duration anticoagulation, even if the initial VTE was provoked by trauma or surgery.

Clinical Risk Factors Common comorbidities include cancer, obesity, cigarette smoking, systemic arterial hypertension, chronic obstructive pulmonary disease, chronic kidney disease, long-haul air travel, air pollution, estrogen-containing contraceptives, pregnancy, postmenopausal hormone replacement, surgery, and trauma. Sedentary lifestyle is increasingly prevalent. A Japanese study found that each 2 h per day increment of television watching is associated with a 40% increased likelihood of fatal PE.

Activated Platelets Virchow's triad of venous stasis, hypercoagulability, and endothelial injury leads to recruitment of activated platelets, which release microparticles. These microparticles contain proinflammatory mediators that bind neutrophils, stimulating them to release their nuclear material and form web-like extracellular networks called neutrophil extracellular traps. These prothrombotic networks contain histones that stimulate platelet aggregation and promote platelet-dependent thrombin generation. Venous thrombi form and flourish in an environment of stasis, low oxygen tension, and upregulation of proinflammatory genes.

Interaction between Venous Thromboembolism and Atherothrombosis Carotid artery plaque doubles the risk of VTE. This observation led to discovery of the broad interaction among VTE, acute coronary syndrome, and acute stroke **(Fig. 279-4)**. These three conditions share similar risk factors and similar pathophysiology: inflammation, hypercoagulability, and endothelial injury. Patients who suffer VTE are more than twice as likely to have a future myocardial infarction or stroke. Conversely, patients with myocardial infarction or stroke are more than twice as likely to suffer a future VTE.