Feasibility of percutaneous catheterization and embolization of the thoracic duct in dogs

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Objective—To develop and determine the feasibility of a novel minimally invasive technique for percutaneous catheterization and embolization of the thoracic duct (PCETD) in dogs and to determine thoricic duct TD pressure at rest and during short-term balloon occlusion of the cranial vena cava (CrVC).

Animals—Fifteen 7- to 11-month-old healthy mixed-breed dogs.

Procedures—Efferent intestinal lymphangiography was performed, and the cisterna chyli was punctured with a trochar needle percutaneously under fluoroscopic guidance. When access was successful, a guide wire was directed into the TD through the needle and a vascular access sheath was advanced over the guide wire. Thoracic duct pressure was measured at rest and during acute balloon occlusion of the CrVC. The TD was then embolized cranial to the diaphragm with a combination of microcoils and cyanoacrylate or ethylene vinyl alcohol.

Results—Successful puncture of the cisterna chyli with advancement of a wire into the TD was possible in 9 of 15 dogs, but successful catheterization was possible in only 5 of 9 dogs. Acute balloon occlusion of the CrVC led to a substantial TD pressure increase in 4 of 4 dogs, and embolization of the TD was successful in 4 of 4 dogs.

Conclusions and Clinical Relevance—PCETD can successfully be performed in healthy dogs; however, this minimally invasive technique cannot currently be recommended for routine treatment of chylothorax, in part because of the technically demanding nature of the procedure. An increase in jugular venous pressure led to an increase in TD pressure, potentially predisposing some dogs to developing chylothorax. (*Am J Vet Res* 2011;72:1527–1534)

Several surgical techniques have been described for the treatment of idiopathic chylothorax in dogs when conservative treatment has failed. These include pleuroperitoneal shunting,[1,2] pleurovenous shunting,[3] thoracic omentalization,[4] TDL,[5–8] mesenteric TD embolization,[9] and pleurodesis,[7] with TDL being the most commonly performed. The reported success rate for TDL alone in dogs is low (50% to 59%)[6,7] and is believed to result from a failure to ligate all branches of the TD at the time of surgery.[5,6] An additional mechanism by which failure or recurrent chylothorax is believed to occur is by the development of collateral lymph vessels around the TDL site, resulting in continued flow of chyle into the thoracic cavity.[10,11] To decrease the rate of collateral lymphatic formation, a combination of TDL and CC ablation via ventral midline laparotomy has reportedly been successful in 7 of 8 dogs with chylothorax.[10]

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Abbreviations

CC	Cisterna chyli
CrVC	Cranial vena cava
CVP	Central venous pressure
JVP	Jugular venous pressure
PCETD	Percutaneous catheterization and embolization of the thoracic duct
TD	Thoracic duct
TDL	Thoracic duct ligation

High CVP is suspected to have a role in the development of thoracic lymphangiectasia and subsequent chylothorax by impeding the outflow of chyle from the TD into the venous system at the lymphaticovenous anastomosis.[12–15] Chronic chylothorax can lead to thickening of the pericardium, which can in turn increase CVP.[14] Because of this, pericardectomy or a combination of pericardectomy and TDL has been recommended to treat chylothorax in dogs and cats.[14] Recent studies[14,16,17] have revealed promising success rates for resolution of chylothorax when these procedures are combined.

Chylothorax has also been associated with thrombosis of the CrVC in dogs, cats, and humans.[18–22] Thrombosis of the CrVC may lead to thoracic lymphangiectasia and subsequent chylothorax directly by physical obstruction of TD emptying into the venous system at the jugulocaval angle or indirectly by increasing CVP.

Thrombosis of the CrVC, however, does not consistently result in chylothorax; this is likely attributable to the presence of collateral lymphatic channels from the TD.[19–21] The development of chylothorax secondary to a CrVC thrombus is likely multifactorial and dependent on the extent of thrombosis and presence and location of collateral TD branches.[20,21]

Thoracic duct ligation is most commonly performed through an invasive thoracotomy approach, which requires prolonged anesthesia and is associated with only moderate success rates.[6,7,23] A minimally invasive thoracoscopic TDL and pericardectomy technique has been described, with success in 6 of 7 dogs with idiopathic chylothorax and 2 of 5 dogs with nonidiopathic chylothorax.[17] Interventional radiology has become the first-line minimally invasive treatment for humans with traumatic chylothorax.[24,25] The first description of successful use of PCETD involved pigs.[26] The same research group later showed that experimentally created chylothorax through iatrogenic TD laceration resolved after PCETD in 5 pigs and 1 dog.[27] Today, some researchers consider PCETD as the standard of care for treating traumatic chylothorax in humans.[25]

Percutaneous catheterization and embolization of the TD in people involves performance of pedal lymphangiography, with the CC catheterized percutaneously under fluoroscopic guidance.[24,25] The catheter is advanced cranially into the TD, and repeated lymphangiography is performed as needed via this access.[24,25] Thoracic duct defects are identified by extravasation of contrast medium. Thoracic duct embolization is performed caudal to the leak by use of a combination of microcoils and cyanoacrylate glue[24] or liquid embolic agents.[25] Success rates of 70%[24] and 71%[25] have been reported for humans undergoing this procedure. Although mesenteric embolization of the CC and TD via laparotomy and mesenteric catheterization has reportedly occurred in a few dogs,[9] PCETD has not been investigated as a minimally invasive technique to treat idiopathic chylothorax in dogs.

