**Vinnytsya National Medical University n.a. Pirogov**

**Chair of endoscopic and cardiovascular surgery**

**« APPROVED »**

on the methodical conference of

department Chair of endoscopic and

cardiovascular surgery

Chief of the department

_____prof. V.V. Petrushenko

« 28 » 08 2017 y.

# METHODICAL RECOMMENDATIONS

FOR INDIVIDUAL WORK OF STUDENTS
AT THE TRAINING BEFORE LESSON

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|---|---|
| *Educational discipline* | Surgery |
| *Module* | 2 |
| Semantic module № | 4 |
| *Topic of the lesson* | Modern methods of diagnostics and low-invasive surgical treatment of heart disease. Acquired heart disease. Classification. Diagnosis. Indications for surgical treatment. Methods of surgical treatment. Complications. |
| *Year* | VI |
| *Faculty* | Medical |

Vinnytsa

1. **Actuality:**

Approximately 3 million myocardial infarctions are recorded annually in the United States, with an accompanying mortality rate of 10% - 15%. Acquired valvular disease is less frequent than coronary artery disease but still accounts for significant morbidity and mortality.

Acquired heart disease takes one of the leading places among the causes of death and disability in the population. Thus, the frequency of defect formation of one or more heart valves in chronic forms of rheumatism is 350-400 cases per 100 thousand population.
Rheumatic fever takes an exceptional place in the overall morbidity. Rheumatic valvular lesion is formed approximately in 35-40 % of patients. Struchkov A. suggests that rheumatic fever should be seen as a systemic connective tissue lesions with preferentially localized pathological process in the heart and development of fibrinoid degeneration, granulomatozis and sclerosis in the valve or tendon fibers.

In most cases mitral valve is affected, in second place - aortic and the third - the tricuspid. Pulmonary valve is usually not affected. It is known that the formation of any valvular heart deformiety leads to circulatory heart failure, and the physiological process of adaptation of the organism goes into several phases and stages (F.Z.Myeyerson).

According to A. Backulev there are such stages of of circulatory disorders:

I stage - the stage of complete compensation. The patient don't complain. Dyspnea does not occur even at exercises.

II stage - the stage of a relative circulatory insufficiency (dyspnea at physical exertion);

III stage - the initial stage of expressed circulatory insufficiency, there is congestion in the lungs and increased venous pressure, enlargement of the liver.

IVstage - the stage of expressed circulatory insufficiency (considerible enlargement of the liver, peripheral edemas, ascites);

V stage - the terminal dystrophic stage of a circulatory insufficiency.

2. **Concrete aims:**

- to recognize heart disease,
- to evaluate the severity of heart defect and its complications,
- to formulate a diagnosis in conformity with the accepted classification of disease,

- to form a scheme of medical and non-medical patient care according to diagnosis,
- to evaluate the efficiency of treatment, and if necessary to carry out its correction,
- to develop a set of measures of primary and secondary prevention of the disease and its complications,
- to evaluate the working capacity of the patient,
- to evaluate the prognosis for life and working ability,
- to maintain patients card

## 3. Basic knowledge, abilities, skills, that are necessary for a training

The student must to know appropriate sections in such subjects as:

| |
|---|
| Anatomy, topographic anatomy |
| Physiology |
| Pathological physiology |
| Microbiology |
| Radiology |
| Internal diseases |
| Pharmacology |
| General surgery |

## 4. Tasks for individual work at the training

### Classification of the pathophysiologic causes of congestive heart failure

*Mechanical abnormalitie:*
Resistance load
Hypertension
Aortic stenosis
Pulmonary stenosis
Flow load (primary and secondary)
Aortic regurgitation Mitral regurgitation Left-to-right shunts

*Resistance to inflow*
Constrictive pericarditis
Cardiac tamponade
Restrictive myocardial disease
Scleroderma
Amyloidosis
Mitral stenosis

*Primary myocardial (muscle) failure*

Cardiomyopathy
Hereditary
Alcoholic
Ischemic
Ischemia (coronary heart disease)
Inflammation
Rheumatic myocarditis
Viral myocarditis
Metabolic
Metal poisons
Myxedema

*Venous congestive states (high output failure)*

Renal failure with fluid retention
Intravenous fluid or blood overload
Chronic severe anemia
Arteriovenous fistula
Cirrhosis

*Arrhythmia*

Severe prolonged tachycardia
Metabolic failure (exhaustion)
Greatly decreased time for filling
Severe bradycardia
Asystole
Ventricular or atrial fibrillation

*Classification of patients with diseases of the heart (New York Heart Association, NYHA)*

**I** - Patients with cardiac disease but without resulting limitation of physical activity. In these patients, ordinary physical activity does not cause undue fatigue.

