





Feeding management in the prevention of production diseases

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Feeding Management in The Prevention of Production Diseases

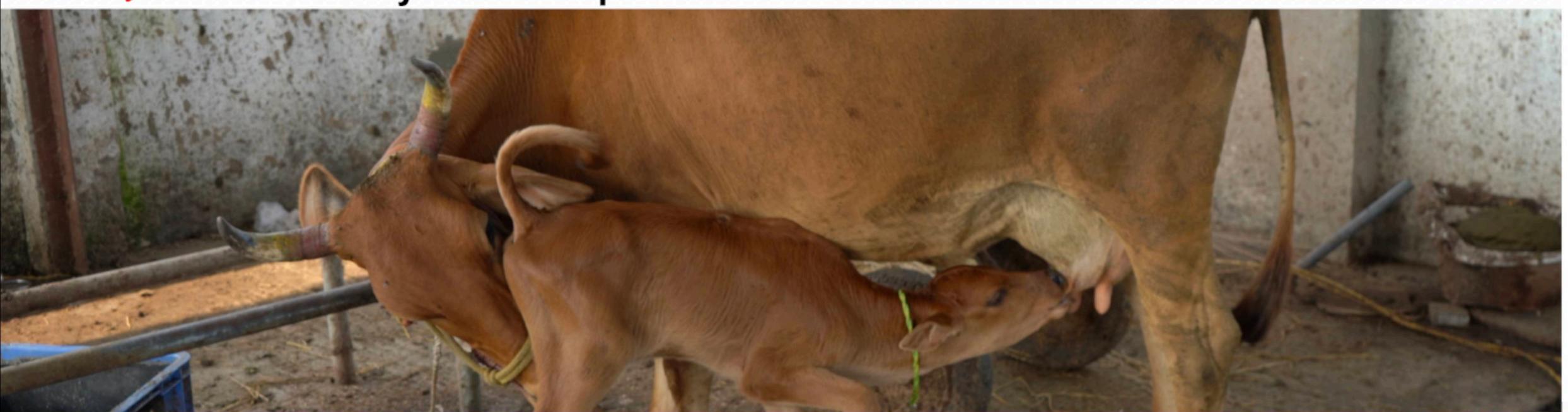


OBJECTIVE

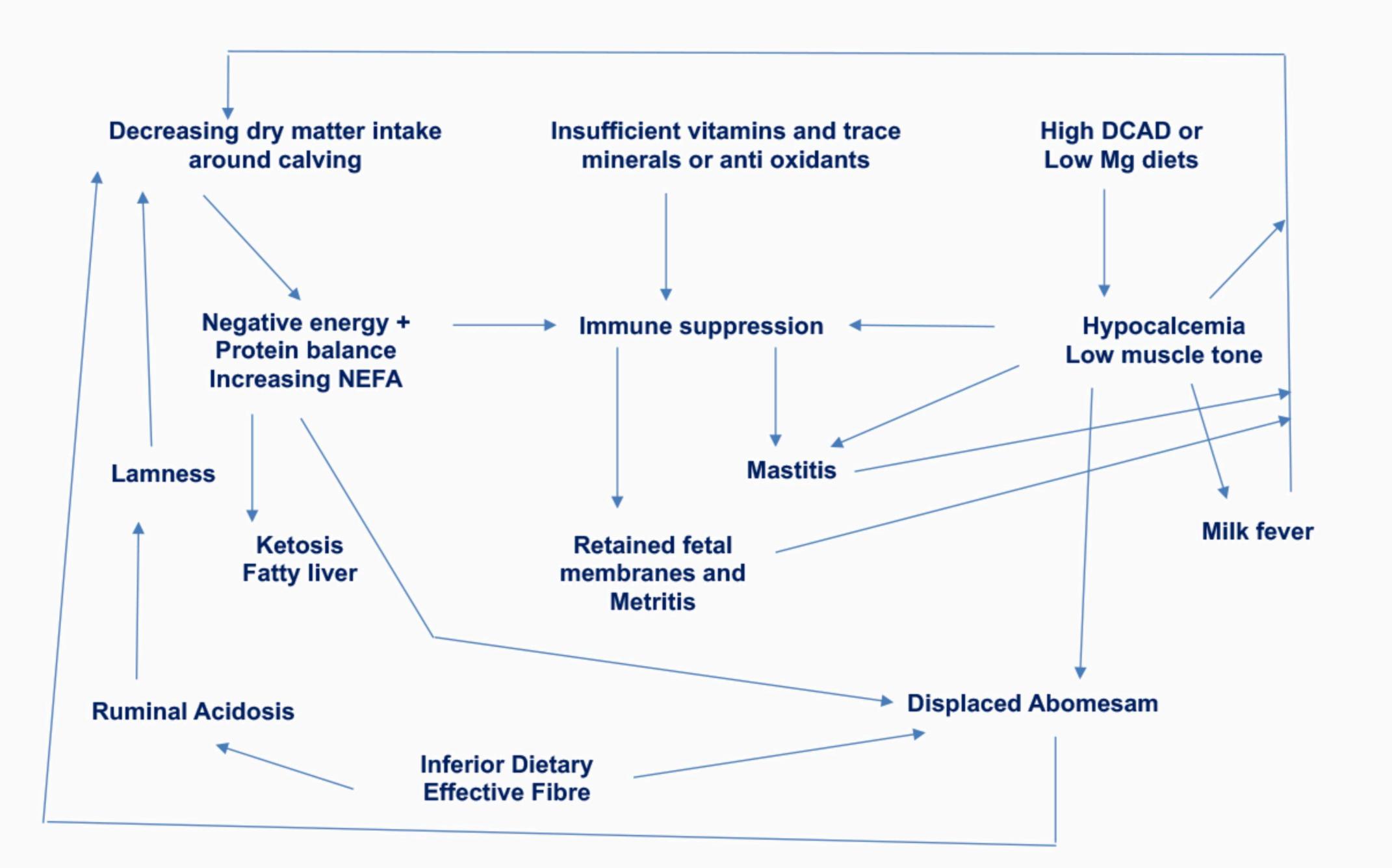
To impart the knowledge on feeding management in prevention of production diseases