The objective of the study reported here was to develop and determine the feasibility of PCETD in dogs. A secondary objective was to determine TD pressure at rest and during acute balloon occlusion of the CrVC to determine whether an increase in CVP leads to an increase in TD pressure.

Materials and Methods

Animals—Fifteen healthy mixed-breed dogs (9 males and 6 females) between 7 and 11 months of age and weighing between 9 and 18 kg were used for the study. The dogs had already been anesthetized for approximately 8 hours for a teaching surgical exercise that was planned to end in euthanasia. Surgical procedures performed included 2 each of cystotomy, splenectomy, gastrotomy, and intestinal resection and anastomosis, without invasion of the lymphatic system or thoracic cavity. The study was performed in accordance with the Canadian Council for Animal Care and Use Guidelines and was approved by the Ontario Veterinary College Animal Care Committee.

All dogs selected for the study had undergone presurgical physical examination and hematologic analysis, and results were unremarkable. Dogs had been premedicated on the morning of surgery with acepromazine maleate[a] (0.05 mg/kg, IM) and morphine[b] (0.2 mg/kg, IM) or hydromorphone[c] (0.05 mg/kg, IM). Anesthesia was induced with thiopental[d] (10 mg/kg, IV) to effect and maintained with isoflurane[e] in oxygen (1% to 2%). All dogs received crystalloid fluid treatment at a surgical rate (10 mg/kg/h, IV). Dogs that had hypotensive episodes or that required additional cardiovascular support were not selected for the study.

Efferent intestinal lymphangiography—The celiotomy incision performed as part of the teaching exercise was reopened, and Balfour retractors were applied to improve visualization of the abdominal cavity. An efferent intestinal lymphatic vessel was identified in the mesoduodenal region and cannulated with a 24-gauge, 0.75-inch over-the-needle catheter. The catheter was sutured in place, and an extension set with a 3-way stopcock primed with iodinated contrast material[f] was attached to the catheter. Laparotomy sponges were packed into the abdomen around the cannulated lymphatic vessel to prevent displacement during transfer of the dog to the angiographic suite, and the abdomen was closed with sutures routinely.

Once transfer to the angiography suite and setup were completed, a lymphangiogram was performed by slowly injecting iodinated contrast medium into the cannulated lymphatic vessel under fluoroscopic guidance. Lymphangiograms were assessed visually to determine the size and location of the CC. The maximum width of the CC was compared with the width of the body of L2, and the location was recorded as partially, completely, or not located within the rib cage.

CC puncture and TD catheterization—With the dog positioned in right lateral recumbency, the CC was punctured as caudally as possible at its greatest dimension with a 21-gauge, 15-cm trochar needle[g] under fluoroscopic guidance. The stylet was removed from the diagnostic needle, and a 0.018-inch, 40-cm stainless steel floppy-tipped guide wire[h] was introduced into the needle and advanced into the TD. If puncture of the contralateral wall was suspected, the needle was gently pulled back while attempting to advance the wire until it moved cranially within the CC and the TD.[25] The needle was then removed, and a 4F, 10-cm coaxial introducer and dilator[h] primed with saline (0.9% NaCl) solution was advanced over the guide wire under fluoroscopic guidance. With the coaxial introducer in place, a 0.035-inch, 180-cm hydrophilic wire[i] was advanced into the TD and the coaxial introducer was removed. A 4F, 11-cm sheath[j] primed with saline solution was advanced over the guide wire with the aid of fluoroscopy into the TD (**Figure 1**).

In the final 7 dogs, a technical modification was made to obtain TD access in 2 steps rather than in 3. Once the CC was punctured with the diagnostic needle, a 0.018-inch guide wire[k] was advanced into the TD. Access to the TD was achieved with a percutaneous access kit[l] that was primed with saline solution and advanced directly over the guide wire into the TD with fluoroscopic guidance. After removing the introducer and stiffening cannula, a hemostasis valve[m] primed with

saline solution was fitted to the sheath to prevent backflow and create a closed system.

Instrumentation for pressure measurement—The ventral cervical region was aseptically prepared. The right jugular vein was percutaneously catheterized with a 20-gauge, 1.88-inch over-the-needle catheter. A 0.021-inch guide wire was placed through the catheter and into the jugular vein with the tip just cranial to the jugulocaval angle. This access was used to place a 4F, 11-cm vascular access sheath primed with saline solution. The sheath was flushed and sutured into place. The left jugular vein was accessed as described for the right jugular vein except that a 9F, 11-cm vascular access sheath[n] primed with saline solution was advanced into the jugular vein to a level cranial to the jugulocaval angle. The sheath was inserted to an appropriate length without entering the jugulocaval angle. The 9F vascular access sheath was flushed and sutured into place. A straight-tipped 4F, 100-cm end-hole catheter[o] primed with saline solution was advanced through the 4F sheath to the level of the distal jugular vein under fluoroscopic guidance. This catheter was used to measure JVP as a surrogate for CVP. Invasive arterial blood pressure was monitored through a previously placed 20-gauge, 1.88-inch over-the-needle catheter placed in the left or right dorsal pedal artery.

