Hemoptysis in Cattle

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Hemoptysis, the coughing-up of blood or blood-stained sputum, can be a dramatic clinical sign in cattle. A minor degree of hemoptysis may infrequently be observed in animals with right-sided infective endocarditis, but, in our experience, massive hemoptysis is virtually indicative of one disease–thrombosis of the posterior vena cava (PVCT, caval thrombosis). This condition has been known to clinicians and pathologists in Europe for over 80 years (4,10,11,12,-14,16) and is now recognised as an increasing problem in the United States of America (Jensen, R., unpublished results). PVCT is a relatively frequent cause of respiratory distress in individual adult cattle admitted to this school and 23 cases were encountered in 273 adult cattle (aged two years or over) with clinically significant respiratory disease that were examined at necropsy during the period 1969-1974 (3,12). PVCT is also an important and increasingly common cause of cardiovascular disease in adult cattle. To date, 54 cases have been seen in this school and these represent approximately 20% of all adult cattle admitted with cardiovascular disease, although the most prevalent syndromes are still traumatic pericarditis and infective endocarditis (each responsible for approximately 40% of such admissions).

Rubarth (11) said, in reference to PVCT, "once recognised, the stigmata are so characteristic that further cases and the real cause of death will be readily detected." Accordingly, the clinical signs of PVCT have been detailed below and related to the underlying lesions. Three typical case histories, each illustrating a different aspect, have also been summarised.

Clinical Signs

PVCT may present in two forms. The most important and common is a respiratory syndrome which follows pulmonary arterial thrombo-embolism and the formation and rupture of pulmonary arterial aneurysms (3,12). Much less frequently, cases are encountered in which the main presenting sign is gross abdominal distension due to hepatomegaly and massive ascites (12,14), but in such cases respiratory signs and pulmonary arterial lesions are also found. It has been reported that "sudden death" may also result from PVCT (11,14) and that animals may unexpectedly be found dead in the stable or at pasture without anything unusual having been noticed (11). In our series, death often occurred unexpectedly, usually soon after an episode of severe intrapulmonary hemorrhage or hemoptysis, but this was consistently preceded by other respiratory signs of at least several days duration. It seems likely that in cases of "sudden death" these signs have passed unnoticed.

A series of 12 cases of PVCT presenting with a respiratory syndrome has been described (12); this article is based on that report and our subsequent investigations of a further 20 cases. Almost all the cattle were female and in the age range one to eight years, although younger animals aged one to two and one half years were particularly frequently affected. In those cases in which background information was available, ill-health had been obvious for between three and 120 days, but in most the period was less than 28 days. The history was always one of a respiratory disturbance, but in a very small proportion of cases very severe thoracic pain was the actual reason for referral. Hemoptysis had not been detected in most cattle before admission.

On admission, a respiratory disturbance characterized by tachypnea (respiratory rate > 30 per minute), hyperpnea (increased depth of respiration) and cough was present in every animal. Fever (rectal temperature > 102.5°F) was also found in a minority of cases. These signs were of little help in establishing a diagnosis, but three features were found sufficiently frequently to make this fairly straightforward, particularly when they occurred together. These signs were: 1. anemia, manifested as mucosal pallor, hemic murmur and lowered packed cell volume (range 10-21.5%; normal 28-35%); 2. hemoptysis; and 3. widespread rhonchi. Other useful signs, which were common but not detected in every case, were thoracic pain, hepatomegaly, which was considered to be present if the liver was found to project into the right para-lumbar fossa, and melena. Finally, signs of congestive cardiac failure, including jugular distension and brisket edema, were observed in a few animals affected by chronic cor pulmonale.

Radiographic examinations were made on some animals as an adjunct to research and student teaching. The chest radiographs often reveal only a non-specific, irregular, focal or diffuse increase in lung density and lung markings (Fig. 1), but sometimes areas of embolic infarction and collapse are signified by small, discrete opacities peripherally (7). In a few cases, large, discrete, spherical opacities may be seen to form during the course of the disease

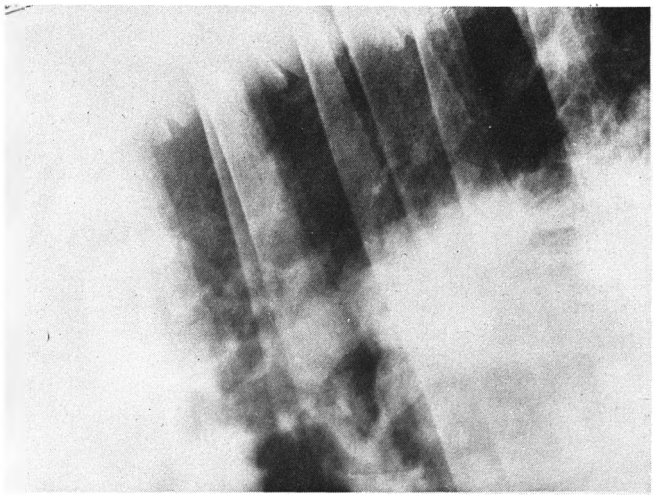

Figure 1. A standing lateral view of the dorsal-caudal thorax of a case of thrombosis of the posterior vena cava (Case A). There is an irregular increase in lung density in the ventral-caudal portion of the lung, largely ventral to the main caudal lobar bronchus. The remainder of the field shows some increase in lung markings.

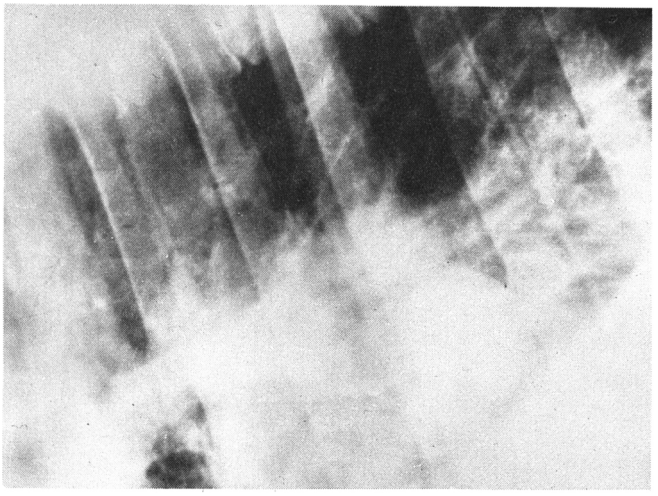

Figure 2. A similar radiograph of Case A taken five days after Fig. 1. There is a similar increase in lung density to Fig. 1, but, in addition, a discrete, spherical opacity (arrows) is apparent immediately ventral to the main caudal lobar bronchus and immediately cranial to the line of the diaphragm. This is an intra-pulmonary hematoma.

