Musculo-skeletal examination and diagnosis of the downer cow

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Abstract

There are many primary conditions that cause a cow to become a downer, some of which are injuries to their musculo-skeletal system. Most downers, no matter their primary cause, also suffer secondary musculo-skeletal damage from the recumbency and this damage often becomes the main reason for them failing to rise. Thus, it is vital to be able to carefully examine the musculo-skeletal system of a downer. This paper will discuss techniques for the examination of the back, pelvis, hind and fore limbs. It will include static and motion palpation, sensory and motor function tests. Neuropathies of the sciatic and femoral nerves can be graded to quantify their level of damage. The use of enzyme tests as a prognostic guide to the ‘compartment syndrome’ will also be discussed.

Introduction

Recumbency is a state of ‘lying down’. It is important to differentiate between a down cow and a downer cow: A down cow is a cow that is recumbent and so is unable to stand up unassisted. Some, however, may be able to stand and even walk around after being lifted but are still unable to stand up unassisted. A down cow is different to a downer cow: The term downer cow is often a vague term and can mean different things to different people: It has been described as “a cow that is unable or unwilling to stand for a variable period of time; persistent (greater than 12 hours), intractable recumbency appears to be the common thread of such cases” (van Metre 2001). Others define it as “a general term that applies to any periparturient cow that is in sternal recumbency and is unable to rise, but where the reason for the recumbency is unknown” (Parkinson and Vermunt et al. 2010) or as “any cow that is down in sternal recumbency for more than 24 hours with no evidence of systemic illness” (Cox 1981). Cox’s definition includes cows other than periparturient ones and includes cows where the diagnosis is known. For the purposes of this paper, I consider downers to be cows that have been down for more than 12 hours irrespective of whether the original cause is known or not and without systemic illness. They are, thus, bright and alert. This fits with Van Metre and Cox’s definitions and only differs from Parkinson’s in cases where the primary diagnosis is known.

Downers can be thought of in terms of up to three phases: the initial cause of the recumbency; the secondary damage from the initial recumbency; and possible tertiary damage.

The primary condition can include a multitude of possibilities which can be grouped into four major categories: metabolic, acute systemic illness, musculo-skeletal or miscellaneous. The paper will focus on the primary musculo-skeletal causes and on the secondary and tertiary damage from the recumbency. It will discuss the examination protocol used in our practice for downer cows when looking for this musculo-skeletal damage, be it the primary or subsequent damage. This damage maybe to muscles, nerves or other structural components. Nerve injuries are further differentiated into the specific neuropathy (sciatic, peroneal, tibial, femoral, obturator, brachial plexus and radial).

The secondary damage is commonly to either the hamstring muscle group or to the peripheral sciatic nerve and its branches. Other neuropathies, including brachial plexus or radial nerve of the forelimb can occur. A variety of additional complications, such as dislocated hips and ruptured gastrocnemius muscles are an occasional
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finding. Secondary damage is due directly to the effects of recumbency whereas tertiary ones follow on from secondary damage. Tertiary conditions could include a ruptured gastrocnemius muscle or dislocated hip from a cow unable to rise due to the secondary muscle or nerve damage already sustained following the primary recumbency. Hip dislocations are interesting as they can be primary, secondary or tertiary. Kidney failure from an excess of myoglobinuria is a tertiary problem following from the secondary compartment syndrome. Other secondary problems would be mastitis from lying in an unhygienic environment or pneumonia from lack of shelter.

The important thought process with all downer cows is to consider not only the initial cause of the recumbency but also any subsequent damage, which follows from the recumbency. The time the recumbent cow has been down prior to the veterinarian’s first examination will influence whether treatment is more appropriate for the primary condition or the secondary complications.

Examination

When examining a recumbent cow, regardless of whether she is a downer at this stage or not, a full clinical examination should be done. This is important to help sort out which of the many possible primary diagnoses she fits into. The techniques I will outline focus on looking for primary musculo-skeletal causes but even when the diagnosis is of a different condition I will still use these techniques to look for secondary damage. For example, a cow recumbent with Protein-Energy Deficiency, which may have a good prognosis of recovering from this primary disease, will have a different prognosis if she has other damage, such as secondary muscle or nerve damage. It goes without saying that it is vital to look for and to find these complications as much as it is to correctly diagnose the primary condition. As with all clinical examinations, it isn’t finished until a thorough examination has been completed and one mustn’t stop when symptoms of one disease are found in case there are other disease processes occurring. This is particularly true for downer cases as many veterinarians don’t tend to think of the secondary effects of the recumbency as they only focus on the symptoms of the primary disease.

