TABLE 19-1 TYPICAL CLINICAL FEATURES OF MAJOR CAUSES OF ACUTE CHEST DISCOMFORT

System	Condition	Onset/Duration	Quality	Location	Associated Features
Cardiopulmonary					
Cardiac	Myocardial ischemia	*Stable angina:* Precipitated by exertion, cold, or stress; 2–10 min *Unstable angina:* Increasing pattern or at rest *Myocardial infarction:* Usually >30 min	Pressure, tightness, squeezing, heaviness, burning	Retrosternal; often radiation to neck, jaw, shoulders, or arms; sometimes epigastric	S_4 gallop or mitral regurgitation murmur (rare) during pain; S_3 or rales if severe ischemia or complication of myocardial infarction
	Pericarditis	Variable; hours to days; may be episodic	Pleuritic, sharp	Retrosternal or toward cardiac apex; may radiate to left shoulder	May be relieved by sitting up and leaning forward; pericardial friction rub
Vascular	Acute aortic syndrome	Sudden onset of unrelenting pain	Tearing or ripping; knifelike	Anterior chest, often radiating to back, between shoulder blades	Associated with hypertension and/or underlying connective tissue disorder; murmur of aortic insufficiency; loss of peripheral pulses
	Pulmonary embolism	Sudden onset	Pleuritic; may manifest as heaviness with massive pulmonary embolism	Often lateral, on the side of the embolism	Dyspnea, tachypnea, tachycardia, and hypotension
	Pulmonary hypertension	Variable; often exertional	Pressure	Substernal	Dyspnea, signs of increased venous pressure
Pulmonary	Pneumonia or pleuritis	Variable	Pleuritic	Unilateral, often localized	Dyspnea, cough, fever, rales, occasional rub
	Spontaneous pneumothorax	Sudden onset	Pleuritic	Lateral to side of pneumothorax	Dyspnea, decreased breath sounds on side of pneumothorax
Non-cardiopulmonary					
Gastrointenstinal	Esophageal reflux	10–60 min	Burning	Substernal, epigastric	Worsened by postprandial recumbency; relieved by antacids
	Esophageal spasm	2–30 min	Pressure, tightness, burning	Retrosternal	Can closely mimic angina
	Peptic ulcer	Prolonged; 60–90 min after meals	Burning	Epigastric, substernal	Relieved with food or antacids
	Gallbladder disease	Prolonged	Aching or colicky	Epigastric, right upper quadrant; sometimes to the back	May follow meal
Neuromuscular	Costochondritis	Variable	Aching	Sternal	Sometimes swollen, tender, warm over joint; may be reproduced by localized pressure on examination
	Cervical disk disease	Variable; may be sudden	Aching; may include numbness	Arms and shoulders	May be exacerbated by movement of neck
	Trauma or strain	Usually constant	Aching	Localized to area of strain	Reproduced by movement or palpation
	Herpes zoster	Usually prolonged	Sharp or burning	Dermatomal distribution	Vesicular rash in area of discomfort
Psychological	Emotional and psychiatric conditions	Variable; may be fleeting or prolonged	Variable; often manifests as tightness and dyspnea with feeling of panic or doom	Variable; may be retrosternal	Situational factors may precipitate symptoms; history of panic attacks, depression

blood flow and coronary arterial oxygen content. When myocardial ischemia is sufficiently severe and prolonged in duration (as little as 20 min), irreversible cellular injury occurs, resulting in MI.

Ischemic heart disease is most commonly caused by atheromatous plaque that obstructs one or more of the epicardial coronary arteries. Stable ischemic heart disease **(Chap. 293)** usually results from the gradual atherosclerotic narrowing of the coronary arteries. *Stable angina* is characterized by ischemic episodes that are typically precipitated by a superimposed increase in oxygen demand during physical exertion and relieved upon resting. Ischemic heart disease becomes unstable most commonly when rupture or erosion of one or more atherosclerotic lesions triggers coronary thrombosis **(Chap. 291e)**. Unstable ischemic heart disease is classified clinically by the presence or absence of detectable myocardial injury and the presence or absence of ST-segment elevation on the patient's electrocardiogram (ECG). When acute coronary atherothrombosis occurs, the intracoronary thrombus may be partially obstructive, generally leading to myocardial ischemia in the absence of ST-segment elevation. Marked by ischemic symptoms at rest, with minimal activity, or in an accelerating pattern, unstable ischemic heart disease is classified as *unstable angina* when there is no detectable myocardial injury and as *non–ST elevation MI* (NSTEMI) when there is evidence of myocardial necrosis **(Chap. 294)**. When the coronary thrombus is acutely and completely occlusive, transmural myocardial ischemia usually ensues, with ST-segment elevation on the ECG and myocardial necrosis leading to a diagnosis of *ST elevation MI* (STEMI, see **Chap. 295**).

Clinicians should be aware that unstable ischemic symptoms may also occur predominantly because of increased myocardial oxygen demand (e.g., during intense psychological stress or fever) or because of decreased oxygen delivery due to anemia, hypoxia, or hypotension. However, the term *acute coronary syndrome*, which encompasses unstable angina, NSTEMI, and STEMI, is in general reserved for ischemia precipitated by acute coronary atherothrombosis. In order to guide therapeutic strategies, a standardized system for classification of MI has been expanded to discriminate MI resulting from acute coronary thrombosis (type 1) from MI occurring secondary to other