(Case A; Fig. 2); these are hematomata.

The animals were not treated while they were in the hospital and eventually they either died or were slaughtered on humane grounds; the period in the hospital ranged from one to 32 days. Throughout this time, respiratory signs generally worsened and hemoptysis, pallor, rhonchi and thoracic pain became more accentuated in some cases and appeared for the first time in others. The majority of animals died following a bout of massive hemoptysis and in most of the remainder failing health was due in large part to the effects of repeated episodes of hemorrhage.

Three examples of PVCT have been detailed below. Animal A presented with respiratory signs, anemia and hemoptysis, animal B with respiratory signs and thoracic pain without anemia (although this developed subsequently) and animal C was an apparent "sudden death."

Clinical and Pathological Findings: Selected Cases

(i) Case A

This was a thin, four-year-old Friesian dairy cow with a history of a painful respiratory disorder and "nasal bleeding" of 21 days duration.

On admission, the presenting signs were dullness, mild respiratory distress and marked thoracic pain, both at rest and after percussion. Tachypnea (45 per minute) and hyperpnea were present and there was frequent productive coughing and mild hemoptysis. Slight tachycardia (80 per minute), mucosal pallor (PCV 19%) and hepatomegaly were noted. No adventitious lung sounds were detected on auscultation and the rectal temperature was normal. A diagnosis of PVCT was made. Thoracic radiographs were obtained for research purposes; these revealed only a non-specific increase in lung density, particularly in the ventral caudal lung lobes (Fig. 1).

No treatment was given and in the following seven days there was marked deterioration with further episodes of hemoptysis (sometimes massive, involving the loss of several liters of blood), increasing pallor (PCV fell to 10%) and increasing thoracic pain. Rhonchi became detectable and were widespread in both lung fields. Further thoracic radiographs five days after admission revealed the presence of a discrete round opacity, approximately 15 cm diameter, in the caudal lung field; this was identified as an intra-pulmonary hematoma (Fig. 2). Blood pressure and gas measurements were also made and these were as follows: right atrial and ventricular systolic pressures were 7 and 76 mm Hg, respectively; pulmonary arterial pressure was 76 mm Hg systolic, 46 mm Hg diastolic; carotid artery pressure was 135 mm Hg systolic, 80 mm Hg diastolic; systemic arterial $PaCO^2$ and PaO^2 were 36.2 and 83 mm Hg, respectively. These results revealed that there was pulmonary arterial hypertension, since our accepted normal values of pulmonary arterial pressure are < 45 mm Hg systolic and < 25 mm Hg diastolic (5). The animal was slaughtered on humane grounds seven days after admission.

At post-mortem examination, a large rough-surfaced white thrombus was found to occlude the hepatic portion of the PVC; no organisms were recovered after aerobic and anerobic culture of material from the thrombus. There was hepatomegaly as a result of chronic venous congestion of the liver; no hepatic abscess was present. A large intra-pulmonary hematoma was found in the right caudal lobe about a ruptured saccular aneurysm of a segmental pulmonary artery. Many large and small berry and saccular aneurysms had formed in lobar and segmental branches of the pulmonary artery at sites of arteritis; these were present in all lobes but were most numerous caudally. Casts of clotted blood filled many bronchi and scattered lung segments were distended with aspirated blood; the blood had originated from saccular aneurysms which had ruptured into the bronchi. There was widespread pulmonary arterial thrombo-embolism, predominantly of segmental and smaller branches in all lobes. The pulmonary arterial trunk was dilated and its diameter exceeded that of the aorta: there was also right ventricular hypertrophy and the ratio of the right ventricular muscle mass to the total ventricular and septal mass was 0.32.

(ii) Case B

This was a seven-year-old Friesian cow with a history of dullness, anorexia and a syndrome resembling very severe traumatic reticulitis which had been present for three weeks.

On admission, the presenting signs were dullness, respiratory distress and severe discomfort due either to anterior abdominal or thoracic pain; the cow was thin and febrile (102.9°F). Tachypnea (50 per minute), hyperpnea, an expiratory grunt and a frequent non-productive cough were noted. Rhonchi were detected in the right caudal lobe area and percussion of this same region produced coughing and pained grunting. Tachycardia (90 per minute) was present but the mucosae were of a normal pink colour (the PCV at this stage was 23.5%). The cow had a markedly reduced abdominal volume and ruminal contractions were weak and infrequent. There was hepatomegaly and percussion and palpation over both the

liver and xiphoid areas apparently caused great pain. The animal walked stiffly, stood with both elbows abducted and hardly ever lay down. Following an initial, slight improvement, there was a marked deterioration on the 14th day after admission. At this stage, respiratory distress became marked, rhonchi became widespread on both sides of the thorax, hemoptysis suddenly occurred and the mucosae were found to be very pale. The PCV at this stage was found to be 14%. Eighteen days after the beginning of severe respiratory signs, the animal died as a result of a severe episode of hemoptysis.

At post-mortem examination, a white thrombus which almost completely occluded the PVC was found in the region of the diaphragm. The ventral wall of the vein was thickened and, on section, the central area of the thrombus was found to be purulent. There was chronic venous congestion of the liver and hepatomegaly. A large amount of clotted blood was found in the trachea and larger bronchi and there was thrombo-embolism in the pulmonary circulation. Numerous white thrombi were also present and several of these were obviously suppurative and associated with small aneurysms. There were several thin-walled abscesses containing white, mucoid pus at the posterior edges of the caudal lobes. In addition, an area of hemorrhagic necrosis, 3 cm in diameter, was found in the left caudal lobe. Many lobules had a dark mottled appearance due to intra-alveolar accumulation of aspirated blood. There was also a focus of suppurative pneumonia in the cranial lobe of the right lung and a moderate degree of diffuse interstitial emphysema. A coagulase-positive staphylococcus was isolated from the vena cava thrombus and from the embolic pulmonary lesions.