When examining a cow that has been recumbent for more than one day the emphasis on the examination is more on the secondary damage and less on the primary cause of the recumbency.

If examining a cow that has been recumbent for a period of time that was initially bright and alert but is now depressed, the likelihood of a complicating pneumonia, mastitis or kidney failure needs to be considered. Mastitis is a risk due to lying in conditions, which are often unhygienic, combined with the risk of leaking milk due to the pressure on the udder from the recumbency. Pneumonia is an occasional finding in cows being nursed without adequate shelter or from aspiration whilst cast in lateral recumbency. Pneumonia and mastitis are possible secondary complications of downers. Myoglobinuria is a common finding in downers due to secondary muscle damage. High levels of myoglobin can cause kidney failure, which would be regarded as a tertiary complication. Perhaps some cows that are accused of ‘sulking’ and ‘throwing-it-in’ are actually suffering from renal problems?

Please ‘take it as read’ that the normal clinical examination has been performed and I will now discuss the detailed musculo-skeletal examination:

Observation of the patient is an important aspect of the examination as noting the posture and responses of the animal during the examination may give some clues: Is the cow bright and alert? Is she in sternal or lateral recumbency? Is she trying to get up, and if so, does the strength of the legs vary from each other? Is she in a normal sitting position, or are any of the legs in an abnormal position? When she tries to stand up, which way do the legs tend to go? Can she change sides or is one hind leg weaker than the other? Are there any obvious swellings?

With the cow in sternal recumbency a back examination is carefully performed. The back is examined for any obvious swellings and palpated for painful areas. This is best done using a blunt probe, such as a vacutainer tube. The probe is run lightly over the lumbar and pelvic musculature seeking acutely painful areas. If none are found the probe is then used to provoke the area more vigourously, as the thick bovine skin often masks mildly painful areas. Pain responses must be repeatable and consistent to be significant and may help localise the affected area. Lack of pain in an area doesn’t preclude trauma in this area.

The vertebral column is then carefully palpated for misalignments and instability. This is done in two parts: static palpation and motion palpation. Static palpation involves observing and feeling the dorsal spinous processes of the thoracic, lumbar and sacral regions and the lateral processes of the lumbar vertebrae. This procedure is to analyse the relationship of these osseous processes to their neighbouring segment to assess if there is any damage. The dorsal spinous processes should follow a natural curvature when the cow is in sternal recumbency, which is
altered if there are fractures of them. The displacement may only be one centimetre from the normal position, so careful examination is required. The relative position of the processes may change when the cow is rolled onto her other side, so this should also be carefully observed. The lateral processes are noted relative to their neighbouring processes and if this orientation changes when the cow is rolled from side-to-side. Any suspected mal-alignment must take into account variations that occur from previous injuries, as it is not uncommon to find old fractures of lateral spinous processes.

Motion palpation is also used to identify areas of vertebral instability. The clinician uses the heels of both hands to push on each vertebral segment several times, in a dorso-ventral direction, to set up a rocking motion. The lumbar region is tested, with particular emphasis in the mid-lumbar area. This is repeated along each of the segments. The amount of normal movement varies slightly along the spinal column with maximum movement at the mid-lumbar region, which has the minimum amount of bracing from the ribs and pelvis. The test is designed to identify any increase in movement compared to normal, which indicates damage to the facet joint.

The lumbo-sacral ligament is assessed for damage. Whilst this is not a common injury, it does occur and should always be included in the examination. Motion palpation of it is performed by standing behind the cow, when she is in sternal recumbency and placing a hand on each tuber coxae and rocking in a ventro-anterior direction. In a normal cow there should only be a small amount of movement at the lumbo-sacral joint. If this ligament is damaged there will be increased movement, which can be both seen and felt. Palpation of the ventral joint space of L6–S1 per rectum whilst rocking on the tuber coxae with the other hand can also help assess it. The space will open and close slightly if this joint has been damaged. In severe cases the movement is obvious but it is easily missed in the more subtle cases. This damage may be secondary damage from crawling or from inappropriate lifting with hip clamps.

