Article

Chylothorax associated with thrombosis of the cranial vena cava

Ameet Singh, Brigitte A. Brisson

Abstract — This study reviewed confirmed cases of concurrent chylothorax and cranial vena caval (CrVC) thrombosis in dogs and cats, and determined predisposing factors for the development of chylothorax associated with CrVC thrombosis. The extent and location of the thrombus, the treatment regime, and the outcome are described. In all 4 cases, implantation of a jugular device was a predisposing factor to thrombosis of the CrVC, and there was extensive thrombosis of the CrVC extending from at least 1 jugular vein to just cranial to the heart. Chylothorax resolved in 3 of the 4 cases after medical and/or surgical intervention. The development of chylothorax concurrently with thrombosis of the CrVC in dogs and cats is likely dependent on the extent and location of the thrombus. Veterinary patients with indwelling jugular devices that develop acute respiratory signs should be assessed for chylothorax associated with thrombosis of the CrVC.

Résumé — Chylothorax associé à une thrombose de la veine cave crânienne. Cette étude a examiné des cas confirmés de chylothorax concomitant et de thrombose de la veine cave crânienne (VCCr) chez les chiens et les chats et a déterminé les facteurs prédisposants pour le développement de chylothorax associé à la thrombose de la VCCr. L'étendue et l'emplacement du thrombus, le régime de traitement et le résultat sont décrits. Dans les 4 cas, l'implantation d'un dispositif jugulaire était un facteur prédisposant à la thrombose de la VCCr et il y avait une thrombose importante de la VCCr s'étendant d'au moins 1 veine jugulaire jusqu'à crânialement au cœur. Le chylothorax s'est résorbé dans 3 des 4 cas après l'intervention médicale et/ou chirurgicale. Le développement du chylothorax simultanément avec une thrombose de la VCCr chez les chiens et les chats dépend probablement de l'étendue et de l'emplacement du thrombus. Les patients vétérinaires avec des dispositifs jugulaires à demeure qui développent des signes respiratoires aigus devraient être évalués pour le chylothorax associé à la thrombose de la VCCr.

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Introduction

Chylothorax is characterized by the accumulation of chyle within the thoracic cavity as a result of a disturbance to the thoracic duct (TD) and/or its tributaries (1). Chyle is an irritant and its accumulation within the thoracic cavity can lead to inflammation and fibrosis of the thoracic wall lining, pleural surfaces, and pericardial sac (2). Chylothorax has been described in dogs, cats, rats, and humans (3). It has been suggested that lymphangiectasia of the mediastinal and pleural lymphatics leads to extravasation of chyle into the thoracic cavity (4). In veterinary patients a predisposing cause is rarely identified and chylothorax is most often deemed idiopathic (5). Other reported causes of chylothorax include trauma, cardiac disease, dirofilariosis, neoplasia, cranial vena cava (CrVC) thrombosis, fungal granulomas, and congenital abnormalities of the thoracic duct (6–10). In humans, chylothorax is most commonly a result of neoplasia or trauma, particularly cardiothoracic surgery (11).

Recent clinical reports on chylothorax have suggested that increases in venous hydrostatic pressure may lead to chylothorax (5,12,13). It has been speculated that increased venous pressure impedes emptying of the TD and its tributaries into the venous system, resulting in the development of thoracic lymphangiectasia and subsequent chylothorax (5,12–15). Cardiac disease leading to right heart failure and increased venous pressures is also a reported cause of chylothorax in the dog and cat (3,7,12).

Thrombosis of the CrVC can lead to increased systemic venous pressure and, depending on the anatomic location of the thrombus, could obstruct lymphatic outflow from the thoracic duct into the venous system (3,5,12). The incidence of CrVC thrombosis in dogs and cats is unknown. Chylothorax is an uncommonly reported consequence of CrVC thrombosis in veterinary patients. Chylothorax was noted in 3/17 patients

Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Guelph, Ontario N1G 2W1.