**II** - Patients with cardiac disease resulting in a slight limitation of physical activity. These patients are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.

**III** - Patients with cardiac disease resulting in limitation of physical activity. These patients are comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnea, or anginal pain.

**IV** - Patients with cardiac disease resulting in an inability to undertake any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome occur even at rest. If any physical activity is undertaken, discomfort increases.

*Signs and symptoms*

- Dyspnea is due to pulmonary congestion, which is the result of increased left atrial pressure.
- Peripheral edema may be the result of significant right-sided congestive heart failure.
- Chest pain may be caused by angina pectoris, myocardial infarction, pericarditis, aortic dissection, pulmonary infarction, or aortic stenosis.
- Palpitations may indicate a serious cardiac arrhythmia.
- Hemoptysis may be associated with mitral stenosis or pulmonary infarction.
- Syncope may result from mitral stenosis, aortic stenosis, or heart block.
- Fatigue is the result of decreased cardiac output.

*Physical examination*

. Blood pressure should be measured in both arms and legs.

. Peripheral pulses. Pulsus alternans is a sign of left ventricular failure. Pulsus parvus et tardus may be seen with aortic stenosis. A wide pulse pressure with a "water-hammer pulse" is seen with increased cardiac output or decreased peripheral vascular resistance, as in aortic insufficiency or patent ductus arteriosus.

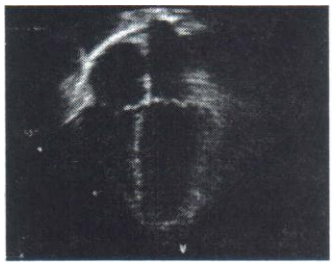
. Neck veins. Central venous pressure may be indirectly inferred from the height of the internal jugular vein filling.

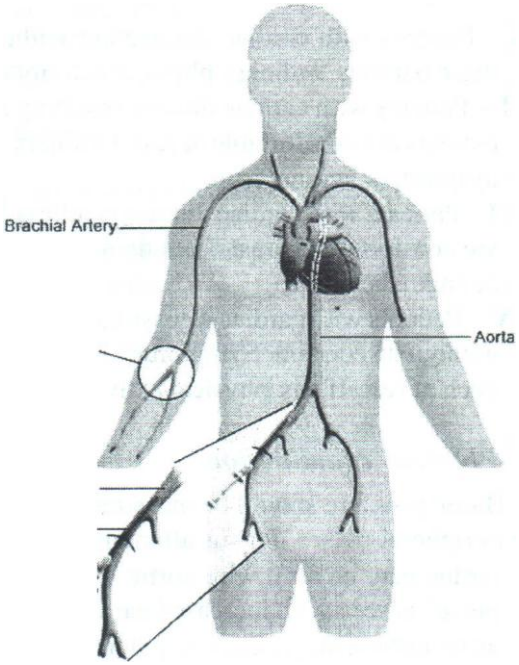
· Inspection and palpation of the precordium. Normally, the apical impulse is appreciated at the midclavicular line, fifth intercostal space. In left ventricular hypertrophy, the apical impulse is increased and displaced laterally. With right ventricular hypertrophy, a parasternal heave is appreciated. Thrills from valvular disease may be felt.

· Auscultation. The quality of heart tones, type of rhythm, murmurs, rales, and gallops are all important.

*Preoperative management*

- Laboratory studies should include such specific to cardiac pathology tests as creatine phosphokinase, lactate dehydrogenase, troponins, ect.
- Chest radiograph, electrocardiogram and echocardiogram are minimal necessary diagnostic tools. CT scan is also beneficial in terms of assessment of lungs, pulmonary arteries, heart and coronary arteries (CT angiography)
- Cardiac catheterization is the definitive preoperative study to measure a pressure in heart chambers and to perform coronary angiography.
- Pulmonary function studies are important in patients with known pulmonary disease.
- Psychological preparation of the patient is an important aspect and should include familiarizing the patient with the postoperative procedures in the ICU.
- Perioperative antibiotics play an important role in the prevention of sepsis.






