

Downer cow syndrome

Definition

Cows that exhibit difficulty rising within the hours following calving most commonly present with hypocalcaemia, although other metabolic or traumatic causes may also be involved. This condition is typically managed through intravenous calcium therapy, which generally allows the animal to regain the ability to stand within approximately 12 hours. When the cow fails to rise within this timeframe, it is generally considered to be developing **downer cow syndrome**.

This syndrome typically affects cows that are initially alert and in sternal recumbency during the periparturient period, yet remain unable to stand despite intravenous calcium administration. It occurs most frequently in the postpartum period. Its etiology is multifactorial, involving metabolic, traumatic, and infectious components. In this context, the etiopathogenesis, epidemiology, diagnosis, as well as the treatment and prevention of this condition will be successively addressed.

Etiopathogenesis

Most authors describe downer cow syndrome as progressing through three distinct stages: primary recumbency, secondary recumbency, and tertiary (or terminal) recumbency. Primary recumbency refers to the initial cause of the inability to stand, the etiologies of which will be detailed in the following section. Secondary recumbency represents a complication arising from the initial condition, typically associated with prolonged recumbency and its consequences. Finally, tertiary recumbency corresponds to the terminal stage, representing the final and invariably fatal outcome resulting from the progression of the first two stages.

A) Etiology of Primary Recumbency

Numerous causes of primary recumbency have been described in adult cattle. However, in periparturient dairy cows, the main etiologies are metabolic, infectious, and traumatic in origin. These causes may act individually or in combination to induce recumbency.

1) Metabolic Origin

Metabolic disorders responsible for downer cow syndrome are numerous. Although they have been described in detail in a previous section, they will be briefly reviewed here. It is important to emphasize that metabolic downer cow syndrome is most often multifactorial, with several metabolic imbalances coexisting and contributing to varying degrees in each case.

a) Hypocalcemia (Milk Fever)

Hypocalcemia is a major cause of postpartum paresis and is characterized by a decrease in blood calcium concentration leading to muscular weakness. Approximately 75% of cows recumbent at calving are hypocalcemic. Calcium plays a critical role in neuronal excitability, nerve impulse transmission, muscle contraction, and membrane stability. Even minor fluctuations in calcemia can alter sodium, potassium, and chloride exchanges, thereby modifying cellular membrane potentials. This leads to a reduced threshold between membrane potential and excitability, resulting in abnormal neuromuscular activity and the development of stage II milk fever, characterized by postpartum paresis.

b) Hypomagnesemia / Hypermagnesemia

Magnesium plays a key role in modulating the severity of hypocalcemia by maintaining electrolyte balance. Hypermagnesemia may exacerbate paralysis by reducing acetylcholine release at the neuromuscular junction. Conversely, hypomagnesemia is frequently observed during late gestation and reduces calcium mobilization by impairing vitamin D activation and decreasing both the synthesis and effectiveness of parathyroid hormone. Consequently, cows with subclinical hypomagnesemia at calving are at a significantly higher risk of developing milk fever and postpartum paresis.

c) Hypophosphatemia

In cases of milk fever, animals with hypophosphatemia rarely regain the ability to stand, even after normalization of blood calcium levels. Hypophosphatemia contributes to prolonged recumbency associated with primary hypocalcemia and is most commonly observed in older, high-producing cows housed under confinement.

d) Hypokalemia

As previously mentioned, hypocalcemia at calving alters transmembrane ion exchange, leading to a shift in intracellular potassium toward extracellular compartments. Potassium is essential for enzymatic activity and muscle contraction; therefore, intracellular potassium depletion results in decreased neuromuscular excitability, leading to flaccid paralysis and recumbency. However, establishing a direct correlation between blood potassium levels and intracellular muscle potassium remains challenging, making the diagnosis of hypokalemia difficult.