(iii) Case C

This five-year-old-Ayrshire dairy cow was treated several times for a "relapsing pneumonia" during a five-week period prior to admission. The cow died suddenly a few hours after entry into the veterinary school, before a full clinical examination could be made, although tachypnea, hyperpnea and hepatomegaly were noted. There was no history of hemoptysis and the PCV at admission was 33.

At necropsy, scars of numerous liver abscesses were found in all lobes, but none were adjacent to the hepatic portion of the PVC. There was massive hepatomegaly as a result of chronic venous congestion of the liver. A rough-surfaced thrombus occluded the entire length of the intra-thoracic portion of the PVC and there was a firm fibrous attachment between this vessel and the pleura covering the accessory lobe of the lung at an area of phlebitis. *Fusobacterium necrophorum* was recovered from the thrombus, the vessel wall in the zone of phlebitis and from the adjacent accessory lobe of the lung. The lobar pulmonary arteries serving the left and right caudal lung lobes were both totally obstructed close to their origins by large septic thrombi; the thrombus was partially organized in the former vessel (Fig. 6) but had only recently formed in the latter. The dependent right caudal lobe was collapsed. There was widespread pulmonary arterial thrombo-embolism in smaller vessels of all lung lobes and many large and small aneurysms resulting from pulmonary arteritis and endarteritis were found in both caudal lobes. There was no blood in the tracheobronchial system and no intra-pulmonary hematomata were discovered. This animal died of acute cor pulmonale as a result of sudden massive pulmonary arterial thrombo-embolism when the pulmonary circulation was already embarrassed by previous episodes of embolism.

Pathogenesis

A thrombus is always found in the posterior vena cava and embolism from this thrombus results in pulmonary lesions (11), which include pulmonary arterial thrombo-embolism, pulmonary arteritis, pulmonary endarteritis and thrombo-arteritis, chronic suppurative pneumonia and the formation of multiple pulmonary abscesses (1). The pulmonary arterial lesions give rise to pulmonary hypertension and the development of aneurysms of this vessel; rupture of these aneurysms causes massive intra-pulmonary or intra-bronchial hemorrhage (2,3). The aneurysms are produced by bacterial infection and inflammation of the arterial wall and are thus classified by some authorities as "mycotic

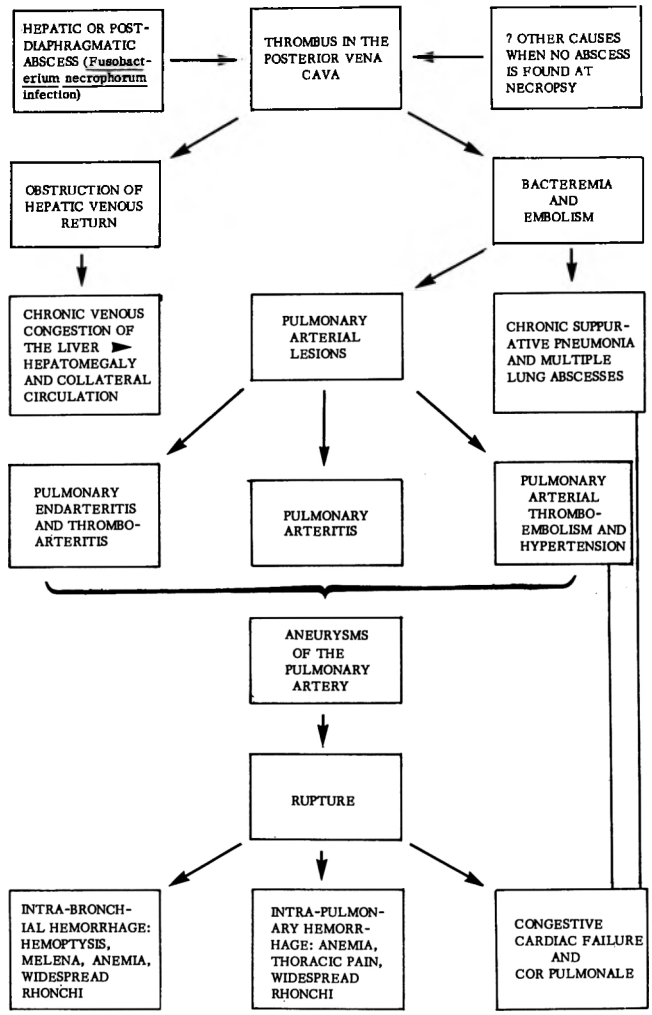

Figure 3. The pathogenesis of thrombosis of the vena cava in cattle.

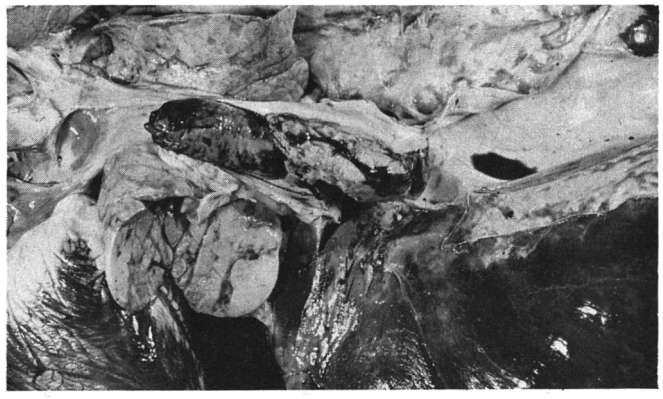

Figure 4. A rough-surfaced thrombus (T) occluding the intra-thoracic portion of the posterior vena cava anterior to the openings of the hepatic veins (arrow). The liver (L) and right atrium (RA) can also be seen.

aneurysms" (6); this term may cause some confusion, since it does not imply fungal infection, and is not used elsewhere in this article. The pathogenesis of the various lesions is summarised in Fig. 3.