When TD access was successful, a 4F, 100-cm end-hole catheter was advanced into the TD through the 4F sheath for TD pressure measurement (in the first 8 dogs) or pressures were measured directly through the percutaneous access set (in the final 7 dogs).

Simultaneous measurements of JVP, peak systolic blood pressure, end diastolic blood pressure, mean arterial blood pressure, TD pressure, and ECG activity for each dog were monitored continuously with a multichannel, digital, hemodynamic data acquisition system with software[p] designed for cardiovascular studies. All pressure channels were calibrated with a mercury manometer before data collection began.

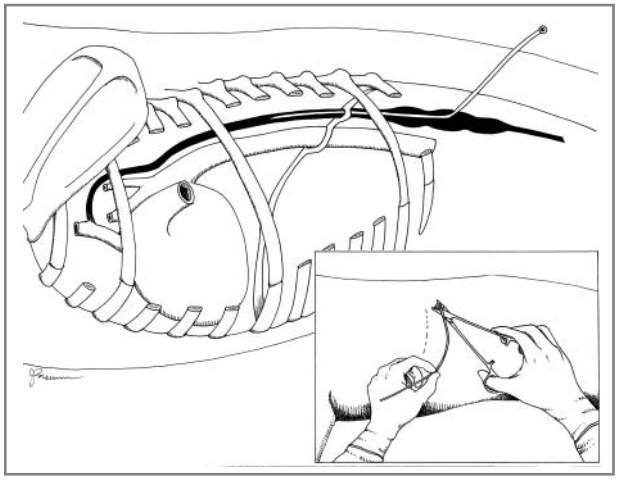

Figure 1—Schematic diagram of a dog positioned in right lateral recumbency and undergoing PCETD. The CC has been percutaneously accessed, and a catheter has been inserted within the TD. Inset—Needle drivers are used to hold the diagnostic needle during initial percutaneous CC puncture. A wire can be fed through the diagnostic needle into the CC and TD by use of fluoroscopic guidance once CC access has been obtained.

After baseline measurements of the aforementioned parameters were obtained, a 25-mm-diameter, 6-cm-long maximal diameter balloon catheter[q] was advanced into the CrVC through the 9F vascular access sheath in the left jugular vein. Under fluoroscopic guidance, the balloon catheter was maximally inflated (by injecting iodinated contrast medium under fluoroscopic guidance) at the level of the lymphaticovenous junction as determined by means of lymphangiography. Occlusion was maintained for 5 minutes. To ensure occlusion of the CrVC, an increase in JVP was confirmed prior to recording TD pressures. After 5 minutes, the balloon was deflated and repositioned further caudal in the CrVC just cranial to its entry into the right atrium to occlude the lymphaticovenous anastomosis junction and any collateral lymphatic channels if present. A baseline measurement was obtained, and the balloon was once again maximally inflated and the increase in JVP confirmed. Thoracic duct pressures were recorded just cranial to the entry of the CrVC into the right atrium after 5 minutes of CrVC occlusion. The balloon was deflated and the catheter removed. For each baseline and 5-minute post–CrVC occlusion measurement, 3 heartbeats were selected within 6 cardiac cycles of the time point and the mean of each variable was calculated.

TD embolization—Initially, TD embolization was achieved by use of a combination of 4 to 8 thrombogenic embolization microcoils[r] and cyanoacrylate glue.[s] Later, a liquid embolic agent[t] was used solely for embolization. Delivery of the liquid embolic agent at the TD embolization site was performed according to detailed product instructions. Briefly, the liquid embolic agent was shaken on a mixer for at least 20 minutes prior to its use to allow for homogenous tantalum radiopacity. The infusion microcatheter[u] was flushed with saline solution. Dimethyl sulfoxide and the liquid embolic agent were prepared in separate 1-mL Luer lock syringes. The catheter (dead space) volume of dimethyl sulfoxide was injected into the microcatheter. Fluoroscopic guidance was used during injection of the liquid embolic agent, which was performed manually at a rate not exceeding 0.3 mL/min until adequate embolization was seen fluoroscopically.[28] When catheterization was deemed impossible or when embolization of the TD was deemed adequate, the dog was euthanized with an overdose of pentobarbital[v] administered IV.

Postmortem examinations were performed by the investigators (AS and BAB). In dogs in which TD catheterization and embolization was achieved, embolization was verified by flushing methylene blue[w] through the mesenteric catheter to identify TD embolization. The TD was then dissected from the surrounding mediastinal tissues and examined.

