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Does Halothane Really Reduce Diaphragmatic Contractility?

To the Editor:—Clergue *et al.*[1] have conducted a complex study of halothane's effect on the diaphragm. They assessed contractility by the pressure developed by the diaphragm. However, pressure development is a complex process that involves several factors that probably changed in the course of their study, so their conclusion that halothane was responsible for the decrease in pressure could be incorrect.

Taking the process of activation in order, the first possibility is that neuromuscular transmission has been affected. In an isolated rat nerve–diaphragm preparation, this effect was only noted with halothane concentrations above 4%,[2] which is not consistent with their findings.

The next step in the process of pressure generation is the development of tension in the muscle fibers. In the dogs studied, hypercapnia was present when halothane was administered. In humans, hypercapnia alone reduces the pressure that the diaphragm can generate, for a given degree of electrical activation.[3] The hypercapnia and reduced contractility were of the same extent as in the dogs studied by Clergue.[1]

The pressure generated in the diaphragmatic fibers for a given degree of electrical activation depends on the length of the contracting fibers and the shape of the diaphragm. By stimulating the diaphragm during airway occlusion and preventing an increase in lung volume, it was assumed that these two factors would be controlled. However, halothane depresses the activity of ribcage muscles more than the diaphragm.[4] When the diaphragm is stimulated in the presence of halothane, the ribcage will be drawn in, and the diaphragm will shorten and descend. If this occurs, it will generate less tension and pressure than it would have done if the contraction were isometric.

If the diaphragm descends, it will increase abdominal pressure. In a study in anesthetized humans,[5] we noted that gastric pressure increased during an occluded inspiration (*i.e.*, when the contraction should have been "isometric") by about one-half that seen during normal inspiration (when the diaphragm descends freely). This is consistent with reduced ribcage stability that has been recognized during anesthesia with volatile agents for years and demonstrated in clinical studies.[6] The influence of this effect in reducing the pressure generated by the diaphragmatic contraction should have been seen by Clergue and his co-workers because they measured gastric pressure. Their control data substantiate the possibility that the relationship between electrical activity and the pressure produced by the muscle was not proportional. During a normal breath, approximately 2.3 units of activation were needed to generate 1 cmH_2O of pressure, at end inspiration. When the airway was occluded, diaphragmatic activity of 21.2 units generated a pressure of 4.9 cmH_2O. This means that during the "isometric" contraction or airway occlusion, the diaphragm needed almost twice as much electrical activity to generate pressure as it needed when it was allowed to contract freely!

These considerations suggest that a satisfactory answer to this question is more likely to be provided by an *in vitro* study than the complex circumstances that are found *in vivo*.

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