(i) Lesions in the liver and posterior vena cava

A rough or smooth-surfaced thrombus is always present in the hepatic or intra-thoracic portion of the PVC between the liver and the right atrium (Fig. 4). Almost all the thrombi form as a result of thrombophlebitis after the wall of the PVC has been infiltrated by a perivascular hepatic or post-diaphragmatic abscess and frequently contain a suppurative core or have a persistent communication with the initiating suppurative lesion. These abscesses are usually solitary and situated immediately adjacent to the PVC; multiple, widespread liver abscesses are not frequently found, at least in our experience. Single abscesses and associated PVC thrombi at various stages of development are also common in livers condemned at local slaughterhouses; unfortunately, these are from animals which have not been examined clinically and whose history is unknown. It is possible that these solitary hepatic abscesses originate from alimentary tract ulcers, particularly of the rumen or abomasum, although such ulcers are not commonly present at the time of necropsy. A few animals have also had septic arthritis, multiple peri-articular abscesses or long-standing interdigital necrobacillosis and it seems likely that such conditions could lead to bacteremia, the formation of a hepatic abscess and ultimately PVCT. Bacteriological examination of the abscess or thrombus at necropsy often yields no significant result, possibly because of previous antibiotic therapy, but *Corynebacterium pyogenes, Staphylococci* or *F. necrophorum* have been recovered from some animals. It is probable that *F. necrophorum* is the main cause of these infections (4). The aetiology of the thrombosis is obscure in the minority of cases in which no abscess is found at necropsy, although the previous presence of an abscess cannot be discounted.

Most thrombi are large and often occlude the lumen of the PVC at or anterior to the openings of the hepatic veins; this gives rise to chronic venous congestion of the liver (Fig. 5), hepatomegaly and the development of a collateral circulation, which utilizes the hemiazygos and mammary veins to return blood to the heart. Gross abdominal enlargement due to ascites and massive hepatomegaly is uncommon and only occurs when the thrombus extends into and obstructs the hepatic veins so that this collateral hepatic venous drainage is blocked. Pulmonary lesions are also present in such cases.

(ii) Lesions in the pulmonary arteries

Lesions are consistently found in the intra-pulmonary branches of the pulmonary arteries and are of particular significance in that they are the cause of the hemorrhage and hemoptysis that often precede death. These lesions include pulmonary arterial thrombo-embolism, pulmonary arteritis, endarteritis and thrombo-arteritis.

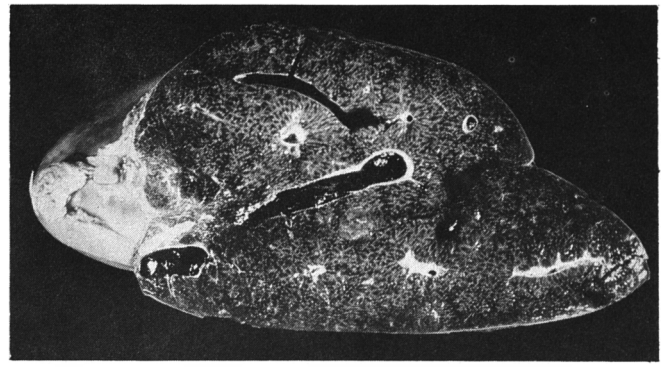

Figure 5. Cross-section of an enlarged liver in which there is chronic venous congestion.

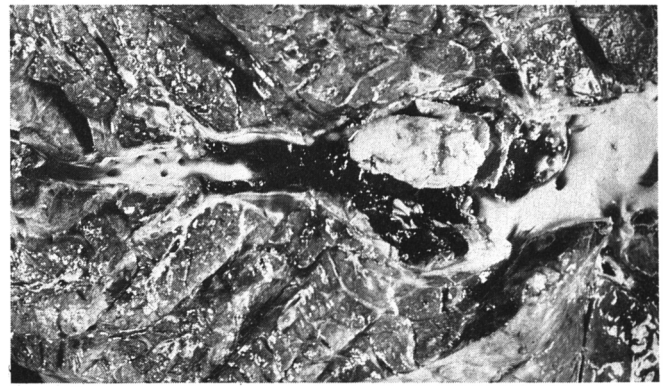

Figure 6. The lobar branch of the pulmonary artery supplying the left caudal lobe is obstructed by an organizing septic thrombus (Case C).

Thrombo-embolism is very evident in intra-pulmonary lobar and segmental branches of the pulmonary artery on macroscopic examination (Fig. 6), but the extent of the lesion is best appreciated in histological sections, in which septic, bland or recanalised thrombi can be seen blocking numerous smaller arteries (Fig. 7). Pulmonary arterial thrombo-embolism is an important lesion because: 1. there is widespread occlusion of the segmental, sub-segmental and smaller arteries by small emboli; 2. fusiform and saccular aneurysms may form at sites of arterial obstruction; and 3. there is sometimes sudden blockage of lobar arteries by large emboli, an infrequent complication which can produce acute clinical signs and even death, particularly when there is already respiratory embarrassment (Case C; Fig. 6). However, the most significant consequence of widespread thrombo-embolism is the development of pulmonary arterial hypertension, which can be detected by direct blood pressure measurement (Case A) or, at necropsy, by such histopathological findings as medial hypertrophy (Fig. 8a) and cellular intimal proliferation (Fig. 8b) in muscular pulmonary arteries and in the arterioles. The increase in pulmonary arterial pressure promotes and accelerates the formation of aneurysms at sites of arteritis, enarteritis or thrombo-arteritis.