Address all correspondence to Dr. Singh; e-mail: amsingh@uoguelph.ca

Table 1. Summary of characteristics of 4 cases (2 dogs and 2 cats) of concurrent chylothorax and thrombosis of the cranial vena cava

Case	Signalment	Jugular device implanted	Duration of implantation (d)	Time between implantation and respiratory compromise (d)	Concurrent illness	Treatment	Outcome
1	5 y, FS, chow chow	Central venous catheter	1	1	Immune mediated thrombocytopenia	TD ligation	Chylothorax resolved 1 d after surgery
2	7 y, FS, Brittany spaniel	Endocardial pacing wire	1409	1392	3° atrioventricular node block	Right atriotomy for removal of the ventricular pacing wire	Intra-operative cardiac arrest
3	4 y, FS, domestic shorthair	Central venous catheter	9	21	Hepatic lipidosis, pancreatitis	TD ligation	Chylothorax resolved 13 d after surgery
4	2 y, MC, domestic shorthair	Vascular access port (VAP)	22	11	None	Medical management	Chylothorax resolved 92 d after removal of the VAP

FS — female, spayed; MC — male, castrated.

in a study describing the manifestations and associated disease syndromes of dogs with confirmed CrVC thrombosis (16). Since this study assessed cases of CrVC thrombosis that developed complications, it was unable to determine how many patients might have developed CrVC thrombosis without clinical consequence and did not specifically describe these 3 cases. Another report described a single case of chylothorax secondary to thrombosis of the CrVC associated with jugular vein catheterization, which resolved after thromobolytic therapy (8). The mechanism for the unpredictable occurrence of chylothorax associated with CrVC thrombosis in veterinary patients has yet to be elucidated.

The objective of this study was to identify and review all cases diagnosed with concurrent chylothorax and thrombosis of the CrVC between January 1999 and December 2008. Further objectives were to determine potential predisposing factors for the development of chylothorax and CrVC thrombosis, to describe the extent of, and location of the thrombus, the treatment regime and outcome in these cases. We hypothesized that chylothorax associated with a CrVC thrombus would occur rarely, develop in association with the presence of a jugular device within the CrVC, and be associated with an extensive thrombus burden within the CrVC.

Materials and methods

Cases

Medical records from the Ontario Veterinary College Teaching Hospital (OVCTH) of the University of Guelph were searched for dogs or cats that were diagnosed with concurrent chylothorax and thrombosis of the CrVC between January 1999 and December 2008. Criteria for inclusion in the study were that medical records were complete and that concurrent CrVC thrombosis and chylothorax were confirmed. The presence of a CrVC thrombus had to have been confirmed by thoracic ultrasonography or echocardiography. Chylothorax had been confirmed in all cases with cytological evaluation of pleural fluid smears prepared as direct smears and after cytospin preparation, and by confirming that triglyceride levels were greater in the pleural fluid than in the serum.

Data collection

The medical records were reviewed, and information retrieved included signalment, history and presenting complaint, clinical signs, concurrent illnesses, laboratory evaluation, diagnostic imaging results, treatment and outcome.

A search of the medical record database from the OVCTH was also undertaken to determine the number of subcutaneous vascular access ports (VAP), central venous (jugular) catheters, and transvenous endocardial ventricular pacing wires that were placed during the study period in order to calculate the incidence of concurrent chylothorax and CrVC thrombosis in the population.

Results

Two dogs (chow chow and Brittany spaniel) and 2 cats (both domestic shorthair) were diagnosed with concurrent CrVC thrombosis and chylothorax during the study period (Table 1). The ages of the affected animals ranged from 2 to 7 y (median 4.5 y). There were 3 females and 1 male, all were neutered. In all cases the primary clinical sign was respiratory compromise characterized by labored breathing and an increased respiratory rate. This prompted further investigation or assessment eventually leading to the diagnosis of pleural effusion.