e) Fat Cow Syndrome and Ketosis

Fat cow syndrome occurs in cows that have not undergone adequate dietary restriction during the dry period. In the days following calving, an imbalance between energy requirements and intake leads to metabolic disturbances. Postpartum paresis associated with fat cow syndrome may result from hepatic lipidosis and ketosis. These conditions can induce a rapid systemic collapse, leading to recumbency that affects both forelimbs and hindlimbs. Studies have shown that approximately 70% of cows developing downer cow syndrome are also affected by fat cow syndrome.

f) Acquired Myodegeneration in Parturient Cows

This condition manifests exclusively at calving and is primarily caused by deficiencies in vitamin E and selenium. Early clinical signs include stiffness of gait, ataxia, followed by recumbency.

g) Malnutrition

This form of paresis is due to generalized weakness resulting from prolonged nutritional deficiencies. Affected animals have been fed diets deficient in protein and energy over an extended period, leading to significant metabolic imbalances. Malnutrition may be further aggravated by parasitism, which causes intestinal malabsorption.

2) Traumatic Origin of Primary Recumbency

Lesions responsible for primary recumbency can be classified into five categories: **neurological, articular, osseous, vascular, and muscular.**

a) Neurological Lesions

Neurological injuries may result from improper obstetrical manipulation or from compression injuries during prolonged or difficult calving. Paralysis arises either from lesions of the **lumbosacral plexus** or from damage to **peripheral nerves**. The most commonly affected nerves include the obturator, sciatic, femoral, common fibular (peroneal), and tibial nerves.

Obturator nerve paralysis may occur when the calf's elbows become lodged against the pelvic inlet during parturition. This results in persistent abduction of the hindlimbs, making it difficult for the cow to stand, particularly on slippery surfaces.

The sciatic nerve, which divides into the fibular and tibial nerves, may be injured at multiple levels. Proximal (pelvic) lesions lead to complete hindlimb paralysis, characterized by extension of the stifle and hock joints and flexion of the distal joints. In such cases, attempts to rise are impossible, and when supported, the animal bears weight on the fetlocks due to distal joint flexion.

Combined lesions of the obturator and proximal sciatic nerves result in a characteristic "frog-like" posture in recumbent cows, with the hindlimbs extended cranially, making rising impossible.

The common fibular nerve may be compressed at the level of the stifle, resulting in flexion of the fetlocks and extension of the stifle, similar to sciatic nerve paralysis.

Tibial nerve paralysis leads to hyperextension of the fetlock, partial flexion of the hock, and loss of cutaneous sensation in the distal regions of the limb.

Femoral nerve paralysis, often following excessive limb abduction, results in atony of the quadriceps femoris muscle. The affected limb assumes a semi-flexed position and cannot support the animal's weight. This condition is less frequent than the previously described nerve injuries.

b) Articular Lesions

Articular lesions occur during difficult calving or traumatic falls. Three types of luxations are commonly associated with downer cow syndrome:

- **Sacroiliac luxation**

Occurs in the periparturient period due to relaxation of the sacroiliac ligaments. The hindquarters remain close to the ground. Although standing may become possible a few days postpartum, the stance is often unstable, with persistent flexion of the hindlimbs.

- **Pubic symphysis rupture**

Frequently observed in two-year-old heifers at their first calving. Affected animals remain in recumbency with the hindlimbs abducted and resting on their medial surfaces.

- **Coxofemoral luxation**

Occurs following falls during calving or attempts to rise. Due to the shallow acetabulum in bovines, this joint is predisposed to dislocation. The luxation is usually unilateral, with the affected limb positioned perpendicular to the body and the plantar surface directed caudally. During attempts to rise, the limb shows exaggerated abduction.

c) Osseous Lesions

Trauma during parturition may be severe enough to induce fractures. The most commonly observed fractures include:

- **Vertebral fractures**

Often resulting from falls during calving, leading to nerve compression and inability to stand or rise.

- **Iliac neck fractures**

Caused by excessive traction during parturition, particularly when the calf's limbs become lodged in the pelvic inlet. These fractures result in collapse of the hip angle.

- **Sacral and femoral head fractures**

These may occur secondary to obturator nerve paralysis.

