

Coenuriasis in Cattle

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Although coenuriasis in cattle is referred to in textbooks, it is less common than in sheep, and is possibly not considered in the differential diagnosis of nervous disorders of cattle in most areas of the United Kingdom. (Greig and Holmes, 1977).

Coenuriasis (local names: Gid, Bendro, Sturdy, Tuatheal) is the disease caused by invasion of the brain and occasionally the spinal cord by the cystic larval stage of *Taenia multiceps*, a tapeworm of the dog and fox (Williams, 1976; Edwards *et al.*, 1979). The localized space-occupying lesion of the CNS usually results in death of the host unless treated surgically.

Prevalence and Epidemiology

Taenia multiceps is widely distributed throughout the U.K., but especially in the hilly, western parts of England and Wales (Herbert *et al.*, 1984).

In a survey of 600 flocks in Dyfed, 3% of sheep under one year developed Gid (Williams and Boundy, 1983). At Carmarthen V.I. Centre, between 1964-1976 117 cases of Gid were recorded in cattle.

Williams (1976) found that about 10% of farm dogs and fox hounds were affected in South and Mid Wales. He considered that these dogs were the major source of infection to ruminants, and that the fox was of little importance in the epidemiology of this parasite.

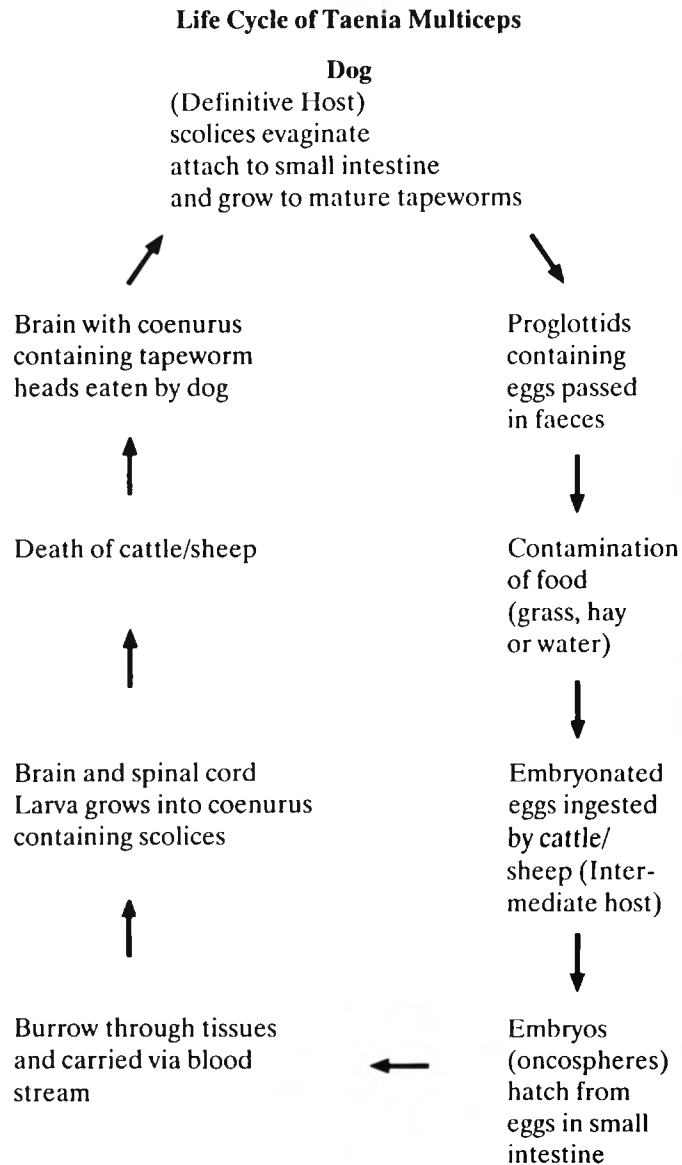
The prepatent period for the tapeworm is six weeks. The ripe proglottids are passed in the dog's faeces daily and the eggs remain viable in the pasture for up to eight weeks. The viability of scolices in coenurus in dead sheep or cattle is thought to be less than 3 days (Williams and Boundy, 1983).