Cardiac catheterization

*Extracorporeal circulation*

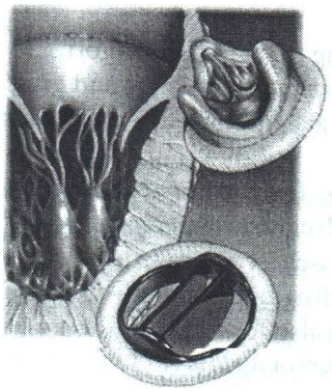
- The rationale for using extracorporeal circulation is to provide the operating team with a motionless heart and a bloodless field in which to work while simultaneously perfusing the different organ systems with oxygenated blood.
- Blood is drained from the right atrium, passed through an oxygenator and a heat exchanger, and pumped back to the aorta.
- Protection of the myocardium during the ischemia induced by the procedure is accomplished by hypothermia and cardioplegia.
- Pathophysiologic effects of extracorporeal circulation include: widespread total body inflammatory response with initiation of humoral amplification systems (coagulation cascade, fibrinolytic system, complement activation, kallikrein-kinin system); release of vasoactive substances (epinephrine, norepinephrine, histamine, bradykinin); retention of both sodium and free water, causing diffuse edema; trauma to blood elements, resulting in hemolysis of red cells and destruction of platelets; respiratory insufficiency, which is usually self-limited.

*Prosthetic valves*

- The three general categories are tissue valves, mechanical valves, and human allograft valves.
- Tissue valves consist primarily of porcine heterografts. These valves usually do not require long-term anticoagulation postoperatively; however, they lack long-term durability, with 10% - 30% failure rate at 10 years.

- Mechanical valves are durable for long periods of time, but they require permanent anticoagulation therapy.

- Human allograft (cryo-preserved) valves do not require anticoagulation and, at the present time, have demonstrated long-term durability. Wider use of this valve is anticipated, especially in the aortic position.



Artificial heart valves

## ***Aortic*** **stenosis**

*Overview*

Congenital bicuspid aortic valves occur in 1% - 2% of the population. They usually develop calcific changes by the fourth decade and symptoms by the sixth decade.

Acquired stenosis results from progressive degeneration and calcification of the valve leaflets.

Patients with a history of rheumatic fever rarely have isolated aortic stenosis but usually have a mixed lesion of stenosis and insufficiency.

Thickening and calcification of the leaflets result in a decreased cross-sectional area of the valve. Symptoms usually begin when the valve area is less than 1.0 cm2 (the normal aortic valve is 2.5 - 3.5 cm2). Critical aortic stenosis imposes a significant pressure load on the left ventricle, which increases left ventricular work, resulting in concentric left ventricular hypertrophy. Eventuany, myocardial decompensation occurs.

*Clinical features*

Classic symptoms are angina, syncope, and dyspnea.

Symptoms usually occur late in the course of the disease and represent myocardial decompensation.

Sudden death is a frequent occurrence in untreated patients at this stage.

Life expectancy averages 4 years after the onset of symptoms.

The classic systolic crescendo-decrescendo murmur is heard best in the second right intercostal space. Radiation of the murmur to the carotid arteries is common. An associated thrill is often appreciated.

A narrowed pulse pressure along with pulsus parvus et tardus is frequently found.

*Visualization data*

Chest x-ray usually shows a heart of normal size. Calcification of the aortic vail may be seen.

Electrocardiogram demonstrates left ventricular hypertrophy.

Echocardiography estimates the degree of stenosis, any associated insufficiency and quality ofleft ventricular function.

Cardiac catheterization is important to measure the pressure gradient across the aortic valve, to calculate its cross-sectional area, and to identify any associated mitral valve or coronary artery disease, which occurs in 25% of the patients.

*Treatment*

Surgical correction is recommended for patients with symptoms of syncope, angina, or dyspnea or with a peak systolic gradient across the aortic valve greater than 50 mmHg or with a valve area less than 0.7 $cm^2$.

Surgery consists of excision of the diseased valve and replacement with one of the prosthetic valves.

## Aortic insufficiency

*Overview*

Myxomatous degeneration, aortic dissection, Marfan's syndrome, bacterial endocarditis, rheumatic fever, and annuloaortic ectasia are common causes.

