SOME OBSERVATIONS ON CHRONIC NASAL DISORDERS IN THE DOG.

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Dedicated to "Spot"

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SUMMARY

A retrospective study of sixty cases of canine nasal neoplasia seen at The University of Glasgow Veterinary School between 1983 and 1985 reached the following conclusions :-

There was no breed predilection; medium to large mesaticephalic dogs were the most commonly affected; the mean age was 9.2 years; there was no sex predisposition; a wide variety of clinical signs was observed, no single one being pathognomonic; carcinomas were more frequent than sarcomas, adenocarcinomas being the most common.

The radiographic features of nasal neoplasia seen on the dorso-ventral intra-oral view were - increased radiopacity together with turbinate destruction although six out of sixty cases did not follow this pattern; vomer erosion and septal deviation were highly suggestive of neoplasia; mineralisation was also indicative of neoplasia but was not typical of any one tumour type.

These findings were related to the literature reviewed.

A prospective study of twenty clinical cases of chronic nasal disorders in the dog seen between October 1985 and May 1986 concluded the following :-

Radiography was the single most useful aid to diagnosis; rhinography provided little additional information; endoscopic examination was useful predominantly in destructive rhinitis and intra-nasal foreign bodies, an alternative to an endoscope would be a large bore auroscope.

None of the biopsy techniques utilized were 100% reliable; treatment of aspergillosis with topical enilconazole achieved good results.

INTRODUCTION AND AIMS

Radiography is a valuable aid to the diagnosis and differential diagnosis of chronic nasal disease in the dog.

A review of the literature was undertaken for two main reasons; firstly, to ascertain the value and reliability of radiography reported by other authors; secondly, to assess whether any other parameters or diagnostic tests had proved useful.

The retrospective survey of sixty cases of nasal neoplasia was carried out to examine the changes seen in neoplasia and compare them to those recorded in other conditions, in particular aspergillosis and chronic hyperplastic rhinitis.

Twenty cases were studied in depth. The aim was to gain personal, practical experience in the radiological diagnosis of all patients presented with nasal disease to Glasgow University Veterinary Hospital during a set period. The usefulness of some other diagnostic techniques, namely endoscopy and rhinography, were assessed.

The purpose was to investigate the possibility of improving the reliability of radiology in the diagnosis of nasal neoplasia by using a number of guidelines.

LITERATURE REVIEW

SECTION I

There are many causes of nasal disease in the dog. The most common presenting feature is nasal discharge, either muco-purulent, sero-sanguinous or a mixture. The nature of the discharge is not disease specific.

This section reviews the conditions which can affect the nasal cavity and its associated anatomical structures. The information is based on the following sources - Lane, 1976; Bedford, 1978; Bedford, 1979; Lane, 1982; O'Brien & Harvey, 1983.

The sequence of disorders reported is based on the frequency with which similar cases were seen at Glasgow University between 1983 and 1985 (Appendix I). On each topic all the texts referred to were broadly in agreement.

Diseases of the Nasal Cavity in the Dog

Intranasal neoplasia.

Although a large number of tumours can arise in the nasal cavity, they are generally classified as:-

a) carcinoma

b) sarcoma

c) other rare types

the most frequent site of origin is the ethmoturbinate region.

Generally the tumour is unilateral initially, but following destruction of the nasal septum it can become bilateral. Extension may also be seen through the overlying bone (causing facial swelling), through the medial wall of the orbit (causing proptosis), through the cribriform plate (causing neurological signs) or through the hard palate. Primary intranasal tumours are usually

malignant, but show little tendancy to metastasize. They do, however, show a high incidence of recurrence following surgical removal.

Nasal Mycoses.

The commonest organism involved is *Aspergillus fumigatus*. *Penicillium spp* and *Cryptococcus neoformans* have also been isolated, but are thought to be far less important.

Aspergillosis is traditionally not thought to be a primary pathogen, but a secondary opportunist invader following tissue damage by virus, trauma, neoplasia or during periods of immuno - incompetance. In man primary fungal disease is recognised as the result of defective cell mediated immunity. Possibly canine rhinomycosis occurs as the result of a similar defect. Since it is also isolated in association with many other opportunist organisms it may be considered both as a primary and secondary invader.

Once infection is established the fine trabecular structure of the turbinate bones is destroyed as the mycelium advances. The infection usually commences in the caudal region of the ventral maxilloturbinate and advances rostrally. Reproductive hyphae produced by the mycelium form a mat with a macroscopic appearance of white jam mould.

In most cases an initial unilateral purulent discharge results. This may be flecked with blood indicating the aggressive nature of the infection. Epistaxis can also occur. Progression of the disease often leads to a bilateral discharge or involvement of the nasal sinuses.

Infiltration of the supporting bones of the nose is thought to be responsible for the pain and facial swelling occasionally seen. Rarely is the animal systemically ill.

Viral Rhinitis.

Adenovirus, herpesvirus, reovirus, influenza and parainfluenza virus have all been implicated as agents in the initiation of acute rhinitis. More specifically; adenovirus is known to cause laryngotracheitis; herpesvirus to cause tracheobronchitis ("kennel cough"); and the distemper virus to produce, amongst other signs, a severe rhinitis.

In many cases the virus only produces a mild or subclinical infection, but this predisposes the animal to secondary bacterial infection which frequently exacerbates and prolongs the initial condition and can produce severe complications.

Bacterial Rhinitis.

Numerous types of bacteria have been identified in the nasal discharge from dogs. However the significance of many of these is hard to evaluate. Bacteria are normally thought to act as secondary pathogens following an initiating cause such as viral infections or trauma.