Walters (1978) found that farm dogs defaecated in fields adjoining the farm buildings, resulting in a heavy build-up on the pasture of worm eggs, which infect young ruminants grazing in the spring. It appears that the younger the animal, the greater the natural susceptibility.

Pathogenesis

(a) Acute or migratory phase

Seven to 14 days after the sheep ingests embryonated eggs, the oncospheres migrate through the CNS. Clinical signs include jerky movements, excess salivation, head aversion, blindness and convulsions. Sudden death may occur from massive infection.



(b) Quiescent phase

During this phase, the larvae settle in the CNS and grow.

(c) Chronic phase

Signs may not occur until 2 to 10 months after ingestion of the eggs. Gradual enlargement of the coenurus causes pressure on nervous tissue resulting in its irritation, atrophy and eventual destruction. The signs vary according to the parts of the CNS pressed.

Materials and Methods

The author applied the tests used by Skerritt and Stal-lbaumer (1984) in sheep, to aid in localization of the coenurus in 6 calves with Gid. The hopping test was done by

lifting one forelimb and causing the calf to pivot on its hind legs.

Diagnosis

Diagnosis is based on a history of Gid on the farm or in the area, age (5-24 months), slow progression of CNS signs usually over a period of months. There are normal total and differential white cell counts, and normal cell content of CSF (lumbo-sacral puncture).

Seriological and skin hypersensitivity tests are unreliable as *Taenia multiceps* shares antigens with other cestode parasites, but a negative test is helpful. X-ray is not useful. Ultrasound was used by Doherty *et al.*, 1989 as an aid to localize a cyst in a lamb, by introducing a mini-probe through a trephine hole. This technique, in association with a detailed neurological examination, may improve the location and surgical removal of gid cysts in cattle.

Differential Diagnosis

<i>Disease</i>	<i>Distinguishing Features</i>
Coenuriasis	Chronic progressive neurological disturbance over 2 to 10 months. Usually unilateral blindness. Age 5 to 24 months
Other space-occupying lesions	
a) Intracranial abscess	Neurological signs usually progress slowly, then become static. Sometimes pyrexia. Neutrophilia. May follow dehorning
b) Intracranial haematoma	Acute onset following trauma
c) Brain tumour CCN	Rare Acute; rapidly fatal unless treated. Convulsions, opisthotonus and bilateral blindness
Listeriosis	Acute, facial paralysis, drooling, etc. Circling. Associated with silage feeding
Lead poisoning	Acute - blindness, Opisthotonus, hyperaesthesia bellowing and convulsions. Chronic - ataxia, blindness, depression. Increased blood lead concentration

Hypomagnesemia

Hyperaesthesia, excess salivation, convulsions, sudden death

BSE

Usually slow progression in cattle 2 years or older

Localization of Cyst

The clinical correlation of signs and sites of cysts is poorly documented in cattle, but in sheep Skerritt (1984), Skerritt and Stallbaumer (1984) and Skerritt (1987) found that the neurological signs can indicate location of gid cysts.

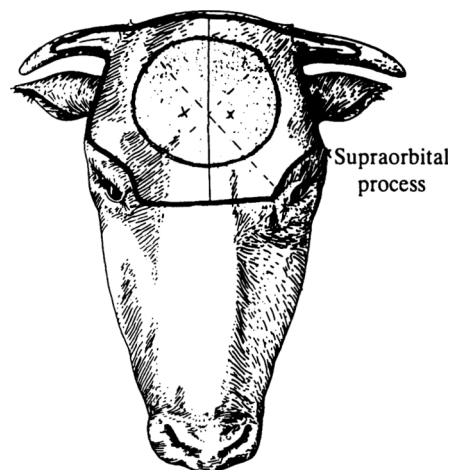
Surgical Anatomy and Site of Incision

In the bovine animal the frontal bone forms the entire roof of the cranium. It is divided into internal and external plates, between which are the frontal sinuses. Softening of the outer plate of the frontal bone occurs less often than in sheep. Due to pressure from the cyst, however, the inner plate is usually softened and pushed against the outer plate, thus partially obliterating the frontal sinus in between.