The pelvic bones are examined whilst doing the rectal, to determine if they are fractured. If the cow has a ventrally dislocated hip the head of the femur may be felt lying under the wing of the ilium or perhaps in the obturator foramen.

The cow is then rolled into lateral recumbency and the hind limb is examined for signs of a dislocated hip, fractured leg, ruptured stifle or torn gastrocnemius muscle/Achilles tendon. These maybe primary, secondary or tertiary conditions. The major leg muscles, particularly the hamstring group, are palpated looking for signs of swelling and oedema. This may indicate compartment syndrome. Blood for muscle enzyme levels may be taken. Compartment syndrome is a secondary effect of recumbency.

The nerve supply to the hind limb is then closely examined: Sensation to the lower leg is assessed using either an 18G hypodermic needle or an electric cattle prodder. The pastern is stimulated at the anterior and caudal aspects. The response is graded into normal, depressed or absent. The cattle prodder is a cruder test, being a stronger stimulus but sometimes this is needed because the hypodermic needle won’t always give a pain response even though the cow has some feeling. Bear in mind that if there is no reaction and the leg is cold from lying in wet conditions it may be misleading. If in doubt, apply the same stimulus to the front legs to compare.

The deep pain response is assessed when the cow is in lateral recumbency by squeezing the inter-digital area with hoof testers. Be careful as to where you are standing, as the normal response may elicit a strong kick response.

A hypodermic needle is used to stimulate the skin of the medial thigh of the leg the cow is lying on to assess saphenous nerve sensory function.

Patellar reflex is assessed in lateral recumbency, using the hoof testers to strike the patellar ligament (Steinfort 1998). The leg needs to be held in a moderately flexed position to be accurate and it does require a little practice. A normal reaction doesn’t have a strong response. It is graded as poor, normal or exaggerated. Striking the patellar tendon causes the quadriceps muscles to contract via a spinal nerve arc. The hamstring group of muscles oppose this contraction so both the femoral and sciatic nerves are involved with the patellar reflex. Femoral nerve damage manifests with a depressed or absent reflex while sciatic nerve damage usually has an exaggerated one. The patellar reflex is a good diagnostic test, which is routinely used in small animal medicine and should always be used when examining recumbent cattle.

The peroneal and tibial nerve reflexes can also be tested. These are variable in cattle so are limited in their usefulness but may be of value in giving a more detailed assessment of the function of the branches of the sciatic nerve. They are used commonly in human medicine. The tendon of insertion of the long digital extensor muscle (extensor digitorum longus) is struck to induce the peroneal reflex. This lies in the middle of the anterior hock crease and the hock needs to be in extension to make good contact. Flexion of the hock occurs if the peroneal
nerve is functional. This is an active effect, which can be felt whilst holding the leg rather than a passive one from just striking the hock joint. The gastrocnemius tendon is used to test the tibial reflex. The hock needs to be flexed and the action will cause the hock to extend if the tibial nerve is functional. The triceps, bicep, cranial tibial and gastrocnemius reflexes in ruminants are unreliable and not recommended for clinical use (Constable 2004).

With the cow still in the lateral position attention is turned to the front leg and it is examined for damage to its bones, joints, ligaments and muscles. An 18G needle is used to stimulate the four quadrants of the pastern to assess its sensation. The triceps reflex can be tested using hoof testers to strike the triceps tendon with the elbow in a moderately flexed position. This is a difficult reflex to interpret.

The cow is then sat up into the sternal position and rolled onto her other side. The static palpation of the back is repeated looking for a change in the relative position of either the dorsal or lateral spinous processes, indicating a fractured back. She is then rolled into lateral recumbency and the second leg examined.

Motor function is assessed in both the recumbent position and ideally, by lifting the cow. The sitting position is noted and when the cow is stimulated with a cattle prodder the function of the legs is observed: Do they go laterally or caudally? How much strength does she have and is one leg stronger than the other? The cow is then lifted using a suitable lifting device, such as hip clamps. Stance is assessed and a particular note is made if there is a proprioceptive deficit, shown by the cow standing on the dorsum of the hoof. This usually won’t be apparent unless the cow is lifted. An assessment is made as to the degree of assistance that the lifting device is giving to the cow and this is factored in when assessing the severity of the condition.

