SNS COLLEGE OF TECHNOLOGY

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Department of Biomedical Engineering

Course Name: Biocontrol System

II Year : IV Semester

Unit IV –Modeling of Biological systems

Topic : Interaction of Pulmonary and Cardiovascular models

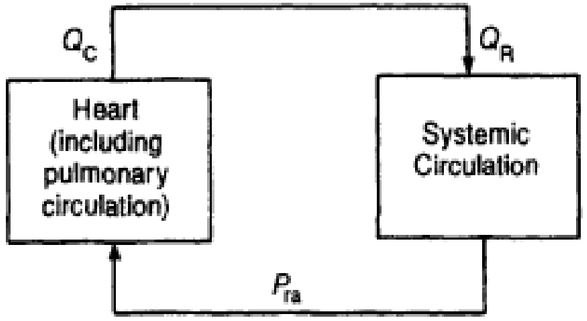
Heart and Systemic Circulation Combined

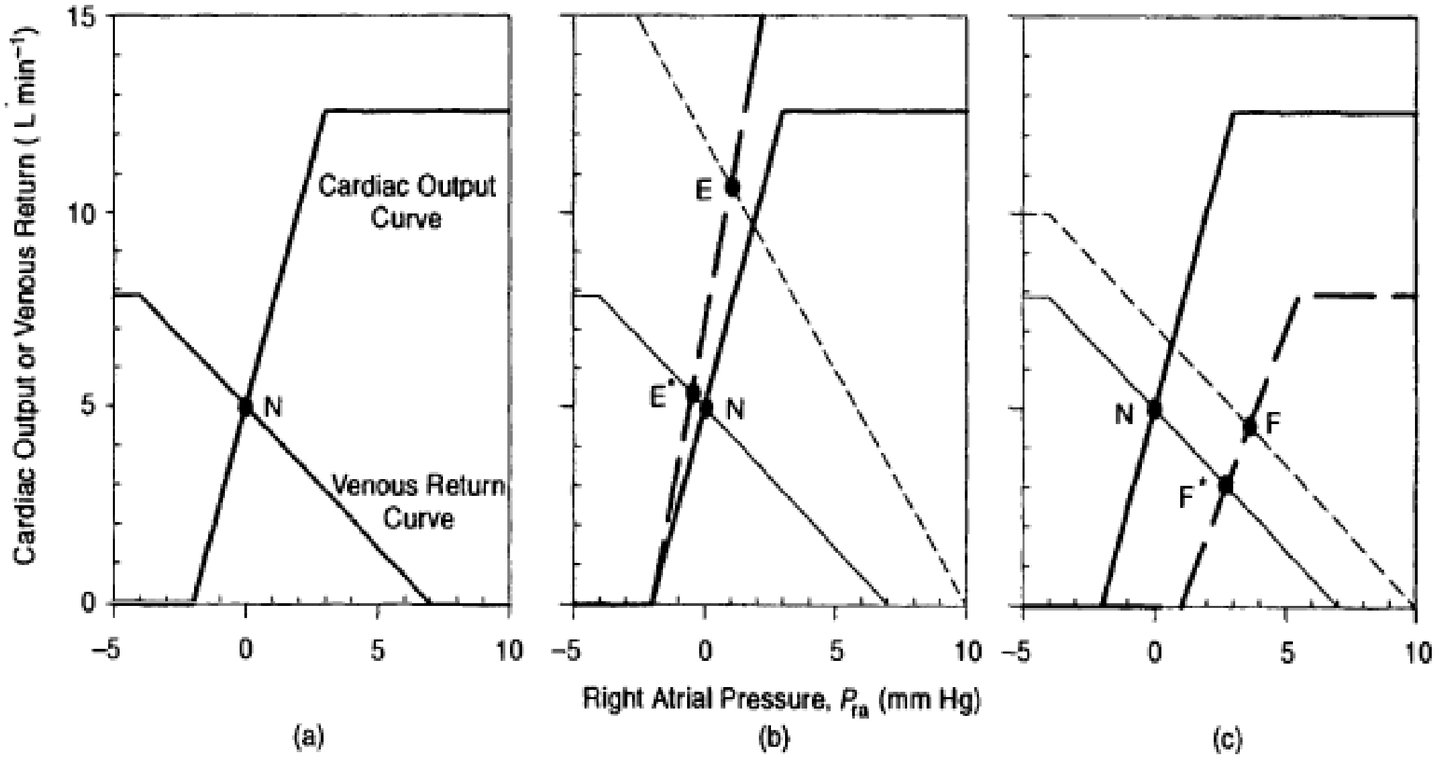
•Consider the "heart" and "systemic circulation“ components of the model together and allowing the system to operate in closed-loop mode.

