Introduction Cardiac output, expressed in liters/minute, is the amount of blood the heart pumps in 1 minute.This is observed in isolated myocardium, but not in living organisms, where the simultaneous decrease in vascular tone (especially with the dihydropyridine derivatives) helps to maintain and even increase cardiac output.Although an excessive increase in the end–diastolic volume may increase myocardial oxygen requirements, this intervention is associated with relatively limited consequences, as compared with catecholamines Effects of calcium entry blockers on cardiac output As calcium is essential to myocardial contraction, calcium entry blockers are expected to decrease the force of contraction of the myocardium.To try to clarify the individual roles and the combined roles of these four factors in generating cardiac output, and hence to facilitate our understanding of the effects of disease processes and therapies on cardiac output, I use a simple analogy that equates cardiac output (that is, the amount of blood pumped by the heart over a period of time) with the speed of a bicycle at a particular time point .Potentially harmful effects of vasopressor agents In contrast to the beneficial effects of vasodilator drugs, the administration of strong vasopressors may decrease cardiac output by increasing afterload, even in individuals with normal cardiac function.Beneficial effects of vasodilating substances Arterial vasodilator therapy results in significant improvements in cardiac output in patients with heart failure by reducing afterload (cycling downhill).Although most clinicians should/will be able to recite the four determinants of cardiac output – heart rate, contractility, preload, and afterload – understanding of the applicability and practical relevance of each of these four components is all too often less well ingrained.The pathophysiology of sepsis involves the release of cytokines, some of which are associated with abnormal calcium handling by the cardiac myocytes [2], leading to reduced myocardial contraction.How to measure cardiac output The amount of cardiac output can be determined through a number of medical procedures, including: o Pulmonary artery catheterization, a procedure in which a catheter is inserted into a blood vessel through a central vein, such as (the femoral vein, the jugular vein in the neck, or others) to reach the right side of the heart and then advance to the pulmonary artery.Some clinical applications Myocardial depression of sepsis Sepsis is usually associated with a normal or high cardiac output; yet many studies have shown that myocardial depression can occur even early in the course of sepsis [1], so why is cardiac output not reduced?As initially demonstrated by Otto Frank and Ernest Starling, an intrinsic property of myocardial cells is that the force of their contraction depends on the length to which they are stretched: the greater the stretch (within certain limits), the greater the force of contraction.This diagnostic procedure can be used to assess the pressure required to fill the chambers of the heart on the right side, estimate cardiac output, study the condition of the heart valves, and determine the amount of resistance of the blood vessels.Effects of inodilating substances Some drugs, such as phosphodiesterase inhibitors (milrinone, enoximone) and levosimendan, exert some inotropic effects in addition to vasodilating effects.Too little pedal power, or impaired contractility, will reduce cardiac output; however, too much effort will result in fatigue, sometimes leading to a complete collapse, with the need to slow down substantially or even to stop.Because of the simultaneous tachycardia and reduced vascular tone, however, afterload is reduced – and cardiac output can therefore be maintained or even increased.Interestingly, this therapeutic approach has been more successful than inotropic stimulation, and has been shown to reduce mortality

rates in this patient population [6,7].