Pleural fluid color ranged from pink to white and all pleural fluids were cloudy with total solids ranging from 25 g/L to 48 g/L. The nucleated cell count ranged from 0.4×10^9 to 17.4×10^9/L. The predominant cell types were mesothelial cells (case 1), non-degenerate neutrophils (case 2), and medium-sized lymphocytes (cases 3 and 4). Triglyceride levels were greater in the pleural fluid (2.15 mmol/L to 2.4 mmol/L) than in the serum (0.5 mmol/L to 0.7 mmol/L) in all cases (reference interval: 0.2 to 1.3 mmol/L), confirming the presence of chylothorax. Bacterial culture was performed on all initial samples of pleural fluid and did not yield any growth.

Two cases (1 and 3) had indwelling central venous catheters in the left jugular vein. Case 2 had a transvenous endocardial ventricular pacing wire in the right jugular vein. Case 4 had a subcutaneous vascular access port (VAP) in the right jugular

vein. The length of time the jugular devices remained in the patients ranged from 1 d to 1409 d (median 15.5 d). Time between placement of the jugular device and development of clinical signs was 1 to 1392 d (median 16 d) (Table 1).

During the study period, 8 VAP, 53 transvenous endocardial ventricular pacing wires and approximately 1020 central venous catheters were placed through a jugular vein. The incidence of chylothorax associated with thrombosis of the CrVC for the VAP, transvenous endocardial ventricular pacing wires and central venous catheters were 13% (1/8), 1.9% (1/53), and 0.2% (2/1020), respectively. The overall incidence of chylothorax associated with thrombosis of the CrVC for all jugular devices was 0.37% (4/1081).

Thrombosis of the CrVC was confirmed with thoracic (mediastinal and cervical) ultrasonography in cases 1, 3, and 4 and with echocardiography in case 2. In case 1 a thoracic ultrasound revealed lack of color flow signal, as measured using color flow Doppler ultrasonography, in the CrVC. Selective and non-selective angiography confirmed a filling defect within the CrVC extending from the jugular veins to the heart. Thoracic ultrasonography in case 3 revealed a large thrombus within the left and right jugular veins at their confluence with the CrVC and extending to the level of the right atrium. In case 4, a thoracic ultrasound confirmed a large thrombus within the CrVC extending to the level of the right atrium and surrounding the distal tip of the VAP. Selective angiography revealed a filling defect comprising most of the CrVC lumen cranial to the heart. Echocardiography performed in case 2 confirmed the presence of a large CrVC thrombus surrounding the transvenous endocardial ventricular pacing wire, extending from the jugular veins into the right atrium and ventricle. Echocardiography, performed in all cases, did not reveal a cardiogenic cause for the chylothorax.

Three of the 4 cases were being treated for a previously diagnosed illness. Case 1 had been diagnosed with primary immune-mediated thrombocytopenia (ITP) and was receiving Prednisone (Novopharm, Toronto, Ontario), 1 mg/kg, PO, q12h and Cyclosporine (Novartis, Dorval, Quebec), 5 mg/kg, PO, q12h therapy. A jugular catheter was placed in the left jugular vein on day 1 of hospitalization and removed 1 d later when the patient developed respiratory distress and chylothorax and CrVC thrombosis were diagnosed. Case 2 was diagnosed with 3° atrioventricular node block based on electrocardiographic (ECG) evaluation. A transvenous endocardial ventricular pacing wire was implanted into the right ventricle via the right jugular vein without complication. The patient was discharged uneventfully 1 d after lead placement and readmitted for signs of dyspnea 1392 d post-implantation. Case 3 was diagnosed with hepatic lipidosis and pancreatitis based on results of clinical signs, abdominal ultrasonography, and laboratory evaluation which included feline specific pancreatic lipase immunoreactivity (104.9 μg/L; reference range: 2.0 to 6.8 μg/L). A left jugular catheter was placed during hospitalization and removed 9 d later. Signs of dyspnea developed 21 d after catheter implantation. Case 4 had a VAP surgically implanted in the right jugular vein as part of a research study evaluating methods of blood collection in healthy feline blood donors at the OVCTH. Signs of dyspnea were noted 11 d after implantation.