- Sacral fractures (L5–L6 region) compress adjacent nerves, leading to rapid onset paralysis.
- Femoral head fractures present clinical signs similar to coxofemoral luxation.

d) Vascular lesions

Vascular lesions responsible for the recumbent cow syndrome are of two main types: **phlebitis** and **hemorrhage**.

Phlebitis of the **femoral vein** or the **utero-ovarian vein** may induce severe systemic clinical signs, including **hyperthermia, shock, and tremors**. In such cases, paralysis and recumbency generally occur later relative to parturition.

Dystocic calving may also result in **internal hemorrhages**, which may be either **abdominal or perivaginal**. These hemorrhages may be caused by tearing or rupture of arteries such as the **uterine, internal iliac, or vaginal arteries**. They may be **acute and rapidly fatal** or may lead to the formation of **hematomas**.

e) Muscular lesions

Muscular lesions are most commonly associated with **falls** or **forceful attempts to stand**. The clinical signs resemble those observed in nerve injuries affecting the same muscles.

Lesions of the **adductor muscles** result in a characteristic posture, with the hind limbs positioned anteriorly in a “**V**” **configuration**. Animals affected in this way retain **cutaneous sensitivity**.

Similarly, rupture of the **gastrocnemius muscle** or the **Achilles tendon (tendon of the hock)** leads to a posture comparable to that observed in **fibular or tibial nerve paralysis**.

f) Compression or rupture of an intestinal loop

This lesion may occur when an **intestinal loop becomes compressed between the fetus and the pelvis** during parturition. This is a severe condition that frequently results in **euthanasia**.

3) Infectious origin

Severe infectious diseases such as **mastitis** and **metritis** may induce **septic shock**, which can lead to the development of recumbency.

The most common mastitis-associated causes are **coliform mastitis**, caused by bacteria such as *Escherichia coli*, *Enterobacter aerogenes*, and *Klebsiella pneumoniae*. These pathogens produce toxins that induce **hyperthermia, hypocalcemia**, and reduce intestinal calcium absorption. The resulting hypocalcemia contributes to recumbency.

Gangrenous mastitis is another cause of recumbency. It is a toxico-infectious condition caused by **staphylococci** or *Clostridium* species. The infection may rapidly lead to **mammary gland necrosis**, causing intense pain and shock, both of which contribute to recumbency.

Puerperal metritis and septicemia may also induce recumbency in cows, although they are relatively uncommon. The causative agents include **coliforms, staphylococci, and streptococci**.

Finally, **parturition-related laminitis** may occur in heavy, obese animals with significant infectious burden. The associated **severe limb pain** makes movement extremely difficult or impossible.

Table 1: Summary of the possible etiologies of recumbent cow syndrome, classified by period of occurrence and frequency of appearance (1 = most frequent)

Onset period	Before calving	Within 4 days after calving	Beyond 4 days after calving
Infectious causes	Abortions (3), septic gestational metritis (4)	Septic metritis with or without retention (2), septic mastitis (3), abortion (5), retained placenta / RPT (7)	Septic metritis with or without retention (1), septic mastitis (2), suppurative arthritis (7), severe advanced pyelonephritis (12)
Metabolic causes	Hypocalcemia (1), grass tetany (5), ketosis (6)	Hypocalcemia (1), grass tetany (8), ketosis-steatosis (9), acidosis (11)	Debility (4), ketosis (8), hypocalcemia (9), hypomagnesemia (10),

			postpartum hemoglobinuria (11)
Traumatic causes	Proximal dislocation (2), severe uterine torsion (7)	Post-calving paralysis (4), dislocation or fracture (6), traumatic gastritis with diffuse peritonitis (7), uterine rupture (10), intestinal crushing (12)	Traumatic gastritis with diffuse peritonitis (3), calving paralysis (5), fracture or dislocation (6)

B) Pathogenesis of Secondary Recumbency

Secondary recumbency results from the **progressive development of irreversible lesions** in a cow that remains in lateral or sternal recumbency beyond a critical time window. These lesions are not directly caused by the initial metabolic or infectious disorder, but by the **consequences of prolonged immobilization**.

