

# African horse sickness



# History

*"If the animal is rode during the Sickness, or urged by driving or otherwise to any degree of speed, he falls at once, literally suffocated by the quantity of frothy matter which fills his trachea and issues in abundance from his nostrils".*

Thomas Perry: District surgeon at Graaff-Reinet 1819

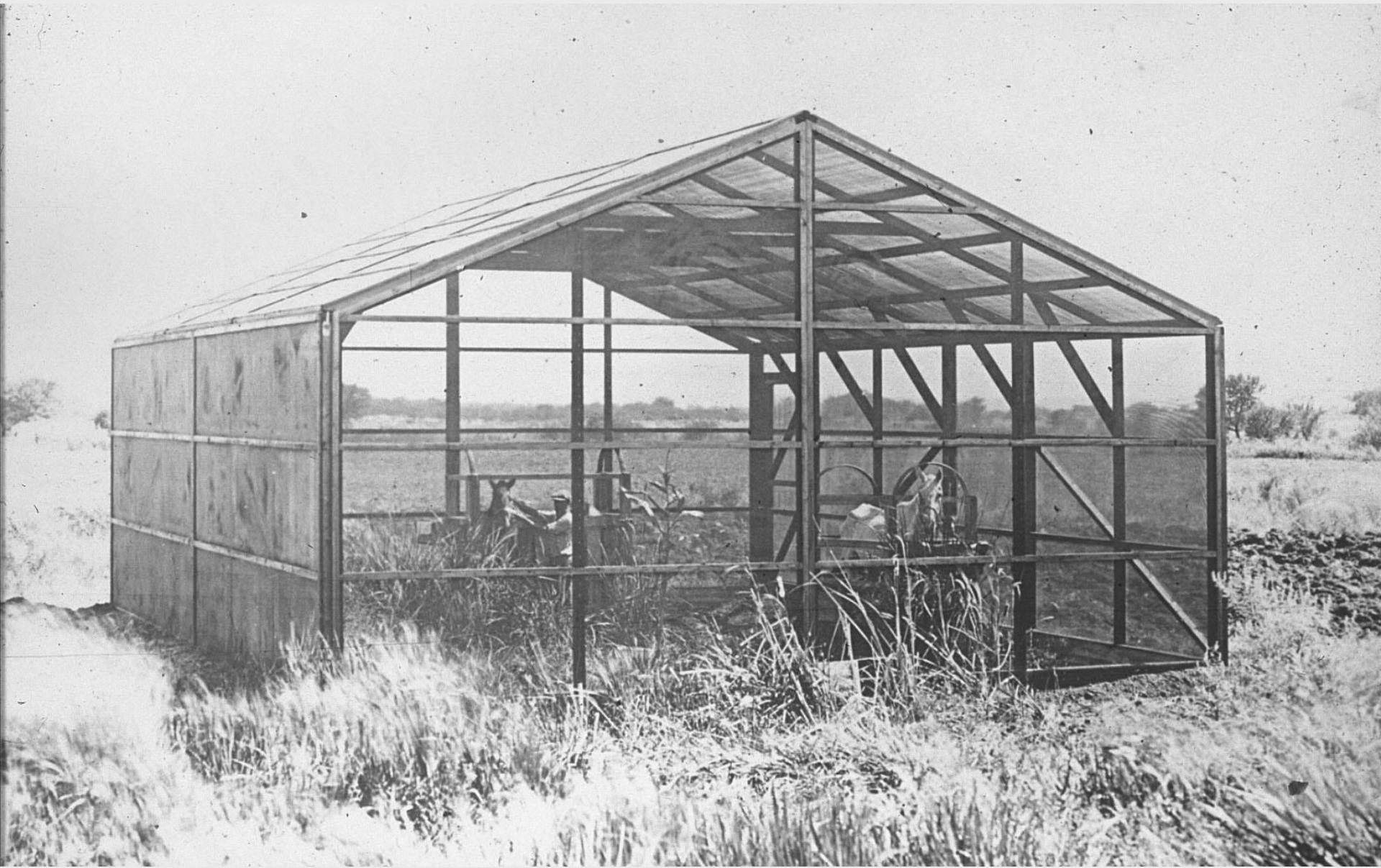
*"He appeared to be in perfect health last night when tied to the wagon wheel to secure him from Hyenas which are numerous here. His death took place about an hour after the symptoms of "The Sickness" were first noticed."*

James Backhouse, Missionary, 1838

T.B. Bayley, 1856 reported that the disease is not contagious, can be prevented by stabling, and that low lying areas should be avoided.