The surgical side and site is determined by the neurological examination.

The position of trephine opening (Figs. 1 & 2) should be 2cm lateral to the mid-line at the intersection of 2 lines drawn from the medial angle of the eye to the base of the opposite horn (in hornless breeds to a point a little above and behind the ear). The thickness of frontal bone in affected cattle at this point is usually 0.5cm to 1.5cm.

Fig. 1 Topography of the Brain and Frontal Sinuses in the Bovine Animal



X Site of Trephine opening
 - - - - - Outline of Brain
 _____ Outline of Frontal Sinuses

Fig. 2 Close view of calf's head showing site of incision



Surgical Treatment

The animal is starved for 12 hours. Crystalline sodium benzyl penicillin (Crystapen, Pitman-Moore) and betamethasone disodium phosphate (Betsolan soluble, Pitman-Moore) are injected intravenously, immediately prior to surgery.

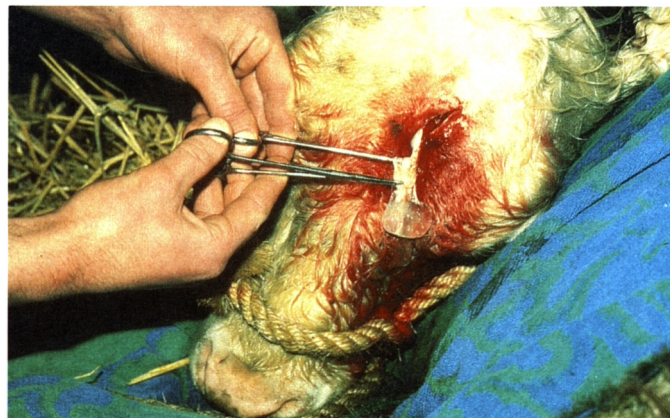
The frontal region of the head is prepared for surgery in the normal way, and the site of incision infiltrated with local anaesthetic. Xylazine 2% (Rompun, Bayer) is given intravenously at 0.5ml per 50kg. This produces recumbency, deep sedation and a useful degree of analgesia.

A 5cm (2") incision is made in the skin and the periosteum is reflected. Craniotomy is done with a 1.5cm trephine and a circular plate of bone is removed. Care is taken to avoid injury to the brain when trephining is lost. Jagged edges to the trephine hole should be avoided as these tend to grip the cyst and make extraction difficult. The meninges, which tend to bulge due to increased intracranial pressure, are incised, taking care to avoid any blood vessels.

At this stage the animal's head is rotated so that the cranium faces downwards. This greatly simplifies the drainage and removal of the cyst. The animal may struggle as the cyst is drained, so the head should be held firmly by an assistant. A 14g needle and cannula is inserted (a dog catheter size 10fg with Luer fitting can be used). The needle is removed and a 60ml syringe is attached to the cannu-

la or catheter and fluid is withdrawn slowly. It is important not to remove too much fluid before attempting to grasp the cyst wall, since the collapsed wall may shrink out of reach into the large cavity that it occupies. Negative pressure maintained by the syringe is usually sufficient to grasp the cyst wall. The cyst wall can be gently held by artery forceps whilst the remainder of the fluid is aspirated (Fig. 3). The skin is sutured in the normal way.

Fig. 3.



Post Operative Treatment

The animal should be kept in a quiet loose-box with no awkward corners where it can become trapped. Ampicillin is given i.m. for 3 days and the sutures are removed at 10 days.

