

OVERVIEW

- Further cases of *H somni* septicaemia in weaned beef calves
- Congenital cerebellar abiotrophy in Aberdeen Angus calves
- Suspected post-operative ileus in a Texel ewe
- Respiratory disease in crows due to *Pasteurella multocida*

GENERAL INTRODUCTION

The mean temperature for December was 0.6°C above the long-term average, mainly due to a high mean minimum temperature. Rainfall was near normal in the west and above normal elsewhere, with Scotland as a whole having 120 per cent of average rainfall. Sunshine was 87 per cent of average with only the Northern Isles and parts of the far north sunnier than average.

DISEASE ALERTS

The following conditions were reported by SRUC VS disease surveillance centres in March 2020. Given similar climatic and production conditions, they could also be important this year.

- **Osteodystrophy in rapidly growing beef fattening animals**
Nutritional osteodystrophy can present with a range of clinical signs which may include lameness, stiffness, joint or epiphyseal swelling, fracture, or gastrocnemius tendon rupture. Cases may come to light several months after introduction of an intensive ration and are a serious welfare issue when they occur. It is important to supply a mineral/vitamin supplement that is appropriate to the ration being fed.
- **Necrotising encephalopathy in neonatal lambs secondary to inadequate ewe nutrition**
Prolonged maternal energy deficiency may lead to neurological disease in neonatal lambs with less severely affected animals slow to stand or suck. Ewes may be in suboptimal body condition and bloods collected in the periparturient period may reveal hypoglycaemia and subclinical ketosis. Histopathology of lamb brains is required to confirm the diagnosis. Assessing dietary energy adequacy by measuring serum beta-hydroxybutyrate three to four weeks pre-lambing is recommended. (See feature article for further information).

CATTLE

Generalised and systemic conditions

A three-year-old Aberdeen Angus cow from a group of 17 was reported to have a poor appetite for around 12 hours before becoming recumbent. Clinical findings of firm faeces and poor gut motility suggested the possibility of hypocalcaemia which was confirmed on biochemistry. Intravenous calcium was administered but death occurred soon afterwards. Postmortem examination found the carcass to be jaundiced and ascitic. A 4 cm diameter firm pale lesion surrounded by a wider area of haemorrhage was identified in the liver and a fluorescent antibody test for *Clostridium novyi* was positive. Lesions of severe extensive acute-subacute necrotising and leucocytoclastic hepatitis with extensive areas of coagulative necrosis and expansion of sinusoids by small gas pockets, bordered by a zone of intense cytoclastic debris accumulation, accompanied by numerous intralobular stout sporulated bacilli, typical of clostridial hepatitis, were detected on histopathological examination. Elsewhere acute zonal periarterial necrotising hepatopathy and moderate mature portal fibrosis were present.

The zonal necrosis was more recent, consistent with a secondary event. The portal fibrosis was most likely secondary to chronic fluke exposure, however there was no evidence of acute fluke migration. The group had been treated with nitroxylnil one week earlier.

Necrotising laryngitis in association with *Histophilus somni* septicaemia was recorded twice during December. The first outbreak of disease occurred in a group of 200 fattening animals purchased from various sources. Ten per cent had been treated with antibiotics and NSAIDs after presenting with lethargy and anorexia. One animal with a history of ataxia and recumbency died and was submitted for investigation of the problem. The carcass exhibited haemorrhages throughout with multiple petechiae on the mucosa of the larynx (Fig 1), trachea and bronchi. Histopathology revealed lesions in multiple tissues, including a severe necrotising laryngitis, consistent with systemic localisation of *H somni*. The second case involved a seven-month-old Aberdeen Angus suckled calf which was submitted for investigation of respiratory and neurological signs. This animal initially developed pyrexia and respiratory signs before progressing to lateral recumbency with opisthotonos and nystagmus. Necrotising laryngitis, patchy lung congestion and polyarthritis were detected on postmortem examination. Histopathology confirmed a thrombotic meningoencephalitis explaining the neurological signs, in addition to multisystemic

leucocytoclastic inflammation including myocarditis typical of *H. somni* septicaemia.