Statistical analysis—A generalized linear mixed model was fitted to the TD pressure data with balloon occlusion as a fixed effect and analyzed by use of commercially available statistical software.[x] To account for repeated measures over time in each dog, various error structures (autoregressive [n = 1], heterogeneous autoregressive [1], Toeplitz [2], heterogeneous Toeplitz [2], and unstructured and unstructured [2]) were tested. Random effects were chosen on the basis

of the Akaike information criterion. Comprehensive residual analyses were performed to determine whether the ANOVA assumptions were met; the residuals were formally tested for normality (Kolmogorov-Smirnov, Shapiro-Wilk, Anderson-Darling, and Cramér-von Mises tests) and were plotted against the predicted values and variables used in the model. Such residual analyses may reveal outliers, unequal variances or other ANOVA-assumption violations, or the need for data transformation. Values of $P < 0.05$ were considered significant.

Results

In all 15 dogs, an efferent mesenteric lymphatic vessel was successfully catheterized and lymphangiography was performed by injecting iodinated contrast material. The mean volume required to fill the CC and TD was 2.9 mL (range, 1.8 to 7 mL). Complete lymphangiographic images including the CC, TD, and lymphaticovenous anastomosis were available for 13 dogs. Procedures were successful in 2 dogs, but the images were not recorded because of technical difficulties. The CC was extremely variable in size, shape, and location. Eight of the 13 CCs assessed on fluoroscopic images were classified as small at their maximum diameter (defined as ≤ 50% width of L2). Most often, the CC was fusiform in shape (**Figure 2**). It was located at least partially within the caudal rib cage in 8 of 13 dogs. Multiple variations in the branching of the TD and its anatomy at the lymphaticovenous anastomosis were observed. Ten of 13 dogs had evidence of filling defects within the CC (**Figure 3**).

The CC was successfully punctured with a diagnostic needle in all dogs, but a guide wire could be advanced into the TD of only 9 of 15 dogs (**Figure 4**). In some dogs, repeated puncture was possible after an initial failed puncture; in others, repeated puncture was impossible because of extravasated contrast medium obscuring visualization or the position of the CC within the rib cage. A wire was advanced within the CC in only 3 of the first 8 dogs and in 6 of the final 7 dogs. In 5 of 9 dogs, a vascular access sheath was successfully advanced over the guide wire into the TD. In 1 of these 5 dogs, the TD was ruptured during insertion of a 4F catheter; pressure measurement and embolization were not attempted in that dog. In 4 of 9 dogs, manipulation of the sheath during attempts to advance it over the guide wire led to the wire backing out of the CC and leakage of contrast material into the retroperitoneal space, which precluded further puncture.

Thoracic duct pressure was measured at rest and after acute balloon occlusion of the CrVC in 2 positions

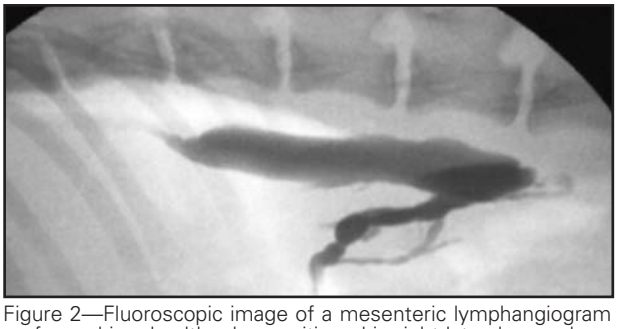

Figure 2—Fluoroscopic image of a mesenteric lymphangiogram performed in a healthy dog positioned in right lateral recumbency. The typical fusiform appearance of the CC is evident.

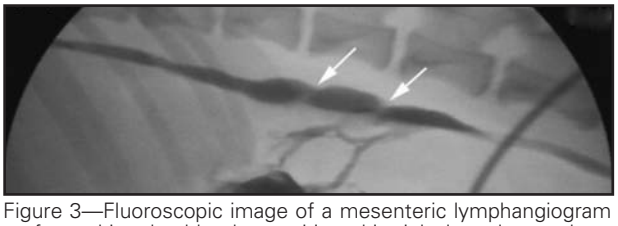

Figure 3—Fluoroscopic image of a mesenteric lymphangiogram performed in a healthy dog positioned in right lateral recumbency. Filling defects are evident within the CC (arrows).

