The Many Causes of Down Cows

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The down cow is a diagnostic challenge because of the multiplicity of factors which can lead to the same general presentation. Recumbency itself makes examination more difficult due to the bulk and weight of modern dairy cows. The superimposition of several causes in a single case make the order of events and the relative importance of various factors difficult or impossible to sort out. On post mortem examination secondary ischemia and muscle tearing, dislocations and fractures may be readily apparent while primary causes remain elusive.

Downers connote different things to different people. Disabled animals in stockyards are often referred to as downers[1] and the humaneness of their treatment has become the focus of considerable public controversy. In dairy practice the term downer is more specific and usually refers to a cow which is in sternal recumbency but cannot rise. In contrast, the cow in lateral recumbency is usually considered a dying animal rather than a treatable entity. Appearances can be deceiving, however. Some downer cows which are well bedded will look almost normal in sternal recumbency, but then when they briefly lie on their sides they will "suddenly" appear much worse. Actually, it is a good sign when a cow can go back and forth between sternal and lateral recumbency voluntarily; many can't do this. One of our experimental downers was found dead one morning and she was still in sternal recumbency with her head off the ground in an almost normal position. Post mortem examination revealed massive DIC (disseminated intravascular coagulation). Apparently she died slowly and went into rigor in the process. Downers in sternal recumbency are further divided into alert and non-alert groups with the former being further divided into creeper and non-creeper types depending on the amount of their movement.[2]

Systemic versus non-systemic and primary versus secondary

To a first approximation the alert downer is the result of local damage while the non-alert downer more typically suffers from systemic problems. This, however, is an oversimplification. Treatment of systemic causes such as parturient paresis can change a non alert downer into an alert one, and persistent systemic problems can lead to regional myoneural injury.[3]

While primary causes of the downer cow problem can be either systemic or local, the secondary effects are always local tissue injury resutling from compression of the limbs or musculoskeletal damage due to struggling to rise or "creep". Regardless of the primary cause of the recumbency, all down animals are subject to tissue compression in the hind limbs which becomes the common "unifying factor" for all downer cow cases. In some cases tearing of the adductor muscles results in hemorrhage, and cows lying in the so called "spread eagle" position are especially susceptible to hip luxation.

Regional damage to muscles causes release of breakdown products into the systemic circulation which leads to elevated levels of enzymes such as creatinine kinase (CK) and myoglobinuria which is seen as brown urine during the first few days of recumbency. In the most severe cases renal damage may result from massive amounts of muscle breakdown products overwhelming the kidney. Renal damage then leads to uremia and other systemic signs of renal failure. Therefore, the interplay of systemic and regional factors can go in both directions. Table 1 summarizes the various factors involved in the downer condition.

Table 1. Factors contributing to the downer cow condition.

	Systemic	Non-systemic (local)
Primary		
	hypocalcemia	calving paralysis
	hypophospahatemia	fractures due to falls
	hypokalemia	lymphosarcoma
	hypomagnesemia	aortic thrombosis
	toxic mastitis or metritis	vertebral abscess
	stress of parturition	vertebral fracture
Secondary		
	renal failure	muscle ischemia and tearing
	DIC	sciatic nerve damage
		hip luxation
		fracture of a femoral head

Systemic causes

An important etiologic clue is when the cow went down with regard to calving. Most dairy downers occur in the periparturient period[2] and are commonly associated with hypocalcemia, but hypophosphatemia[4] may complicate matters. Beyond the periparturient period but during the first month after calving, hypokalemia may occur. After peak lactation, metabolic problems are not likely to be responsible for downers, but a variety of non-systemic causes can lead to the downer condition.

Cows which receive several doses of isofluopredone for treatment of ketosis can become recumbent with hypokalemia due to the mineralcorticoid activity of the drug.[5] For unknown reasons, type 2 muscle fibers are most severely affected. Hypomagnesemia[6] has been mentioned as a cause of the downer syndrome but not widely supported.

Non-systemic primary causes

If dystocia has occurred, then calving paralysis may be a contributing factor as well. Calving paralysis is the preferred term for what is often referred to as obturator paralysis. Bilateral experimental transection of only the obturator nerves will not put a cow down unless the cow is on slippery concrete.[7] These cows will appear normal at a walk on dry concrete, but will spread the hocks wide when running. When they fall on dry concrete they can regain standing, but on a wet surface they have a hard time standing. The larger lumbar root of the sciatic nerve, as well as the obturator nerves, are vulnerable to compression damage between a fetus and the bony pelvic inlet portion of the birth canal. When both the lumbar root of the sciatic and the obturtator nerve are cut bilaterally, the experimental cow will be ataxic and not able to rise to a standing position in most cases.

If dystocia occurred then intrapelvic trauma and exhaustion due to calving could be contributing factors. In one case a 13-year-old Angus cow was seen to be lame on the left hind several days before calving but became a downer at calving.[8] Post mortem examination revealed fractures of the left iliac shaft and floor of the pelvis. Apparently the cow slipped on ice prior to calving, but the mechanical stress of calving worsened the effect of the fractures.