Two major factors determine the transition from primary to secondary recumbency:

- **The duration of recumbency prior to treatment (time factor)**
- **The mechanical pressure exerted on muscles, nerves, and vascular structures (pressure factor)**

These factors act synergistically and determine the prognosis.

1) Pathophysiological role of recumbency duration

The **duration of recumbency** is the central determinant of secondary complications.

Experimental and clinical observations demonstrate a **direct relationship between time spent recumbent and the probability of recovery**. When appropriate treatment (e.g., calcium therapy) is administered **within the first 6 hours**, recovery is generally complete, as structural damage has not yet occurred.

Beyond this period, particularly after **6–9 hours**, **compression-related lesions** begin to develop due to sustained pressure over anatomical prominences.

These lesions include:

- **Muscle ischemia** due to vascular compression → reduced oxygen supply → cellular necrosis
- **Nerve compression** (especially of the obturator, sciatic, and femoral nerves) → impaired motor function
- **Vascular stasis** → reduced perfusion → tissue hypoxia
- **Pressure-induced myopathy** → degeneration of muscle fibers

Once these lesions are established, even after correction of the initial cause (e.g., hypocalcemia), the animal remains unable to stand, leading to **secondary recumbency**.

2) Role of mechanical pressure and tissue damage

Prolonged recumbency exposes specific anatomical regions (muscles, nerves, and soft tissues) to **continuous mechanical pressure**.

This results in a cascade of pathological events:

- **Sustained compression of muscle tissue** → impaired perfusion → ischemia
- **Ischemia** → **necrosis of muscle fibers** → loss of contractile function
- **Compression of peripheral nerves** → conduction block → motor paralysis
- **Capillary collapse** → worsening of local hypoxia

Clinically, these lesions are often **irreversible**, particularly when the duration of recumbency exceeds critical thresholds.

3) Importance of calcium therapy and prognosis

Although hypocalcemia is a common primary cause of recumbency, it is not the limiting factor in recovery once treatment is initiated.

Importantly:

No significant difference in blood calcium levels has been observed between cows that recover and those that remain recumbent.

This demonstrates that:

- The **primary metabolic disorder may be corrected**
- However, **functional recovery depends on the absence of pressure-induced lesions**

Thus, the **time spent recumbent is the key prognostic factor**, not the severity of the initial hypocalcemia.

Clinical implication (this is what separates a good teacher from a great one)

- Early intervention (≤ 6 hours) → **high probability of full recovery**
- Delayed intervention (> 9 hours) → **risk of irreversible lesions** → **secondary recumbency**
- The presence of **compression lesions overrides metabolic correction**

2) Mechanisms underlying the development of decubital lesions

When an animal is in recumbency, the body regions in contact with the ground are subjected to **prolonged and significant pressure**, which directly compromises the integrity of the compressed tissues. This leads to **pressure-induced ischemia** affecting the limbs located beneath the animal.

This ischemia is responsible for two types of clinical manifestations: **local signs** and **systemic signs**.

Local manifestations

Local signs are primarily grouped under **compartment syndrome**. Once established, this syndrome becomes self-perpetuating and leads initially to **muscle damage**, followed by **nerve damage** at a later stage.

The rate of onset and severity of this syndrome depend on:

- the magnitude of the applied pressure
- the elasticity of the fascia
- the degree of muscle encapsulation by the fascia

For example, the **semitendinosus muscle** has a thick fascial envelope and is therefore particularly vulnerable during prolonged recumbency. Other muscles that may be affected include the **adductor muscles**, the **gastrocnemius muscle**, and associated ligaments such as the **ligamentum teres femoris**.

The extent of tissue damage also depends on the animal's position. Lesions are more severe when soft tissues are compressed between a hard surface and underlying bone prominences. These bony prominences are also common sites for **nerve compression**, such as the **common fibular nerve**, which courses superficially around the fibular head.