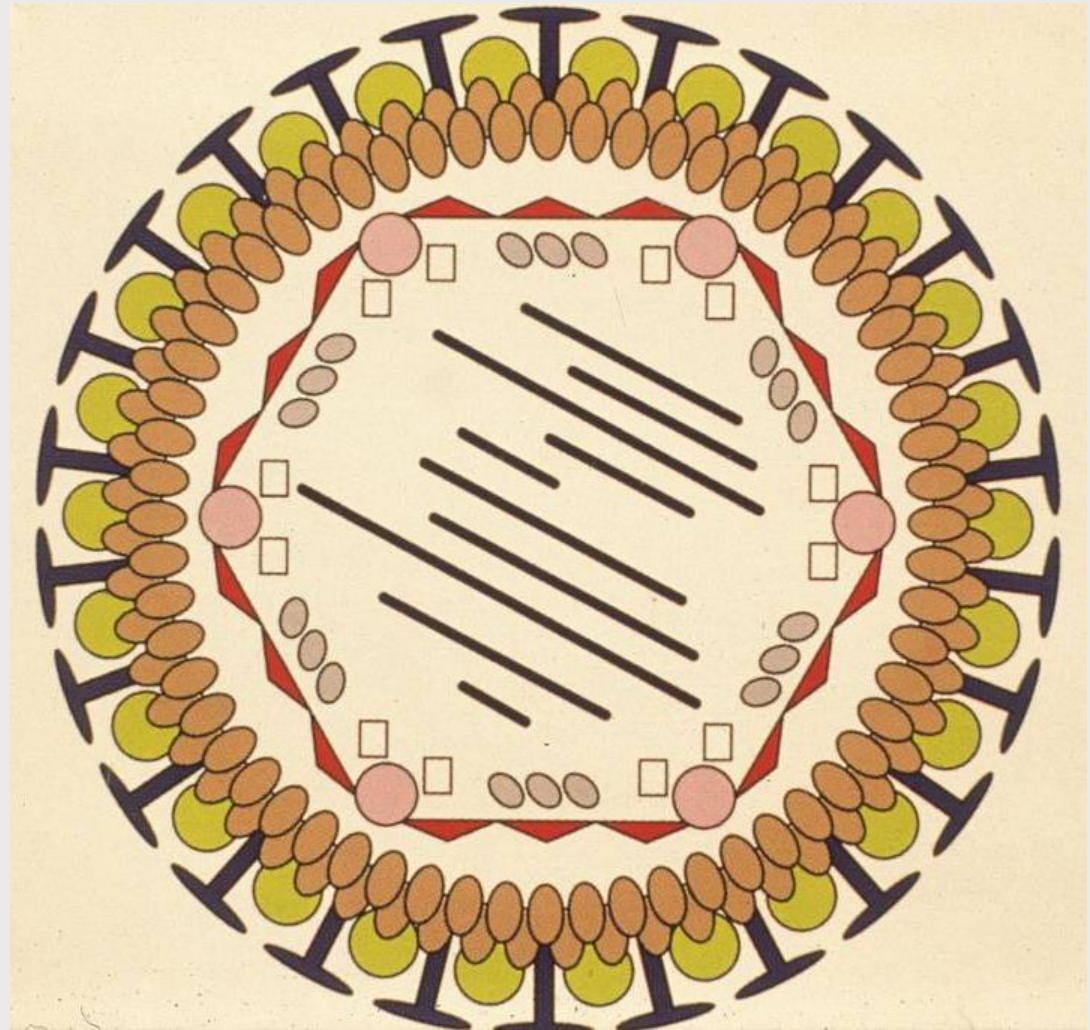
# History

- Before 1900:
  - Caused by dew on the grass (Dew disease)
- 1903: McFadyean
  - Horses protected in mosquito-proof enclosures
- 1905:
  - 1st vaccine developed by Arnold Theiler
  - Simultaneous inoculation of virus and hyperimmune serum from salted horses
  - Used till 1933
- 1931: Alexander
  - Attenuation of AHSV through passage of virus in mouse brains
  - Propagation of virus in chicken embryos
- 1944: Du Toit
  - *Culicoides* -transmitted