Production Diseases

- Related to disturbance of one or more metabolic process in animals.
- Particularly during and after parturition
- Hormonal change
- Moving from non lactating to lactating animals
- Roughage to high concentrate diet
- Major drop in feed intake
- One in 2 dairy cows are prone for metabolic diseases



Production Diseases



Production Diseases

- Milk fever
- Ketosis
- Grass tetany
- Sub acute ruminal acidosis (SARA)
- **Laminitis**
- Fatty liver
- Downer cow syndrome
- Post parturient Haemoglobinurea

- Left displacement of abomesam (LDA)
- Bloat
- Retained fetal membrane (RFM)
- Liver abscesses
- Udder edema
- Mastitis
- Metritis



1.MILK FEVER (Hypocalcemia / Parturient paresis)

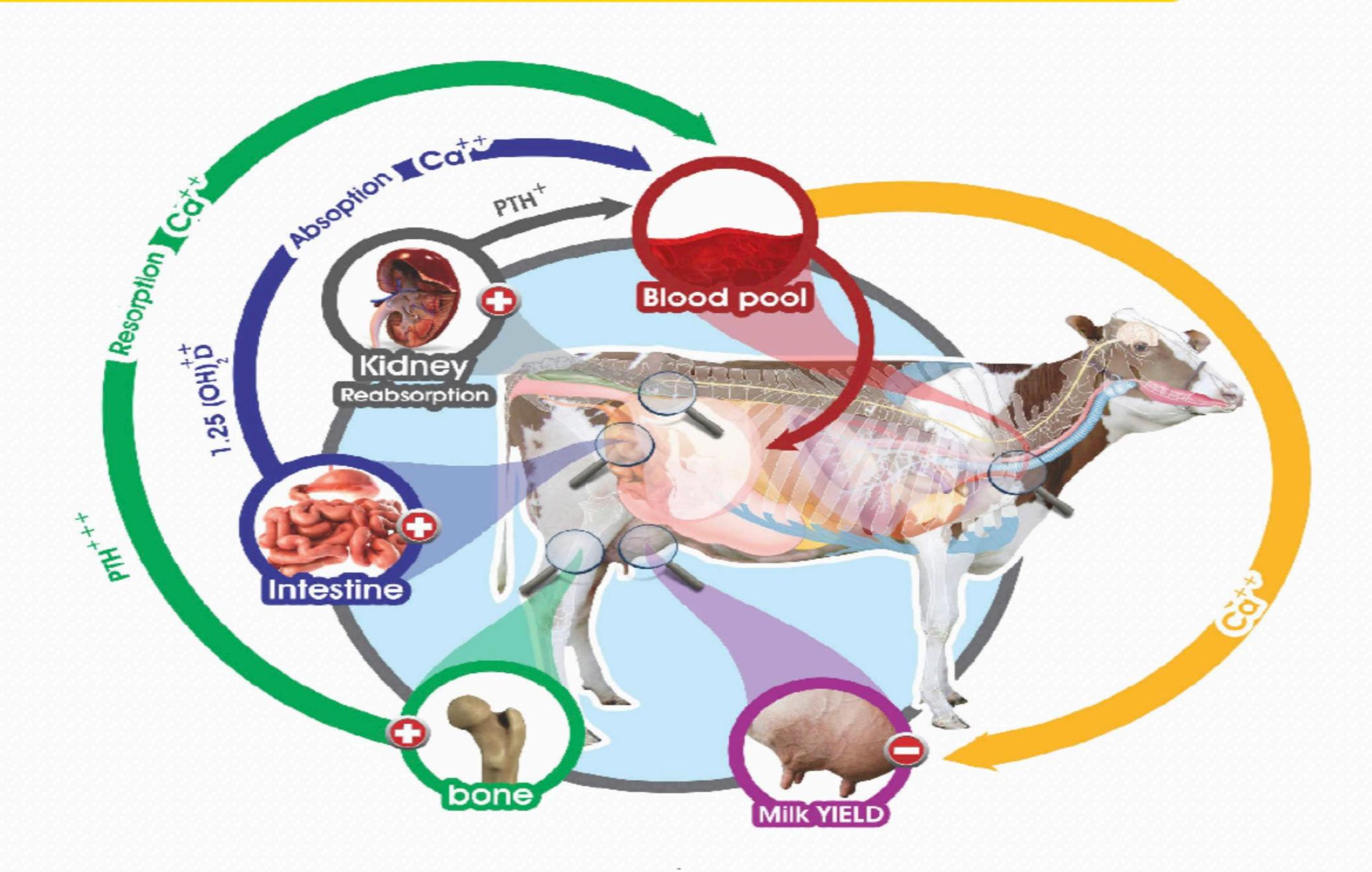
- Caused by a large calcium (Ca) demand, Occurs within 24 hrs of calving.
- Unable to meet the Ca demand at the onset of milk production
- Ca level in blood < 5 mg/dl
- Weakness, recumbency, shock, paralysis, circulatory collapse, coma and death
- 6% incidence rate in dairy cattle; Incidence increases with milk production and age
- Sub clinical hypocalcemia can affect 50% of dairy cows leads to:
 - Decreased dry matter intake
 - -- Ketosis
 - Retained placenta
 - RFM
 - Decreased reproductive efficiency
 - Decreased milk production