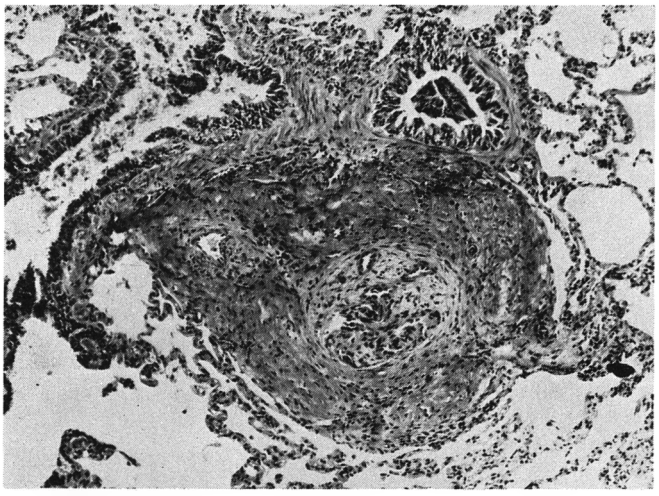

Figure 7. Recanalisation of a thrombus occluding a muscular pulmonary artery. (H & E; x 50).

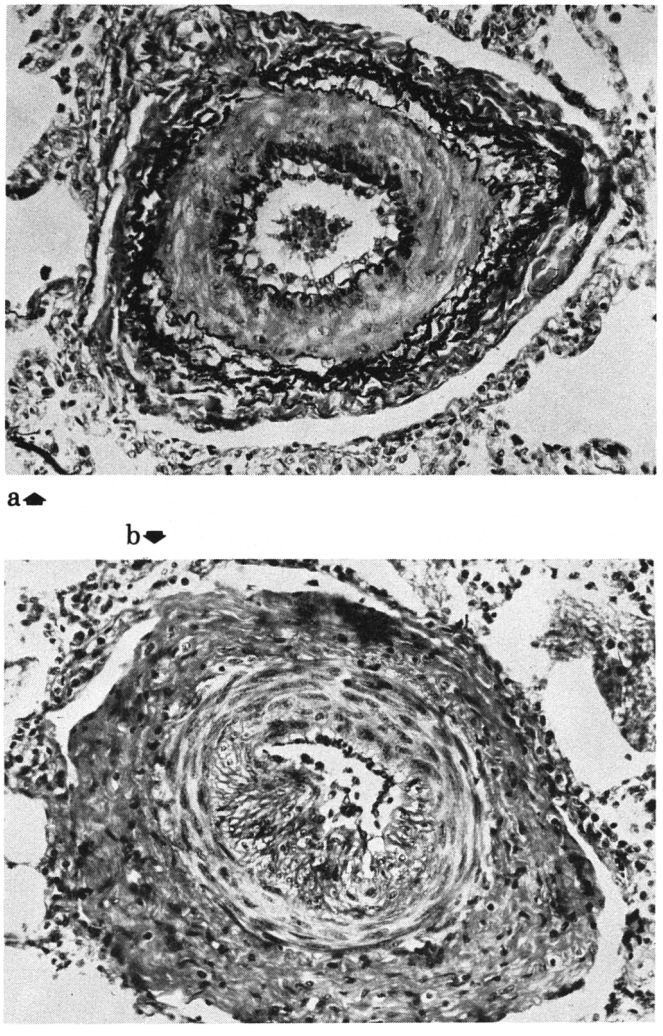

Figure 8. (a) Medial hypertrophy in a muscular pulmonary artery (Verhoeff; x 170); (b) Medial hypertrophy and cellular intimal proliferation in a muscular pulmonary artery (Martius Scarlet Blue; x 170).

Pulmonary arteritis is the most important of these lesions and is initiated by localization of septic emboli in the perivascular connective tissue of lobar and segmental branches of the pulmonary artery (Fig. 9 [i]). Extension of the embolic septic focus erodes the adventia and then the media of the vessel so that a shallow saccular aneurysm forms at the site (Fig. 9 [ii]). Hypertension and progressive inflammatory erosion of the vessel wall result in gradual enlargement of the aneurysm, which becomes deeper and surrounded by a mixture of pus and clotted blood (Fig. 9 [iii] and Fig. 10). The blood is derived largely from ruptured *vasa vasorum,* which have been eroded by perivascular suppuration and torn by movements of the vessel during systole. At the same time, the wall of an adjacent bronchus may be eroded by the perivascular abscess, which may establish a fistulous communication with the bronchial lumen (Fig. 9 [v]). Eventually, the wall of the aneurysmal sac becomes so attenuated that rupture occurs and blood is ejected into the adjacent pulmonary tissue, where it passes into the bronchial system (resulting in hemoptysis) or it forms large hematomata in the interstitium of the lung. Hemoptysis usually develops when an embolic abscess is located between the artery and bronchus in such a way that the bronchial and arterial walls are eroded simultaneously. In the stage immediately before rupture, a saccular aneurysm surrounded by clotted blood extends into an abscess which has formed a fistula into the bronchus or which has eroded the cartilage and muscle of the bronchial wall leaving only the mucous membrane intact (Fig. 9[v] and Fig. 11). Rupture of aneurysms of this type is immediately followed by hemoptysis, since the blood is ejected directly into the bronchial system (Fig. 9[vi] and Fig. 12). At necropsy, casts of clotted blood may be found in the tracheobronchial tree, in the upper respiratory tract and in the alimentary tract and aspirated blood may fill many alveoli. If the suppuration involves only the arterial wall, so that the bronchial wall remains intact (Fig. 9[vii] and Fig. 10), blood ejected from ruptured aneurysms cannot gain access to the bronchial system and gathers in large, lamellated interstitial hematomata (Fig. 9[viii]), which may be 20 cm or more in diameter. This intrapulmonary hemorrhage is the cause of anemia in the absence of hemoptysis. A further uncommon complication of pulmonary arteritis is local dissection of blood within the tissue planes of the media of the artery to form a short, dissecting aneurysm (Fig. 9[iv]), which may give rise to thoracic pain.

