The Department of Medicine and Radiology, the San Diego VA Medical Center, and the University of California, San Diego.

**Reprint requests:** Dr. Slutsky, San Diego VA Hospital V-114, 3350 La Jolla Village Drive, San Diego 92161

**REFERENCES**

12. Nelson T, Slutsky R, Verba J. Effect of patient imaging angle on apparent cardiac volumes and the potential impact on measurement of valvular regurgitant fractions. Submitted for publication
13. Slutsky R, Berger F, Garver F. Abstinence makes the heart grow stronger. Submitted for publication

**Respiratory Muscle Tone and The Control of Functional Residual Capacity**

The study of Cooper and Boswell in this issue (see page 29) stresses the clinical relevance of a question which has been the subject of considerable interest among respiratory physiologists for the past 30 years. Does some activity normally persist in respiratory muscles at end-expiration in man, and if it does, does this activity participate to an appreciable extent in the determination of functional residual capacity (FRC)?

Numerous studies have shown that FRC in supine or seated man is reduced by a mean of 20 percent during anesthesia with or without muscle paralysis. A fall in FRC without visible abnormalities detected on chest x-ray film also appears to be a frequent occurrence in severe head trauma which, like general anesthesia, involves unresponsiveness and a marked decline in voluntary activity. The mechanism of these reductions in FRC, however, remains largely uncertain. To the extent that FRC in normal man at rest is set by a balance of inward recoil of the lung and outward recoil of the chest wall, the reductions in FRC which are seen during anesthesia and analogous states could be due to an increase in lung elastic recoil, a decrease in outward chest wall recoil, or a combination of both changes. Although pulmonary compliance usually falls with anesthesia, the observation that the fall in FRC is coupled with a decrease, not an increase, in transpulmonary pressure at FRC has been interpreted to indicate that the effect of anesthesia primarily responsible for the fall in FRC is a reduction in outward recoil of the chest wall; the reduction in pulmonary compliance, therefore, would be the consequence rather than the cause of the change in FRC. On the basis of these studies, it thus appears that at end-expiration there is normally some degree of involuntary activity ("tone") in human inspiratory muscles which contributes to the maintenance of FRC and is abolished by anesthesia.

Experiments with curare also support the notion that inspiratory muscle tone contributes to the maintenance of FRC in humans breathing at rest. When submaximal neuromuscular block was produced by curare in six awake seated subjects, a mean fall in FRC of 15 percent was measured at the time of maximal weakness. In addition, paralysis caused a parallel shift in the relaxation pressure-volume (PV) curve of the chest wall so that the transthoracic pressure was increased at any given gas volume of the lungs. To the extent that diaphragmatic function was relatively well preserved during paralysis, a loss of "tone" in the inspiratory rib cage muscles was perceived as being the likely mechanism of the reduction in FRC. The abundance of muscle spindles in the external and internal interosseous intercostal muscles and the susceptibility of these spindles to the effects of anesthetic and paralyzing agents support this interpretation. The recent observation that most seated quadriplegic subjects have reduced FRC and decreased transpulmonary pressure at end-expiration is
also consistent with the idea that the inspiratory intercostals normally contribute to maintaining the outward recoil of the chest wall and, with it, the FRC in the seated posture.

The failure of partial paralysis by curare to affect FRC in the supine posture suggests that the role of the intercostal muscles is less in this posture. However, to the extent that the abdomen behaves like a fluid-filled container, and to the extent that active diaphragmatic tension may oppose the cephalad displacement of the abdominal contents into the thorax in the horizontal posture, the diaphragm might be expected to take on, at least in part, the role of the intercostal muscles in maintaining FRC in this posture. Indeed, Froese and Bryan, measuring the configuration and movements of the diaphragm in supine normal subjects, reported a cephalad displacement of the diaphragmatic dome, especially in its dependent part, during total paralysis. Also, Muller et al. described a diminution in the diaphragmatic electromyogram (EMG) at end-expiration during halothane anesthesia and rapid-eye-movement sleep. These changes in diaphragmatic EMG were attributed to changes in muscle spindle activity in the diaphragm.

Most of the published literature is thus consistent with the idea that some involuntary activity is normally present in the “relaxed” inspiratory muscles of awake humans and that this activity maintains the end-expiratory level above the actual resting position of the respiratory system. Some studies, however, have been unable to show any noticeable change in FRC with anesthesia. Such discrepancies, of course, may simply reflect procedural differences, but more troublesome are the recent observations of Hedenstierna et al. who studied the effects of anesthesia and muscle paralysis in 16 supine subjects and found a mean 28 percent fall in FRC without any measurable reduction in chest wall (rib cage and abdomen) dimensions at end-expiration, and those of Kimball et al. who observed that partial curarization in seated subjects could produce a substantial reduction in FRC without affecting the relationship between transthoracic pressure and rib cage volume during voluntary relaxation. These observations clearly suggest that the fall in FRC during anesthesia or muscle paralysis results from a fluid accumulation in the chest or abdomen (ie, central blood pooling) rather than a loss of tone in the inspiratory intercostals. This interpretation has further merit, in the sense that a shift of blood from the extremities into the thorax might also account for the decrease in transpulmonary pressure at FRC and the observed decrease in gas volume of the lungs at any transthoracic pressure. It is important, however, to realize that, while attractive, this is still a hypothesis, and the study of Cooper and Boswell in this issue, by showing that these reductions in FRC may lead to increased venous admixture and profound hypoxemia, emphasizes the need for a clear understanding of the mechanism responsible for the changes which occur during general anesthesia and analogous conditions.

André De Troyer, M.D., Brussels, Belgium;* and James G. Martin, M.D., Montreal, Canada†

REFERENCES


*Erasme University Hospital, Brussels School of Medicine.
†Meakins-Christie Laboratories, Royal Victoria Hospital, McGill University Clinic.
Reprint requests; Dr. De Troyer, Chest Service, Erasme University Hospital, Brussels, Belgium 1070