Causes

- Ca deficiency is due to severe outflow of Ca during initial milk production
- Calcium interacts with other minerals in the blood
 - Incidence may be influenced by levels of:
 - Mg, K, P and estrogen levels
 - Acid-base balance
 - Imbalances high risk for milk fever.
 - High alkaline environment hinders the mobilization of Ca from bone and absorption of Ca from intestine.
 - Inhibit parathyroid glands and renal synthesis of Vit D which restricts blood Ca levels
- Feeding high Ca diets prior to parturition
 - Cow doesn't adapt to mobilizing own Ca reserves



Calcium Homeostasis



Clinical Signs

- ► Stage I Standing
 - Hypertensive, weakness, anorexic, hypersensitive, shaking of head, protrusion of tongue and grinding of teeth
 - Prodromal stage- anorexia, agalactia, rumen stasis, scanty feces, normal temp, heart and respiratory rate



Clinical Signs

► Stage II – Sternal recumbency

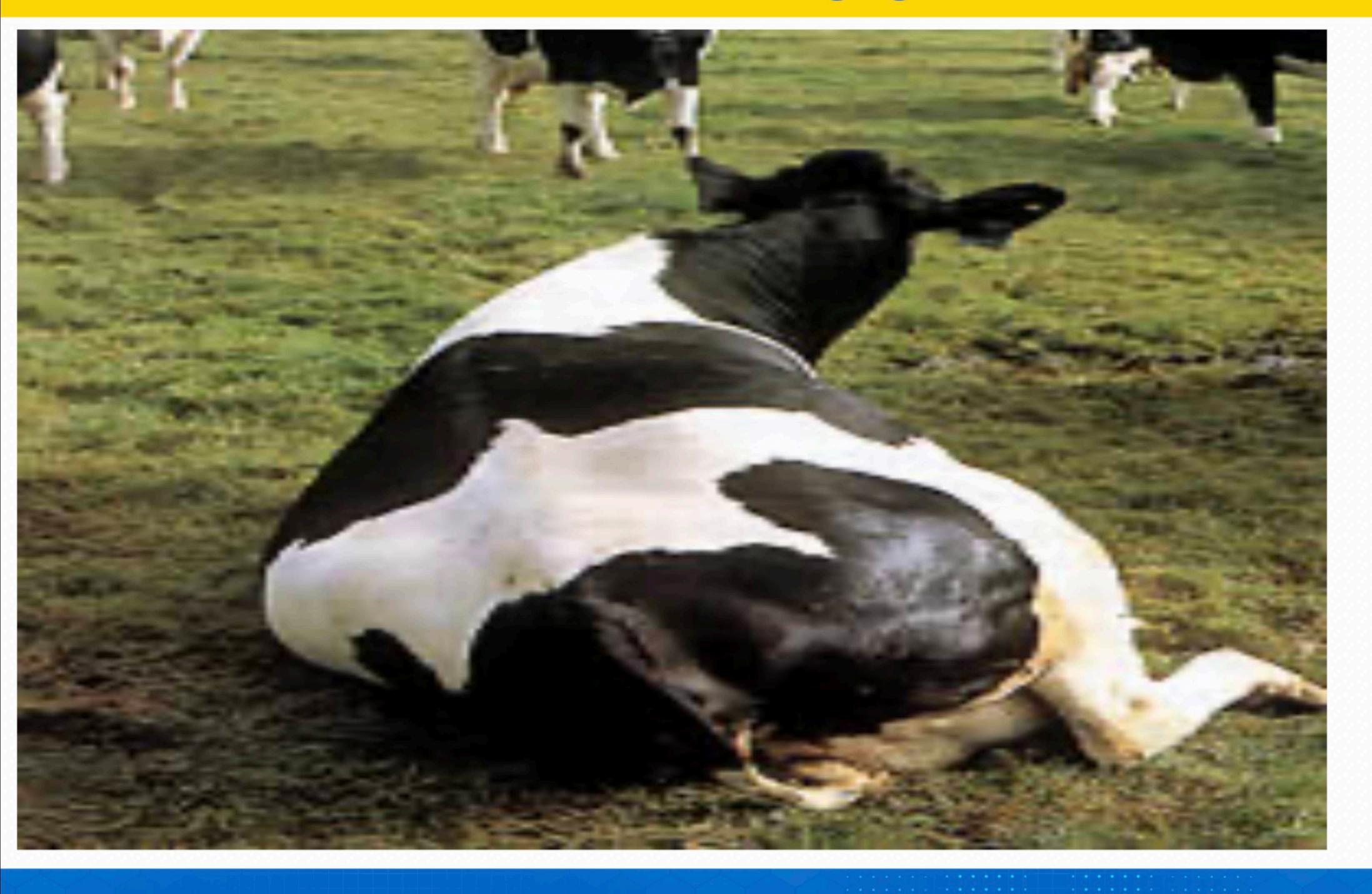
► Flaccid paralysis, lying on sternum, depression, small muscle trem ors, low body temp, cold extremities, muffled heart beat, bloat, dilated pupils, lateral kink of head, subnormal temp 36-38° C and Dry muzzle





Typical position of a milk fever cow.

Note the 'S' neck and the rectum bulging with feces under her tail.



Clinical Signs

- ► Stage III Lateral recumbency
 - Unresponsive, unconscious stage, lying on side with legs stretched out, bloat develops, comatose
 - ► Animals will die if untreated in this stage





Treatment

Stage I

Oral or Intra Venous (IV) calcium salts

- ▶ Oral gels can absorb into the blood in ~15 min
- Oral treatment allows for higher Ca dosage
- May help prevent relapse
- Stage II & III

Must treat with IV Calcium

- Administer slowly over period of 10 min
- May require subsequent treatments

