الفسلجة
مرحلة ثانية

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Cardoivascular System
CARDIAC OUTPUT

Myocardial Contractility

The contractility of the myocardium exerts a major influence on stroke volume.

1. the sympathetic nerves stimulation to the heart, the whole length–tension curve shifts upward and to the left. The positive inotropic effect of norepinephrine liberated at the nerve endings is augmented by circulating norepinephrine, and epinephrine has a similar effect.
2. The parasympathetic nerve stimulation, there is a negative inotropic effect on both atrial and ventricular muscle.

3. Changes in cardiac rate and rhythm

affect myocardial contractility (known as the force–frequency relation. **Ventricular extrasystoles** condition the myocardium in such a way that the next succeeding contraction is stronger than the preceding normal contraction. This **postextrasystolic potentiation** is independent of ventricular filling, since it occurs in isolated cardiac muscle and is due to increased availability of intracellular Ca^{2+}. A sustained increment in contractility can be produced therapeutically by delivering paired electrical stimuli to the heart in such a way that the second stimulus is delivered shortly after the refractory period of the first.

3. Catecholamines exert their positive inotropic effect via an action on cardiac β1-adrenergic receptors, with resultant activation of adenylyl cyclase enzyme and increased intracellular (cAMP).

4. Xanthines such as caffeine and theophylline that inhibit the breakdown of cAMP are predictably positively inotropic.

5. Digitalis and related drugs are positively inotropic, alternatively, due to their inhibitory effect on the Na+–K+ ATPase in the myocardium; where increase intracellular Na+ increases, in turn, the availability of Ca+2 in the cell with its key role in cardiac contraction.

6. Hypercapnia, hypoxia, acidosis, and drugs such as quinidine, procainamide, and barbiturates depress myocardial contractility (negative inotropic effect).

7. (intrinsic depression), as in heart failure reduced myocardiac contractility (negative inotropic effect). The causes of this depression may be due to down-regulation of β-adrenergic receptors and impaired Ca+2 liberation from the sarcoplasmic reticulum.

It could be considered an adaptation to reduce energy expenditure and avoiding cell death in acute heart failure.

8. Loss of myocardium, as in MI, reduces myocardiac contractility.

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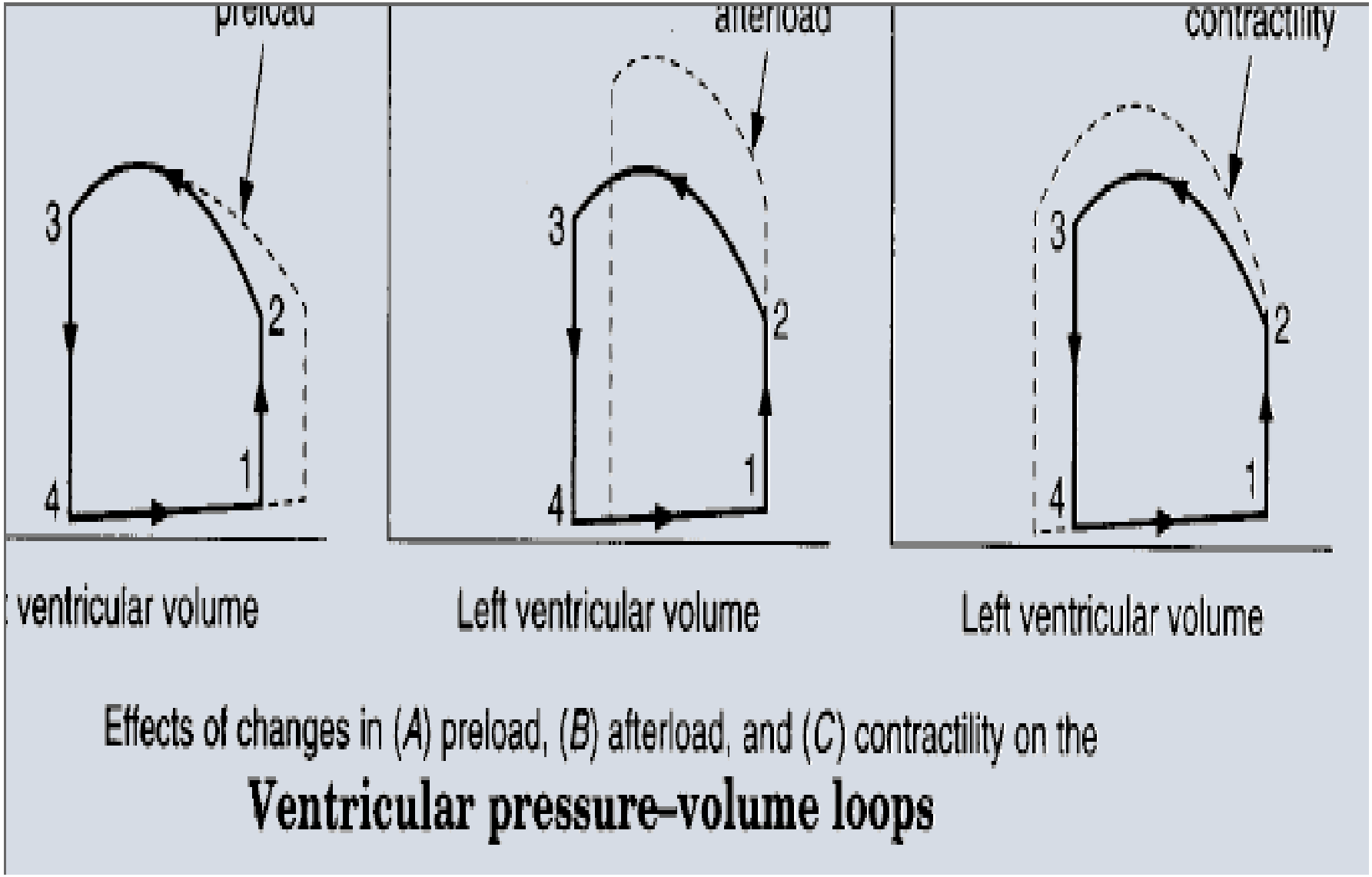