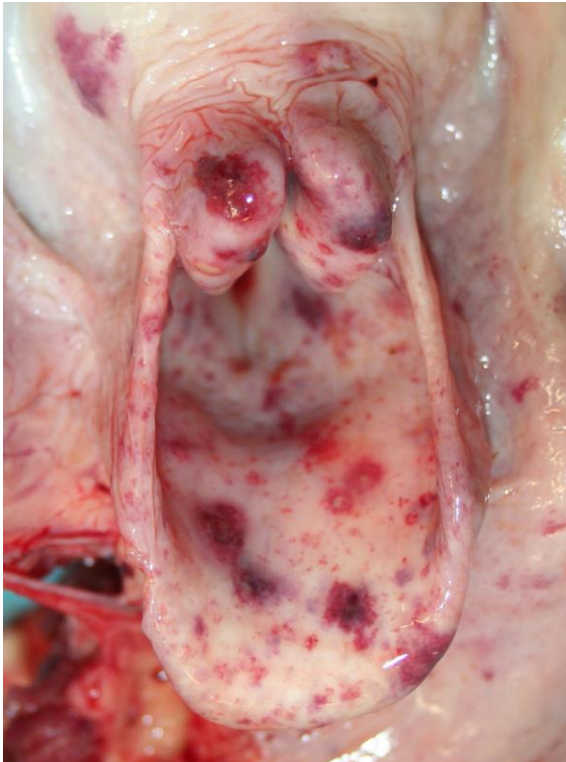


Figure 1 – Laryngeal haemorrhage in a case of *Histophilus somni* septicaemia

Alimentary tract disorders

A herd submitted one beef and two dairy calves following the acute deaths of five calves in three days. The beef and dairy herds were managed separately, but with shared personnel and equipment. All affected calves were less than three days of age and appeared healthy at birth before rapidly becoming cold and recumbent. Postmortem examination confirmed dehydration and watery diarrhoea in all cases; and faecal samples tested positive for *Escherichia coli* K99. Concurrent colisepticaemia was also diagnosed. Histopathology was carried on tissues from one calf and revealed changes consistent with a severe form of K99 *E. coli* colonisation and secondary septicaemia/bacteraemia. SRUC VS commented that generally the *E. coli* strains identified in colisepticaemia and enterotoxigenic *E. coli* are different, but the latter can become systemic in the agonal phase of disease.¹

Respiratory tract diseases

Two, two-week-old, Holstein Friesian heifer calves were presented alive to investigate respiratory disease in young dairy calves. Both calves were normothermic and

moderately dyspnoeic and tachypnoeic. Calf 2 had an additional right sided head tilt and ear droop. Postmortem examination confirmed a diagnosis of pneumonia, *Mannheimia haemolytica* was isolated and respiratory syncytial virus detected by PCR. Examination of the middle ear of calf 2 revealed a purulent otitis media on the right side (Fig 2) consistent with the clinical signs being due to peripheral vestibular disease and cranial nerve dysfunction including compression of the facial nerve (Bell's palsy) secondary to the bacterial otitis. *Mannheimia haemolytica* was also isolated from the affected middle ear and is a recognised cause of otitis media² as a result of ascending infection from the nasopharynx. No evidence of *Mycoplasma bovis* infection was identified in either case.

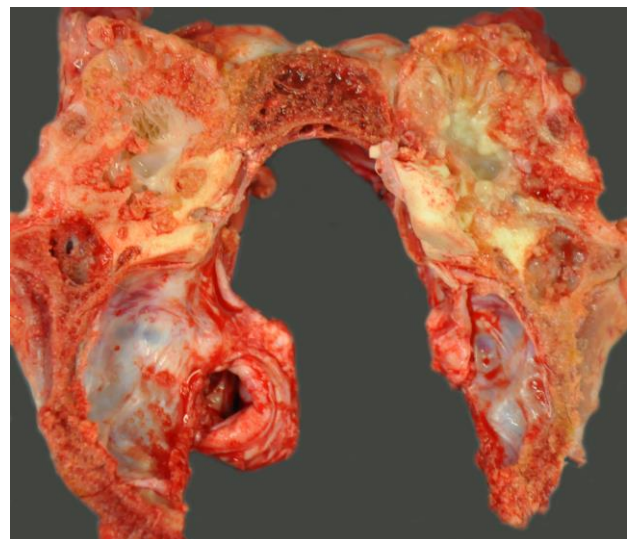


Figure 2 – Otitis media due to infection with *Mannheimia haemolytica* in a neonatal dairy calf