Motor function of the front limbs is also noted here because a radial nerve/brachial plexus injury is a not uncommon secondary problem in a downer and can be an occasional primary condition. If the cow stands on her hind limbs but not on her forelimbs it is important to determine if this is because she ‘won’t’ or ‘can’t’. A chest strap is placed under the cow and she is re-lifted. A radial nerve/brachial plexus injury is now easy to diagnose because of the characteristic stance. They are difficult to diagnose when the cow is recumbent and this is another reason why the extra effort and time taken to lift the cow is worthwhile. It should be part of every standard recumbency examination.

**Diagnosis**

**Traumatic injuries**

The more obvious traumatic injuries, such as dislocated hips, fractured legs etc won’t be covered here as they are adequately covered elsewhere. However, it is worth noting that a ventral dislocated hip and ruptured Achilles tendon are not uncommon secondary or tertiary injuries to ‘downers’. By tertiary injuries, I mean a cow that has become recumbent from her primary cause and has developed secondary muscular or peripheral nerve damage. This secondary damage prevents her from successfully standing but in trying to do so, she falls and suffers extra damage. Such injuries are usually terminal. The prognosis for a cow with a secondary dislocated hip following from other primary damage is hopeless so I no longer try to replace them. Gross muscle tearing in the major muscle groups is often overlooked clinically and can often only be appreciated on post mortem (Cox 1981). This tertiary muscle damage will prevent many downers from recovering.

**Muscular injuries of the longissimus lumborum**

This damage will occasionally cause recumbency. It may have occurred from a primary injury, particularly a ‘bulling’ injury but it may also be secondary from crawling or struggling to stand. These cases will have full nerve function but are very painful when the affected muscles are palpated. They are unable to stand up as it is too painful but if they are lifted they can usually walk around normally for a while. They may deteriorate over a few days as they re-injure the area from continually struggling to rise, or they may improve spontaneously.

**Fractured back**

These can be easily missed unless a full examination is performed as described, above. If examined soon after the injury they have a surprising amount of motor function and may nearly be able to stand. They have full sensory nerve function although this will deteriorate over a few days. The patellar reflex would be expected to be exaggerated bilaterally but it usually isn’t and varies from case to case. The area most likely to fracture is the mid lumbar region. There will be rotation of the adjoining vertebral segments relative to each other. If the fracture is at L2-L3 the lateral process of L2 on one side will be higher than L3 on that side whereas it will be lower on the other side. When the cow is rolled onto her other side this relationship will be reversed. The dorsal spinous processes of L2 and L3 may be slightly deviated laterally to each other and this will change when the cow is
rolled from one side to the other. Motion palpation will reveal increased movement at this joint although the amount of movement is subtle and it does require practice to be competent at this test. These signs prove there is a rotation at the zygo-pophyseal joint thereby meaning that the joint is fractured. Euthanasia is the only option.

**Obturator nerve**

The obturator nerve arises from the fifth lumbar nerve with a smaller branch from the sixth lumbar. It courses along the roof of the pelvic canal under the ventral wing of the sacrum and down the medial aspect of the ilium, exiting via the obturator foramen. It innervates the muscles that provide the main adductor function to the hind limb but note, that the sciatic nerve also has some adductor function (Getty 1975).

If the cow is recumbent with the leg abducted laterally, hip dislocation, ruptured abductor muscles and obturator paralysis must be differentiated. Careful palpation of the hip is required to rule out a dislocation, as it will be a ventral one. Ruptured abductor muscles are usually associated with falling with the legs in the splayed position whereas obturator paralysis will have occurred with a dystocia, although in my experience, less than 5% of calving paralysis’ cases would involve this nerve. The obturator paralysis cow will have normal patellar reflexes and full sensation to the lower limb as the sciatic branches supply all of the sensation below the fetlock. Calving paralysis cases with wide-based stance, increased patellar reflex(es) and decreased sensation to the pastern will have both obturator and sciatic nerve damage. Cows recumbent from obturator paralysis are prone to a secondary dislocation of their hip and need to be monitored for this.