The most frequently affected regions are the **hind limbs**, as they bear the body's weight during recumbency. In addition, **pressure sores (decubital ulcers)** are commonly observed over the **sternal region**.

At the skin level, prolonged compression may lead to **ischemic necrosis**, followed by **secondary bacterial infection**, resulting in ulcerative lesions (escarres) at pressure points.

Systemic manifestations

Systemic signs are grouped under the **crush syndrome** (rhabdomyolysis-related syndrome).

This syndrome results from the **release of intracellular components** from damaged muscle cells into the bloodstream, leading to:

- **Renal damage**
- **Hyperkalemia**, which may induce **cardiac arrhythmias**
- **Increased serum creatine kinase (CK)**, reflecting muscle damage

II) Epidemiology of Recumbent Cow Syndrome (Revised and Strengthened)

Recumbent cow syndrome affects approximately **10% of multiparous cows**. It is associated with **substantial economic losses**, as it primarily affects **high-producing dairy cows with high genetic value**, in which the financial impact of mortality or culling is considerable. Furthermore, the prognosis remains **guarded**.

Only about **33% of affected cows recover**, whereas the remaining animals are removed from production, either by **euthanasia (approximately 4%)** or by **culling at the farmer's discretion (approximately 23%)**.

Risk factors

The occurrence of recumbent cow syndrome is influenced by multiple **host-, environment-, and disease-related factors**, summarized below.

1) Host-related factors

Factor	Predisposition and mechanism
Age	Higher incidence in multiparous and older cows , due to decreased metabolic adaptability and increased susceptibility to metabolic disorders (especially hypocalcemia).
Breed	Holstein > Guernsey > Brown Swiss > Jersey . High-producing breeds, particularly Holsteins, have greater metabolic demands, predisposing them to calcium and energy imbalances.

2) Physiological factors

Factor	Predisposition and mechanism
Postpartum period	Most cases occur within the first week after calving , a period characterized by abrupt metabolic changes (onset of lactation, negative energy balance, calcium mobilization), increasing the risk of metabolic and neuromuscular disorders.

3) Environmental factors

Factor	Predisposition and mechanism
Season	Higher incidence during winter months (December–February) , likely due to reduced exercise, housing conditions, and dietary changes affecting mineral balance.
Housing conditions	Increased risk in tie-stall systems , where restricted movement promotes prolonged recumbency and increases the risk of compression lesions.

4) Disease-related factors

Factor	Predisposition and mechanism
Metabolic and peripartum disorders	Conditions such as hypocalcemia significantly impair neuromuscular function and predispose to recumbency.
Obstetrical complications	Events such as stillbirth are associated with difficult calving, increasing the risk of trauma and prolonged recumbency.

Diagnostic Approach to Downer Cow Syndrome

Step 1: Confirm the definition

- Cow in **sternal recumbency**
- **Alert**
- Unable to stand > **12 hours after IV calcium therapy**

If NOT → you are NOT dealing with true downer cow syndrome
If YES → you should proceed

Step 2: Rapid triage (first 2–3 minutes)

Ask yourself immediately:

Is this systemic / life-threatening?

- Hyperthermia / hypothermia
- Shock signs
- Severe mastitis or metritis
- Toxic appearance

If YES → **infectious cause likely** → **emergency management**

Step 3: Time factor (CRITICAL)

- < 6 hours → prognosis favorable
- 6–12 hours → guarded
- 12 hours → high risk of **secondary recumbency lesions**

This determines urgency, not diagnosis

Step 4: Orientation via anamnesis

Key discriminators:

Finding	Likely orientation
Dystocia / traction	Traumatic
No calving issue + high production	Metabolic
Fever + foul discharge	Infectious
Sudden collapse	Metabolic / vascular

You are not diagnosing yet — you are **ranking probabilities**

Step 5: Clinical differentiation

A. Metabolic

- Alert, normal mentation
- No major asymmetry
- Possible tremors, weakness
- History of hypocalcemia

Think: hypocalcemia, hypophosphatemia, ketosis

B. Traumatic / Neurological

- Asymmetry

- Abnormal limb position
- Inability to bear weight even with assistance
- Reduced or absent sensitivity

Think: nerve damage, fracture, luxation

C. Infectious / Toxic

- Fever
- Depression
- Tachycardia
- Congested mucosa

Think: mastitis, metritis, septicemia

Step 6: Can the cow stand with assistance?

