Discussion of heart murmurs tends to be associated with specialist ward rounds in teaching hospitals, but a good understanding of this clinical sign provides valuable information.

**AUSCULTATING A HEART MURMUR**

**When does it occur?**
- Time the murmur in systole or diastole to the first heart sound and by palpating the upstroke of the carotid artery as systole.
- Is the murmur early, mild, late, holosystolic, or diastolic?

**How loud is it?**
- Grade I — very soft, only heard with special effort
- Grade II — soft, faint, but heard immediately
- Grade III — moderately loud
- Grade IV — so loud that a thrill can be felt
- Grade V — very loud, heard with only part of the stethoscope on the chest wall
- Grade VI — heard with the stethoscope removed from the chest wall.

**Where is it maximal?**
- Apex, left parasternal area, aortic area, pulmonary area.

**Where does it radiate to?**
- Neck, axilla, back.

**Categories of heart murmurs**
There are three broad categories of heart murmurs:
- systolic (murmur begins with S1 or after S1, ends at S2)
- diastolic (murmur begins after S2, ends before S1)
- continuous (murmur continues without interruption from systole through S2 into diastole. Typical in patent ductus arteriosus).

**Systolic heart murmurs**
Systolic murmurs are illustrated in Fig. 1 and are classified as:
- early systolic
- midsystolic
- late systolic
- holosystolic (pansystolic)

**Early systolic murmurs** occur in acute severe mitral regurgitation, tricuspid regurgitation (with normal right ventricular [RV] pressures) and ventricular septal defect (VSD).

**Midsystolic murmurs** occur in aortic sclerosis (often in the elderly), aortic stenosis and pulmonary valve stenosis, but can also be a feature of an innocent murmur (common in children, adolescents and young adults).
A more sinister cause is mitral regurgitation as a result of ischaemic heart disease.

Late systolic murmurs. The classic cause is mitral valve prolapse, also associated with one or more systolic clicks.

Holosystolic murmurs are classic murmurs of mitral regurgitation and high-pressure tricuspid regurgitation. A distinctive and diagnostically important feature of tricuspid regurgitation is that the murmur becomes louder with inspiration (Carvallo’s sign). The murmur of VSD becomes louder with inspiration as a result of ischaemic heart disease.

Mid-diastolic murmur. There is a clear interval after S2 before the murmur starts, the best example being mitral stenosis.

As a general rule it is necessary to refer a patient with a heart murmur if any of the features mentioned below are present. The more features present, the higher the risk. These features can be interpreted as being indicative of significant or pathological heart murmurs.

Symptoms
- Effort intolerance, reduced exercise capacity or dyspnoea more than grade I (New York Heart Association).
- Orthopnoea, or increase in number of pillows used at night, or paroxysmal nocturnal dyspnoea (PND).
- Oedema of the ankles.
- Chest pain (angina or angina-like).
- Syncope or pre-syncope could be an important symptom of a severe aortic valve lesion.

Clinical signs
- Blood pressure. Low blood pressure, a very narrow pulse pressure (systolic blood pressure minus diastolic blood pressure), or a very wide pulse pressure (> 50 mmHg) could help in the evaluation of heart murmurs.
- Pulse. If the pulse is irregular, consider atrial fibrillation.

• Elevated jugular venous pressure (JVP). This is a sign of heart failure. The patient should be supine at 45°. External jugular pressure can be elevated without right heart failure, but internal jugular pressure is more accurate as a predictor of heart failure. The venous column in the internal vein is very difficult to palpate, but the pulsations may readily be seen.

• Abdomino-jugular reflux sign. This is a useful sign to diagnose early heart failure. A positive sign is defined by an increase in JVP of greater than 3 cm, sustained for longer than 15 seconds. During the examination the patient is supine with the head elevated to 45°. The bladder of a sphygmomanometer is inflated, pressing the bulb 1 - 3 times, after which the bulb must be closed and the partially inflated bladder flat laid flat on the upper abdomen. With the open hand the examiner presses the bladder until the mercury column is at 20 mmHg and keeps it there for 15 seconds while watching the JVP. The patient should breathe normally and not hold his/her breath.

• Left parasternal heave. A left parasternal heave palpated with the ball of the hand implies RV hypertrophy, indicative of pulmonary hypertension — a complication of valvular disease, especially mitral valvular disease. Occasionally this left parasternal heave may be caused by a massively enlarged left atrium (especially in mitral valvular disease).

• Loud P2. A loud P2 is indicative of pulmonary hypertension.