All patients were evaluated at the time of jugular device placement. The complete blood (cell) count (CBC) was normal in cases 2 and 4. Case 1 had a hematocrit of 22% (reference range: 39% to 56%) and a platelet count of 16 (reference range: 117 to 418 $\times$ 10^9/L). Case 3 had a hematocrit of 25% (reference range: 39% to 56%). Serum biochemical evaluations were normal in cases 1, 2, and 4. Biochemical analysis confirmed that case 3 had evidence of cholestasis. Prothrombin time (PT), partial thromboplastin time (PTT), and fibrin degradation products were measured in case 1 and were within normal limits. Activated clotting time (ACT) was measured in case 3 and was within normal limits.

Medical management of chylothorax in case 4 consisted of removal of the VAP (22 d after implantation), pleural evacuation (q4h via thoracostomy tube for 2 wk then intermittent thoracocentesis 2 to 3×/wk for 2 wk and then 1–2×/wk for 2 mo), dietary modification and administration of a benzopyrone (Rutin; Shoppers Drug Mart, North York, Ontario), 250 mg, PO, q12h. Resolution of the chylothorax was documented 92 d after removal of the VAP. This cat remains normal 8 y after resolution as assessed by thoracic radiographs and clinical findings.

Surgical removal of the transvenous endocardial ventricular pacing wire and organized thrombus was performed in case 2, 1409 d after implantation. Total venous occlusion and cardioplegia were used intraoperatively to perform a right atriotomy allowing removal of the transvenous endocardial ventricular pacing wire. An epicardial pacing wire was placed on the right ventricular free wall. This dog died in the immediate postoperative period as a result of malfunction of the temporary pacing wire and prolonged cardiac arrest.

In case 1, en-bloc TD ligation was performed 24 d after diagnosis (23 d after removal of the jugular catheter) and the chylothorax resolved immediately after surgery. In case 3, medical management consisting of intermittent thoracocentesis, dietary modification, benzopyrone therapy (250 mg, PO, q12h) and low molecular weight heparin (Tinzaparin; Leo Pharma, Richmond Hill, Ontario), 100 U/kg, SQ, q12h, was performed for 28 d. Thoracocentesis was required every 1 to 5 d. Although a thoracic ultrasound performed 44 d after the onset of chylothorax revealed evidence of blood flow through both jugular veins and CrVC, the chylothorax persisted. Thoracic duct ligation was performed 63 d after chylothorax was first diagnosed. Chylothorax resolved in this case 13 d after surgery, and the cat remained asymptomatic 1 y later.

Discussion

Cranial vena caval thrombosis has been reported as a cause of chylothorax in dogs and cats (8,16). All cases reported in this study were diagnosed with CrVC thrombosis and chylothorax after implantation of a jugular device in the right or left jugular vein for an unrelated condition. Although chylothorax is presumed to have developed secondary to CrVC thrombosis in all cases in this study, this cannot be confirmed. All caval thrombi in this study were extensive and involved 1 or both jugular veins and most of the length of the CrVC to the level of the right atrium. Resolution of the chylothorax was seen in the 3 cases that survived to discharge.

In 2 previous studies, ligation of the TD at its entry into the venous system at the level of the jugulocaval angle did not lead to chylothorax (14,15). This was thought to be a result of redirected lymphatic flow through collateral channels that enter the CrVC caudal to the TD itself. Further experimental work revealed that ligation of the CrVC caudal to the entrance of the azygous vein caused chylothorax in 70% (14) and 54% (15) of dogs and 61% (15) of cats. Blalock (15) also reported that dogs whose TD was ligated mid-thorax prior to CrVC ligation did not develop chylothorax. These findings provide evidence for the presence of collateral lymphatic channels within the thoracic cavity and could explain why some animals developed chylothorax and others did not (15).