The underlying pathologic process may be a fibrosis and shortening of the valve leaflets (as occurs in rheumatic fever), a dilatation of the aortic annulus (as occurs in Marfan's syndrome), or myxomatous degeneration of the leaflets.

Aortic insufficiency imposes a significant volume load on the left ventricle in accordance with Starling's law of the heart. This extra load leads to early left ventricular dilatation. If uncorrected, this dilatation may lead to left ventricular failure with pulmonary congestion. Secondary mitral insufficiency may occur at this stage.

*Clinical features*

There is a greater variability in time between the onset of aortic insufficiency and the appearance of symptoms than occurs with aortic stenosis.

Early symptoms include palpitations secondary to ventricular arrhythmias, and dyspnea on exertion.

Later, severe congestive heart failure is seen. Death results from progressive cardiac failure.

The characteristic diastolic murmur is heard along the left sternal border. The duration of the murmur during diastole often correlates with the severity of the aortic insufficiency.

The murmur radiates to the left axilla.

The pulse pressure is often widened. Short, intense peripheral pulses ("water-hammer pulses") are characteristic.

Vi*sualization data*

· Chest x-ray shows left ventricwar dilatation.
· Echocardiography estimates the degree of insufficiency and quality of left ventricular function.
· Cardiac catheterization with aortography is used to quantitate the degree of aortic insufficiency, estimate the valve cross-sectional area, and determine the presence of associated.coronary artery stenosis.

*Treatment*

· Surgery is recommended whenever ventricular decompensation is demonstrated. Patients at this stage тау or тау not have significant symptoms.
· Valve replacement with a prosthesis is the indicated therapy.

## Mitral stenosis

*Overview*

· Although only 50% of patients report a history of rheumatic fever, this condition is thought to Ье the cause of mitral stenosis in virtually a11 cases. Congenital defects causing adult mitral stenosis are very rare.
· The time interval between the episode of rheumatic fever and the manifestation of mitral stenosis averages between 10 and 25 years.
· The underlying pathologic changes are fusion ofthe commissures and thickening ofthe leaflets with or without shortening of the chordae tendineae.
· The normal cross-sectional area ofthe mitral valve is 4-6 ст2. In mild mitral stenosis the area is reduced to 2 - 2.5 $cm^2$; in moderate stenosis, to 1.5 - 2 $cm^2$; and in severe stenosis, to 1 – 1,5 $cm^2$.
· Pathophysiologic changes induce: increased left atrial pressure, pulmonary hypertension, atrial fibrillation, decreased cardiac output, increased pulmonary vascular resistance.

*Clinical features*

· Dyspnea is the most significant symptom: it indicates pulmonary congestion secondary to increased left atrial preasure.
· Other manifestations include: paroxysmal nocturnal dyspnea and orthopnea, chronic cough and hemoptysis, pulmonary edema, systemic arterial embolization from a left atrial thrombus.

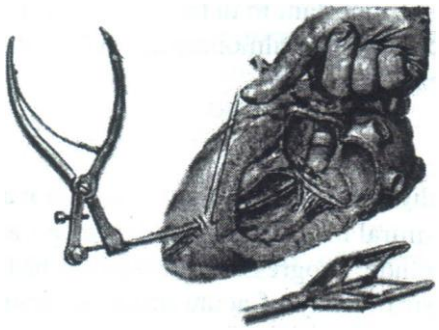
· Long-standing pulmonary hypertension may result in right ventricular failure and secondary
tricuspid regurgitation. · The typical patient is thin and cachectic. · Auscultation reveals the classic triad of an apical diastolic rumble, opening snap, and loud first heart sound

*Visualization data*

· Chest x-ray typically shows a prominent pulmonary vasculature in the upper lung fields.
The cardiac silhouette may be normal or may show a double density of the right heart
border. A lateral chest x-ray with a barium swallow may detect left atrial enlargement.
· Electrocardiogram may be normal or may show P-wave abnormalities, signs of right
ventricular hypertrophy, and right axis deviation. · Transthoracic and transesophageal echocardiography evaluate valvular and ventricular function. · Cardiac catheterization is used to calculate the mitral valve cross-sectional area, the
mitral valve end-diastole pressure gradient, pulmonary artery pressure, and any associated
valvular or coronary artery disease.