**Sciatic nerve**

The sciatic nerve originates from the L6, S1 and S2 nerve roots. It is closely aligned to the ventral surface of the sacrum before exiting the pelvic canal and coursing around the greater trochanter. At this level, it gives off several branches of various sizes (ramus muscularis and rami musculares), which radiate to serve the muscles of the hip and upper thigh. The cranial gluteal nerve, arising from L6 and S1 and the caudal gluteal nerve, arising from S1 and S2 also innervate these muscles. These nerves control the range of movement of the hip (Getty 1975). The sciatic nerve splits into the peroneal and tibial nerves at the level of the mid-thigh. The peroneal nerves down the anterior part of the lower limb providing sensation to this aspect of the leg and motor function to the muscles which flex the hock and extend the digits (Getty 1975). The tibial nerve courses down the posterior part of the lower limb providing its sensation and innervating the muscles which extend the hock and flex the digits (Getty 1975). There is a communicating branch between the dorsal axial and plantar axial nerves, which supplies the axial region of the digits III and IV (Hall 1971).

The sciatic nerve is the nerve commonly involved with calving paralysis, in my experience. It can also be damaged by falling or from a ‘bulling’ injury. It will range in symptoms from the cow walking with a mild ataxia through to recumbency with total paralysis of one or both legs. There will be a loss of sensation to the lower limb and an associated proprioceptive defect, which is evident when the cow is lifted. Some cases sit with the hind limb in extension forward, such that the hoof is near the head. This is due to loss of extension function of the hip. The patellar reflex is usually exaggerated with cases of sciatic nerve damage. This is due to the lack of dampening of the quadriceps contraction by the weakened hamstring group. If the sciatic damage is associated with dystocia the two legs are usually affected unevenly with one being more severe than the other one. This will show by different sensory function to the two pasterns, different degrees of patellar reflex abnormalities and different motor functions. The more affected leg is usually the leg that she was sitting on during the birth. During their nursing they will tend to sit on the more severely affected leg because as they try to move the stronger leg will push her over onto her weaker leg. This weaker leg then becomes prone to secondary damage.

I class distal sciatic nerve injuries into four classes: The first degree has loss of motor function but retaining full sensory function at the patern. They show a proprioceptive defect when lifted and a normal or exaggerated patellar reflex, which differentiates them from a femoral or obturator nerve injury. They have a good prognosis with an expected recovery time of 3 or 4 days. The second grade involves loss of sensation to the peroneal branch of the sciatic nerve, manifesting as decreased sensation on the anterior aspects of the pastern. Prognosis is fair with an expected recovery time of a week to 10 days. Grade 3 has a loss of sensation to the tibial nerve as well, so that there is no superficial sensation to the lower limb. The deep pain reflex will, however, still be present. Prognosis is guarded with a 2-4 week recovery. Grade 4 has a loss of the deep pain reflex as well as no sensation at the pastern. Prognosis is very poor with nursing of a month or more required.

Cows with Grade 4 calving paralysis must be differentiated from those with hypothermia and from a form of ‘nerve shock’ that can occur immediately after delivery. If the hind legs are cold and have no feeling, it is important to check the front legs also and if they are the same then a Grade 4 diagnosis cannot be made. ‘Nerve shock’ applies to some cows where the calf has been stuck violently, but only for a short time. When they are
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These expected recovery times also assume that they are nursed properly so that secondary myopathies and/or neuropathies don’t extend them. If this occurs, a Grade 1 sciatic nerve from damage in the pelvic canal from a dystocia may become a Grade 2 or 3, due to this secondary peripheral damage. This is more likely to occur in heavy animals, in animals lying on a hard surface or animals not swapping from side to side, either voluntarily or with assistance.

The grading system can be explained by damage to the nerve roots that form the peroneal branch occurring before the tibial nerve roots are affected. Whilst the L6 and S1 nerve roots supply both the peroneal and tibial nerves (Getty 1975), it is possible that the roots forming the peroneal nerve are more exposed than those forming the tibial nerve. This would mean that more prolonged trauma is needed to affect the deeper tibial nerve roots. This is consistent with my observations and is the basis of my grading system for sciatic nerve injuries. I have never seen a case of calving paralysis with sensation to the dorsal aspect of the pastern without it also being present on the plantar aspect. Conversely, I have never seen a calving paralysis case lacking plantar pastern sensation but still having dorsal pastern sensation. The communicating branch between the peroneal and tibial nerves only supplies the axial region of digits III and IV not the dorsal or plantar aspects of the pastern (Hall 1971) and doesn’t complicate this grading system.