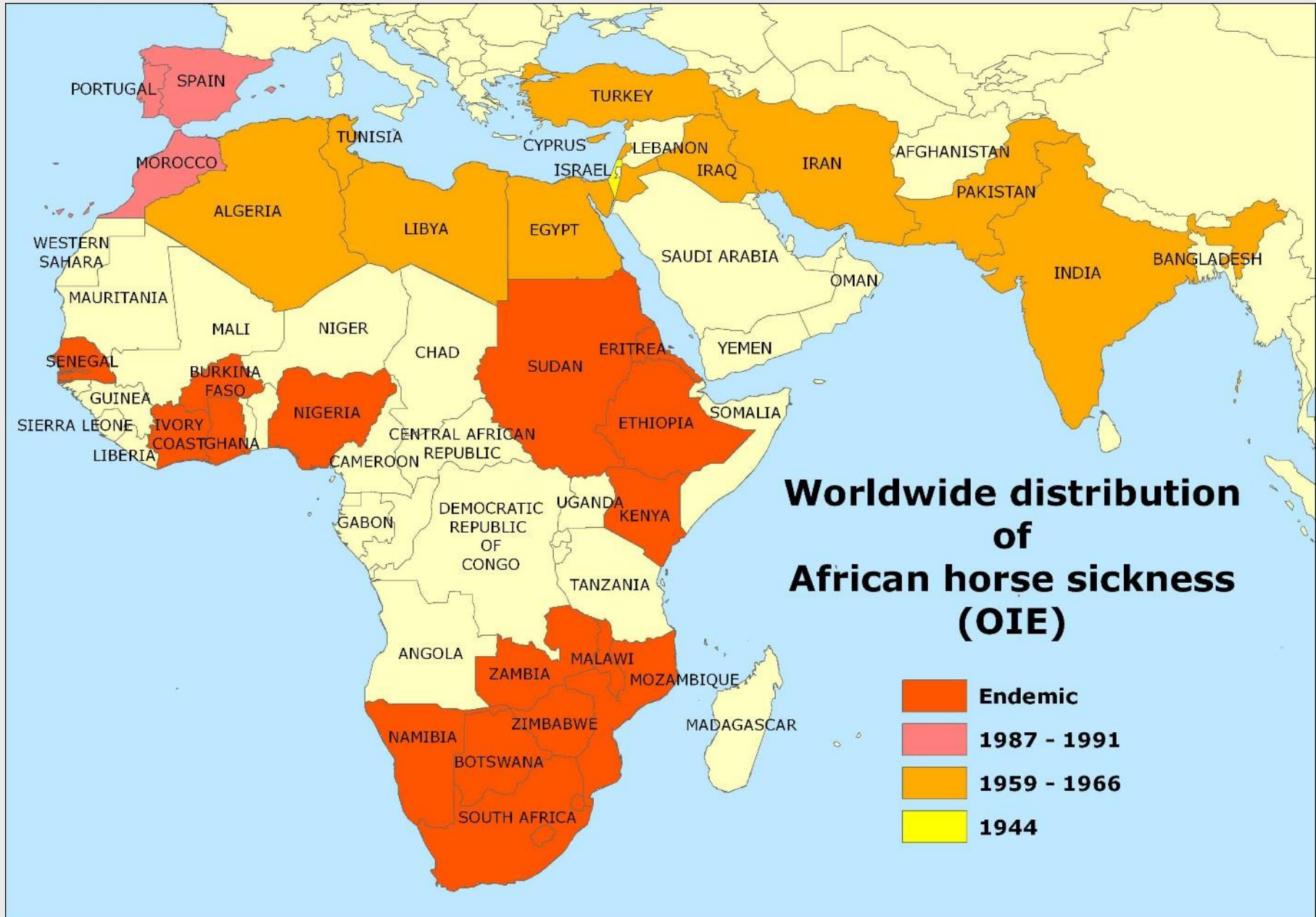


# Aetiology

- Family: Reoviridae
- Genus: *Orbivirus*
- 10 segments of dsRNA
- 9 serotypes
- Cross-protection between some serotypes:
  - 1 and 2
  - 3 and 7
  - 5 and 8
  - 6 and 9
- Modified live vaccine strains
  - Mouse and tissue passage
  - Plaque size on tissue culture



# Recent outbreaks



# Epidemiology

## Outbreaks

- Heavy rains followed by warm, dry spells
- late summer
- Frost

## Host susceptibility

- Horses: 70-95% mortality
- Mules: 50-70% mortality
- Donkeys: resistant
- Zebras: resistant (not long-term carrier)
- Dogs: susceptible