Surgical or Post-Surgical Complications

There is usually a dramatic reversal of the pre-operative neurological deficits, and the CNS signs diminish gradually over a few days. However, **hyperexcitability** is a common post-operative development but usually improves over 2 to 4 weeks.

Haemorrhage

The risk can be reduced by avoiding the mid-line when trephining or pushing the needle across the mid-line, where the dorsal sagittal sinus lies in the falx cerebri. All probing should be done gently to avoid laceration of the brain.

Cerebral oedema

This is recognized by depression, stupor and dilated pupils. Betamethasone given prior to surgery reduces the risk of this complication, but if it occurs, then betamethasone can be repeated every six hours.

Infection

This is avoided by cleanliness and antibiotic cover.



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Failure to extract a cyst or more than one cyst

There may be a temporary remission and a second attempt at surgery must be done later. The author prefers to remove the whole cyst, although De Villiers (1950) achieved a 60% success rate in sheep by drawing off the fluid only.

Results

Three of the 6 calves were seen on one farm where they occurred sporadically. Prevalence of Gid in lambs on these farms varied between 1 and 8%. Dogs were not routinely wormed and in two cases the farm dog slept with the calves.

Gid occurred in various breeds at 5-24 months.

In all cases the nervous signs were slowly progressive with grazing and sucking becoming increasingly difficult. Separation from the herd occurred and the animals became easy to catch and showed little interest in their environment. They appeared dazed with a vacant expression and tended to stand still with the head pushed against a wall. On being moved from this position, circling continued until they again became trapped in a corner. There was progressive paresis and loss of weight.

All the calves tended to collide with obstacles and had normal pupillary reflex which suggested a cerebral lesion. Papilloedema was seen in both eyes in case No.1 and also in case No. 6 before the second attempt at surgery. In both cases the animal appeared totally blind. The author could not decide whether papilloedema occurred in the other cases.

Five calves were operated on successfully. Calf No. 5 died suddenly before surgery. Calf No. 1 regained its feet within 24-hrs of removal of cyst despite having been recumbent for 3 weeks previously. All the cases except one had a single cyst in either the left or right cerebrum. No cerebellar or spinal cysts were diagnosed.

Calf No. 6 appeared to recover well after the first operation when 50ml of fluid plus cyst was removed from the left cerebrum. Six weeks later the heifer's condition deteriorated, she became blind in both eyes and circled in either direction but with head aversion to the right. It was initially thought that part of the cyst had been left and re-grown, but an inspection through the initial trephine hole revealed no abnormality. The animal was then trephined on the right side and 350ml of fluid was aspirated. Unfortunately, the cyst collapsed and was not extracted. Following surgery this animal showed signs of cerebral oedema with increased intracranial pressure probably caused by the trauma of removing such large quantities of fluid. Betamethasone was given every 6 hours until remission of signs. This heifer made a full recovery but had remaining hyperexcitable when approached, until slaughtered four months after the second operation.

TABLE 1 Clinical signs

Calf No	1	2	3	4	5	6	
Circling (wide) left or right	L	R	R	L	R	L	Circled to both L and R before 2nd op.
Head aversion left or right	L	—	R	—	R	—	Head aversion to R before 2nd op.
Grinding of teeth	Yes	—	—	Yes	Yes	Yes	
Snoring	Yes	—	—	—	—	—	
Recumbency	R i g h t side for 3 weeks before surgery						
Ataxia	Yes	Yes	Yes	Yes	Yes	Yes	
Paresis	Yes	—	—	Yes	Yes	Yes	

Cerebellar signs such as head tremor, opisthotonus and dysmetria were not seen. However, calf No. 1, which had been recumbent for 3 weeks before it was first examined, had nystagmus in both eyes.