This grading system is crude but fairly reliable when they are assessed on the first day of the calving paralysis. It can help estimate the recovery time and give guidance to the farmer as to whether it is worth starting treatment and nursing or if euthanasia should be elected. It is also very useful when re-examining a case seen previously to assess secondary damage. If the grade has deteriorated since the initial visit then secondary damage has occurred to the sciatic nerve or its branches, distal to the original injury. This finding helps lead a discussion with the farmer on adequate nursing care.

This secondary damage to the peripheral sciatic nerve was well demonstrated in an experiment where 16 cows were anaesthetised for a period of six, nine or 12 hours maintaining them in right sternal recumbency on a rubber mat (Cox and McGrath et al. 1982). The position was to mimic the typical milk fever position. Eight of the cows were able to stand within three hours of completion of anaesthesia but the other eight remained recumbent and became downers. The right pelvic limb of the downers was rigid and obviously swollen but this was also apparent in some of the ambulatory cows. Creatinine kinase enzymes were markedly elevated at 12 and 24 hours for all cows and there was no significant difference between the recumbent and the ambulatory groups. By day 2 and 4, the enzyme levels had decreased markedly in the ambulatory group and remained significantly higher in the downer group, as one would expect. However, muscle damage did not explain why some animals could stand at the end of the anaesthesia and others couldn’t. They found that the most marked and consistent gross change at necropsy of the downer cows was damage to the right sciatic nerve in the region caudal to the proximal end of the femur. Peroneal damage was also found in some of them at the level of the lateral proximal tibia. In contrast, normal clinical function of the right pelvis limb in the ambulatory cows indicated minimal sciatic nerve damage. The damage to the sciatic nerve could be due to direct pressure on the nerve from the surface they are lying on but it can also be due to pressure from the swollen muscle bellies that it is passing through.

This experiment (Cox and McGrath et al. 1982) shows how important secondary damage is in determining the outcome of a downer cow. In this case the cause of the primary recumbency, the anaesthetic, was completely resolved but within three hours of its completion some cows were unable to rise due to the secondary damage that had already occurred. Some cows had been recumbent for as little as six hours before succumbing to these secondary effects.

Femoral nerve

The femoral nerve arises from L5, with contributions from L4 and possibly L6. Its main motor function is to the quadriceps muscles, which act as strong extensors of the stifle joint. It also innervates the sartorius muscle, which flexes the hip and adducts the limb. It provides sensory supply to the medial thigh region via the saphenous branch (Getty 1975).

Femoral nerve injury as a primary condition is usually associated with a bulling injury or other trauma to the lumbar region of the back. It is not primarily associated with calving paralysis, as the nerve doesn’t pass through the pelvic canal. It is a common secondary complication of any recumbent cow, especially milk fever cases. It is caused by the cow struggling to rise and hyper-extending the lower lumbar region, which tears the femoral
nerve roots. Femoral nerve injuries are easy to diagnose if a full examination, as described, is performed but if it isn’t many cases of it will be missed. A cursory examination of a cow with a femoral nerve injury will reveal no abnormalities other than the recumbency. A more careful examination will reveal a depressed or absent patellar reflex and a tendency for the legs to slip out behind her when she tries to stand. She will have full sensory function to the distal limb as it is innervated by the sciatic nerve not the femoral nerve. If they are left in the paddock they tend to crawl around swapping from side to side with their legs in a frog-leg position. This hyper-extended back causes ongoing damage to the femoral nerve roots.

I use four grades to quantify femoral nerve injuries: Grade 1’s are recumbent but are able to stand and walk normally when lifted. Their patellar reflexes may be normal, depressed or absent. When they try to stand without assistance, their legs tend to go out behind them. Grade 2 cases are recumbent with depressed or absent patellar reflexes and a tendency for the legs to go out behind them when they try to stand. If they are lifted they will be able to stand independently and can often walk a few steps before they stumble and fall, often with their legs extending out behind. Grade 3’s are unable to stand when lifted and have no patellar reflexes. They are able to keep their legs under them in the normal sitting position at rest unlike the Grade 4, which sit in the full frog leg position and are unable to correct it.

Cattle must be nursed in a way to prevent them re-injuring their back as they crawl around or when they fall after being lifted. If not, a Grade 1 or Grade 2 can quickly deteriorate to a Grade 4, from which there is no recovery. In this scenario, a primary Grade 1 femoral nerve injury, which has a good prognosis, has suffered secondary damage to its femoral nerve and it is this secondary damage that becomes the limiting factor in its recovery. Whilst the damage is to the same nerve there is still the pattern of secondary damage on top of the primary damage.

