Beer and the Respiratory Muscles: The Adverse Effects of Ascites

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The acquisition of a beer belly is the fate of many a physiologist. Fortunately, far fewer of us pursue beer drinking to the point of liver failure and the development of ascites. Ascites is defined as the presence of excess protein-containing fluid in the peritoneal cavity, which can range in amount from <100 ml up to as much as 25 liters. The most common cause is alcoholic cirrhosis, but ascites can result from many other conditions including liver failure of other etiologies (such as viral hepatitis), cardiac disease (in particular heart failure and constrictive pericarditis), hypoalbuminemic states (including nephrotic syndrome and chronic malnutrition), various malignancies (such as ovarian and primary liver cancer), end-stage renal failure, and pancreatitis. Peritoneal dialysis is another setting in which excess fluid is found in the peritoneal space, although the amount of dialysate used per cycle (1-3 liters) is at the low end of the amount that can be found with ascites caused by disease.

The cardinal respiratory symptoms experienced by humans with ascites are dyspnea and exercise limitation. Pulmonary function testing reveals particularly reduced lung volumes including vital capacity and total lung capacity (2,3). In addition there may be changes in diaphragm length, radius of curvature and strength (5,10). Large-volume paracentesis or diuresis reduces the load on the respiratory muscles, augments lung volumes, alters diaphragm curvature, shortens abdominal muscle length, and reduces the magnitude of diaphragm force needed to maintain ventilation (2-5). Human work on respiratory muscle consequences of ascites or peritoneal dialysis has usually been conducted over a small range of changes in peritoneal fluid volume, whereas it is subjects with large amounts of ascites who have the greatest amount of dyspnea and the largest reductions in lung volumes.

Animal models have been used previously to study ascites and the respiratory system. Hubmayr et al. (6) infused 60-100 ml/kg body weight of fluid acutely into the peritoneal space (equivalent to 4-7 liters of ascitic fluid in a 70 kg person). They found that peritoneal fluid expanded the lateral and anterior-posterior dimensions of the lower rib cage, moved the diaphragm cranially, and lengthened the diaphragm when passive. Active diaphragm contraction attenuated the effects of peritoneal fluid on diaphragm length, particularly for less dependent areas of the diaphragm.
In the present issue of the Journal, Leduc and De Troyer report the third in their series of investigations about the effects of ascites on the respiratory muscles in a canine model (7-9). They infused up to 200 ml/kg of fluid into the peritoneal space (equivalent to 14 liters of ascitic fluid in a 70 kg person), which is two to three times the volume used by Hubmayr et al. (6). Respiratory system responses at high ascites volumes were found to differ qualitatively as well as quantitatively from those at low to intermediate fluid volumes.

The first study (8) found that abdominal elastance increased with ascites once fluid volumes exceeded 50 ml/kg. Transdiaphragmatic pressure in response to phrenic nerve stimulation remained stable as ascites volume increased to 100 ml/kg but fell by ~45% with progressively higher ascitic fluid volumes. Ascites lengthened the unstimulated diaphragm, but only by <5%, and displaced its dome cranially. During active diaphragm contraction, ascites impaired muscle shortening considerably (by ~65%), decreased the caudal movement of the diaphragm during contraction substantially (by ~75%), and increased the circumference of the lower rib cage. The second study (7) used computed tomography to more closely examine changes in diaphragm length and shape. In this study, there was a trend for transdiaphragmatic pressure to increase with intraperitoneal infusion of fluid in amounts below 100 ml/kg, whereas with large volume infusions it decreased significantly (by ~25%). The unstimulated diaphragm lengthened with increasing ascites, but by no more than 13%. The radius of curvature of the diaphragm did not change at fluid volumes up to 50 ml/kg, but then increased progressively thereafter, augmenting by ~50%.

The third study, reported presently (9), is directed towards abdominal muscle function. With peritoneal fluid infusion, the length of unstimulated internal oblique muscle increased progressively, by up to ~60%. During electrical stimulation of the transversus abdominus and the internal oblique, intra-abdominal pressure was increased two to three-fold by ascites volumes up to 100 ml/kg, but then decreased progressively with higher fluid volumes. Simultaneously measured airway pressure did not change significantly with low amounts of peritoneal fluid, but decreased when the volumes of ascites was increased further. Contraction-induced muscle shortening was very low (~6%) at high ascites volumes.
Thus what emerges from these three studies (7-9) is that ascites impairs the respiratory actions of the diaphragm by a combination of increased load and changes in diaphragm geometry. Concomitant changes in diaphragm muscle length may improve muscle force but only partially compensate for the adverse effects of load and altered geometry. In contrast, for the abdominal muscles the impairment is due to increased load, heightened diaphragm elastance reducing the actions of the abdominal muscles on the lung, and, at high ascitic fluid volumes, considerable muscle elongation leading to unfavorable positioning on its length-tension relationship. The role of changes in abdominal muscle shape with ascites was not investigated in the animal models.

As acknowledged by the authors (7-9), these studies used a model in which fluid was introduced rapidly into the peritoneal space. In contrast, many diseases leading to ascites are considerably more chronic, in that ascites develops and increases in amount over the course of weeks to months. Some studies on the effects of peritoneal dialysis suggest that the human respiratory system is able to compensate for the chronic presence of modest amounts of fluid in the peritoneal space (10,11), but data are lacking about whether such compensation occurs with large amounts of disease-induced ascites. Models of emphysema-induced chronic hyperinflation in small animals have demonstrated compensatory changes in respiratory muscle structure involving the adding or dropping of sarcomeres, which can restore the muscle back towards the optimal position on its length-tension relationship (1). Such a change with chronic ascites, if it were to occur, would be especially important for the abdominal muscles. Plasticity of rib cage structure has also been found in the hamster model of emphysema (12). Stiffening of the lower rib cage with chronic ascites (preventing the ascites-induced expansion) would be particularly advantageous for the maintenance of normal diaphragm geometry and thus function. Whether compensatory alterations in abdominal muscle sarcomere number or lower chest wall stiffness occur awaits further study.

Another difference between the animal model used by Hubmayr, Leduc and colleagues (6-9) and humans with ascites is that clinical ascites is often due to a disease with systemic effects. The underlying disease may directly impair respiratory muscle structure and function and/or pulmonary gas exchange by
mechanisms in addition to those produced directly by ascites, which may in turn contribute to dyspnea and exercise limitation. In support of this, Flintrop et al. (5) found in subjects with ascites that hand grip strength was reduced to a comparable extent as that of maximal inspiratory force, and furthermore that inspiratory and expiratory muscle force were not improved by paracentesis. Nonetheless clinical studies have also demonstrated the improvement in dyspnea and pulmonary function tests that occur with removal of ascitic fluid (2-5), indicating that the mechanical effects of the fluid in the peritoneal space are indeed important clinically. The studies in a canine model of ascites (6-9) have provided considerable insight into the mechanisms by which large amounts of intraperitoneal fluid impairs the ability of the respiratory muscles to maintain adequate ventilation.

REFERENCES


