
Early theories about pulmonary ventilation regulation during exercise centered singularly on arterial P{sub}CO{sub}2, arterial blood pH, or reflex stimulation originating from muscle receptors. Dejours believed that no factor by itself, but rather a multiplicity of interacting factors, regulated breathing during exercise. He hypothesized that exercise hyperpnea depended on humoral (chemical) and neurogenic stimuli that varied their contributions depending on the phase of exercise and recovery.

The figure presents Dejours’ observations that the time course of pulmonary minute ventilation (V{sub}E{sub}) during the transitions from rest to exercise to recovery followed a consistent pattern. V{sub}E{sub} increases within the same ventilatory cycle, coinciding with the start of exercise. Some 10 to 20 seconds later, ventilation volumes slowly increase to an eventual steady state. Minute ventilation declines abruptly when exercise stops, remains fairly constant for 20 to 30 seconds, and then decreases progressively to the resting value.

Dejours concluded that ventilatory dynamics in exercise combine rapid (fast component) and slow (slow component) responses that progress in defined stages during exercise and recovery. He proposed that different physiologic factors control the fast and slow components. Two factors contribute to the fast component: (1) cerebral input from afferent impulses from the brain’s psychomotor area to the respiratory center in the medulla and (2) extrathoracic mechanoreceptor stimulation from “proprioceptors” in active body segments. Two mechanisms also modulate the slower component of the ventilatory response. The first, a reflex, originates from muscle chemoreceptors sensitive to progressive physiochemical changes within active muscle as exercise progresses. The

**Pulmonary minute ventilation (V{sub}E{sub}) during mild exercise and recovery (inset graph).** Portion B of inset represents the immediate, rapid increase when exercise begins; ST reflects the more gradual rise to a steady state; F indicates the quick fall when exercise stops; S represents the slower return of ventilation to the preexercise level. The main graph shows the contribution of these ventilatory response components to oxygen consumption. Neurogenic and humoral components both increase with the intensity of the preceding exercise; the fast component at the start of exercise increases with exercise intensity much less than the progressive increase in neurogenic and humoral controls. (Adapted with permission from Dejours P. The regulation of breathing during muscular exercise in man. A neuro-humoral theory. In: Cunningham DJC, Lloyd BB, eds. The regulation of human respiration. Oxford, England: Blackwell, 1963.)
second factor represents a humoral mechanism. Dejours’ belief in humoral control developed from experiments that occluded leg blood flow. Restricting venous return during leg exercise caused \( V_e \) to decline below resting levels, thus demonstrating ventilatory dependence on blood-borne (humoral) chemicals produced in active tissues.

Dejours stressed the interrelationship between the fast and slow components in exercise ventilation. Reflex and cortical factors initiated the rapid rise in ventilation at exercise onset. Subsequently, humoral factors, and possibly progressive neurogenic output, modulated the slower rise in ventilation during the first minutes of exercise. The latter steady-state response during exercise probably related to (1) increases in reflex drive through local physical and chemical changes at the peripheral mechanoreceptors and (2) positive interactions between neurogenic and humoral drives. Ventilation decreases precipitously when exercise stops and neurogenic input ceases. Ventilation then becomes regulated exclusively by humoral factors from the recovering musculature.

The studies of Dejours formed the basis for explaining pulmonary ventilation during exercise and recovery. Subsequent research (see Fig. 14.4) provides additional factors to explain exercise hyperpnea and provides a more comprehensive model for ventilatory control.