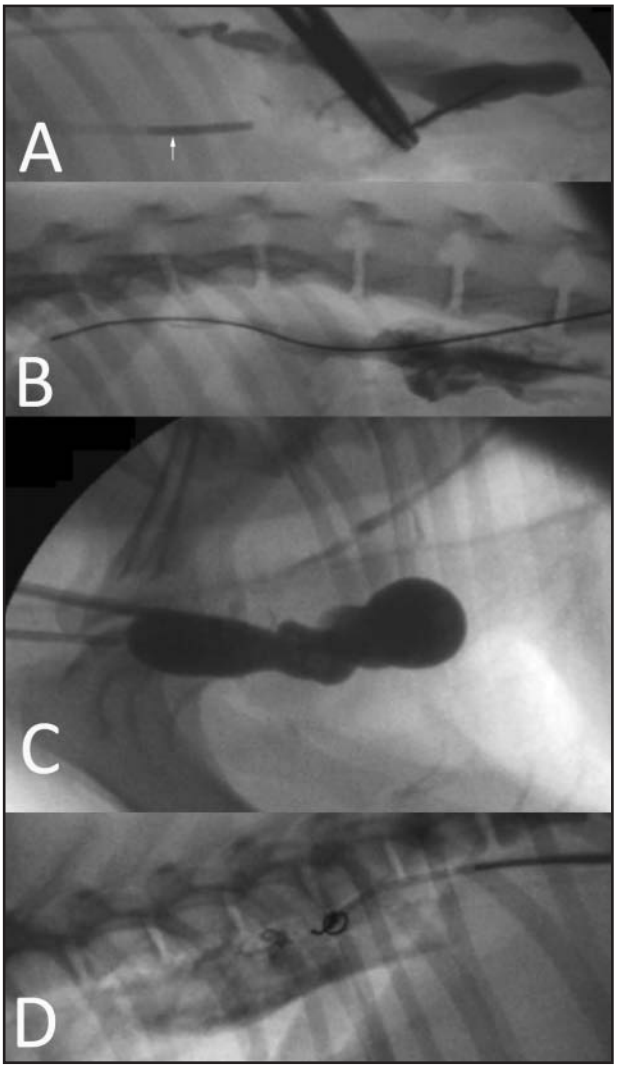

Figure 4—Representative fluoroscopic images from a mesenteric lymphangiogram in a healthy dog positioned in right lateral recumbency. A—The CC has been percutaneously punctured with a diagnostic needle. Needle drivers are used to hold the diagnostic needle to limit radiation exposure to the operator's hands. A marker catheter (1-cm marks) has been placed within the esophagus (arrow). B—The CC has been successfully accessed. A guide wire has been advanced into the TD. C—A balloon catheter has been inflated with contrast agent and is obstructing TD outflow at the lymphaticovenous anastomosis. The cranial TD anatomy is seen at the level of the lymphaticovenous anastomosis. D—The TD is outlined after embolization with microcoils and cyanoacrylate glue. One large and several small microcoils are visible within the TD cranial to the diaphragm.

in 4 dogs (Figure 4). The median pressure increase in the TD after 5 minutes of acute CrVC balloon occlusion at the level of the lymphaticovenous junction and further caudal in the CrVC just cranial to its entry into the right atrium was 3.1 mm Hg (range, 0.47 to 16.02 mm Hg) and 3.12 mm Hg (range, 0.71 to 9.07 mm Hg), respectively. The median increase in JVP after 5 minutes of acute CrVC balloon occlusion at the level of the lymphaticovenous junction and further caudal in the CrVC just cranial to its entry into the right atrium was 20.07 mm Hg (range, 13.76 to 22.95 mm Hg) and 27.19 mm Hg (range, 22.97 to 31.23 mm Hg), respectively. Once the TD pressure data were analyzed statistically, a logarithmic transformation was required. Baseline and occlusion JVPs were significantly ($P < 0.001$) different. A significant ($P < 0.001$) effect of JVP on TD pressure was also found. The TD pressure increased significantly ($P = 0.022$) in all dogs during acute balloon occlusion, compared with baseline TD pressure.

All 4 dogs in which TD catheterization was achieved underwent TD embolization with a combination of thrombogenic microcoils and cyanoacrylate (2 dogs) or the liquid embolic agent (2 dogs; Figure 4). In the first dog, cyanoacrylate glue injection was performed quickly followed by immediate postembolization lymphangiography. Glue emboli in the right atrium, ventricle, and pulmonary artery were noted at postmortem examination in this dog. In the second dog, cyanoacrylate embolization was performed by slow injection to prevent embolization into the heart and lungs, which resulted in the microcatheter used for delivery becoming glued into place. In the final 2 dogs in which TD embolization was performed, delivery of the liquid embolic agent occurred without complication. Complete embolization was confirmed by mesenteric injection of methylene blue in all 4 dogs on the basis of absence of flow across the embolized TD segment at postmortem examination.

In the remaining 6 dogs in which CC puncture and wire advancement were not possible, ≥ 1 of the following was encountered: bending of the flexible diagnostic needle through the musculature, an inability to angle the needle between the ribs to allow insertion in a cranial direction, and an inability to advance the wire beyond the filling defects identified within the CC on lymphangiography.

Discussion

Findings of the present feasibility study suggested the novel technique for PCETD can be performed successfully in healthy dogs and holds promise as a minimally invasive technique for the management of dogs with idiopathic chylothorax. However, because of the low number of successful embolizations achieved here, PCETD cannot be recommended for routine use in clinical practice. Obtaining CC access was technically demanding, and further modification of equipment and techniques will likely improve the success rate of the procedure in the future.

A considerable amount of learning was involved in puncture and wire advancement into the CC, with success in 6 of the final 7 dogs, compared with only 3 of the first 8 dogs. Without successful CC puncture, PCETD cannot be attempted, making puncture the most critical step of the procedure. With surgeon experience and technique modifications, CC puncture and wire advancement became more straightforward.