TABLE 2 Visual and postural deficits

Calf No.	1	2	3	4	5	6
Blink reflex	No response	No response R	No response L	No response R	No response L	No response R Totally blind 6 weeks after 1st op.
Unilat. blind folding	Not done	Covering caused total blindness	Covering caused total blindness	Covering caused total blindness	Covering caused total blindness	Covering of L caused total blindness
Hopping test	Not done Recumbent	Deficit R	Deficit L	Deficit R	Deficit L	Deficit R
Walking up and down steps		Deficit R	Deficit L	Deficit R	Deficit L	Deficit R initially then L after operation.
Side of operation	L	L	R	L	R	L Re-operated 2 mos. later R

TABLE 3 Summary of neurological examination

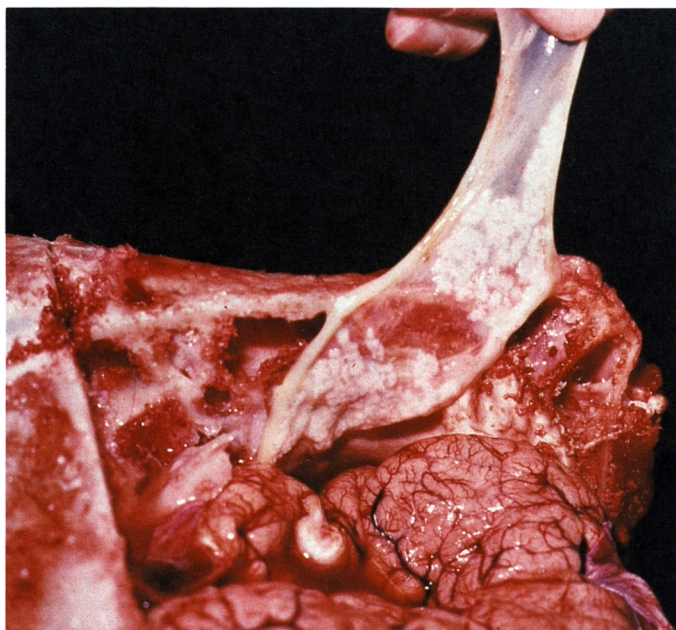
Clinical sign	Interpretation (situation of cyst)
Dull sound on percussion Slight softening or pain elicited on digital pressure of frontal bone	Ipsilateral cerebrum
Circling (wide) and head aversion	Ipsilateral cerebrum
Unilateral blindness	Contralateral cerebrum

Bilateral blindness Suggests large cyst
 Postural deficits Contralateral cerebrum

Post mortem findings of calf No. 5

A large coenurus cyst was found in the right cerebrum covered only by the meninges and a thin layer of brain parenchyma. The gyri above the cyst were flattened due to pressure. Herniation of the caudal cerebrum ventral to the tentorium cerebelli had occurred together with compression and coning of the cerebellum. The cranium overlying the coenurus had undergone softening and thinning.

Fig. 4 Coenurus



The semi-translucent cyst may contain between 70 and 1200 tapeworm heads (scolices), which develop from its inner wall. The cyst may contain 100 to 350 ml of fluid. Above figure shows cyst being teased from the brain of dead calf No. 5.

Post mortem findings of heifer No. 6.

A small cavity was found in the left cerebrum and a large cavity in the right cerebrum, with no connection between them. The remains of a degenerated cyst was found in the larger cavity which proved that the animal can get better even if the cyst is not removed.

Prevention and Control Measures

These are similar to those for the control of hydatidosis. Walters (1977) observed that Gid was virtually eliminated on farms where he used routine anthelmintics against *Echinococcus spp.*

1. **Do not:**
 - a) feed uncooked ruminant heads, raw meat or offal to dogs.
 - b) allow calves and lambs to graze heavily infected pasture e.g. following sheep dog trials or use by hounds.
2. **Do:**
 - a) dispose of ruminant carcasses properly.
 - b) strictly control disposal of offal and heads from abattoirs and knackers.
 - c) confine farm dogs when they cannot be supervised.
 - d) take action against stray dogs.
 - e) persuade farmers and kennels with hounds or gundogs to give an effective cestocide every six weeks, or at least dose during the danger period for calves and lambs. Praziquantel (Bayer, Droncit) is most effective against both immature and mature forms. Since cestocides are not ovicidal dogs should be confined for 24 hours after treatment and the faeces buried.