Effects of changes in (*A*) preload, (*B*) afterload, and (*C*) contractility on the

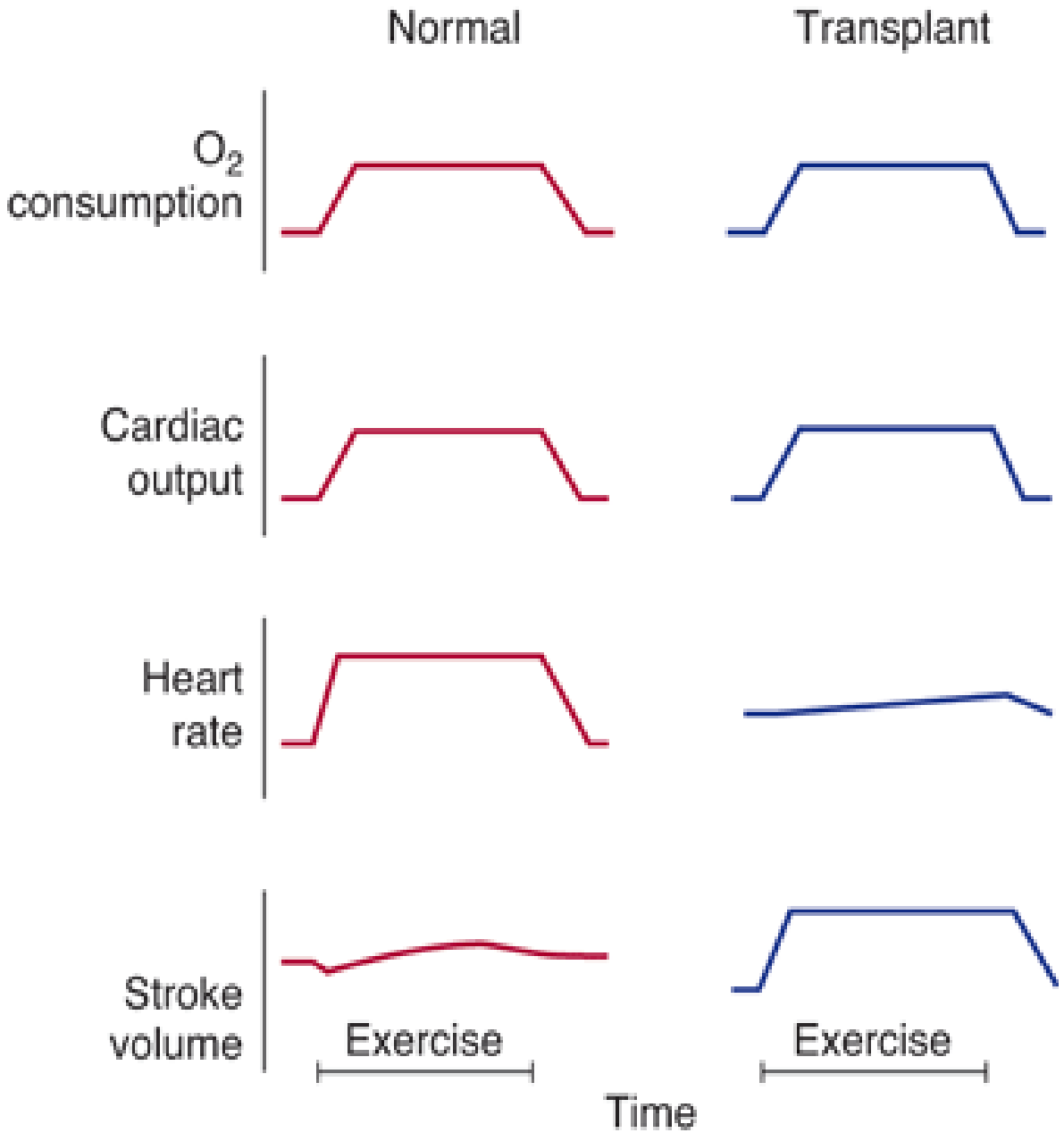
Ventricular pressure–volume loops

Integrated Control of Cardiac Output

The mechanisms listed above operate in an integrated way to maintain cardiac output.

During muscular exercise, there is increased sympathetic discharge, so that myocardial contractility is increased and the heart rate rises. The increase in heart rate is particularly prominent in normal individuals, and there is only a modest increase in stroke volume.

However, patients with transplanted hearts are able to increase their cardiac output during exercise in the absence of cardiac innervation through the operation of the Frank–Starling mechanism. Circulating catecholamines also contribute. If venous return increases and there is no change in sympathetic tone, venous pressure rises, diastolic inflow is greater, ventricular end-diastolic pressure increases, and the heart muscle contracts more forcefully. During muscular exercise, venous return is increased by the pumping action of the muscles and the increase in respiration. In addition, because of vasodilation in the contracting muscles, peripheral resistance and, consequently, afterload are decreased. The end result in both normal and transplanted hearts is thus a prompt and marked increase in cardiac output.


Normal
Transplant
O_2 consumption
Cardiac output
Heart rate
Stroke volume
Exercise
Exercise
Time