• Cardiomegaly. The normal apex beat (most lateral, inferior pulsation on chest) should be in the 5th intercostal space (ICS) in the midclavicular line. If the apex beat is more inferolateral, cardiomegaly must be considered.

• High-risk signs on auscultation:
  - murmur of grade III or more
  - holosystolic murmur
  - murmur radiating to the neck (carotids), axilla or back
  - diastolic murmurs
  - gallop rhythm.
• Signs suggestive of infective endocarditis. There are numerous minor signs of infective endocarditis, e.g. anaemia, fever, splinter haemorrhages, haematoma. A positive blood culture is a major sign of infective endocarditis.
• Signs suggestive of embolism. Stroke in a young person, haematuria, or absent pulses in a patient with a heart murmur are indicative of embolism.
• ECG signs:
  • no P-waves, with irregular R-R intervals (atrial fibrillation)
  • LV or RV hypertrophy
  • ST-segment changes or T-wave inversions.
• Chest X-ray signs:
  • cardiomegaly (cardiac size more than 50% of intrathoracic diameter)
  • valvular calcifications (best seen on lateral radiograph)
  • any sign of pulmonary oedema (Kerley’s B lines, interstitial lines, etc.)
  • any sign of pulmonary hypertension (large pulmonary cone or large pulmonary artery).

BEDSIDE MANOEUVRES

The accuracy of the cardiac physical examination for valvular lesions is enhanced by the use of simple bedside manoeuvres that temporarily alter cardiac haemodynamics.

Valsalva manoeuvre

Remove the bulb of a sphygmomanometer and place the rubber tubing leading to it into the patient’s mouth. The patient blows into this tube until the mercury column is 40 mmHg and continues blowing to keep the mercury at 40 mmHg for 15 seconds while the doctor listens to the heart. The Valsalva manoeuvre will reduce the intensity of all left-sided murmurs except in the case of:
  • hypertrophic obstructive cardiomyopathy
  • mitral valve prolapse.
Caution should be exercised when performing the Valsalva manoeuvre in patients with ischaemic heart disease.

Isometric exercise

This is performed with a sustained isometric handgrip, using a sphygmomanometer. Roll up the bladder and fixate it. Close the tubing system at the bulb, inflate it with 1 - 3 pumps and then close the bulb. The patient squeezes the rolled-up bladder with the non-dominant hand maximally, while the doctor reads the level of the mercury. The patient repeats the manoeuvre, but the pressure is kept constantly at 50 - 70% of the maximum, while the doctor listens to the heart. During this manoeuvre, there will be a depression of the vagus tone and an increase in sympathetic tone, which will cause tachycardia and elevate blood pressure and cardiac output. All left-sided regurgitant murmurs may decrease. If there is underlying left ventricular (LV) dysfunction in ischaemic heart disease, an S4 or S2 may be elicited by the manoeuvre. Mitral regurgitation may be elicited if there is an underlying ischaemic papillary muscle.

Respiration

Inspiration increases venous return, which will increase RV volume. Therefore, right-sided S3 or S4 and the systolic murmur of tricuspid regurgitation will increase with inspiration. This also holds true for the murmur of tricuspid stenosis, pulmonary stenosis and pulmonary regurgitation.

Postural changes

The simplest is to let the patient move from a standing to a squatting position while the examiner listens to the heart (the latter usually stays seated while listening to the same area). If the patient is physically unable to do this, he/she remains supine while the examiner bends the patient’s knees passively towards the abdomen. Standing produces the same physiological results as the Valsalva manoeuvre. With squatting the effects are the opposite. Squatting causes a right-sided murmur and aortic and mitral regurgitation to increase, but mitral prolapse and hypertrophic obstructive cardiomyopathy (HOCM) to decrease.

GENERIC COMPLICATIONS OF VALVULAR DISEASE

• Pulmonary hypertension.
  Patients with mitral valve diseases [mitral stenosis, mitral regurgitation] are particularly prone to develop an increasing severity of pulmonary hypertension.
• Cardiac failure. Valvular heart disease is an important cause of heart failure, which will lead to right heart failure.
• Infective endocarditis. A heart murmur, especially of valvular origin, is a high-risk situation as a result of which a patient can easily, during bacteraemias, develop infective endocarditis.
• Dysrhythmias. A pulse, irregular in rhythm and volume, is indicative of atrial fibrillation and is a common complication of valvular disease, especially mitral valve disease (stenosis and regurgitation).
• Embolism, usually presenting as stroke or peripheral arterial disease.