Nervous system disorders

Two Aberdeen Angus calves were submitted to investigate an issue with severe congenital neurological disease affecting two of four calves born to a group of 10 heifers calving ahead of the main herd. Both calves presented with a slight head nod and had difficulty in maintaining sternal recumbency. They were unable to stand when assisted and had complete loss of proprioception in all four limbs. No abnormalities were observed on postmortem examination. Neuropathology detected cerebellar cortical degeneration involving multifocal vacuolation of the basal molecular layer, scattered Purkinje neuronal fine cytoplasmic vacuolation, chromatolysis and nuclear pyknosis, occasional Purkinje axonal spheroids and an impression of paucity of internal granule layer neurones. Pestivirus involvement was considered unlikely and excluded by additional testing. A diagnosis of congenital cerebellar atrophy was

reached. Congenital or neonatal onset of neurological signs associated with cerebellar cortical degeneration is recorded in Angus and Angus cross calves however the clinical and neuropathological features in these cases differed in some respects, for example no seizures were reported. However, the characteristics and progressive nature of the lesions were consistent with an inherited condition. Accidental inbreeding had occurred with three of the heifers found to be granddaughters of the sire used.

Renal diseases

A four-year-old shorthorn cow was found dead with no history of clinical illness. Severe bilateral pyelonephritis was identified on postmortem examination (Fig 3). The renal pelvises and proximal ureters were distended with haemorrhagic, purulent urine and bacteriology produced a pure, profuse growth of *Corynebacterium renale*. *C. renale* is a common inhabitant of the lower reproductive tract and is an opportunistic pathogen that takes advantage of tissue damage and/or immunosuppression with ascending infection occurring most frequently in the periparturient period. Sudden death is an unusual presentation for pyelonephritis and despite chronic renal pathology the cow was in good condition and had been eating. The same condition was diagnosed on a second holding in a 13-year-old beef cow that had been purchased three weeks earlier for fattening. This animal was more typical, being thin with a history of malaise and reduced appetite in the week prior to death. Cases of pyelonephritis are usually sporadic, but it is worth checking that access to water is adequate as poor intakes in housed cattle is a possible predisposing factor.



Figure 3 – Pyelonephritis due to *Corynebacterium renale* in a four-year-old beef cow

SMALL RUMINANTS

Alimentary tract disorders

Post-operative ileus was suspected in a two-year-old Texel ewe that had died a few days after an embryo transfer flushing procedure. Postmortem examination findings included marked distension of the large intestine by liquid contents (Fig 4) and haemorrhage within the adrenal glands. Haemorrhages in the adrenal glands in humans may be associated with acute illness or stressful events such as surgery and are thought to be the result of sympathetic system activation.³ There is experimental evidence in sheep that postoperative ileus is mediated by the sympathetic nervous system, as ileus secondary to laparotomy and surgical procedures was prevented by splanchnicectomy. Stress-mediated changes were therefore thought to be the most likely explanation for the unusual findings in the ewe.

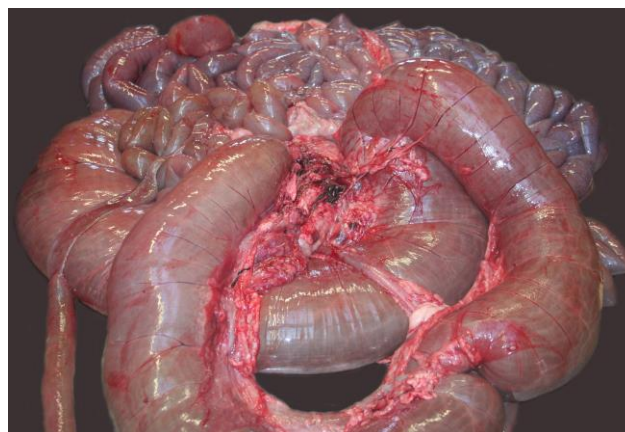


Figure 4 – Distension of the large intestine in a ewe with suspected post-operative ileus

