TABLE 7-2 CHARACTERISTIC PHYSICAL, ELECTROCARDIOGRAPHIC, AND CHEST RADIOGRAPHIC FINDINGS IN CHRONIC ACQUIRED VALVULAR HEART DISEASE

CAUSE	PHYSICAL FINDINGS*	ELECTROCARDIOGRAM	RADIOGRAPH
Aortic stenosis	Pulsus parvus et tardus (may be absent in older patients or in patients with associated aortic regurgitation); carotid *shudder* (coarse thrill) Ejection murmur radiates to base of neck; peaks late in systole if stenosis is severe Sustained but not significantly displaced LV impulse A_2 decreased, S_2 single or paradoxically split S_4 gallop, often palpable	LV hypertrophy Left bundle branch block is also common Rare heart block from calcific involvement of conduction system	LV prominence without dilation Post-stenotic aortic root dilation Aortic valve calcification
Aortic regurgitation	Increased pulse pressure Bifid carotid pulses Rapid pulse upstroke and collapse LV impulse hyperdynamic and displaced laterally Diastolic decrescendo murmur; duration related to severity Systolic flow murmur S_{3G} common	LV hypertrophy, often with narrow deep Q waves	LV and aortic dilation
Mitral stenosis	Loud S_1 OS S_2-OS interval inversely related to stenosis severity S_1 not loud and OS absent if valve heavily calcified Signs of pulmonary arterial hypertension	LA abnormality Atrial fibrillation common RV hypertrophy pattern may develop if associated pulmonary arterial hypertension is present	Large LA: double-density sign, posterior displacement of esophagus, elevation of left mainstem bronchus Straightening of left heart border as a result of enlarged left appendage Small or normal-sized LV Large pulmonary artery Pulmonary venous congestion
Mitral regurgitation	Hyperdynamic LV impulse S_3 Widely split S_2 may occur Holosystolic apical murmur radiating to axilla (murmur may be atypical with acute mitral regurgitation, papillary muscle dysfunction, or mitral valve prolapse)	LA abnormality LV hypertrophy Atrial fibrillation	Enlarged LA and LV Pulmonary venous congestion
Mitral valve prolapse	One or more systolic clicks, often midsystolic, followed by late systolic murmur Auscultatory findings dynamic Symptoms may include tall thin habitus, pectus excavatum, straight back syndrome	Often normal Occasionally ST-segment depression or T wave changes or both in inferior leads	Depends on degree of valve regurgitation and presence or absence of those abnormalities
Tricuspid stenosis	Jugular venous distention with prominent *a* wave if sinus rhythm Tricuspid OS and diastolic rumble at left sternal border; may be overshadowed by concomitant mitral stenosis Tricuspid OS and rumble increased during inspiration	Right atrial abnormality Atrial fibrillation common	Large RA
Tricuspid regurgitation	Jugular venous distention with large regurgitant (systolic) wave Systolic murmur at left sternal border, increased with inspiration Diastolic flow rumble RV S_3 increased with inspiration Hepatomegaly with systolic pulsation	RA abnormality; findings are often related to the cause of the tricuspid regurgitation	RA and RV enlarged; findings are often related to the cause of the tricuspid regurgitation

LA, Left atrium; LV, left ventricle; OS, opening snap; RA, right atrium; RV, right ventricle; S_{3G}, S_3 gallop.
*Findings are influenced by the severity and chronicity of the valve disorder.

common causes of acute severe aortic valve regurgitation. Congenitally abnormal (most commonly bicuspid) aortic valve leaflets often lead to chronic severe aortic regurgitation (see Table 7-1).

Clinical Presentation

Acute Severe Regurgitation

Patients may have symptoms related to their underlying pathology, such as fever and malaise from endocarditis or severe chest pain due to aortic dissection. In addition, they are likely to suffer progressive signs and symptoms of cardiogenic shock, including tachycardia and hypotension caused by severely impaired cardiac output and fulminant pulmonary edema due to markedly elevated filling pressures. In general, the more rapidly the severity of regurgitation evolves, the less well it is hemodynamically tolerated.

Chronic Severe Aortic Regurgitation

When severe regurgitation develops slowly over months to years, compensatory mechanisms such as LV remodeling lead to chamber dilatation and increased compliance, permitting even large regurgitant volumes to be well tolerated. At onset, the symptoms are typically exertional, including dyspnea and fatigue. Orthopnea and occasionally chest pain can develop in the absence of epicardial coronary disease.