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In Reply:—I thank Koblin for calling attention to another example of the delayed return of spontaneous circulation. The mechanism postulated in that report, however, does not appear to be the most likely explanation of the event we described.[1] Our patient[2] probably did have some increase in intrathoracic pressure as discussed by Martens *et al.*[1] The tracheal tube, initially advanced into the bronchus in an attempt to staunch the pulmonary hemorrhage, was withdrawn when blood flow continued from both in and around the tube in the bronchial position. Airway pressures during manual ventilation were not excessive, although bilateral chest tubes were placed to rule out the possibility of tension pneumothorax. Our patient made no spontaneous respiratory efforts once support was discontinued, but some decrease in intrathoracic pressure, no doubt, still occurred, perhaps augmenting passive ventricular filling. Whether this augmentation can explain the return of spontaneous circulation after the cessation of all cardiac electrical activity is questionable. An improvement in ventricular filling certainly would enhance forward flow in an empty but beating heart. However, how this filling would stimulate the completely quiescent myocardium—the total absence of both cardiac electrical and mechanical activity—is not readily apparent. There had been no electrical cardiac activity—as evidenced by two separate concurrently functioning electrocardiogram systems—for several minutes at the time the efforts were terminated. This situation spontaneously reversed.

Hill raises another important issue. The sensitivity of current clinical brainstem testing as a marker for cessation of brain function is a critical question and the spontaneous movement of a patient so diagnosed would be a concerning event. However, those individuals who breathe spontaneously after the discontinuation of mechanical ventilatory support fail the so-called apnea test and therefore are not eligible for organ donation in our institution. Further, the harvesting process does not occur in patients receiving ongoing cardiopulmonary resuscitation. It is reserved primarily for individuals who have been successfully resuscitated to the only clinical endpoint currently available, the restoration of spontaneous circulation. Those who cannot be successfully resuscitated are not usually candidates for organ donation. The problem comes when hemodynamic function is restored in the apparent absence of neurologic function. This latter issue, the reliability of our assessment of irreversible neurologic "death," is really the crux of the point raised by Hill and remains a significant issue.

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The Proper Study of Anesthetized Humans: I

To the Editor:—Barnas *et al.*[1] recently published data describing how changes in mean lung volume affect respiratory system mechanics measured during sinusoidal forcing. The authors assume that these results obtained in healthy, awake, seated subjects can be applied to anesthetized recumbent patients. To understand why this assumption may not be valid, a bit of history is required. Pulmonary physiologists

always have been interested in measuring the passive mechanical properties of the respiratory system. Recognizing the difficulty in achieving complete muscular relaxation of the chest wall in awake subjects, investigators used muscle paralysis and general anesthesia to examine what they believed to be the true passive mechanical properties of the respiratory system.[2] However, it soon became apparent that anesthesia *itself* changed these properties.[3,4] This finding was unfortunate for the physiologists, as it meant that the anesthetized state was not an appropriate model of the "relaxed" respiratory system while awake. Of course, the converse is also true, so that it cannot be assumed that fundamental mechanical behavior of the lung, such as the dependence of mechanical properties on lung volume, is not altered by anesthesia.

As an example of this principle, the relationship between lung volume and pulmonary resistances depends on airway smooth muscle tone.[5] Joyner *et al.*[6] found that halothane changes this relationship in dogs when smooth muscle tone is present, because halothane reduces smooth muscle tone; halothane did not change this relationship when tone was absent. Because airway smooth muscle tone is present in awake human subjects,[7] it is likely that halothane-induced reductions in this tone would minimize the effect of lung volume on lung resistances during halothane anesthesia.

The recognition that the anesthetized state may profoundly affect the mechanical properties of the respiratory system spawned many years of fruitful investigation that attempted to explain the mechanisms responsible for such changes. The fact that these mechanisms still are not fully understood attests both to their complexity and to the inherent difficulty in making these measurements in anesthetized subjects. It seems ironic that in the face of all these years of effort, the paper of Barnas *et al.* and other recent publications[8,9] seem to revert to the practice of applying results obtained in awake patients to the anesthetized state. This approach certainly simplifies the experiments, but the findings of our predecessors have not left this option available. To paraphrase Alexander Pope, ". . . the proper study of [anesthetized] mankind is [anesthetized] man."

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The Proper Study of Anesthetized Humans: II

To the Editor:—Recently, Barnas *et al.*[1] made the point that the contradictory results regarding the change in airway resistance when lung volume is increased above functional residual capacity (FRC) may have been due to the use of techniques with varying tidal volume and frequency.[1] More than 10 yr ago, we used a fixed-volume (58 ml), fixed-frequency (3 Hz) technique to examine the effect of lung volume on airway resistance in both awake and anesthetized humans.[2,3]

We always found a hyperbolic relationship between lung volume and resistance; there was never an increase in resistance above FRC, but in most cases we limited the volume increase above FRC to 1 liter or less. Resistance values at FRC in normal subjects were the same as those described by Barnas *et al.* However, increased bronchomotor tone, occurring either spontaneously or after the administration of histamine, was associated with a high airway resistance and a change in shape of the curve, so that in these circumstances there was always a large fall in resistance with increasing volume above FRC.[4] Barnas *et al.* need not speculate from dog experiments about the effects of anesthetics on airway resistance in humans. We showed that both halothane and enflurane caused significant bronchodilatation in patients, whereas isoflurane was less likely to produce a consistent bronchodilatation.[5] Of considerable interest was the observation that, in patients with neuromuscular blockade, halothane, and isoflurane produced a significant increase in expiratory reserve volume (ERV) of about 130 ml. When FRC during anesthesia happens to be at the upper end of the resistance hyperbola, this increase in