## Culicoides-borne

### *c. Imicola*

- Breed in damp soil rich in organic matter, widespread, associated with increased rainfall, active at night, tend not to enter buildings
- Virogenesis in the midge – 8 days

### *c. Bolitinos*

- Breed in cattle, buffalo, wildebeest dung, cooler areas, not dependent on rainfall and inclined to enter buildings

# Pathogenesis

- Initial lymph node
- Primary viraemia
- Target organs – lung, heart, spleen
- Replication – microvascular endothelial cells, monocyte-macrophages
- Vasculitis – oedema
- Secondary viraemia
  - Up to 21 days in horses
  - Up to 40 days in zebras



# Clinical signs

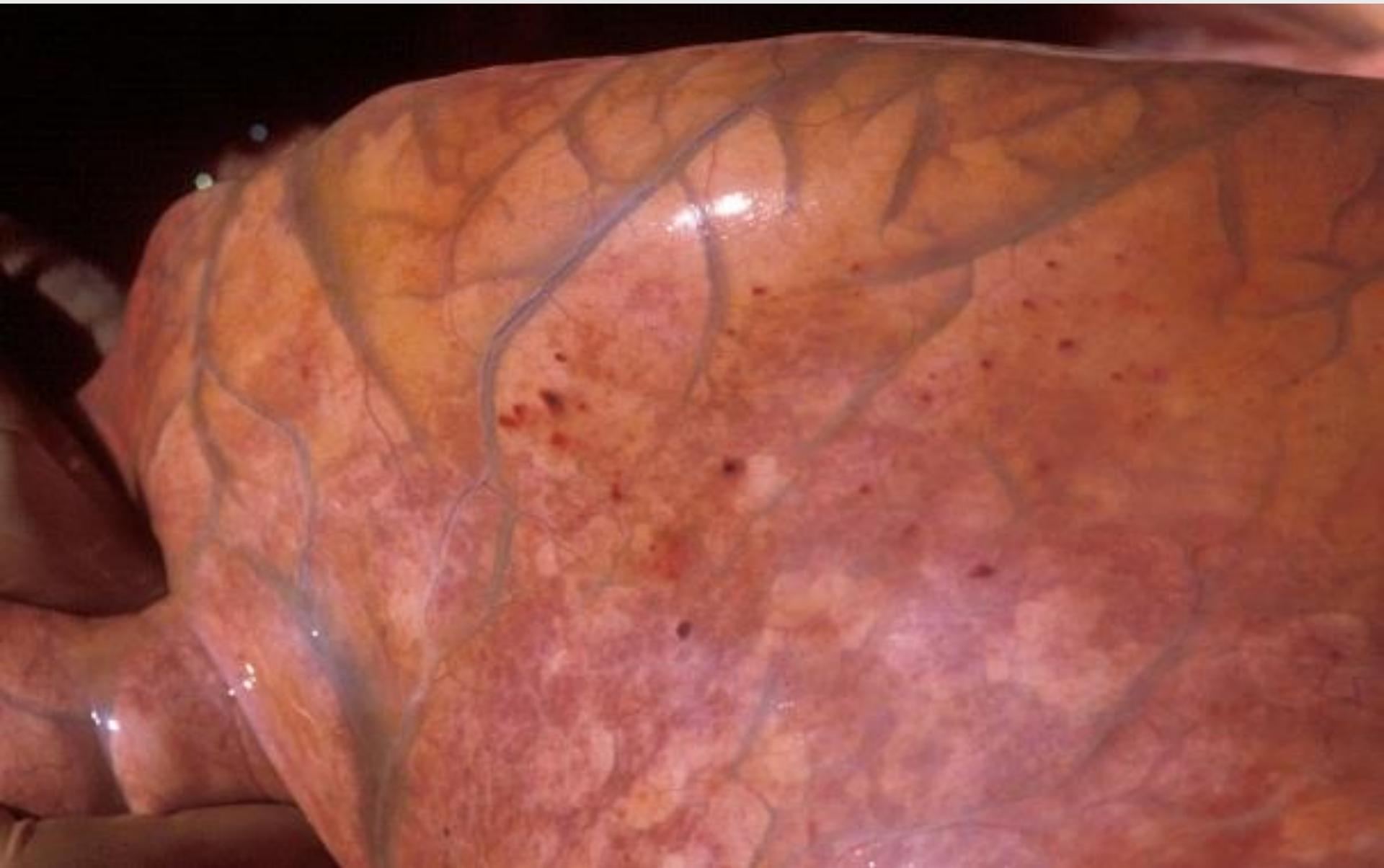
## **Horse sickness fever form**

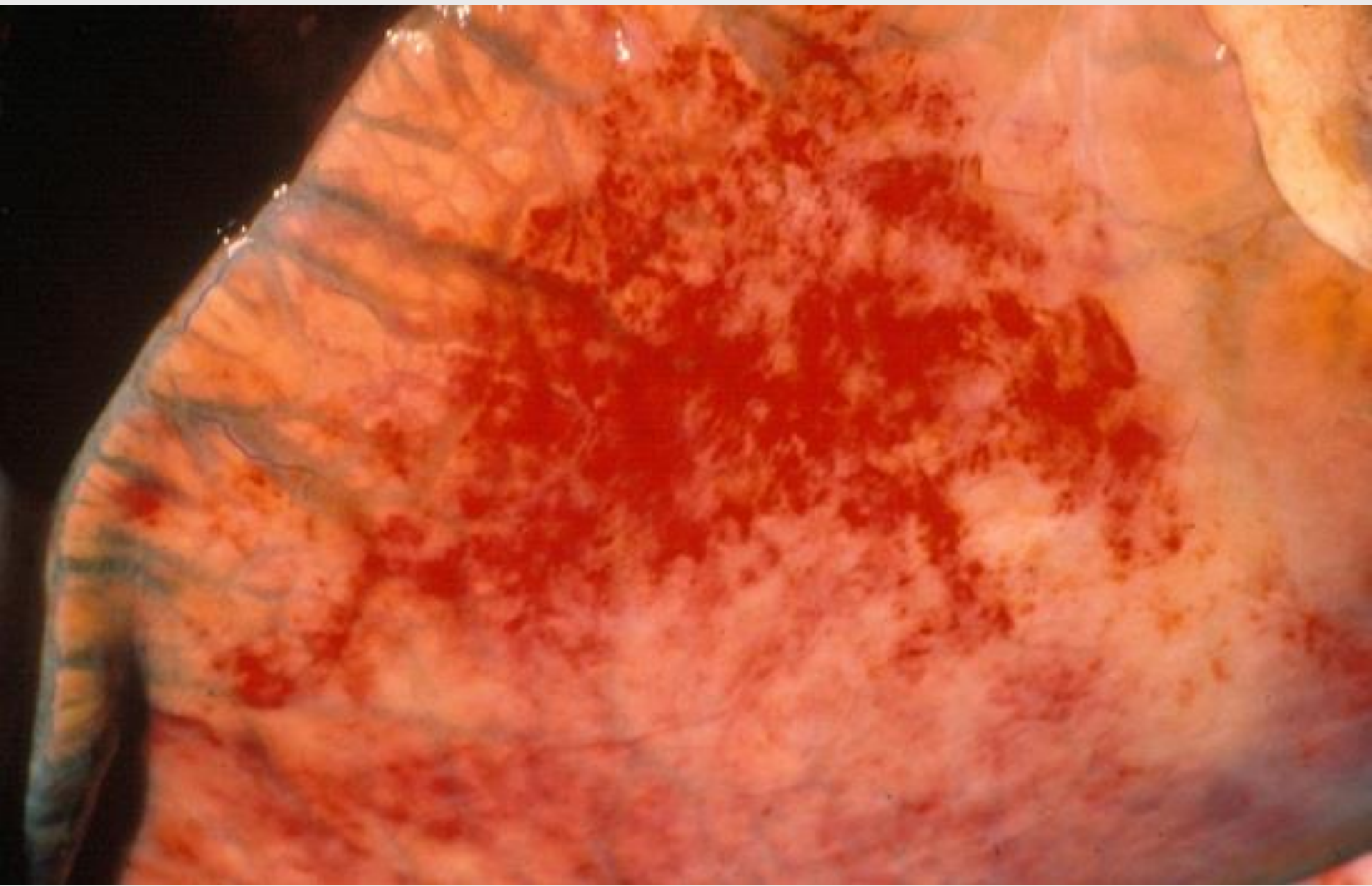
- Vaccinated horse, donkeys, zebra
- Fever only clinical signs
- Subclinical infections