Should respond with in 30 minutes of treatment and be standing





Prevention and Control

- Good nutritional management practices in the pre and post parturient period
- Maintenance of Ca : P ratio
- Oral Calcium drenching 50-60% efficacy
- ► Acidification of the diet
 - DCAD balance (Na+K)-(CI+S)
- Low Calcium ration during pregnancy (20g per day)
- Vitamin D and its analogue administration



2.Ketosis

- Most frequently happens in first 6-8 wks after calving; Very often affects first calf heifers or older cows
- ▶ 40 % herd affecting this disease 50 % cow under sub clinical to 20 % clinical
- Decreased blood glucose level to 25 mg/dl from 50-60 mg/dl; 60-85 % available glucose utilized for milk production
- Increase in free fatty acid (FFA) and triglycerides in plasma
- Decreased liver glycogen and increased liver lipid content (30 % Vs 7 % normal value)
- Deficiency of ACTH and cortisone causes ketosis



Acetonemia / Ketosis

Cause

- Occurs when intake doesn't meet requirements of the animal
- Excessive amounts of ketone bodies (acetone, aceto acetate and B-hydroxy butyrate (BHBA) found in urine, blood and milk
- Butyrate to BHBA in rumen epithelium
- ▶ Blood ketone: 10 mg to 50 mg/dl and low blood sugar (25 mg/100ml)
- BHBA: Aceto acetate ratio Normal : 1:10.5 ; Ketosis: 1:3.6 to 1:4.3
- Animal mobilizes its own fat reserves
- Mobilized fat transported to liver and oxidized to Acetyl CoA
- Acetyl CoA oxidized to Aceto acetyl CoA via TCA cycle. It depends on adequate supply of oxalo acetate from the precursor propionate.
- Deficient of oxalo acetate limits Acetyl CoA oxidation and converted to Aceto acetate and BHBA
- Deficiency of ACTH and oxaloacetate
- Shortage of glucose precursors propionate AA and lactate

Can use a Keto Stick to test urine for ketone bodies