Pulmonary endarteritis and thrombo-arteritis are encountered much less frequently than pulmonary arterial thrombo-embolism and pulmonary arteritis. Endarteritis, the result of septic micro-emboli entering the wall of the pulmonary artery via the *vasa vasorum,* can give rise to large fusiform aneurysms up to 15 cm in diameter (1). In this instance, numerous small abscesses form in the arterial wall beneath the endothelium and extension of these results in weakening and aneurysmal dilatation of the affected

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Clinical synopsis. Response: visible in 24-48 hours; average recovery in 3-4 days. *Precautions:* veterinarian should be aware of the possible side effects of dexamethasone such as suppression of inflammation, reduction of fever, increased protein degradation and its conversion to carbohydrate leading to a negative nitrogen balance, sodium retention and potassium diuresis, retardation of wound healing, lowering of resistance to many infectious agents such as bacteria and fungi, reduction in numbers of circulating lymphocytes. *Contraindications:* animals with severe renal functions, impairments and untreated infections. *Warnings:* Milk taken from dairy animals during treatment and for 72 hours after the latest treatment must not be used for food. Clinical and experimental data have demonstrated that corticosteroids administered orally or parenterally to animals may induce the first stage of parturition when administered during the last trimester of pregnancy and may precipitate premature parturition followed by dystocia, fetal death, retained placenta and metritis.

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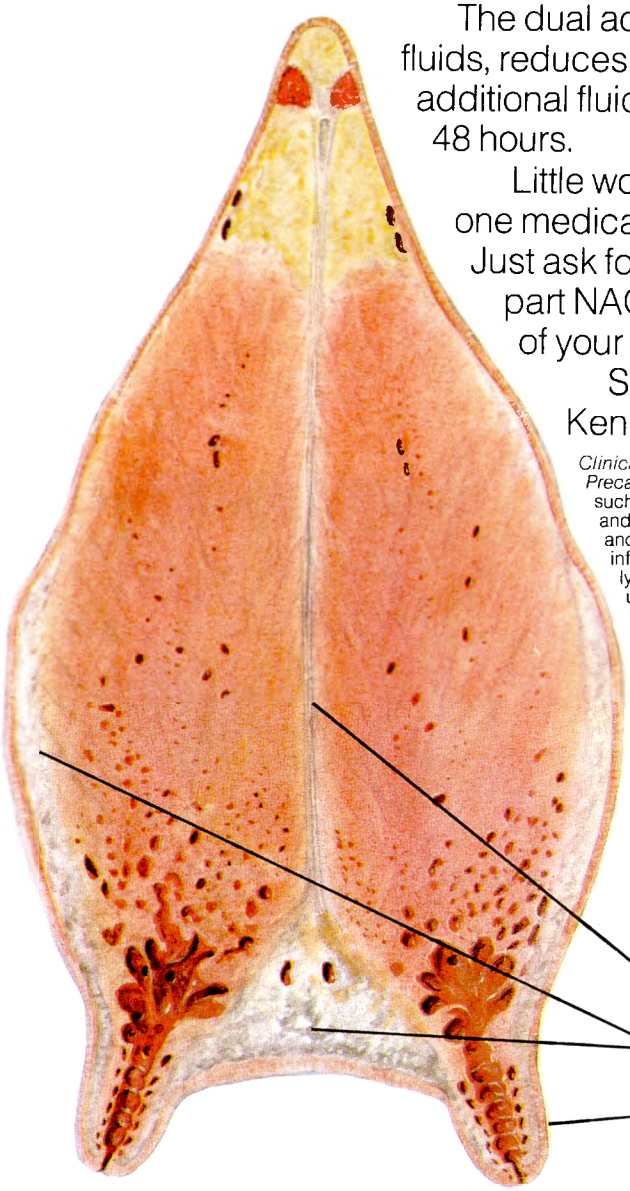

*Each bolus contains 200 mg. trichlormethiazide and 5 mg. dexamethasone.

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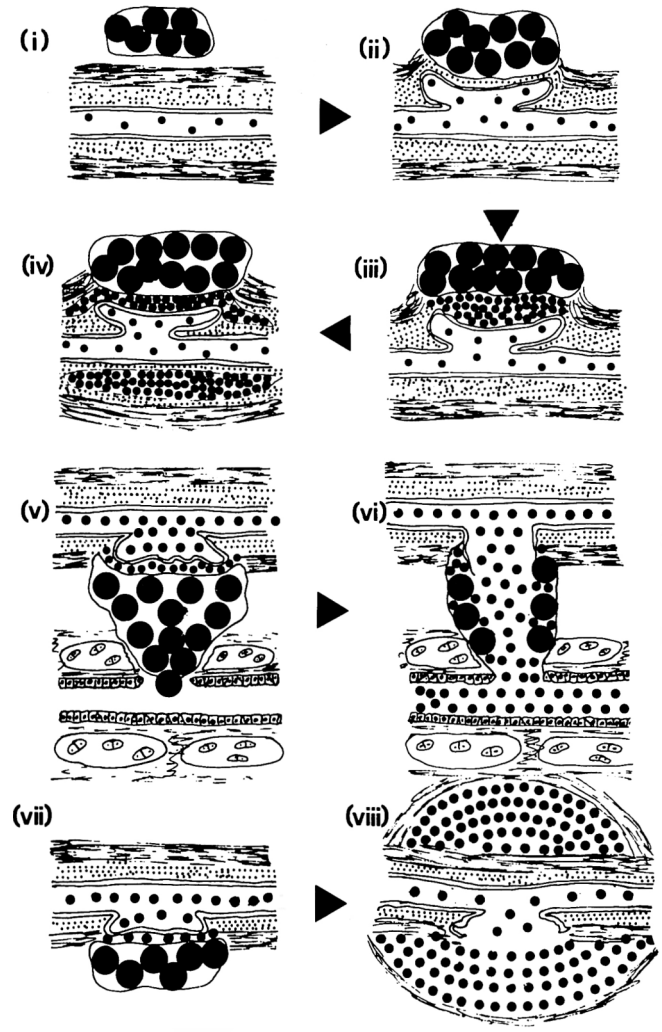

Figure 9. Diagrammatic representation of the pathogenesis of hemoptysis and hematomata resulting from pulmonary arteritis. (i) An embolic abscess is situated adjacent to the pulmonary artery. The endothelium, intima, media and adventitia making up the arterial wall are indicated. (ii) Extension of the abscess results in erosion of the adventitia and media of an arc of the pulmonary artery and the formation of a saccular aneurysm at the site. (iii) Erosion of vasa vasorum in the wall of the pulmonary artery results in intramural and perivascular hemorrhage between the base of the aneurysm and the surrounding abscess. (iv) There is dissection of blood between the tissue planes of the media in the vicinity of the aneurysm and a dissecting aneurysm is formed. (v) A perivascular abscess erodes the wall of the bronchus to form a communication with the lumen. At the same time, it erodes an arc of the wall of the pulmonary artery to form a saccular aneurysm surrounded by clotted blood derived from eroded vasa vasorum. (vi) Rupture of the aneurysm in (v) results in hemoptysis and the contents of the abscess are flushed into the bronchus. (vii) A perivascular abscess which results in pulmonary arteritis and the formation of a pulmonary arterial aneurysm, but which does not erode the accompanying bronchus. (viii) Massive lamellated hematoma in the interstitium as a result of rupture of the lesion described in (vii) above. The blood cannot gain access to the bronchus (cf. vi).