It has been speculated that impeding outflow of the TD and its collateral channels would result in chylothorax (13–15). Numerous reports of chylothorax secondary to venous hypertension as a result of congestive heart failure, constrictive pericarditis, blastomycotic granuloma, and other etiologies are found in the literature (3,7,12). Kramer et al (17) speculated that critically located thrombosis of the superior vena cava at the entry of the major collateral lymphatics into the venous system would cause backflow of chyle into tributaries of the TD and could explain the inconsistent development of chylothorax secondary to thrombosis of the CrVC in human pediatric patients. Once pleural and thoracic lymphatics become engorged, chyle leaks across the parietal pleura, eventually leading to chylothorax (17). A similar anatomic pathophysiology may exist in canine and feline patients.

Lymphangiography of the canine TD confirms a great variety of anatomic differences along the duct and at its entry into the jugulo-caval angle (18,19). Kagan et al (18) demonstrated the many variations in the anatomy of the canine TD by performing lymphangiography in 20 dogs and showing that only 1 dog had a single lymphatic vessel that extended from the cisterna chyli cranially into the thorax terminating at the jugulo-caval angle. The remaining dogs had multiple collateral lymphatic branching off the TD and various locations of termination of the TD into the venous system. This variation in the anatomy of the canine TD and its collateral supply is likely an important factor in explaining why only a small number of patients with CrVC thrombosis develop chylothorax.

If a CrVC thrombus extends from the jugulo-caval angle (where the TD enters the venous system) to the right atrium it will obstruct not only TD drainage but also drainage of the major collateral lymphatics. Such massive thrombosis will likely obstruct most lymphatic drainage into the venous circulation, possibly leading to thoracic lymphangiectasia and subsequent chylothorax (17). All 4 cases in our study had extensive CrVC thrombosis obstructing a significant portion of the CrVC, likely preventing collateral lymphatic drainage. Ultimately, the key factor in the development of chylothorax associated with a CrVC thrombosis may be the location of the thrombus and obstruction of collateral lymphatic drainage. This may explain why only a small percentage of patients with CrVC thrombosis develop chylothorax as the thrombus must be extensive and in a precise anatomic location in order to obstruct collateral lymphatic flow into the venous circulation.

Jugular catheters have been associated with the development of CrVC thrombosis (20–22). In this study, the jugular implants were placed in the right or left jugular vein at equal frequency suggesting that side does not play a role in the development of concurrent CrVC thrombosis and chylothorax. Placement of jugular devices can alter blood flow, damage vascular endothelium and activate the coagulation cascade which can lead to a prothrombotic state (20–22). In the cases presented here, concurrent diseases that lead to prothrombotic conditions (immune-mediate thrombocytopenia and pancreatitis) may also have contributed to the development of CrVC thrombosis. Several studies in human patients with long-term jugular venous access have determined that the catheter tip position was an important risk factor for developing CrVC thrombosis (21). Catheter tips that were positioned in the brachiocephalic vein or in the cranial part of the superior vena cava were associated with a higher risk of thrombosis. At the OVCTH, it is assumed that most central venous catheters placed in the jugular vein extend into the CrVC; however, tip position is rarely confirmed after placement. Both the VAP and transvenous endocardial ventricular pacing wire were confirmed to extend into and beyond the CrVC, respectively.

Chylothorax associated with thrombosis of the CrVC was a rarely recognized event with a mean incidence of 0.37% (4/1081) amongst all cases of jugular devices implanted. As the number of jugular catheters placed was based on the number of catheters purchased during that period, this number is likely an overestimate of the number of catheters actually placed. Regardless, the rate of concurrent chylothorax and CrVC thrombosis was low in this study.