## **Peracute to acute “dunkop”/pulmonary form**

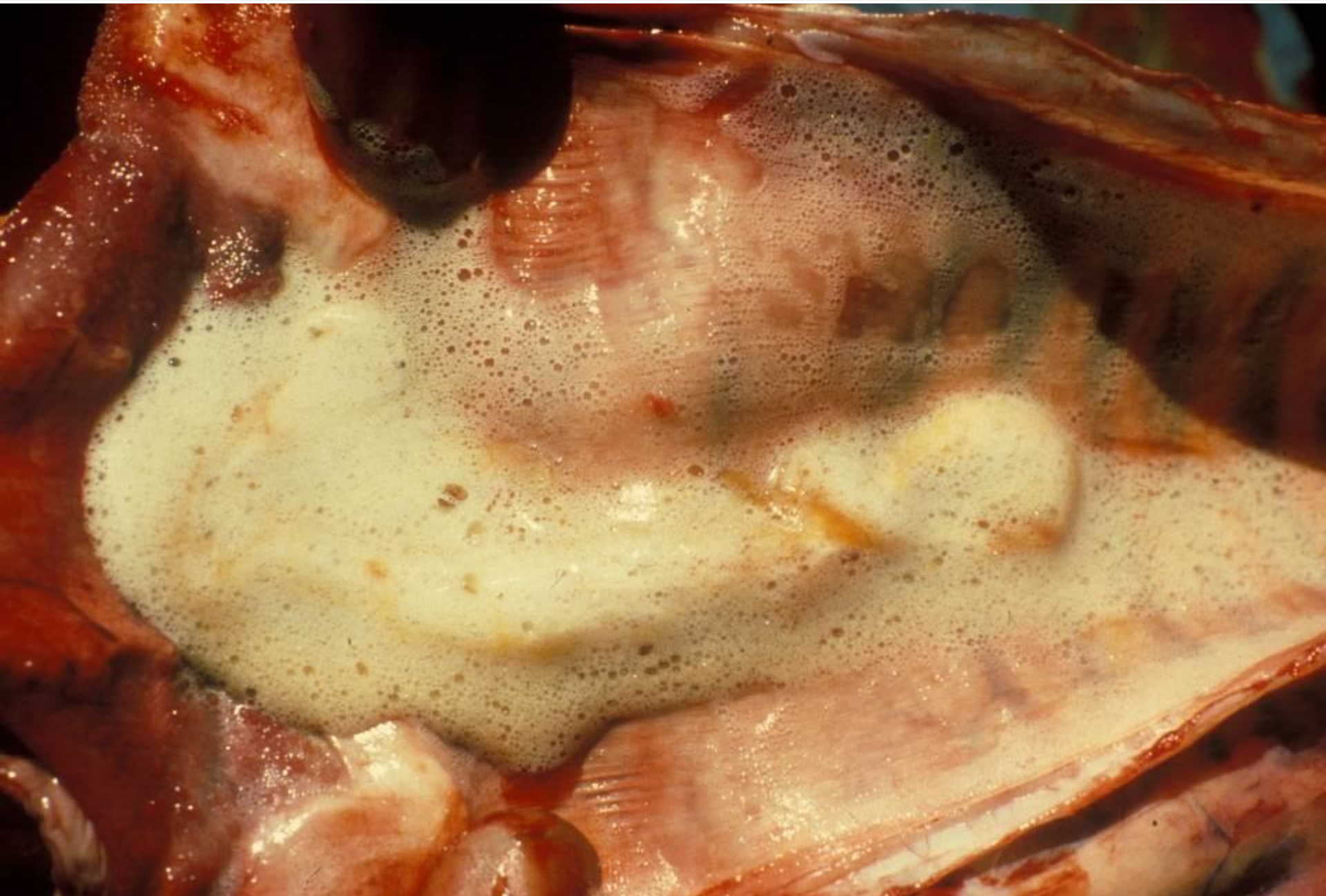
- Unvaccinated horse, foals, dogs
- Fever
- Very rapid onset of severe dyspnoea: due to lung oedema, hydrothorax
- Sometimes frothy discharge from nostrils
- Rapid deaths (<24 hours)
- Less than 5% recovery