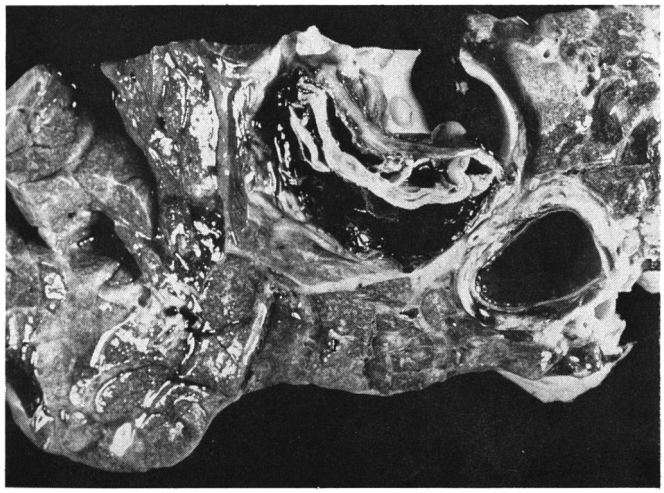

Figure 10. Cross-section through a saccular aneurysm that has formed in an arc of the wall of a lobar pulmonary artery at a focus of arteritis. Pus mixed with clotted blood is present beneath the necrotic wall of the aneurysmal sac. Abscesses covered by endothelium can be seen projecting into the lumen of the artery (the artery has been opened during the necropsy). The lobar bronchus is also marked; its wall is not affected by the perivascular suppuration. This figure corresponds to the lesion in Fig. 9 (vii).

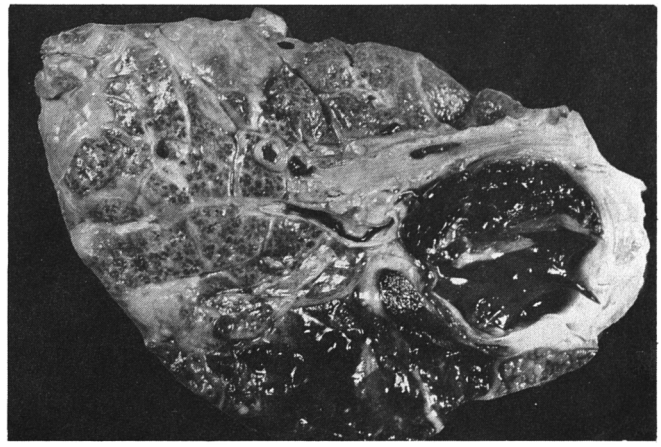

Figure 11. A saccular aneurysm opens, via a narrow neck (arrows), from a lobar branch of the pulmonary artery (L). The aneurysmal sac is filled with blood and is surrounded by a large volume of clotted blood (C) and pus. The wall of the accompanying bronchus (B) has been eroded so that only the mucous membrane remains. This figure corresponds to the lesion illustrated in Fig. 9 (v). This figure is reproduced from Breeze, et al. (3) by kind permission of the Editorial Board of the Journal of Pathology.

length of artery. The term thrombo-arteritis implies that thrombosis is secondary to an inflammatory reaction in the wall of the artery. This process does occur in PVCT but at the present time its importance has not been ascertained. This is because many of the pulmonary lesions are so complicated and advanced at the time of necropsy that the sequence of events cannot be determined accurately.

(iii) Lesions in the lungs and heart

Chronic suppurative pneumonia, pleurisy and the formation of multiple pulmonary abscesses are frequently observed. Cor pulmonale develops when there is severe pulmonary arterial hypertension resulting from widespread pulmonary thromboembolism and chronic suppurative pneumonia, occurring either together or separately. In such cases, there is right ventricular hypertrophy and dilatation of the pulmonary arterial trunk so that its diameter

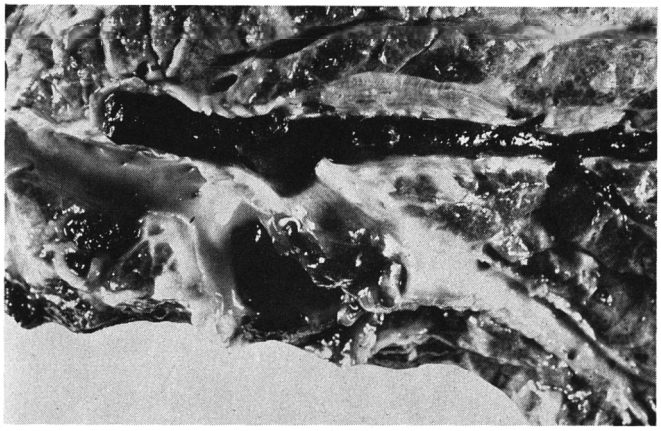

Figure 12. A lobar branch of the pulmonary artery (L) in which there is a saccular aneurysm (S) surrounded by an abscess (A), which has also eroded the wall of the bronchus (B). The bronchus contains a cast of clotted blood as a result of rupture of the aneurysm. This lesion corresponds to that shown diagrammatically in Fig. 9 (vi). This figure is reproduced from Breeze, et al. (3) by kind permission of the Editorial Board of the Journal of Pathology.

exceeds that of the aorta at the same level. In a proportion of cattle, clinical signs of congestive cardiac failure may result from chronic cor pulmonale. A concomitant right-sided infective endocarditis has been noted in approximately 10% of PVCT cases (15) but this has not been recorded in our series.