MANAGEMENT OF VALVULAR DISEASE

When a cardiac murmur is heard during routine auscultation of the heart the following questions should be asked:
• Is it systolic or diastolic?
• Are there any features of a high-risk patient?
• Could it be benign?
• Are there any complications?

If it is not a benign murmur, a diagnosis is necessary. For this purpose referral to a specialist physician or cardiologist may be necessary.

Preventive measures

Primary prevention

Prevention of primary attacks of rheumatic fever depends on prompt recognition and adequate treatment of group A streptococcal (GAS) tonsillopharyngitis. Eradication of GAS from the throat is essential with the administration of appropriate antibiotics, penicillin being the antibiotic of choice. Although it has a narrow spectrum of activity, it has a longstanding proven record and is the least expensive.
Dosage regimens are the following:

**Benzathine penicillin G**
- 600,000 units once intramuscularly for patients < 27 kg.
- 1.2 million units once intramuscularly for patients > 27 kg.

**Penicillin V**
- Children: 250 mg 3 times/day per mouth for 10 days.
- Adults: 500 mg 3 times/day per mouth for 10 days.

**Penicillin allergy**
- Erythromycin 40 mg/kg/day (2 - 4 times/day) per mouth for 10 days.

The newer macrolides may be used, or an oral cephalosporin for 10 days. The following antibiotics should not be used to treat GAS: tetracyclines, trimethoprin-sulfamethozazole, chloramphenicol.

**Secondary prevention**
After the acute treatment, secondary prophylaxis should be considered. Patients who have had a previous attack of rheumatic fever or who have rheumatic valvular disease are at high risk of recurrent attacks of endocarditis. These patients should therefore receive continuous antimicrobial prophylaxis. Medical practitioners should consider each individual situation when determining the duration of continuous prophylaxis. Rheumatic fever with proven carditis and residual valvular disease should receive prophylaxis for at least 10 years after the last attack of rheumatic fever, and perhaps until the age of 40 years, sometimes lifelong. Patients with rheumatic fever with carditis but without valvular disease, should receive prophylaxis for 10 years or well into adulthood (whichever is longer). Patients with rheumatic fever without carditis only need prophylaxis for 5 years or until the age of 21 years, whichever is longer. Successful prophylaxis depends primarily on patient compliance and adherence to the regimens.

Dosage regimens are the following:
- penicillin V 250 mg twice a day per mouth
- for penicillin allergy — erythromycin 250 mg twice a day per mouth.

**Prevention of infective endocarditis**
Patients with valvular heart disease (rheumatic or degenerative in origin) require additional short-term antibiotic prophylaxis before certain dental or surgical procedures to prevent the possible development of infective endocarditis. Patients with a history of a previous episode of infective endocarditis are at particularly high risk of again developing endocarditis. Those with prosthetic valves are also in a high-risk category. It is very important to realise that antibiotic regimens in use to prevent recurrences of rheumatic fever are inadequate to prevent infective endocarditis. Whenever a patient with a valvular disease needs to undergo a dental, surgical or invasive diagnostic procedure the general practitioner should seek advice from a specialist with regard to the best prophylaxis for infective endocarditis.

**Medical management**
In the vast majority of patients with valvular disease, the decision whether to replace the valve will be taken by the cardiologist/specialist physician. There are, however, patients for whom medical therapy is the only option. There are many frail older patients with symptomatic valvular disease (mainly degenerative) in whom the risks of surgical intervention are prohibitive and for them medical treatment is the only option. It may also be possible to influence the natural history of valvular disease using medical management. Many of the landmark trials on the treatment of heart failure included patients with valvular disease and heart failure.

**What can be done?**

**Symptom control.** Diuretics can relieve the symptoms of congestion but must be used cautiously and in low doses so as not to influence the haemodynamics of valvular disease negatively. Beta-blockers, cautiously used in low doses, can by slowing the heart rate improve ventricular filling and often relieve symptoms, especially during exercise.

**Atrial fibrillation** is a common complication of many valvular diseases; it will lead to deterioration in cardiac function and it can cause embolism, especially stroke. Early anticoagulation with warfarin to a target International Normalised Ratio (INR) of 2 - 3.1 is nearly always warranted. To control the rate, digoxin, beta-blockers or rate-limiting calcium channel blockers (especially verapamil) are effective. A resting pulse rate of below 90/min and absence of any pulse deficit should be seen as adequate rate control.

**IN A NUTSHELL**

The use of ACE-inhibitors, as in the original heart failure landmark trials, should always be considered and may preserve ventricular function.