Conclusions

The findings of Skerritt and Stallbaumer (1984) in sheep is confirmed by the author in cattle: the success of surgical treatment is dependent on the location of the coenurus, its localization by interpretation of clinical signs, and the degree of damage produced by the coenurus and by surgical interference.

If the animal is not in good condition, slaughter should be considered, otherwise surgery is well worth attempting.

Many of the cases occur in calves which have never been outside and where infected dogs have slept either on hay bales or in calf pens. Farm dogs and foxhounds are the major sources of infection and if they are regularly treated with an efficient cestocide, the losses from Gid could be significantly reduced.

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Abstracts:

A new diarrhoeic syndrome with ataxia in young Charolais calves: clinical and microbiological studies

J. Espinasse, H. Navetat, M. Contrepolis, D. Baroux
F. Schelcher

Veterinary Record (1991) 128, 422-425

A new diarrhoeic syndrome was examined clinically in 19 one to two-week old Charolais calves. It differs from other digestive disorders in calves of this age in the discrete diarrhoeic signs, the absence of dehydration and the presence of signs of ataxia. The microbiological study carried out for three consecutive years in 58 sick calves and nine healthy control calves demonstrated the special role of *E coli* possessing virulence markers from septicaemic strains (CS31A, Col V). The clinical signs could be the result of bacteraemia with subacute *E coli* endotoxaemia.

Calf neonatal enteritis is always accompanied by diarrhoea and dehydration and associated with anorexia and asthenia to different degrees (Blood and Radostits 1989). In France (deRycke and others 1986) and other countries (Snodgrass and others 1986) the pathogenic agents most frequently involved are rotavirus, coronavirus, enterotoxinogenic *Escherichia coli*, salmonella and cryptosporidium.

A new diarrhoeic syndrome, in which asthenia and ataxia are the predominant clinical signs, has been prevalent in the Charolais area since the winter of 1986. Mucoïd diarrhoea, sometimes preceded by constipation, appears before or after the nervous signs, but dehydration is rarely observed. The author's studies were carried out during three calving periods in the winters of 1986/87, 1987/88 and 1988/89, to determine the clinical and microbiological characteristics of the syndrome.

Replication of bovine viral diarrhoea virus in the bovine reproductive tract and excretion of virus in semen during acute and chronic infections

P.D. Kirkland, S.G. Richards, J.T. Rothwell, D.F. Stanley

Veterinary Record (1991) 128, 587-590

Five mature bulls were studied during an acute transient infection with bovine viral diarrhoea virus (BVDV). The bulls had been infected experimentally by the intranasal instillation of blood and serum from a cow which was a persistent carrier of the virus. Infection was confirmed by the demonstration of a low titred viraemia in four of the five animals and by the seroconversion of all five. Semen samples were collected from each bull on four occasions between seven and 14 days after infection. The virus was isolated from the semen of three of the five bulls and from nine of 12 batches of semen from them. In contrast to other studies of the infection of semen, BVDV was isolated with similar efficiency from raw, unprocessed semen and from diluted, extended semen. The titres of virus in the semen ranged from 5 to 75 TCID₅₀/ml. The infection did not appear to affect the quality of the semen. Shedding of virus continued after the end of the period of viraemia and appeared to be a consequence of the replication of the virus in the reproductive tract and its subsequent excretion in the seminal fluid. Virological studies of the reproductive tracts of these bulls suggested that the most productive sites of virus replication were the seminal vesicles and the prostate gland. Con-current studies in a persistently infected bull supported these findings.