(iv) Causes of the main clinical signs

Intra-pulmonary hemorrhage causes anemia and intra-bronchial hemorrhage is responsible for hemoptysis, anemia and melena, which is associated with the swallowing of coughed-up blood (melena may also result from concurrent ulceration of the abomasum or rumen). The anemia is hemorrhagic in type and is usually normocytic and normochromic, becoming macrocytic as the number of reticulocytes increases. Rhonchi are probably produced by widespread distortion of the bronchial lumina by: 1. casts of clotted blood; 2. pus in lobes affected by suppurative pneumonia; and 3. peribronchial aneurysms and abscesses. Thoracic pain may result from intra-pulmonary hematomata, suppurative pneumonia and pleurisy, and dissecting aneurysms.

Diagnosis

Three features are of particular value in making a clinical diagnosis of PVCT. These are 1. anemia; 2. hemoptysis; and 3. widespread rhonchi. Thoracic pain, hepatomegaly and melena, when present, are also useful (12). Hemoptysis should be distinguished from epistaxis, which might occur in infectious bovine rhinotracheitis for example. We have encountered minor hemoptysis in two cases of right-sided infective endocarditis, but, with these uncommon exceptions, we regard hemoptysis as a virtually pathognomonic sign of PVCT. Widespread rhonchi are detectable in many affected cattle and the diagnostic possibilities when these sounds are scattered over a wide lung area are generally limited to PVCT, parasitic bronchitis and diffuse fibrosing alveolitis (1,9). When other findings are considered, there is little difficulty in differentiating PVCT from the latter two conditions. The differential diagnosis of the more important respiratory diseases we recognise in adult cattle in Britain, including PVCT, has been considered more fully elsewhere (1,13).

Diagnosis of PVCT at necropsy presents no problem, providing the PVC is opened. This is best achieved by removing the larynx, trachea, lungs, heart, liver and diaphragm together, then opening the vascular system; this procedure preserves the relationships of the organs and facilitates demonstration of the lesions. Finally, Mackey (8) has stated that hemoptysis may result from the rupture of pulmonary vessels associated with rupture of lung abscesses or forceful respirations. We have not encountered this type of case except where the pulmonary abscesses were secondary to PVCT and suggest that hemoptysis may be wrongly attributed to the presence of primary lung abscesses because the PVC is not examined.

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Practice Tip

DHIA Records

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My practice tip is how a dairy practitioner uses DHIA records. I am in an exclusive dairy practice composed of reproduction and herd health programs. I encourage my dairymen to the point of insistence that they be on official test. We receive permission from our dairymen to have DHIA records mailed to us from the computer center and the following is an explanation of how we use them.

When these records come in, I usually glance over them and get an idea of whether this client is improving or has a few gray areas, etc. I look at the number of cows on test, the percentage in milk, how much milk is being obtained, and the number of pounds of milk per milking cow. I like to keep up with this on a month-by-month basis. Rolling herd averages for milk and butterfat and the percent of butterfat are important.

The next part of this sheet is economic information. It is important for me as a practitioner to keep up with my client's feed cost. How much is he getting for his milk? You can tell whether he is making money or not.

The other part of this sheet concerns breeding information. The left-hand side deals with cows that have not been bred. The important area here is the cows that are open over 100 days. The right-hand portion of this deals with cows that have been bred but have not been bred long enough to determine pregnancy. The important area here is the number of cows bred three or more times, the percent problem cows and the number of cows that are 100 days or more open and you are still breeding. It also gives the total of the number of cows in the breeding herd.

This section of the DHIA record deals with the pregnant animals in the herd. We have the number of pregnant animals, the average days open, or projected calving interval, and conception information. Also the breeding conception for the past 30 days. This part of our record gives us a comparison of our first-calf heifers as compared to our older cows. Heifers with a 14-2 ME and older cows with a 16-39 ME. We can tell here whether he has some inferior heifers milking this year.

We also have information on the mastitis test. Cows to be milking for the next six months, cows to be dry and cows to calve. Average days dry, consistency of lactation, then we have a summary for the past 12 months, the herd average for the last 12 months and we can see whether he is improving or regressing.

From the mastitis test we get the CMT scores; the scores were 2's and 3's on the right-hand column. What do we do with these? We look at them to see this client's problem areas, then we give them to our secretary. We have had some forms printed up. We put these in a folder and keep each summary record for the previous 12 months in this folder.

We like to keep a record and have information on this client's feeding program–whether he is feeding corn silage, all hay, what percent grain ration he is feeding, etc., whether they are feeding a blended ration. We like information on his vaccination status, and time of vaccination, as to month of year or stage of lactation, etc., and we have these for six years. We have the secretary plot the number of milkings plus the percent in milk month by month and here we have a five-year basis. We can look back on most of these herds for the past three or four years and see what is going on. We plot the rolling herd average for milk and for butterfat.

The second sheet is calving interval and HRS index number. Days open and days dry, and the other part is breeding for conception and cull rate. On the bottom we have a blank for comments. Any important or severe management changes made, we'd like to record here. Maybe he hired a new herdsman or fired his old one, or he put in a new feeding system, or built a new dairy, and it might severely affect management.

Before we go out to visit a herd, say on Wednesday, maybe Tuesday or Monday night we'll pull out this folder, sit down, look over it, and we can see whether he is making progress or going backward. We can spot problem areas before they get serious.

The problem I've always had before is I would go on the farm and things would get out of hand. I wouldn't have time to go over our records and so forth, and this way I know what he is doing and if he has a problem, I can bring it up and say, "Look, Joe, you've got too much mastitis, looks like," or "You're not catching your cows in heat," or "You are having conception problems. What's the problem?" It has been a big help to me in my relationship with my clients. If any of you are interested in these forms, please contact me and I'll help you out.

If you wish a copy of your client's records, first ask your client for his permission. The North Carolina state dairy extension does our computer records, charges the client $2.50 a month for this extra copy and they will mail directly to us. We ask the client for his permission, then we notify the state dairy extension leader, giving him a list of clients we wish to receive extra copies on, and in a couple of weeks we get copies and they continue on.