In this study, a large range of time elapsed between jugular device implantation and development of clinical signs related to chylothorax (Table 1). The time was 1 d for case 1 and 1392 d for case 2. Case 1 was diagnosed with immune-mediated thrombocytopenia that may have hastened thrombosis due to systemic inflammation and vasculitis. Presumably, it was the anatomic location of the thrombus and its extensive nature which led to the very rapid development of chylothorax in this case as most lymphatic flow into the venous circulation was likely obstructed. Despite case 2 having a large thrombus extending from the right ventricle and right atrium to the jugular veins, thrombus formation was likely a slow process given the time between implantation of the lead and development of clinical signs of chylothorax. This is supported by the chronic nature of the organized thrombus noted at surgery and postmortem examination. Lymphangiography was unfortunately not performed in either of these clinical cases and thoracic lymphangiectasia was therefore not confirmed.

If chylothorax develops secondary to CrVC thrombosis, it should theoretically resolve once the lymphatic outflow obstruction has been removed. Supporting this is a report of successful resolution of chylothorax secondary to CrVC thrombosis in a dog using recombinant tissue-plasminogen activator (8). In contrast, clinical evidence of chylothorax persisted despite ultrasonographic evidence of resolution of the CrVC thrombus in case 3. Incomplete thrombus resolution could explain the lack of response for several days after documentation of flow within the

CrVC. In addition, it has been shown that in human patients with venous hypertension, the liver can act as a reservoir for lymphatic fluid by dilating its lymphatics and interstitial spaces (23). One potential reason for persistent accumulation of chyle in case 3 after resolution of the CrVC thrombosis could be a compensatory increase in lymphatic outflow due to emptying of the hepatic lymph reservoir (23). Thoracic lymphangiectasia may also have persisted as a result of chronic lymphatic outflow obstruction and could have lead to persistent chylothorax beyond the time of resolution of the CrVC thrombus. Severe thoracic lymphangiectasia likely takes days to weeks to resolve and may have coincided with resolution of chylothorax 13 d after TD ligation in this case.

In case 4, chylothorax resolved 82 d after diagnosis and initiation of medical management consisting of intermittent thoracocentesis, dietary modifications and removal of the VAP. Greenberg and Weisse (13) reported a case of chylothorax secondary to inadvertent ligation of the left brachiocephalic vein in a cat which underwent spontaneous resolution with medical management alone. The authors hypothesized that chylothorax was a result of impaired drainage of lymph into the venous system secondary to ligation of the left brachiocephalic vein which could potentially lead to similar hemodynamic alterations as a CrVC thrombosis. In an experimental study, which supported this theory, TD ligation performed at the level of the 6th or 7th intercostal space in cats demonstrated the establishment of collateral lymphatic circulation to either the right lymphatic duct or the azygous vein within 7 and 66 d of ligation, respectively (24). Although a thoracic ultrasound was not performed to determine if the CrVC thrombus actually resolved in this case, development of collateral lymphatics could have occurred.

A major limitation to this study was that systemic venous pressure measurements were not available before and after resolution of the effusion of chyle. Previous experimental work examining the effect of central venous pressure (CVP) on TD drainage and pressure found that an elevated CVP was a major impediment to the TD emptying into the venous circulation (25,26). In these studies, CVP was experimentally elevated through massive crystalloid infusion (25) and asbestos pericarditis (26); however, the long-term effect of elevated CVP (development of chylothorax) was not evaluated in either study. Furthermore, the authors of the present paper have documented that acute balloon occlusion of the CrVC led to immediate and significant elevations in CVP in 4/4 dogs and thoracic duct pressure in 3/4 dogs (unpublished data). The fact that chylothorax does not develop in all cases of congestive heart failure, a condition in which CVP is consistently elevated, suggests that an elevated CVP is not the only factor involved in the development of chylothorax secondary to thrombosis of the CrVC, and that other factors are likely to exist (12). One such factor is likely the anatomic variations of the TD and its tributaries between patients.