Musculo-Skeletal conditions

Six weeks after moving to winter grazing 20 per cent of a group of 165 Scottish blackface and mule hogs were reported to be dull and ill thriven. One blackface hogg was euthanased for investigation and the remainder were housed. The carcass weighed 17 kg and was very thin with no body fat. Significant pneumonia due to *Mannheimia haemolytica* and *Trueperella pyogenes* was detected but not considered to be the main issue. The femoral cortex was thinner than average with a diameter of 1.5 to 2 mm and the frontal bone was paper thin and translucent. The brain and growth plates were grossly normal. Copper deficiency and lead toxicity were ruled out as the cause of the osteoporosis. Histopathology detected established abomasitis and enteritis consistent with a chronic response to parasitism despite the worm burden being low at the time of death. Evidence of a vacuolar encephalomyelopathy was also considered to be

secondary to metabolic disturbances associated with the gastroenteritis. Examination of the frontal bone and long bones confirmed pronounced osteoporosis with premature closure of growth plates and growth arrest lines in the proximal diaphyses. These findings indicated that the bones had stopped growing at least twice; most likely as a result of protein deficiency/general under-nutrition. Osteoporosis of the frontal bone in ill thriven hogs has been recognised for many decades.⁴

Renal diseases

A beltex tup with a two-week history of rapid weight loss was euthanased and an on-farm postmortem examination performed. Prior to death the urea value was noted to be elevated at 115 mmol/l (reference range 4-8 mmol/l) and the albumin result of 28 g/l was slightly low (reference range 30 to 40 g/l). Both kidneys were reported to be pale and swollen and representative sections were fixed in formalin. A severe chronic global, diffuse glomerulopathy together with variable plasmalymphocytic and neutrophilic tubulointerstitial nephritis was described on histopathology. Abundant tubular luminal hyaline cast formation was evident particularly within the medulla and a Congo red preparation viewed under polarised light revealed apple green birefringence particularly within glomeruli. These findings confirmed glomerular amyloidosis as the cause of the nephropathy. Amyloidosis occurs secondary to chronic inflammation the source of which was not clear in this case.

Skin diseases

Samples were submitted from two separate outbreaks of lower leg lesions during December. In the first case affected sheep were reported to be lame. Biopsies were taken and histopathology confirmed epidermal hyperplasia and pronounced dermal vascular proliferation suggestive of orf, however no lesion typical of parapoxvirus was detected. PCR testing for parapoxvirus DNA was not carried out. There was also evidence of superficial bacterial infection and *Streptococcus dysgalactiae* was detected in pure culture from one of three swabs and in mixed growth from another. In the second outbreak lesions were present between the coronary band and fetlock of affected sheep. Spread within the flock had occurred over a period of three months but lameness was not reported and there was no response to foot bathing. Both ulcerative and granulomatous lesions were described. Histopathology found extensive dermal vascular proliferation and granulation tissue indicating that the lesions were well-established. Purulent exudation extended beyond the surface into deeper tissues, suggesting that bacterial infection was contributing to the lesions. *Staphylococcus*

aureus was isolated but parapoxvirus DNA was not detected by PCR testing. Outbreaks of similar lesions have previously been reported⁵ and the aetiology remains to be clarified.

BIRDS

Pigeons

Fixed and fresh viscera were submitted following the death of eight adult pigeons from a group of 25. Affected birds had reduced appetites with associated weight loss and green diarrhoea. Histological evidence of interstitial nephritis with tubular degeneration and necrosis plus lymphocytic pancreatitis were highly suggestive of pigeon paramyxovirus (PPMV). The Animal and Plant Health Agency (APHA) was notified and further testing was initiated. The clinical presentation in outbreaks of PPMV can be variable and classical neurological signs such as torticollis and paralysis may not be observed. PPMV is a notifiable disease and suspect cases, including incidents where the possibility of PPMV cannot be excluded, should be reported immediately to APHA.

