CLINICAL PATHOLOGY

Examination of skin scrapings or swabs for parasitic, bacterial or other agents is essential. Culture and sensitivity tests for bacteria are advisable to enable the best treatment to be selected. Skin biopsy may be of value in determining the causal agent. In allergic or parasitic states thereis usually an accumulation of eosinophils in the inflamed area. In mycotic dermatitis organisms are usually detectable in the deep skin layers although they may not be cultivable from superficial specimens

TREATMENT

Primary treatment must be to remove the noxious physical or chemical agent from the environment or to supplement the diet to repair a nutritional deficiency

The choice of a suitable treatment for infectious skin disease will depend upon the accurate identification of the etiological agent

Supportive treatment includes both local and systemic therapy. Local applications may need to be astringent either as powders or lotions in the weeping stage or as greasy salves in the scabby stage. The inclusion of corticosteroids or antihistamine preparation is recommended in allergic states and it is desirable toprescribe sedative or anesthetic agents when pain or itching is severe If shock is present, parenteral fluids should be administered. If the lesions are extensive or secondary bacterial invasion

is likely to occur, parenterally administered antibiotics or antifungal agents may bepreferred to topical applications. To facilitate skin repair, a high protein diet or the administration of protein hydrolysates or amino acid combinations may find a place in the treatment of valuable animals. The use of vaccines as prophylaxis in viral and bacterial dermatitides must not be neglected. Autogenous vaccines maybe most satisfactory in bacterial infections.

An autogenous vaccine is particularly recommended in the treatment of

staphylococcal dermatitis in horses and bovine udder impetigo in which long and repeated courses of treatment with penicillin produce only temporary remission.

An autogenous vaccine produces a cure in many cases.

**PHOTOSENSITIZATION**

ETIOLOGY AND EPIDEMIOLOGY

If photosensitizing substances (photodynamic agents) are present in sufficient concentration in the skin, dermatitis occurs when the skin is exposed to light.

Photodynamic agents are substances that are activated by light and may be ingested preformed (and cause primary photosensitization)or be products of abnormal metabolism (and cause photosensitization due to aberrant synthesis of pigment or be normal metabolic products that accumulate in tissues because of faulty excretion through the liver (and cause hepatogenous photosensitization) . Faulty excretion through the liver may be due to hepatitis, caused in most instances by

poisonous plants, or to biliary obstruction caused by crystal-associated cholangiohepatopathy, or rarely by cholangiohepatitis or biliary calculus.

**Primary photosensitization**

Photosensitization due to the ingestion of exogenous photodynamic agents usually occurs when the plant is in the lush green stage and is growing rapidly. Livestock are affected within 4-5 days of going onto pasture and new cases cease soon after the animals are removed. In most cases the plant responsible must be eaten in large amounts and will therefore usually be found to be a dominant inhabitant of the pasture. All species of animals are affected by photodynamic agents, although susceptibility may vary between species and between animals of the same species.

Photosensitizing substances that occurnaturally in plants include:

**Photosensitization due to a berrantpigment synthesis**

The only known example in domestic animals is inherited congenital porphyria, in which there is an excessive production in the body of porphyrins, which are photodynamic.

**Hepatogenous photosensitization**

The photosensitizing substance is in all instances phylloerythrin, a normal end product of chlorophyll metabolism excreted in the bile. When biliary secretion is obstructed by hepatitis or biliary duct obstruction, phylloerythrin accumulatesin the body and may reach levels in the

skin that make it sensitive to light.

Although hepatogenous photosensitization is more common in animals grazing grep.pasture, it can occur in animals fed entirely on hay or other stored feeds and in animals exposed to hepatotoxic chemicals e.g. carbon tetrachloride. There appears to be sufficient chlorophyll, or breakdown products of it, in stored feed to produce critical tissue levels of phylloerythrin in affected animals.

The following list includes those substances or plants that are common causes of hepatogenous photosensitization.

The individual plants are discussed in more detail in the section on poisonous plants.

* Plants containing hepatotoxins
* Plants containing steroidal saponins
* Congenitally defective hepatic function
* Photosensitization of uncertain etiology

**Clinical finding**

**1-General signs**

 These commence with intense irritation and the animal rubs the affected parts,often lacerating the face by rubbing it in bushes. When the teats are affected the cow may kick at them and walk into ponds to immerse the teats in water, sometimes rocking backwards and forwards as if to cool the affected parts. In nursing ewes there may be resentment towards the lambs sucking, and heavy lamb mortalities due to starvation may result.

Local edema is often severe and may cause drooping of the ears, closure of the eyelids and nostrils, causing dyspnea, and dysphagia due to swelling of the lips. An early sign is increased lacrimation, with the initially watery discharge developing into athicker, serous discharge accompanied by blepharospasm and swelling of the eyelids.

Initial erythema of the muzzle is followed by fissuring, then sloughing of the thick skin.

**2-Skin lesions**

 Skin lesions are initially erythema, followed by edema and subsequently weeping with matting then shedding of clumps of hair, and finally gangrene. They have a characteristic distribution, restricted to the

unpigmented areas of the skin and to those parts which are exposed to solar rays. They are most pronounced on the dorsum of the body, diminishing in degree down the sides and are absent from the ventral surface. The demarcation between lesions and normal skin is very clear-cut, particularly in animals with broken-colored coats.

Predilection sites for lesions are the ears, conjunctiva, causing opacity of the lateral aspect of the cornea, eyelids, muzzle,face, the lateral aspects of the teats and, to alesser extent, the vulva and perineum. In solid black cattle dermatitis will be seen at the lips of the vulva and on the edges of the eyelids, and on the cornea. Linear erosions often occur on the tip and sides of the tongue in anin1als with unpigmented oral mucosa. In severe cases the exudation and matting of the hair and local edema causes closure of the eyelids and nostrils.In the late stages necrosis or dry gangrene of affected areas leads to sloughing of large areas of skin.

**3-Systemic signs**

These include shock in the early stages,due to extensive tissue damage. There isan increase in the pulse rate with ataxia and weakness. Subsequently a considerable elevation of temperature (41-42°C,106-107°F) may occur.

**4-Nervous signs** These including ataxia, posterior paralysis and blindness; depression or excitement are often observed. A peculiar sensitivity to water is sometimes seen in sheep with facial eczema: when driven through water they may lie down in it and have a convulsion.

**5-Liver insufficiency**

Signs are described elsewhere and may accompany photosensitive dermatitis when it is secondary to liver damage.