•Assume the following parameter values, which have been chosen so that the model provides a first approximation to the human cardiovascular system under normal resting conditions: f = 72 beats min-I, Co = 0.035 LmmHg", Cs = 0.0007LmmHg-1, Pp1=-4mmHg, RA=19.2mmHgminL-1, Ry=0.4mmHgminL-1, CA=0.028 LmmHg-1, Cy = 0.5LmmHg-1, Pms = 7mmHg, PA = 100mmHg. Under such conditions, the cardiac output and venous return curves are as shown in Figure.

•The intersection between the two curves yields the steady-state operating point, labelled N. This is established at a cardiac output of 5 L min-1 and Pm of 0mm Hg.

Heart and Systemic Circulation Combined





Heart and Systemic Circulation Combined

•At the onset of exercise, the tensing of the muscles involved plus an increase in venomotor tone produces a decrease in venous compliance, thereby raising Pms.

•Sympathetic stimulation leads subsequently to an increase in heart rate, which elevates the slope of the cardiac output curve, and increased vasomotor tone. Then, local vasodilation of the muscular vascular beds produces a marked decrease in peripheral resistance.

•The new steady-state cardiac output is now increased to "-J 10.5 L min-I, twice the resting value, while Pm remains relatively unchanged (point E)

Heart and Systemic Circulation Combined

• The next model is used to predict the steady-state values of cardiac output and Pra in heart failure following myocardial infarction.

•To represent the reduction in effectiveness of the heart as a pump, we assume that Cs is increased and CD is decreased by 30%.

•If this were the only effect of heart failure, cardiac output would decrease by 40% to about 3 L min-I (point F*). At the same time, Pra would rise by app 3 mm Hg

Regulation of ventilation

$$k = \frac{V_{STPD}}{V_{BTPS}} = \frac{P_B - 47}{863}$$

•The volumetric fractions, $F_{ICO\,2}$ and F_{ACO2} , can be converted into their corresponding partial pressures, P_{ICO2} and P_{ACO2} , using Dalton's law:

$$P_{ICO_2} = F_{ICO_2}(P_B - 47), \qquad P_{ACO_2} = F_{ACO_2}(P_B - 47)$$

$$P_{ACO_2} = P_{ICO_2} + \frac{863\dot{V}_{CO_2}}{\dot{V}_A}$$

$$P_{AO_2} = P_{IO_2} - \frac{863\dot{V}_{O_2}}{\dot{V}_A}$$

Respiratory Controller

•The controller part of the system includes the chemoreceptors, the neuronal circuits in the lower brain involved in the generation of the respiratory rhythm as well as the neural drive to breathe, and the respiratory muscles.

•The controller response to CO2 has been shown to be linear over the physiological range. In the absence of vigilance, such as during sleep, the controller output falls rapidly to zero (i.e., central apnea occurs)whenPaC0 2 decreases slightly below normal awake resting levels. Exposure to hypoxia (i.e., when Pa0 2 decreases below 100mmHg) leads to an increase in the CO2 response slope as well as the ventilatory controller output.