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A TECHNICAL MODIFICATION TO IMPROVE EXPERIMENTAL PRODUCTION OF HEPATOGENIC ASCITES

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ABSTRACT

Constriction of the thoracic inferior vena cava is a useful experimental maneuver to reproduce massive ascites. Unfortunately, the margin of safety of this technique is narrow in that too much constriction overly restricts venous return with subsequent shock and death, and lesser constriction is often associated with extensive venous collateralization via the azygos system and failure to sustain hepatic congestion. By combining azygos vein ligation with 50% constriction of the supradiaphragmatic inferior vena caval circumference at the time of the initial thoracotomy, we have found that intense hepatic congestion is sustained and that dogs consistently develop massive ascites within 2-3 weeks.

Hepatic venous outflow block is commonly used experimentally to reproduce ascites and to simulate some features of congestive heart failure, Budd-Chiari syndrome and hepatic cirrhosis. The usual approach involves constricting the inferior vena cava (approximately 50% circumference) just above the diaphragm (1). Although often successful, this technique carries a narrow margin of safety in that excessive constriction compromises venous return and cardiac output leading to early death, while inadequate constriction fails to promote intense hepatic congestion and ascites. A variety of ancillary maneuvers have been tried to improve the ascites/death ratio. Typically, they involve wrapping the vena cava with inflammatory stimulants (e.g. cellophane, aluminum bands, umbilical tape) to induce more gradual and progressive caval narrowing with more sustained hepatic sinusoidal hypertension. Nonetheless, ascites formation remains inconsistent in appearance and volume and some operative mortality persists.

Using this experimental model in the dog, we observed that a key factor interfering with maintenance of vena caval hypertension and hepatic congestion (the sine qua non for excess hepatic lymph and ascites formation) was development of a collateral venous network bypassing the site of caval constriction. This venous plexus involved large intercostal and lumbar tributaries below the ligature that gained access to the normotensive superior vena cava and right atrium via dilated azygos and hemiazygos veins (*Fig. 1*). Interruption of this route of venous decompression at the time of initial inferior venal caval constriction uniformly produced massive ascites formation within 14-21 days.

TECHNIQUE

Via a standard postero-lateral thoracotomy under general endotracheal anesthesia, the inferior vena cava is freed from the phrenic nerve and isolated with an umbilical tape just above the diaphragm. Because the dog's thoracic inferior vena cava is approximately 4cm in length, caval isolation is readily accomplished without opening the pericardium. After 50% constriction of caval circumference, the azygos vein is ligated superiorly near its entrance into the superior vena cava and inferiorly along the postero-lateral chest cage. Care is taken to dissect close to the azygos vein to avoid injury to the thoracic duct which

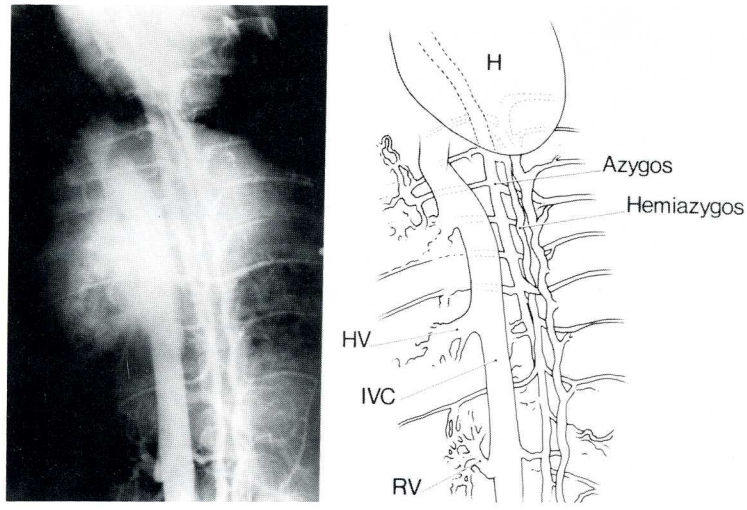

Fig. 1: Vena cavagram (left) with schematic outline (right) in dog 21 days after 50% constriction of the thoracic inferior vena cava demonstrating extensive collateralization via a dilated azygos and hemiazygos plexus of veins bypassing the site of supradiaphragmatic caval constriction (poorly seen in this x-ray). Ascites was minimal. HV-hepatic vein; IVC-inferior vena cava; RV-renal vein; H-heart.

courses between the azygos vein and the aorta. After closure of the chest, air is fully aspirated using an indwelling thoracostomy tube and 3-way stopcock which are removed prior to awakening the dog from anesthesia.

COMMENT

Although supradiaphragmatic inferior vena caval constriction is an excellent experimental method to reproduce hepatogenic ascites, spontaneous venous compression via the azygos vein and its tributaries is sometimes sufficient to decongest the liver and ascites fails to appear. Clinically, too, fibrous atresia and "obstructive webbing" of the inferior vena cava are associated with the Budd-Chiari syndrome (2), but in some patients with isolated anomalous inferior vena cava syndrome, collateralization via the azygos vein (so-called azygos substitution or continuation) acts to decompress the splanchnic viscera and hepatic congestion with ascites are absent (3). Whereas reoperation and further experimental constriction of the thoracic inferior vena cava is possible to recongest the liver, the procedure requires reopening the thorax with considerable inconvenience, morbidity and mortality. On the other hand, by simply ligating the azygos vein both inferiorly, where the bulk of intercostal veins enter, and superiorly, where the azygos vein empties into the superior vena cava, venous decompression is averted, vena caval constriction is safely limited to 50% circumference, hepatic congestion is sustained, and massive ascites consistently develops within three weeks. It is likely that interruption of the azygos vein at its juncture with the superior venal cava is alone sufficient to prevent hepatic decongestion, but concomitantly ligating the inferior segment of the azygos vein ensures restriction of potential venous collateral pathways for decompression.

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