YES → better prognosis

NO → suspect:

- severe nerve damage
- fractures 7, 9 groupe 8
- advanced secondary lesions

Step 7: Evaluate secondary recumbency

Signs:

- Muscle swelling / hardness
- Pressure sores
- Pain on manipulation

If present → **compartment syndrome + crush syndrome** → **prognosis worsens sharply**

Step 8: Prognostic decision

Situation	Prognosis
Early + metabolic	Good
Delayed but reversible cause	Guarded
Traumatic severe	Poor
Secondary lesions advanced	Very poor

III) Diagnosis of Downer Cow Syndrome

The diagnostic approach to downer cow syndrome aims to identify the **cause of primary recumbency** and to assess the **severity of secondary recumbency lesions**. Given the wide range of possible etiologies, a **rigorous and methodical approach** is required.

The diagnostic process is based on two main steps: **anamnesis** and **clinical examination**.

A) Anamnesis

A thorough anamnesis is essential and often allows significant narrowing of differential diagnoses. It helps:

- orient toward **metabolic, traumatic, or infectious causes**
- evaluate the **duration and progression of recumbency**
- assess **prognosis and management feasibility**

Two stages must be distinguished: the **initial visit** and **follow-up evaluations**.

Table : Anamnesis during the first visit

Parameter	Key Questions
Duration of recumbency	< 12 h?
Evolution	Attempts to stand? Changes side spontaneously? Assisted repositioning?
Animal	Age? Economic value?
Production stage	Calving date? Periparturient?
Previous condition	Body condition? Previous similar episodes?
Medical history	Concurrent diseases?
Calving conditions	Dystocia? Forced extraction? Duration? Calf alive? Size?
Onset circumstances	Cow slipped? Trauma suspected?
Environment	Bedding type? Other animals affected?
Farmer factors	Time available? Motivation? Reliability?
Treatments already given	Calcium? Antibiotics? Anti-inflammatories?

Table : Anamnesis during follow-up visits

Parameter	Key Questions
Treatments administered	Calcium? Antibiotics? Anti-inflammatories?
Evolution	Attempts to stand? Appetite?
Management	Regular repositioning?
Clinical progression	Skin lesions? Deterioration/improvement?
Farmer compliance	Instructions followed?
Farmer factors	Time available? Motivation? Reliability?

B) Clinical Examination

Following anamnesis, a **complete clinical examination** must be performed. Its objectives are to:

- identify the **underlying etiology**
- detect **complications of recumbency**
- evaluate the **prognosis**

The examination must follow a **logical progression**, from general observation to targeted assessments.

1) General and distant examination

Initial evaluation should include:

- posture, head position, ear movements
- level of consciousness and responsiveness
- tail movements
- ability or attempts to stand

These elements help distinguish:

- **alert downer cows** (typical of downer cow syndrome)
- from **systemically depressed animals**

2) General clinical examination

A full systemic evaluation includes:

- rectal temperature
- heart and respiratory rates
- digestive motility
- hydration status (sunken eyes, skin elasticity)
- mucous membrane color
- feces and urine output
- signs of metabolic imbalance

3) Mammary and reproductive examination

This step aims to detect infectious causes:

- mastitis (milk examination)
- metritis (vaginal discharge, vaginoscopy if needed)

4) Orthopedic examination

Performed in two stages:

a) Examination in recumbency:

- limb position and angulation

- pain, swelling, heat
- suspicion of fractures or muscle injuries

b) Examination in standing position (if possible):

(using lifting devices such as a hip lifter)

- weight-bearing ability
- muscle tone
- limb abduction/adduction
- abnormal mobility or crepitation
- ability to move once standing

5) Neurological examination

- evaluation of cutaneous sensitivity
- detection of peripheral nerve deficits

Table : Summary of clinical examinations

Type of examination	Key elements
Distant exam	Posture, consciousness, behavior
General exam	Vital signs, hydration, digestion
Mammary/reproductive	Mastitis, metritis
Orthopedic	Limb position, fractures, muscle damage
Neurological	Sensitivity, nerve deficits

The information obtained from both the anamnesis and the clinical examination generally allows the elimination of numerous differential diagnoses and a significant narrowing of the diagnostic hypotheses.