Forelimb neuropathies

The nerves to the forelimb emanate from the brachial plexus, which is formed by the roots from the ventral branches of the last three cervical (C6–8) and first two thoracic nerves (T1–2). The brachial plexus gives rise to several nerves: suprascapular, subscapular, pectoral, musculocutaneous, axillary, radial, ulnar and median (Getty 1975).

The radial nerve innervates the triceps muscle and the extensors of the carpus and digital joints. When sectioned experimentally, an extensor paralysis was produced with flexion of the elbow, carpal and phalangeal joints (Vaughan 1964). At the walk, no weight was borne and the claws were dragged along the ground. There were areas of reduced sensation on the anterior aspect of the metatarsus and digits.

The median and ulnar nerves innervate the flexors of the carpus and the superficial and deep digital flexors. Sectioning of both nerves simultaneously produced little abnormality in the standing position but at the walk the leg was advanced stiffly with marked hyperextension of the carpal, fetlock and pastern joints. There was an absence of sensation on the posterior aspect of the limb below the elbow (Vaughan 1964).

Forelimb neuropathies may involve the entire brachial plexus or be confined to just the radial nerve. Damage to the brachial plexus can occur from prolonged compression between the scapula and ribs associated with lateral recumbency. This involves damage to all of the nerves of the plexus, leading to total paralysis of the forelimb. Radial nerve paralysis is usually a complication of recumbency with pressure causing trauma to the radial nerve as it passes over the humerus. Brachial plexus paralysis can be differentiated from radial nerve paralysis on the basis of the ability to bear weight (Parkinson and Vermunt et al. 2010). Whilst cattle appear unable to bear weight in both cases, those with radial nerve paralysis can bear weight on the limb if it is manually placed directly under them whilst they are lifted, unlike those with brachial plexus paralysis that can’t. This is because the muscles of the shoulder are still functioning in a radial nerve case.

After sectioning the radial nerve in an experimental animal there were areas of reduced sensation on the anterior aspect of the metatarsus and digits (Vaughan 1964). However, it is my experience that cows still have sensation to the dorsal pastern in cases of radial nerve paralysis. Cases with brachial plexus also have sensation to the pastern, both dorsally and plantarly, so I don’t rely on an absence of this to make my diagnosis. It seems the nerves are damaged enough to affect motor function but not sensory function. The cows need to be lifted to enable a diagnosis damage to be made.

Non-responsive milk fever

Such cases are cows which have failed to stand after the metabolic disorder has been corrected and show no
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other obvious abnormalities. The typical case has a history of suffering from milk fever when first found by the farmer early in the morning, was treated with calcium but was still recumbent that afternoon, by which time the vet is called. She doesn’t show any signs of calcium deficiency and intravenous calcium fails to improve her. Further clinical examination fails to reveal any other abnormalities. Many of these cows have the early stage of compartment syndrome, which at this stage is probably similar to a person’s leg ‘going to sleep’ when sitting in a chair for too long. Effective physiotherapy is required (Poulton 2010) and if applied early the cow will be able to stand. If not, she will develop more serious secondary muscle and/or nerve damage and become a true downer, as shown by Cox’s experiment (Cox and McGrath et al. 1982). Non-responsive milk fever cases are differentiated from milk fever cases that have suffered secondary femoral nerve damage whilst trying to rise from the initial recumbency when a full musculo-skeletal examination is done, on the basis of patellar reflexes. However, they will appear the same if a less complete examination is done.

Compartment syndrome

The hamstring group of muscles (biceps femoris, semitendinosus and semimembranosus) are particularly prone to damage in a downer cow. This damage can occur within as little as 3–6 hours of lying in sternal recumbency, especially if the surface is hard and/or the animal is heavy (Cox 1982).