Wild birds

A member of the public reported finding five dead carrion crows (*Corvus corone*) and two were submitted for postmortem examination. White mucus was found with the pharynx and there was a severe fibrinous air sacculitis in both. A fibrinous pericarditis was also noted in one bird (Fig 5). Both birds were very thin and no food was detected within their digestive tracts. Small numbers of tapeworm were present in the small intestine and considered an incidental finding. Bacteriology was carried out on completion of testing to exclude infection with avian influenza virus, and *Pasteurella multocida* was cultured from the air sac of one bird. This is a recognised cause of respiratory disease in corvids and losses can be high due to the large numbers of birds congregating at roosts.⁶



Figure 5 – Fibrinous pericarditis due to *Pasteurella multocida* infection in a carrion crow

MISCELLANEOUS

Deer

A seven-month-old, female, red deer calf was found dead and presented for postmortem examination. Thirteen deaths had occurred in the three weeks since the group of 130 were weaned and housed and the keeper reported a lot of coughing. The carcass was thin with a severe, predominantly cranioventral pneumonia characterised by multiple, often coalescing, firm, slightly raised areas and *Dictyocaulus* spp were detected in the bronchi. Other findings included a 10 cm length of necrotic colon with secondary peritonitis, a single rumen ulcer and several renal infarcts. Histopathology confirmed a granulomatous pneumonia associated with numerous lungworm larvae and eggs and multifocal mycotic pneumonia. Fungal hyphae were also identified in association with the rumen ulcer and renal infarcts indicating systemic mycosis. Thrombosis associated with fungal colonisation was also considered to be the cause of the devitalised colon wall which was the ultimate cause of death. The rumen ulcer was thought to be the most likely source of the mycotic infection. The calves had been treated with an ivermectin/triclabendazole product eight weeks earlier which would have allowed re-infection with lungworm prior to housing. Further anthelmintic treatment was recommended.

References:

- 1 Acres SD. Enterotoxigenic *Escherichia coli* Infections in Newborn Calves: A Review. *J Dairy Sci* 1985; 68(1): 229-56
- 2 Duarte ER, Hamdan JS. Otitis in cattle, an aetiological review. *J Vet Med B* 2004; 51: 1-7
- 3 Simon DR, Palese MA. Clinical update on the management of adrenal haemorrhage. *Curr Urol Rep* 2009; 10: 78-83
- 4 Nisbet DI, Butler EJ, Bannatyne CC, Roberston JM. Osteodystrophic diseases of sheep 1. An osteoporotic condition of hoggs known as double scalp or cappel. *J Comp Path* 1962; 72: 270-80
- 5 van der Burgt GM, Ward T, Codner J. Unusual skin lesions in sheep. *Vet Rec* 2011; 168(21): 569-70
- 6 Strugnell BW, Dagleish MP, Bayne CW *et al.* Investigations into an outbreak of corvid respiratory disease associated with *Pasteurella multocida*. *Avian Path* 2011; 40(3): 329-36