In the 6 dogs in which CC puncture and wire advancement were not possible, a complication encountered was bending of the flexible diagnostic needle through the sublumbar musculature, preventing sufficient angulation of the needle between the ribs to allow insertion within the CC. When humans undergo PCETD, they are in a supine position, similar to dorsal recumbency in veterinary patients.[24,25] Humans have a much shorter dorsoventral dimension, which minimizes potential interference from intra-abdominal structures. When TD catheterization and embolization in pigs was first performed as a model for humans, the pigs were positioned in dorsal recumbency and the CC was accessed through a similar ventrodorsal approach.[26] A custom-made 16-gauge, 10-cm-long, blunt stainless steel cannula was inserted IP and used as a stiffening guide through which the diagnostic needle was inserted to prevent it from bending during CC puncture.[26] A refinement of this instrument is now available for use in humans. A similar instrument may have been useful to prevent inappropriate bending of the thin diagnostic needle when attempting CC puncture in the present study.

Dogs have a much shorter lateral-to-lateral dimension than humans do, and the canine CC is intimately associated with the dorsal aspect of the abdominal aorta.[29] For these reasons, we chose to obtain CC access through a lateral approach with the dogs in lateral recumbency. Additional research on this technique could focus on the feasibility of PCETD with the dog positioned in dorsal recumbency. Ventral access would remove the need for the needle to travel through the sublumbar musculature and might improve the rate of successful access into the CC. Conversely, a ventral approach would require a longer and more flexible needle to be used and might complicate puncture of the CC because of its location dorsal to the aorta. Complications related to inadvertent puncture of the aorta or gastrointestinal tract are possible, but did not happen when this technique was performed in pigs and humans when the needle was angled cranially above the transverse colon and to the right of midline to avoid the aorta.[25,30]

Advancing a vascular access sheath over the wire was the second limiting step to this procedure, with successful advancement in only 5 of 9 dogs. In several dogs, advancement was complicated by interference of the abdominal soft tissues, which caused the sheath to bend. Bending of the vascular access sheath prevented further advancement, led to the wire backing out of the TD or CC, or resulted in rupture of the CC or TD. This problem was compounded by the fact that 2 sequential sheaths had to be inserted over floppy wires of increasing diameter. Success rates for advancing the access sheath over the wire improved over the course of the study and with use of the percutaneous access set instead of the regular vascular access sheath. The hydrophilic coating of the percutaneous access set allows it to glide easily through the tissues, and its tapered dilator and stiff inner cannula allow direct access over a

0.018-inch wire, removing the need for a vascular access sheath to first be inserted and thus eliminating an entire step. In addition, the cannulated stainless steel guide contained within the percutaneous access set stiffens the sheath and prevents bending during insertion though the body wall and sublumbar musculature. This modification allowed for less manipulation within the TD, decreasing the chance of iatrogenic rupture.

A 71% success rate was recently reported for PCETD in the treatment of traumatic chylothorax in humans.[25] The authors of that report state that the ability to catheterize the CC and TD is the single most important factor related to the success rate of PCETD. Factors stated to affect successful catheterization in people include operator experience, the patient's body condition, and the ability to see lymphatic anatomy.[25] Interestingly, those authors also describe a high degree of learning associated with this technique, which is similar to our experience.

Lymphangiography revealed variability in the size and location of the CC. Eight of 13 dogs had a small CC that was ≤ 50% the width of L2 in the lateral fluoroscopic image. Subjectively, initial needle puncture was easier with larger CCs. Despite this limitation, successful puncture of several small CCs was achieved in the final 7 dogs. Accessing small lymphatic channels (2 to 3 mm) is also possible in humans without a CC.[30]

Eight of 13 dogs had a CC that was partially located within the rib cage. When the CC was located under the ribs, a perpendicular approach through an intercostal space was necessary because the ribs prevented caudocranial angulation of the diagnostic needle. A perpendicular approach to the CC made advancement of the wire in a cranial direction within the TD difficult and advancement of the vascular access sheath over the wire impossible. Caudocranial angulation of the needle during initial puncture was easiest when the CC was located caudal to the rib cage; this allowed cranial angulation of the wire and vascular access sheath, facilitating CC access. Careful examination of the CC size and location during preoperative lymphangiography will be important for selecting dogs to undergo PCETD.

In 10 of 13 dogs, lymphangiography revealed what appeared to be filling defects within the CC. To our knowledge, such filling defects in dogs have not previously been described. On the basis of a previous anatomic study[31] and the fact that our guide wire appeared to bounce against the fluoroscopically visible defects, preventing cranial advancement of the wire in some dogs, we hypothesized that these defects might represent lymphatic valves. However, such valves were reportedly not identified in a study[31] of canine CCs in which transmission electron microscopy was used. Furthermore, valves within the CC should theoretically open from a caudal to cranial direction and should allow guide wire advancement within the CC and TD. Sacculation of the CC of humans has been reported and was believed to result from the confluence of lymphatic vessels at the CC[32]; this could not be confirmed in the dogs of the present study. Another possible explanation would be that the filling defects in the study dogs resulted from vertebral artery branches creating indentations on either side of the distended CC. This would result in less contrast material in those areas and could explain the fluoroscopic appearance of filling defects. However, this possibility does not entirely explain why guide wire advancement was not possible in some dogs unless the indentation created by the vessels physically obstructed the TD at that level. Second-plane fluoroscopy would have allowed determination of the width of the CC in a lateral-to-lateral plane but was not performed in our study. Postmortem examination of 4 dogs revealed some degree of indentation of the CC by the vertebral arteries in dogs with successful embolization. Preoperative lymphangiographic examination will be important for identifying the number and location of CC filling defects because they may prevent successful catheterization.