# Clinical signs

## **Subacute or “dikkop”/cardiac form**

- subcutaneous oedema of head and neck
- mortality rate - 50%





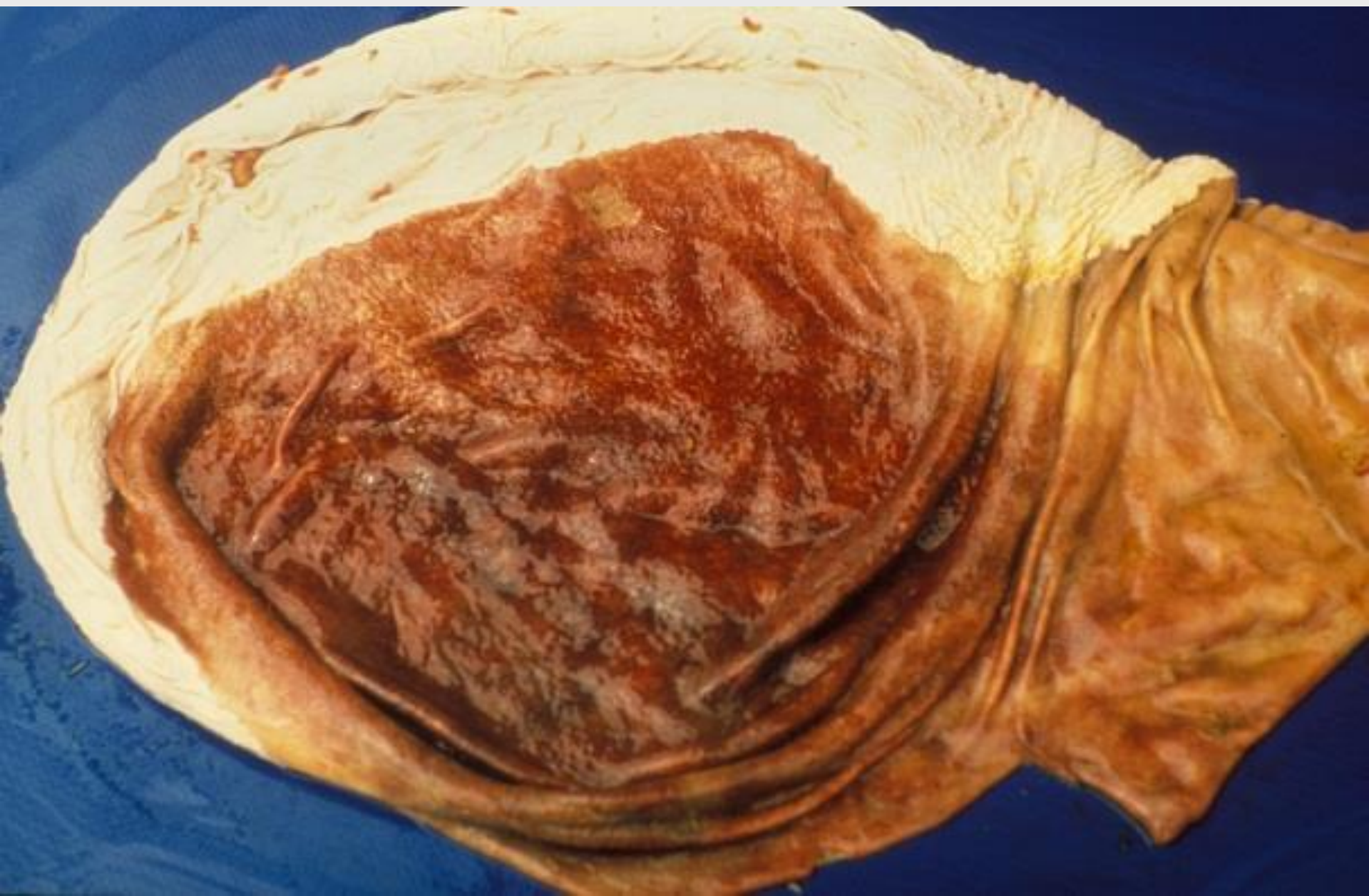






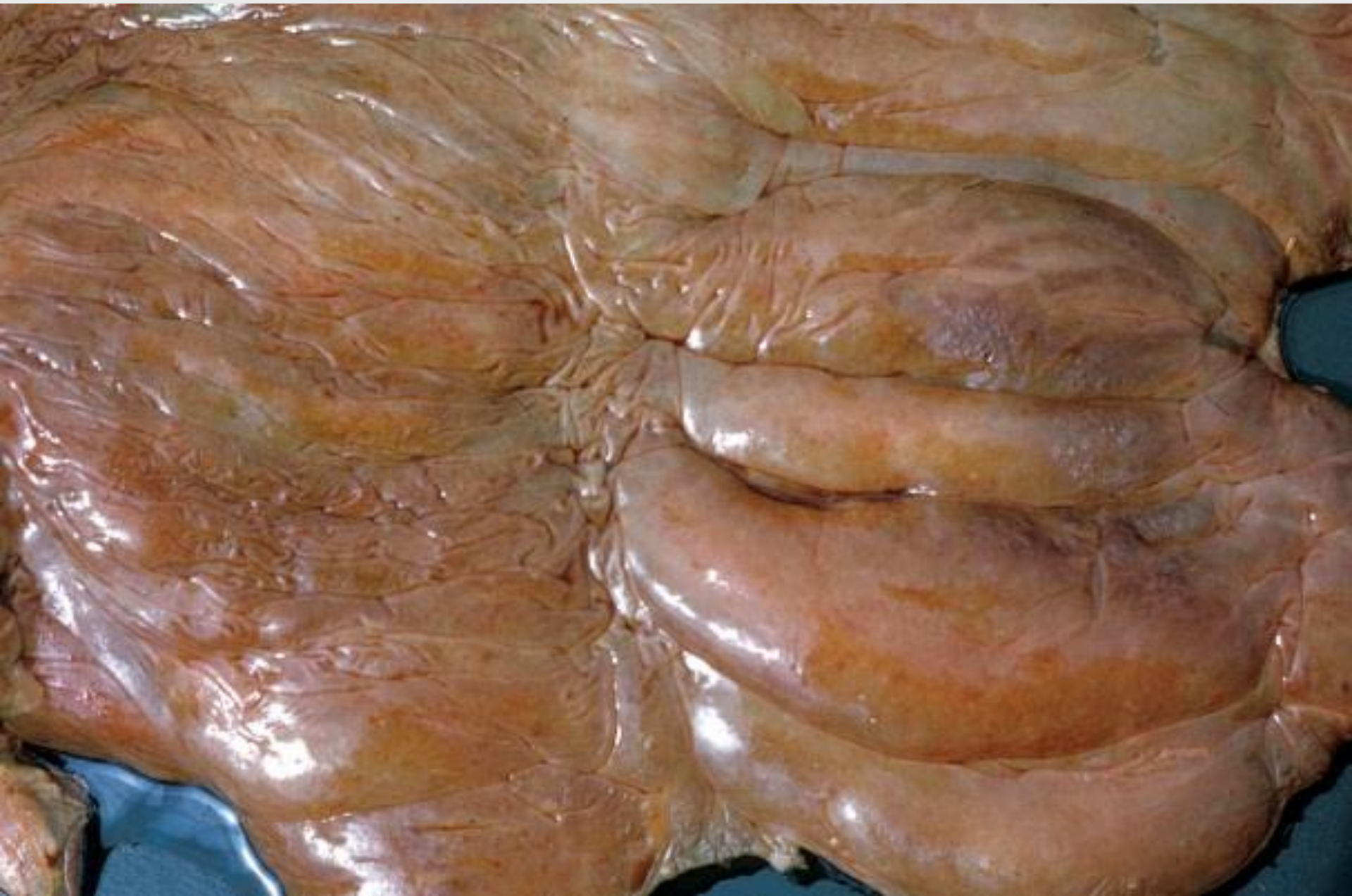














# Diagnosis

- Clinical signs
- Virus isolation – heparin blood
- RT-PCR – EDTA
- Serology-serum
- Organs – lung, spleen on ice for isolation/PCR, or formalin for IHC

# Differential Diagnosis

- Equine encephalosis
- Oedema – purpura haemorrhagica and equine viral arteritis, swelling in AHS does not extend to legs
- Piroplasmosis

# Control

## Vector control

- Stabling
- Fans
- Netting on windows and door impregnated with insecticide
- Topical insecticide/repellents on the horse
- High-lying areas

## Vaccination

- Vaccination - bottle 1 and 3 weeks later bottle 2, repeat annually late winter, early spring
- Bottle 1 - serotypes 1, 3, 4
- Bottle 2 - serotypes 2, 6, 7, 8
- Cross-reaction between 1 and 2, 3 and 7, 5 and 8, 6 and 9

## Rest and therapy

**AHS Controlled Area**

- AHS Free Zone
- AHS Surveillance Zone
- AHS Protection Zone