However, the etiological diagnosis of downer cow syndrome remains complex due to the wide variety of potential causes and the possible coexistence of multiple conditions. A structured and methodical diagnostic approach is therefore essential to guide clinical reasoning and prioritize the most likely hypotheses.

B) Additional Examinations

A wide range of additional examinations can be used in the etiological diagnosis and prognostic evaluation of downer cow syndrome. However, these tests are **rarely performed as a first-line approach**, as they are often not readily available in field conditions.

When performed, they frequently allow:

- confirmation of the initial etiological hypothesis
- refinement of prognosis
- adaptation of therapeutic management

The main complementary examinations are summarized in the following Table

Table : Complementary examinations for diagnosis and prognosis in downer cow syndrome

Examination	Diagnostic criteria	Advantages / Limitations
Blood tests		
Calcium measurement	↓ Calcium → hypocalcemia	Useful for early diagnosis

Electrolytes (P, Mg, K)	Imbalances	Prognostic value within first 96 h
Urea / Creatinine	↑ → renal failure (pre-renal shock) or muscle damage	Interpretation may be difficult
Creatine kinase (CK)	>200 IU/L → muscle damage; >10,000 IU/L → very poor prognosis	Reflects severity of recumbency lesions
AST (ASAT)	↑ (45–110 IU/L) → hepatic or muscular damage	Non-specific
Milk analysis		
California Mastitis Test (CMT)	Positive → mastitis	Rapid, field test
Ketone bodies	Positive → ketosis / hepatic involvement	Easy to perform
Urine analysis		
Myoglobin / Hemoglobin	Present → muscle damage or hemolysis (e.g. babesiosis)	Rapid and easy
Ketone bodies / bile pigments	Present → hepatic disorder	Field applicable
Imaging		
Radiography	Fractures (proximal bones), spinal lesions	Limited to hospital setting
Ultrasonography	Muscle/tendon lesions, masses	Limited use in bovine practice
Other tests		
Cerebrospinal fluid (CSF) analysis	Spinal cord disorders	Rarely used
Abdominal / thoracic paracentesis	Excess fluid → peritonitis / pleuritis	Low specificity

Additional, more specific tests may be performed once an etiological diagnosis has been established. For example, in cases of mastitis-related recumbency, bacteriological analysis can be used to guide targeted antimicrobial therapy.

IV) Treatment and Prevention of Downer Cow Syndrome

A) Prognosis

Assessment of prognosis is a critical step in the management of downer cow syndrome, as it directly influences therapeutic decisions. However, establishing a reliable prognosis remains challenging due to the multifactorial nature of the condition and the influence of external factors such as farmer motivation and available resources.

Prognosis primarily depends on the underlying cause of primary recumbency. Conditions such as fractures, spinal cord injuries, and severe septic processes (e.g., acute mastitis or metritis with hypothermia) are associated with a poor prognosis, whereas metabolic disorders such as hypocalcemia, when treated early, generally carry a favorable outcome.

Muscle damage is a major determinant of prognosis. The presence of myonecrosis significantly reduces the likelihood of recovery. Measurement of creatine kinase (CK) activity can assist in evaluation: values exceeding 12,000 IU/L are generally associated with a poor prognosis, although this threshold remains debated.

In practice, prognosis relies on five main factors:

- Epidemiological data
- Clinical signs and behavior
- Evolution over time
- Biochemical parameters
- Farmer commitment

Approximately 50% of affected cows recover within four days, whereas recovery after seven days of recumbency is крайне rare.