Lymphosarcoma when it invades the vertebral canal is most commonly found in the lumbar region.[9] The predilection for this region is probably due to metastasis from the uterus and some other abdominal viscera. Venous drainage from the uterus, pelvic viscera and descending colon is via the caudal vena cava. Anastomotic connections between the caudal vena cava and the vertebral venous sinuses in the floor of the vertebral canal have been demonstrated by positive contrast radiology (venography). Blood in the vertebral venous sinuses can return to the caudal vena cava or go to the heart via the azygous (hemiazygous in cattle) vein(s). During the abdominal press as in coughing, defecation, urination or labor, blood can be forced from the caudal vena cava and into the vertebral venous sinuses. Once the brief abdominal pressure is past, blood in the vertebral venous sinuses will flow to the point of lowest pressure and hence some of it may return "from whence it came". If the flow of blood in the venous sinuses reverses, there is a brief moment when the velocity is zero. At that time the probability of a wandering metastatic cell adhering to the inner surface of the vertebral venous sinuses is enhanced greatly. *[The right heart predilection of bovine lymphosarcoma may be due to brief flow irregularities there].*

A quick and easy way to visualize this lesion is to remove the lumbar part of the axial skeleton by transverse section at L1 and 6. The lumbar vertebrae should be put in a freezer for a few hours and then sectioned with a bandsaw transversely. After cooling, the fat around the dural tube becomes hard making it easier to differentiate from the softer light brown to pink lymphosarcoma masses which compress the cord and spread laterally into the intervertebral foramina. For confirmation, an impression smear can be made and stained with new methylene blue.

A less common cause of the downer condition not related to calving is aortic thrombosis. A saddle thrombus is one that gets "hung up" at the bifurcation of the aorta to form the iliac vessels. Such a thrombus has been reported[10] to be the cause of a downer cow. Although this is only a single reported case, others may have gone unreported because so few downers are subjected to thorough post mortem examination; and when they are, such a lesion is easily over-looked.

Longitudinal section of the vertebral column with a bandsaw may reveal a vertebral fracture in the lumbar region or an abscess. An abscess of a vertebral body may be the result of infectious material finding its way into the region and "settling down" via the mechanism mentioned above for vertebral lymphoma. Such an abscess can result in a so-called pathologic fracture and subsequent injury to the roots of the lumbosacral plexus and the cauda equina (the bovine cord ends at the level of the cranial part of the sacrum).

Non-systemic secondary causes

Experimental induction of the downer cow condition has been used to study the effect of muscle and nerve compression in a controlled laboratory trial free of the numerous variables present in clinical cases.[3] Healthy non-pregnant cows were anesthetized with halothane anesthesia for 6, 9 and 12 hours to mimic the effects of

recumbency in parturient paresis. All experimental cows were positioned on a hard rubber cow mat with the right hind limb under the body . Blood gases were monitored throughout the anesthetic period, and positive pressure ventilation with a mechanical respirator was used to maintain normal blood gases. Within 30 minutes after the end point of anesthesia, the cows were attempting to rise. Half (8 of 16) of the experimental subjects were able to rise within 3 hours after the end of anesthesia, but some of these showed temporary peroneal nerve paralysis on the previously down hind limb. Several cows which were not able to stand were able to rise on their forelimbs into an unusual bovine "dog sitting" position. All the others were able to stand on their forelimbs when lifted with clamps on the tuber coxae. These observations correlate with numerous experiences with clinical cases. That is, during the first few days of recumbency or longer, most alert downers can stand on their forelimbs when lifted with hip clamps. This observation clearly shows the importance of myoneural damage to the hind limbs due to recumbency. It is impossible to explain such an observation as a result of systemic problems alone. When the experimental downer cows were lifted after a day of recumbency the hind limb which was down during the anesthetic recumbency was swollen markedly in most cases.

There are two reasons for the sparing of the forelimbs. The first is that in sternal recumbency the forelimb is never under the body as one hind limb usually is. The second is that the brisket is a modification of the sternum specifically to bare weight during sternal recumbency and it works very well. Since most of the fore weight is borne by the brisket, the forelimbs are spared from compression damage.

Post mortem examination of experimental and clinical downer cows reveal similar lesions which are for the most part localized in the upper hind limb. This is because the upper parts of the hind limb are compressed against the pelvis while the more distal parts are positioned under the belly which is soft. While the abdomen is soft, it is also heavy and this weight on the common peroneal nerve can cause compression damage where the nerve crosses the stifle joint, especially the lateral surface of the head of the fibula.[11] For this reason, knuckling over on the fetlock, a sign of peroneal damage is often seen in recovered downer cows. This deficit is usually temporary, but it can be permanent.[11]

When a cow is in lateral recumbency, the forelimbs are no longer protected from compression damage. The brachial plexus can be compressed between the wide flat ribs of the cranial bovine thorax and the medial side of the shoulder joint. This can be prevented by pulling the forelimbs forward so that the shoulder joint is not in direct contact with the rib cage. At Minnesota we saw a successful teat repair go sour when the patient came off the table with the down forelimb paralyzed. Despite considerable time and effort working with this cow, she never recovered and was euthanized.[2]

Gross pathology of the hind limb includes pale ischemic muscle mixed with hemorrhagic torn muscles, but the torn muscles are limited to the medial thigh region where the adductors lie. Discoloration and scar tissue around the sciatic nerve is primarily in the region caudal to the hip joint where the nerve wraps around the upper end of the femur and is vulnerable to compression against the femur at this point.[2]

Sciatic nerve damage also can occur due to dislocation of the femur if the head of the femur moves cranio-dorsally towards the greater sciatic foramen. More often, however, the head of the femur moves ventral and into the obturator foramen.[12-14] Once it forms a bony false acetabulum, the hip will be stabilized again. This has been observed to happen within 42 days.[14]

The amount of muscle function loss and the location of the dysfunctional muscles determine the ability to stand or become recumbent by default. While muscle damage may be more striking in appearance on post mortem examination, a smaller lesion to the sciatic nerve will have more profound consequences due to the wide distribution of the nerve. Therefore, subtle differences in position during the initial recumbency will determine the location and severity of lesions and the ensuing deficits. Minimizing this damage should be the aim of both prevention and treatment efforts.

**** In memory of Dr. John Stephen Onapito, a fine clinician and scientist who died soon after returning to his native Uganda. Probably, he was the only U.S. graduate student to complete a PhD thesis on the downer cow problem.***

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