The compressed muscles swell as lymphatic fluid and venous blood becomes trapped within the fascial compartments of the muscle. These hydrostatic forces progressively reduce the arterial blood supply causing an ischaemic myonecrosis. This in turn leads to further swelling, which causes more ischaemic myonecrosis and a destructive cycle ensues. The semitendinosus has the thickest fascial boundaries and is most at risk of this damage. Conditions associated with hypotension, such as milk fever, potentiate the risk of this syndrome because the reduced blood pressure is less able to perfuse the swollen muscle areas. Cows that tend to lie on the same side are more prone to damage to their muscles of their lower limb. This occurs in conditions like calving paralysis, where there is more damage to the nerves of one leg than to the other. The stronger leg will push the cow onto the weaker leg each time she tries to rise. Bilaterally conditions, such as a back injury affecting the femoral nerve, tend to swap from side to side and thus have less risk of muscle damage. The weight of the animal, the surface they are resting on and whether they are rolled manually are other important factors.

Trauma from the animal trying to rise can also cause haemorrhage within the muscle bellies further contributing to the increased pressure (Cox 1981).

A range of blood tests was conducted on 433 periparturient recumbent cows submitted over two years by veterinary practitioners from which they had information on the cows’ outcomes (Clark and Henderson et al. 1987). Their paper evaluated the biochemical and haematological results as predictors of likely outcome.

Their preferred model used serum urea, aspartate amino transferase (AST) and days sampled, from days 0 to 7. They fitted probability of recovery contours using the log of these two enzymes with the curves representing 5, 10, 30, 50 and 70% recovery. The probability contours were adjusted according to the number of days sampled. Creatinine phosphokinase (CK) was not included, as it didn't significantly improve the model. However, if there was evidence of liver damage (elevation of glutamate dehydrogenase GDH) they used a CK – urea model. These probability curves are very interesting but they only tell part of the story as there may be other primary or secondary damage that will have a more significant impact on the outcome.

Predictions of non-recovery using a ‘critical’ line representing a less than 5% probability of recovery were also looked at (Clark and Henderson et al. 1987): For CK this threshold varied each day, as shown in table 1. Critical levels are highest initially (50 times upper limit of normal reference range) and reduce to 10 times normal levels at seven days recumbent. For AST, the critical level was 890U/l, representing 7.4 times the upper limit of normal reference range (25–120 U/l at 30 degrees C). This was constant over the first seven days of recumbency.

These critical levels to predict non-recovery are very useful as a strong case can be made to euthanase cows with levels above these critical values.
Musculo-skeletal examination and diagnosis of the downer cow

Proceedings of the Society of Dairy Cattle Veterinarians of the NZVA, 2012

<table>
<thead>
<tr>
<th>Days recumbent</th>
<th>Critical CK level</th>
<th>Times normal (0-327 U/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5</td>
<td>12,200</td>
<td>33</td>
</tr>
<tr>
<td>1</td>
<td>18,600</td>
<td>50</td>
</tr>
<tr>
<td>2</td>
<td>16,300</td>
<td>44</td>
</tr>
<tr>
<td>3</td>
<td>14,000</td>
<td>38</td>
</tr>
<tr>
<td>4</td>
<td>10,900</td>
<td>29</td>
</tr>
<tr>
<td>5</td>
<td>8,500</td>
<td>23</td>
</tr>
<tr>
<td>6</td>
<td>6,200</td>
<td>17</td>
</tr>
<tr>
<td>7</td>
<td>3,900</td>
<td>10</td>
</tr>
</tbody>
</table>

Table 1. Critical CK levels relative to days recumbent (Clark and Henderson et al. 1987)

Records

It is important to document the findings of the musculo-skeletal examination. The advantage of this is that if the cow is revisited at a later date it is easy to compare findings and to determine if there has been an improvement or a deterioration suggesting secondary damage. This may help explain why the cow has failed to recover from the initial recumbency and helps lead the discussion with the farmer on adequate nursing care, which is so important in the downer cow syndrome.

Conclusion

Management of recumbent cows is a complex issue: The treatment and care of them is labour intensive and expensive but unnecessary culling is an economic loss.

It is important that the clinician conducts a thorough and complete examination of them to fully understand the disease process or processes occurring. This needs to be thought of in terms of the primary cause of the recumbency, the secondary damage from the recumbency and possible tertiary complications, being either further gross damage to the musculo-skeletal system or organ system diseases, such as mastitis or renal problems.

Detailed examination techniques including static and motion palpation, sensory and motor function tests and blood enzyme analysis must be performed. Interpretation of these findings requires an understanding of the functional anatomy of the musculo-skeletal system and of the pathogenesis of the downer cow syndrome.

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