Clinical indicators of poor prognosis include:

- Lateral recumbency
- Anorexia and reduced water intake
- Hyperesthesia (non-metabolic origin)
- Persistent prostration

Conversely, signs such as spontaneous attempts to rise or the ability to change position are associated with a more favorable outcome.

Monitoring disease progression is essential. Improvement in appetite, alertness, and posture suggests a favorable prognosis, whereas deterioration or prolonged recumbency (>4 days) indicates a poor outcome.

Biochemical monitoring is not required initially but may be useful in cases with stagnant evolution. Worsening renal parameters, persistent electrolyte imbalances, or increasing CK levels are unfavorable indicators.

Finally, farmer involvement is a decisive factor, as intensive nursing care is essential for recovery.

B) Treatment

Management of downer cow syndrome involves three complementary approaches:

- Supportive treatment
- Treatment of primary recumbency
- Prevention and management of secondary recumbency

1) Supportive Treatment

The primary objective is stabilization of the animal.

Shock must be addressed first, whether septic, hypovolemic, or traumatic. Treatment involves intravenous fluid therapy with electrolyte solutions, glucose supplementation, and, when necessary, cardiostimulant agents such as heptaminol or nikethamide.

Pain management is essential, as pain reduces both appetite and the ability to stand. Non-steroidal anti-inflammatory drugs (NSAIDs) are commonly used, although their administration carries a risk of abomasal ulceration.

Given the frequent occurrence of hepatic dysfunction, lipotropic agents (choline, methionine, cysteine, vitamins B12 and B1) are recommended to support liver function.

Metabolic stimulation may be achieved using insulin, vitamin E, selenium, or glucocorticoids. Glucocorticoids also contribute to reducing milk production, thereby decreasing metabolic demand. Similarly, intramammary air insufflation has been described as a method to reduce lactation.

In anorexic animals, ruminal activity should be stimulated using yeast and mineral supplementation.

2) Treatment of Primary Recumbency

Treatment depends on the underlying etiology.

- **Metabolic causes:** correction of electrolyte imbalances (calcium, phosphorus, magnesium)
- **Infectious causes:** antimicrobial therapy and management of septic shock
- **Traumatic causes:** supportive and anti-inflammatory treatment

In cases of severe nerve damage or compartment syndrome, dimethyl sulfoxide (DMSO) may be used due to its anti-inflammatory and anti-edematous properties.

When prognosis is extremely poor, euthanasia must be considered early to avoid unnecessary suffering and economic loss.

3) Management of Secondary Recumbency

Management focuses on preventing complications associated with prolonged recumbency.

Nursing care

Environmental management is essential:

- Spacious, clean, dry bedding
- Non-slip but soft flooring
- Easy access to feed and water

Animal management includes:

- Repositioning every 3–6 hours
- Limb massage and passive mobilization
- Regular milking to prevent mastitis

In cases of hypothermia, external warming should be provided.

Farmer involvement is crucial, as successful management requires intensive and continuous care.

Assistance to standing

Mechanical assistance should only be attempted in animals capable of supporting their weight.

Methods include:

- Hip lifters (Bagshawe clamp)
- Slings or harness systems
- Mobile lifting devices (e.g., Paralift)
- Water flotation therapy

Each method has advantages and risks, particularly regarding tissue damage and stress.

Animals should be monitored closely after lifting, and movement should occur in a secure non-slip environment.

If the animal fails to stand after approximately two weeks, euthanasia should be strongly considered.

C) Prevention

Preventive measures focus on reducing the incidence of primary recumbency and limiting progression to secondary recumbency.

Key strategies include:

- Balanced nutrition during the peripartum period
- Proper housing and calving conditions
- Early treatment of metabolic disorders
- Adequate obstetrical management

Prevention of secondary recumbency relies on early intervention, proper nursing care, and rapid correction of underlying disorders.

In herds with a high incidence, a comprehensive herd health audit is recommended.