Investigating congenital neurological presentations in lambs - 1

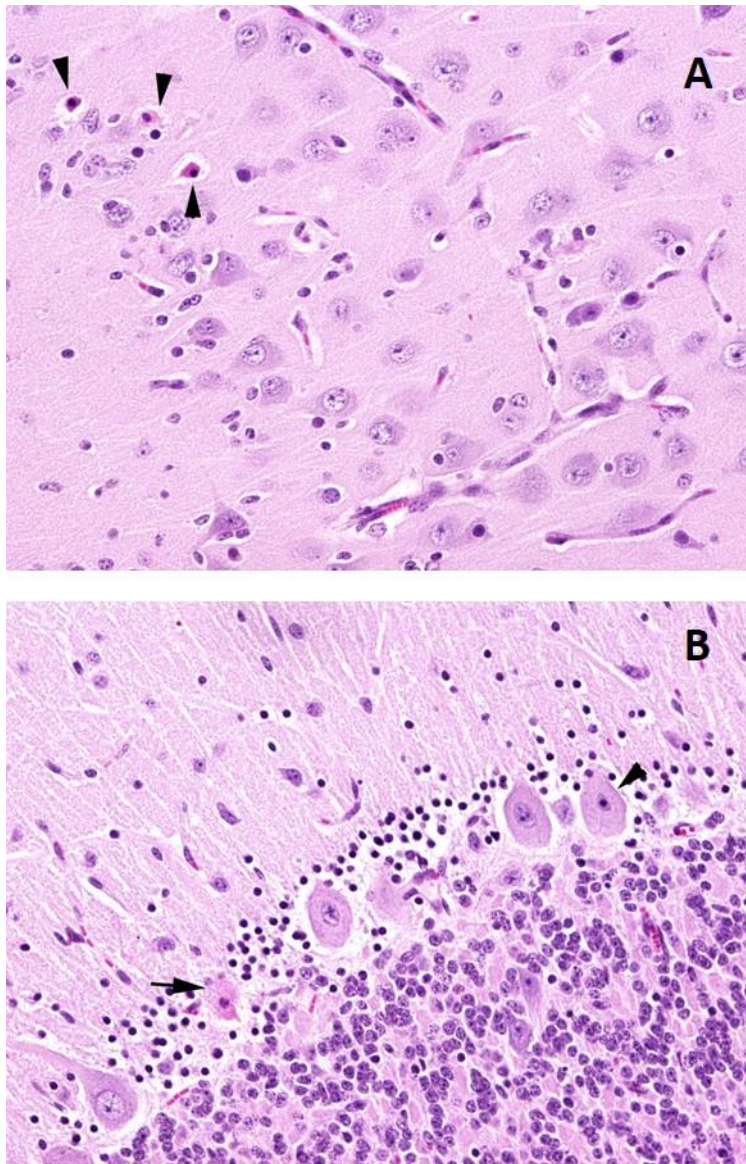
In-utero copper deficiency (congenital swayback) and the teratogenic effects of transplacental Border disease virus infection are well recognised causes of neurological presentations in lambs. However, in a neuropathological survey of neonatal lambs, superficial laminar cerebrocortical neuronal necrosis (SLCCN) was detected more frequently than these conditions (1). This article summarises the clinical and laboratory investigation of SLCCN. The differential diagnoses and investigation of other causes of congenital neurological presentations in lambs will be included in a second article.

Lambs with SLCCN often present with other lesions, most commonly cerebellar Purkinje neuronal necrosis (Fig A), with deep laminar cerebrocortical neuronal necrosis, vacuolar leucoencephalopathy, brainstem neuronal chromatolysis and long fibre tract degeneration being present in a smaller proportion of cases. The severity of the clinical signs is correlated with the extent and severity of the neuropathology. The most severely affected lambs were stillborn or extremely weak, preventing the detection of other neurological disease. The less severely affected lambs were often weak, dull, unable to stand, fell to one side and some had a head tremor. A number of these lambs succumbed to secondary infections. In some flocks, lambs that were born with neurological signs (usually dullness, tremor and ataxia) that resolved over several days, and occasionally presumed affected surviving lambs had residual non-progressive cerebellar deficits.

The flocks involved reported less than 10 per cent morbidity. All lambs were from multiple births with triplets being over-represented, however variation in the severity of the clinical signs between sibling lambs was common. In two flocks, maternal serum beta hydroxybutyrate (BHB) concentrations were determined within 24 hours of parturition and were markedly elevated in four ewes that had given birth to affected lambs, however BHB concentrations were within the reference range at intervals greater than one day post-partum. The presence of maternal ketonaemia together with the clinical observations indicate that in utero energy deprivation consequent to late gestational maternal negative energy balance is the likely cause of the necrotising encephalopathy in at least some of these lambs. In one flock, maternal illness resulting in inanition was the explanation, however in most flocks management factors were considered to be the underlying cause. Checking ewe BHB levels three to four weeks pre-lambing is a useful guide to dietary energy sufficiency.

In all cases of neurological diseases in lambs, the nature of the neurological deficits, information on the clinical progression in lambs, the presence or absence of clinical signs in siblings, clinical examination of the dams, blood samples (plasma and serum) from dams within 24 hours of parturition and neuropathological examinations are helpful when investigating the cause. Analysis of BHB levels in dams within 24 hours of parturition is especially important in the investigation of lambs with suspected SLCCN.

1. Congenital necrotising encephalopathy in lambs. Scholes SFE and Watson PJ. *Veterinary Record* 2004 (154) 32



Typical neuropathological findings in lambs born to ketotic ewes.

A: Superficial laminar cerebrocortical necrosis with individual necrotic neurones (arrowheads) in the superficial laminae of the frontal cortex

B: Individual Purkinje neuronal degeneration (arrowhead) and necrosis (arrow) in the cerebellar vermis.