The combination of microcoils and cyanoacrylate glue for embolization of the TD was first reported after recanalization of the TD was performed in a human in whom microcoils were used as the sole method of embolization.[33] In a recent study[25] of PCETD in 109 humans treated for traumatic chylothorax, embolization with microcoils alone also resulted in higher failure rates, compared with the failure rate when microcoils were combined with liquid embolic agents. Although the combination of microcoils and embolic glue resulted in excellent embolization of the TD as judged by postmortem examination of 2 dogs of the present study, the larger number of steps required to pack multiple microcoils within the TD and the unpredictable rate of polymerization of cyanoacrylate glue were of concern.

The nonadhesive liquid embolic agent used in this study is composed of an ethylene-vinyl-alcohol copolymer dissolved in dimethyl sulfoxide and suspended micronized tantalum powder to provide contrast for visualization during fluoroscopy. It is approved by the US FDA to treat intracranial arteriovenous malformations in humans.[34] This agent has several advantages over other embolic agents when used for the treatment of arteriovenous malformations.[34] A unique feature is its cohesive property, which prevents embolization into the heart and lungs and the microcatheter tip from being glued within the vessel.[35] Thoracic duct embolization in the last 2 dogs of the present study was easier technically, and fewer complications were encountered when the liquid embolic agent was used instead of microcoils and cyanoacrylate glue. The agent was also used in 18 of 71 human patients in a recent study[25] and should be investigated further for performance of TD embolization in dogs.

At some institutions, humans affected by low-output (< 1,000 mL/d) traumatic chylothorax are initially managed conservatively.[36] Some authors suggest that high-output chylothorax warrants surgical TDL with open or video-assisted thoracoscopy.[36] Recently, PCETD has been described as an alternative to thoracotomy or video-assisted thoracoscopy for TDL and, in some institutions, has become the initial treatment of choice for traumatic chylothorax in humans because of its minimal invasiveness.[24,25] The development of a minimally invasive technique for PCETD in dogs affected by idiopathic chylothorax could remove the need for invasive thoracotomy required for TDL and

pericardectomy. In the present study, a laparotomy was performed to catheterize a mesenteric lymphatic vessel for lymphangiography. Direct mesenteric lymphangiography was chosen for this study to ensure adequate anatomic visualization and to allow for repeated injection during the developmental stages of this technique. Popliteal lymphangiography and laparoscopic or ultrasound-guided mesenteric lymphangiography are minimally invasive alternatives to direct mesenteric catheterization in dogs.[37–40] Because repeated injection of contrast medium can be performed via the TD catheter once access has been obtained, these techniques could be used in combination with PCETD to achieve a completely minimally invasive technique. Video-assisted laparoscopy could perhaps be used to perform direct mesenteric catheterization for lymphangiography or the catheterization of the CC or TD for embolization.

In dogs, a high CVP is a potential cause of thoracic lymphangiectasia and subsequent transmural leakage of chyle, resulting in chylothorax.[5,6] Chylothorax has been experimentally created by ligation of the CrVC distal to the entrance of the azygous vein.[41,42] In contrast, experimental TDL at the level of the lymphaticovenous anastomosis does not lead to chylothorax in dogs.[41] The presence of compensatory lymphaticovenous communications caudal to the site of entry of the TD is likely responsible for the absence of secondary chylothorax in these instances in some dogs.[41] Furthermore, chylothorax secondary to thrombosis of the CrVC is also reported rarely and appears to occur only with massive jugulocaval thrombosis that occludes outflow of the TD and any collateral lymphaticovenous communications caudal to the site of TD entry into the CrVC.[20,21] Acute balloon occlusion of the CrVC was performed at 2 anatomic locations in the present study in an attempt to simulate massive CrVC thrombosis, leading to an increase in CVP. Obstruction of the CrVC with a balloon catheter at the level of the lymphaticovenous junction was intended to obstruct emptying of chyle into the venous system at the level of lymphaticovenous anastomosis. Obstruction of the CrVC just cranial to its entry into the right atrium was intended to also obstruct emptying of any collateral lymphaticovenous communications present. Because of the length of the balloon available for the study, both balloon positions obstructed the TD entry site and most of the CrVC.