*Treatment*

· Surgery is recommended for all patients with symptomatic mitral stenosis. The choice of operative approach depends on the extent of these changes.
· Closed or open mitral valve commssurotomy should be attempted for a patient with simple fusion of the commissures and minimal calcification, although a later prosthetic valve replacement may be necessary.
· Mitral valve replacement is required for patients with severe disease of the chordae tendineae and papillary muscles.
. Permanent anticoagulant therapy is especially important for mechanical prosthetic valves in the mitral position because this therapy prevents thromboembolization.



Scheme of closed mitral valve commssurotomy

## Mitral insufficiency

*Overview*

. Mitral insufficiency is usually due to rheumatic fever. Other causes include: myxomatous degeneration, papillary muscle dysfunction or rupture secondary to coronary artery disease, and bacterial endocarditis.

· The pathogenesis in mitral insufficiency secondary to rheumatic fever is similar to that in mitral stenosis. Why insuficiency predominates in some patients and stenosis in others is not understood.

· Pathophysiologic changes include: increased left atrial pressure during systole; lateappearing pulmonary vascilar changes, including increased pulmonary vascular resistance; increased left ventricular stroke volume.

*Clinical features*

. Many years may elapse between the first evidence of mitral insufficiency and the development of symptoms.

. In general, symptoms occur late in the course of cardiac decompensation, and include dyspnea on exertion, fatigue, and palpitations.

· Physical examination reveals a holosystolic blowing murmur at the apex that radiates to the axilla, accompanied by an accentuated apical impulse. The duration of the murmur in systole correlates with the severity of the disease.

· Atrial fibrillation may be present.

*Visualization data*

· Chest x-ray demonstrates an enlarged left ventricle and atrium.

· Electrocardiogram shows evidence of left ventricular hypertrophy in 50% of cases.

. Echocardiography estimates the degree of stenosis, any associated insufficiency and quality of left ventricular function.

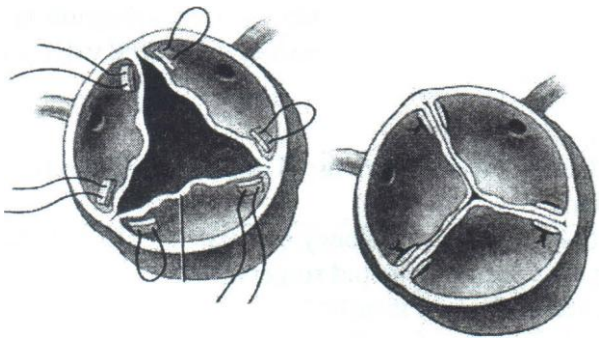
· Cardiac catheterization is important to determine left ventricular function, the degree of the mitral valve insufficiency, the pulmonary artery pressure, and any associated valvular or coronary artery disease.

*Treatment*

· Medical treatment with digitalis, diuretics, and vasodilator agents has a significant place in the treatment of stable mitral insufficiency and in preparation for surgery.

· Surgical indications include: progressive congestive heart failure; progressive cardiac enlargement; mitral insufficiency of acute onset, as from ruptured chordae tendineae; disease in more than one valve.

. When possible, mitral valve repair and annuloplasty are performed. The valve is replaced if necessary.



**Scheme ofvalvuloplasty**

## Tricuspid stenosis and insufficiency

*Overview*

. Organic tricuspid stenosis is almost always due to rheumatic fever and is most commonly found in association with mitral valve disease. Isolated tricuspid disease is rare.

. Functional tricuspid insufficiency is the result of right ventricular dilatation secondary to pulmonary hypertension and right ventricular failure. Functional insufficiency is more common than organic tricuspid valve disease.

· Tricuspid insufficiency is sometimes seen in the carcinoid syndrome, secondary to blunt trauma or secondary to bacterial endocarditis in drug addicts.

. The pathogenesis in tricuspid stenosis secondary to rheumatic fever is similar to that in mitral valve disease.

. Elevation of right atrial pressure secondary to tricuspid stenosis leads to peripheral edema, jugular venous distention, hepatomegaly, and ascites.

*Clinical features*

· Moderate isolated tricuspid insufficiency is usually well tolerated.

· When right -sided heart failure occurs, symptoms (edema, hepatomegaly, ascites) develop.

. Tricuspid insufficiency produces a systolic murmur at the lower end of the sternum.

· Tricuspid stenosis produces a diastolic murmur in the same region.

· A prominent jugular venous pulse may be observed.

. The liver may be pulsatile in tricuspid insufficiency.