Jugular catheters and other jugular devices can lead to thrombosis of the jugular veins and CrVC. Extensive thrombosis of the CrVC was associated with the development of chylothorax in all 4 patients in the present study. Thrombi that form in a strategic location within the CrVC and obstruct the TD and major collateral lymphatic outflow have been associated with the development of chylothorax in human pediatric patients (17). A similar mechanism likely exists for the inconsistent development of chylothorax secondary to CrVC thrombosis in dogs and cats. Veterinary patients with central venous catheters that develop acute respiratory signs should be assessed for chylothorax secondary to thrombosis of the CrVC. The etiology of this syndrome is complex and likely multifactorial but resolution appears common. CVJ

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Book Review
Compte rendu de livre

"Pigmented Spectacles." Conversations with Dr. Ian Ayrton Earle Kirby

Chesterton D. 5 Snowberry Court, Caledon Village, Ontario L7K 0B5, 180 pp. $16.00 CDN.

The first 3 chapters of this book are about Earle's early years growing up in St. Vincent with an excellent description of Caribbean island life in the 1920s and 1930s. These pages also describe his close relationship with his parents, grandparents, aunts and uncles who impressed on him that he was special and had much to offer to his small island home. They instilled a pride in his ancestry and heritage that stayed with him for life. As a child, Earle was free to roam the hills, valleys, and forest with his friends and he developed a lifelong interest in the island's plant, bird, and animal life. He respected the education he received under the Cambridge University system and he excelled at school. Earle loved learning about everything.

Earle received a scholarship to attend the Imperial College of Tropical Agriculture and Chapter 4 is about his 3 years in Trinidad, the war years, and his work with the St. Vincent Ministry of Agriculture when he returned home. Earle received a scholarship to attend the Ontario Veterinary College in Guelph and Chapter 5 describes his 4 years at OVC, graduating in 1952, the last year of the 4-year Veterinary Degree course. Here, Earle reminiscences about his experiences in Canada, Guelph, and his education at OVC. Comments from several of his classmates of their impressions of Earle are included and we read too about a college romance.

Chapter 6 is about Earle's veterinary work in St. Vincent as Chief Veterinary Officer, his irritation with bureaucracy, and his developing interest in the archeological history of St. Vincent as he was shown the many artifacts farmers had found.

Earle's marriage to Monica, their family life, their interest in the welfare and education of the children on the island are outlined in Chapter 7. Earle tried to pass his pride in his heritage on to the children and encouraged them to work to protect the bird and animal species of the island, particularly the St. Vincent parrot.

In 1957, Earle was accepted for a post graduate degree at the Royal Veterinary College, School of Tropical Medicine in Edinburgh, Scotland as detailed in chapter 8. He also attended a 10-day workshop in Denmark.

Chapters 9 and 10 revolve around Earle's deepening interest in the history and the archeological evidence of the prehistory of St. Vincent that continued full time after his retirement from the Ministry. Earle was convinced that people from Mali in West Africa had settled in the area prior to 1400 AD and that the Black Caribs descended from them. He wrote papers on his discoveries, spoke at meetings, and was involved in the founding of The St. Vincent Archeological and Historical Society. He also developed the St. Vincent Archeological Museum.

The people of St.Vincent recognized "Doc Kirby" for his efforts to protect the environment, his willingness to help and guide others, and his promotion of the history of the islands. Earle received the 'Order of the British Empire' and was honored in St. Vincent with a stamp in 'The Year of the Elder.' He was awarded, posthumously, the Euan P. McFarlane Award for Outstanding Environmental Leadership in the Insular Caribbean. The final 2 chapters include this recognition and testimonials from friends.

The book is available from David Chesterton (d.chesterton@sympatico.ca) for $16.00 in Canada, including taxes, packaging, and shipping.

About the Author. David Chesterton decided someone should write a biography of Dr. Ian Ayrton Earle Kirby. He first met Dr. Kirby in 1972 when he escorted students from Humber College to the Islands. For the next 17 years he and his wife Anne visited Earle when sailing in St. Vincent and the Grenadines. He and Anne then led Elderhostel groups to St. Vincent for several years and always visited the Botanical Gardens (the oldest in the Caribbean) and the museum to talk to Earle.

Reviewed by ***Richard Julian, DVM,*** *Professor Emeritus, Department of Pathobiology, OVC, University of Guelph, Guelph, Ontario N1G 2W1.*