Thoracic duct pressure increased by a median of 3.1 mm Hg in 4 of 4 dogs in which pressure measurements were obtained during occlusion of the CrVC. In 1 dog, TD pressure increased by 16.02 and 9.07 mm Hg after CrVC occlusion for 5 minutes at the level of the lymphaticovenous junction and further caudal in the CrVC just cranial to its entry into the right atrium, respectively. One can speculate that this dog would be at risk for developing thoracic lymphangiectasia and subsequent chylothorax if collateral lymphaticovenous communications did not develop following CrVC thrombosis. A similar study[43] involving sheep was conducted by measuring TD pressure after progressive balloon occlusion of the CrVC. That study demonstrated that TD pressure increases proportionally to external jugular vein pressure in awake sheep. Our findings were not in complete agreement with those findings because the TD pressure increased in all dogs of the present study but not proportionally with JVP. The dogs had been anesthetized for approximately 8 hours, which may have affected their TD and blood pressures, but this possibility is not supported by the unremarkable baseline JVPs. In addition, we cannot confirm that a 5-minute CVC occlusion time was sufficient to create a maximum increase in TD pressure. Increasing the duration of occlusion could have increased the TD pressure further until a certain point at which it might have stabilized or decreased, depending on compensatory mechanisms. The 5-minute occlusion period was chosen because of concerns that long-term acute CVC occlusion would have deleterious cardiovascular effects; however, this did not occur.

The dogs used in the present study were clinically normal and were used primarily for undergraduate surgical exercises. There was no evidence of pleural effusion or thoracic lymphangiectasia in any dogs. However, dogs with chylothorax may have a tortuous TD or thin-walled TD or CC that would make initial CC puncture and wire and sheath advancement more challenging, compared with healthy dogs, and this possibility should be considered if this technique is to be performed in such dogs.

Collateral organ damage was not reported in any experimental cases in the first report[30] of PCETD or when this technique was performed in humans with chylothorax.[25,33,44] Because a full postmortem examination was only performed in the dogs that underwent successful CC puncture and TD embolization in the present study, we are unable to comment on any collateral organ damage that may have occurred during our attempts to access the CC percutaneously in the dogs in which it was unsuccessful. On the basis of previous studies and the fact that major collateral organ trauma was not observed in the 4 dogs that underwent postmortem examination, it can be speculated that PCETD is a safe technique.

Although PCETD was successfully performed in a portion of the dogs in the present study, this minimally invasive technique cannot be recommended for the routine treatment of dogs with idiopathic chylothorax. Modifications to the technique, including the use of a more rigid small-bore needle that would allow more consistent puncture of the CC and a novel vascular sheath that would allow for easier advancement over the guide wire into the TD, may improve the success rate of PCETD and allow it to be used in clinical practice. Our findings also suggest that lymphangiography prior to PCETD is of utmost importance because the location, morphology, and size of the CC and branching of the TD may preclude the use of this technique.

a. Acepromazine maleate (10 mg/mL), Ayerst Laboratories, Montreal, QC, Canada.
b. Morphine (15 mg/mL), Sandoz Canada Inc, Boucherville, QC, Canada.
c. Hydromorphone (2 mg/mL), Sandoz Canada Inc, Boucherville, QC, Canada.
d. Thiopental sodium USP, Hospira Healthcare Corp, Vaughn, ON, Canada.
e. Isoflurane USP, Baxter Corp, Mississauga, ON, Canada.
f. Omnipaque 350 mg of I/mL, GE Healthcare Canada Inc, Mississauga, ON, Canada.
g. AccustickTM Introducer Needle, Boston Scientific Corp, Watertown, Mass.
h. Mini-Access Kit 4F, Infiniti Medical, Malibu, Calif.

i. Weasel wire, Infiniti Medical, Malibu, Calif.
j. 4F 11-cm vascular access sheath, Infiniti Medical, Malibu, Calif.
k. Cope Mandril Guidewire, Cook Inc, Bloomington, Ind.
l. Neff Percutaneous Access Set, Cook Inc, Bloomington, Ind.
m. Check-Flo Performer Assembly, Cook Inc, Bloomington, Ind.
n. 9F, 11-cm vascular sheath, Cook Inc, Bloomington, Ind.
o. Beacon Tip Royal flush plus light flow catheter, Cook Inc, Bloomington, Ind.
p. Powerlab Data acquisition system with LabChart, version 7.0, ADInstruments Ltd, Bella Vista, NSW, Australia.
q. 25-mm × 6-cm maximal diameter balloon catheter, Infiniti Medical, Malibu, Calif.
r. Complex Helical Platinum Coil, Target Therapeutics Inc, Fremont, Calif.
s. Vetbond Tissue Adhesive, n-butyl cyanoacrylate, 3M Health Care, Saint Paul, Minn.
t. Onyx, donated by ev3 Endovascular Co, Irvine, Calif.
u. Echelon-10 Microcatheter, ev3 Endovascular Co, Irvine, Calif.
v. Euthansol, Schering-Plough Animal Health, Pointe-Claire, QC, Canada.
w. Methylene blue, SABEX Inc, Boucherville, QC, Canada.
x. PROC MIXED, SAS, version 9.2, SAS Institute Inc, Cary, NC.

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