*Visualization data*

. Chest x-ray shows enlargement of the right heart, which may also be reflected on the electrocardiogram.

. Echocardiography estimates the amount of tricuspid valve pathology.

. Echocardiography estimates the degree of stenosis or insufficiency and condition of heart chambers.

· Cardiac catheterization is the most accurate guide to diagnosing tricuspid disease. It should include evaluation of any associated aortic or mitral valve lesions.

*Treatment*

· Isolated tricuspid disease, especially tricuspid insufficiency, may be well tolerated without surgical intervention.
· In mild to moderate tricuspid insufficiency associated with mitral valve disease, opinion varies concerning the need for tricuspid surgery.
. In the case of extensive tricuspid insufficiency associated with mitral valve disease, the consensus is that either tricuspid repair or tricuspid valve replacement is appropriate.
· Tricuspid stenosis, when significant, is remedied by commissurotomy or valve replacement.
· Valve excision alone may be indicated in certain cases of tricuspid insufficiency secondary to endocarditis, especially in intravenous drug abusers.

**Pulmonic valve disease**

· Acquired lesions of the pulmonic valve are uncommon. The carcinoid syndrome, however, may produce pulmonic stenosis.
· Surgical repair or replacement of the valve is carried out when warranted by the degree of dysfunction.

**Multiple valvular disease**

. More than one valve may be involved in rheumatic fever, as indicated in the foregoing discussions.
· Abnormal physiologic responses to multivalvular disease may be additive but usually reflect the most severely affected valve.
. Treatment involves repair or replacement of all valves with significant dysfunction.

**REVIEW TESTS**

1. A 78-years-old previously healthy man is admitted to the ER with complaints of angina, dyspnea, and near syncope. Electrocardiogram is normal, and a loud systolic *murmur* is heard in the second right intercostal space with radiation to the carotids. What's the most appropriate tactics of managing the patient?

A. Myocardial infarction
B. Pericarditis
C. Mitral regurgitation
D. Aortic stenosis
E. Aortic insufficiency

2. A 72-years-old woman was admitted to ER with unstable angina. Cardiac catheterization reveals severe triple vessel coronary artery disease. What's the most appropriate tactics of managing the patient?

A. Coronary artery bypass surgery
B. Observation
C. Medical management (e.g., nitrates, ß-blockers)
D. Percutaneous coronary angioplasty
E. Administration of tissue plasminogen activator (e.g., streptokinase)

3. Cardiac tamponade is a surgical emergency and should be managed by immediate:

A. Surgical exploration
B. Pericardiocentesis
C. Infusion of fluid
D. Injection of NSAIDs

4. A 4-years-old boy is evaluated for a systolic murmur upon auscultation of the chest. Chest radiograph demonstrates cardiomegaly and rib notching. Physical examination reveals diminished femoral pulses. A 40 mm differential exists between upper and lower extremity blood pressures. Give your presumable diagnosis:

A. Patent ductus arteriosis
B. Coarctation of aorta
C. Atrial septal defect
D. Bilateral common femoral artery stenosis
E. Aortic stenosis

5. An isolated large atrial septal defect results in:

A. Right-to-left shunt
B. Left-to-right shunt
C. No shunt
D. Bidirectional shunting

6. All of the following lesions BUT ONE have a right-to-left shunt in the presence of normal pulmonary vascular resistance:

A. Tetralogy of Fallot
B. Ventricular septal defect
C. Tricuspid atresia
D. Pulmonic stenosis and atrial septal defect
E. Complete atrioventricular canal

7. A l-week-old severely cyanotic infant is most likely to have:

A. Aortic stenosis or partial anomalous pulmonary venous drainage with atrial septal defect
B. Transposition of the great vessels, tetralogy of Fallot, or truncus arteriosus
C. Coronary arteriovenous fistula or pulmonary stenosis

8. Tetralogy of Fallot includes all BUT ONE of the following lesions:

A. Ventricular septal defect
B. Pulmonic stenosis
C. Hypoplastic left ventricle
D. Overriding aorta
E. Right ventricular hypertrophy

9. All of these defects have a left-to-right shunt except:

A.Atrial septal defect
B. Patent ductus arteriosus
C. Tetralogy of Fallot
D. Ventricular septal defect

*Correct answers:* I - D, 2 - A, 3 - B, 4 - B, 5 - B, 6 - B, 7 - B, 8 - C, 9 - C.