary tumor
       c. Primary adrenal neoplasms
       d. Ectopic adrenocorticotropic hormone (ACTH)–secreting carcinoid tumor
       e. Pharmacologic glucocorticoid use

Explanation

The answer is **e**. (Brunicardi, pp 1394-1395.) The most common cause of Cushing syndrome is iatrogenic, via administration of synthetic corticosteroids. Cushing syndrome refers to the clinical manifestations of glucocorticoid excess due to any cause (Cushing disease, administration of exogenous glucocorticoids, adrenocortical hyperplasia, adrenal adenoma, adrenal carcinoma, ectopic ACTH-secreting tumors) and includes truncal obesity, hypertension, hirsutism, moon facies, proximal muscle wasting, ecchymoses, skin striae, osteoporosis, diabetes mellitus, amenorrhea, growth retardation, and immunosuppression. Cushing disease is caused by hypersecretion of ACTH by the pituitary gland. This hypersecretion, in turn, is caused by either a pituitary adenoma (90% of cases) or diffuse pituitary corticotrope hyperplasia (10% of cases) because of hypersecretion of corticotropinreleasing hormone (CRH) by the hypothalamus.

5. The adrenal gland is composed of two distinct organs, the adrenal cortex and the adrenal medulla. Choose the WRONG statement about adrenal physiology.

     A. The cortex is divided into three functional zones: the outer glomerulosa, the intermediate fasciculata, and the inner reticularis.
     B. Zona glomerulosa produce mineralocorticoids

C. Zona fasciculata produce glucocorticoids
D. Zona reticularis produce sex steroids
E. Only mineralocorticoids are absolutely required for life

4. Individual tasks for applicants for higher education on the topics:

1. Treatment of adrenal adenoma.
2. Tactics in the treatment of patients with mixed adrenal tumors

5. List of recommended literature:

Basic

1. SABISTON: TEXTBOOK OF SURGERY: THE BIOLOGICAL BASIS OF MODERN SURGICAL PRACTICE, TWENTY FIRST EDITION Copyright © 2020
2. Gozie Offiah, Arnold Hill//RCSI Handbook of Clinical Surgery for Finals. 4th ed. 2020
3. Цигикало О. В. Clinical Anatomy and Operative Surgery=Клінічна анатомія і оперативна хірургія.. Підручник для ВМНЗ IV р.а.: Рекомендовано МОЗ: 2020/ 528 с.
4. Березницький Я. С. (за ред.) General Surgery=Загальна хірургія. — 2-ге вид. Підручник для ВМНЗ III—IV р.а.: Рекомендовано ДУ"Центр.метод.каб.з вищої мед.освіти МОЗ України": 2020/ 328 с.
5. Christian de Virgilio, Areg Grigorian//Surgery: A Case Based Clinical Review. 2nd Ed. 2020
6. JANE C. ROTHROCK// Alexander's Care of the Patient in Surgery. 16th Ed. 2019
7. RUTHERFORD'S VASCULAR SURGERY AND ENDOVASCULAR THERAPY, 9th ed. Volume 1, Volume 2. Copyright © 2019 by Elsevier, Inc.
8. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, 11th Ed. Copyright © 2019 by Elsevier Inc.
9. SEIDEL'S GUIDE TO PHYSICAL EXAMINATION: AN INTERPROFESSIONAL APPROACH. Copyright © 2019 by Elsevier, Inc.

Additional.

1. Hamilton Bailey's Physical Signs: Demonstrations of Physical Signs in Clinical Surgery, 19th Edition Paperback – Import, by John S.P Lumley (Author), Anil K. D'Cruz (Author), Jamal J. Hoballah (Author), Carol E.H. Scott-Connor (Author) 25 Feb 2016
2. Schwartz's Principles Of Surgery With DVD Hardcover – 2014 by F. Charles Brunicardi (Author), Dana K. Andersen (Author), Timothy R. Billiar (Author), David L. Dunn (Author), John G. Hunter (Author), & 2 More

6. Electronic informative resources

1. http://moz.gov.ua – Міністерство охорони здоров'я України
2. www.ama-assn.org – Американська медична асоціація / American Medical Association
3. www.who.int – Всесвітня організація охорони здоров'я
4. www.dec.gov.ua/mtd/home/ - Державний експертний центр МОЗ України
5. http://bma.org.uk – Британська медична асоціація
6. www.gmc-uk.org - General Medical Council (GMC)
7. www.bundesaerztekammer.de – Німецька медична асоціація
8. http://medforum.in.ua/partners- Асоціація хірургів України
9. http://endoscopy.com.ua/ - Асоціація ендоскопічних хірургів України
10. http://thoracic-surgery.com.ua/ - Асоціація торакальних хірургів України
11. https://youcontrol.com.ua/ - Асоціація судинних хірургів України