Bacteria commonly isolated include *Staphylococcus spp*, *Streptococcus spp*, and *Proteus spp*. However it is now thought possible that a few bacteria can act as primary pathogens as well as in association with other organisms. *Bordatella bronchiseptica*, is one example of this.

Chronic Hyperplastic Rhinitis.

Prolonged irritation and stimulation of the nasal mucous membranes can result from conditions such as bacterial or viral infection or foreign body rhinitis if left untreated. Irreversible hyperplastic changes of the epithelium, particularly the mucous glands occur so that even if the initiating factors are removed the rhinitis may persist due to glandular hypersecretion.

Polyps are rare, but can occur associated with chronic hyperplastic changes. These are benign fibrous masses which expand within the nasal chamber and may have visible protruberences in the nasopharynx or from the nares. They may be difficult to diagnose radiographically, but respond well to surgical removal.

Allergic Rhinitis.

A rarer cause of nasal discharge is an allergic response. The patient is usually a younger animal. The clinical signs may show some seasonal occurrence. Sudden onset sneezing with a serous discharge when associated with exposure to a potential allergen is suggestive of an allergy. The presence of eosinophils in the nasal secretions is also a possible indicator. In certain cases diagnosis can be made using intra-dermal skin testing for hypersensitivities to inhaled allergens.

Allergic rhinitis has been reported as the cause of

recurrent explosive epistaxis in one case. It is an immunoglobulin E (IgE) - mediated hypersensitivity resulting from the reaction of the release of vasoactive mediators such as histamine, bradykinin, serotonin. These act on the respiratory tract causing smooth muscle spasm, increased capillary permeability and oedema which may be seen as a serous discharge. Decreased ciliary action of the overloaded ciliary mucous membranes interferes with the normal protective function of the respiritory epithelium. The resultant damage to the nasal epithelium makes it more susceptible to trauma and epistaxis.

Cryoglobulinaemia as a cause of epistaxis.

Hyperglobulinaemias are most commonly seen in association with lymphoproliferative disorders such as those caused by neoplasia (multiple myeloma, lymphosarcoma). It is generally thought that the protein is normal qualitatively, but simply present in excessive amounts. Those proteins that are macroglobulins can cause an increase in serum viscosity leading to a number of abnormalities such as circulatory disturbances and haemorrhagic tendencies.

Macroglobulins that have the characteristic of precipitation or gel formation at below body temperature are referred to as cryoglobulins.

Clinical signs in these animals include cold intolerance with circulatory impairment of peripheral extremities due to cryoprecipitation. Occasionally such animals present primarily with epistaxis. Diagnosis is made using immunoelectrophoresis.

Other causes of epistaxis.

i) Immunologically mediated thrombocytopaenia (I.M.T). This can occur as the result of several types of immune interactions with platelets which results in their destruction.

Clinical signs are compatible with those resulting from a poor clotting ability, haemorhagic tendency and subsequent anaemia. Normally the animal presents with wide-spread petechial and ecchymotic haemorrhages, but, very rarely, epistaxis may be the only obvious presenting sign.

Diagnosis is confirmed by demonstrating that anti-platelet antibody is present.

ii) Inherited coagulation defects.

Haemophilia is the commonest defect in dogs resulting from a deficiency in factor VIII. It is inherited as a sex-linked recessive gene. Other rare defects also occur.

Clinical signs are of spontaneous excessive bleeding usually as a consequence of trauma albeit mild. Hence epistaxis could be a presenting sign.

iii) Acquired coagulation defects.

The commonest is caused by Warfarin poisoning and to a lesser extent by the coumorals. Warfarin causes partial or complete deficiency primarily of factor VII and prothrombin. This can result in haemorrhage of varying severity and is often fatal. Epistaxis may be the sole sign, but it would normally be associated with other clinical features.

Idiopathic rhinarial ulceration.

Some dogs producing a nasal discharge may persistently lick their noses and consequently develop nasal excoriation.

An intractable ulcerative condition is occasionally seen in older dogs of various breeds and German Shepherd Dogs of any age. This is normally bilateral. Signs include chronic persistent sneezing, nasal discharge and sporadic epistaxis.

The ulceration may be progressive and extensive erosion of the alar cartilage can lead to distortion or even collapse of the nostrils. The etiology is unknown.

Other conditions affecting the nostrils.

i) Nasal solar dermatitis.

This condition is commonly seen in Border Collies and Shetland Sheepdogs.

Normally the lesion is located in the dorsum of the nose which shows depigmentation, erythema, erosion and ulceration. In severe cases there is excessive involvement of the lateral alar cartilage with haemorrhage.

It has been suggested that it is due to an indirect photosensitivity reaction of multifactorial etiology. This lesion is occasionally premalignant.

ii) Squamous Cell Carcinoma.

This is the most common neoplasm of the nostril. It is locally malignant and invasive causing tissue destruction, haemorrhage and discharge exacerbated by secondary bacterial infection.

Foreign Bodies.

Foreign bodies are an infrequent cause of chronic nasal discharge. If the offending object gains entry via the external nares and lodges in the rostral nasal conchae an immediate violent nasal irritation is set up leading to paroxysms of sneezing and possible head banging or nasal frenzy. This may be sufficient to dislodge the object. However, the symptoms may persist due to inflammatory reaction and possibly secondary bacterial infection. In severe cases, where damage is extensive, stenosis of the airways can occur.

Grass awns are one particular type of foreign body. These have a seasonal and geographical incidence. Less frequently needles are chewed through the soft palate; chicken bones or wood may pass forward from the naso-pharynx or bones caught between the upper carnassial teeth can result in pressure necrosis through the hard palate. If the foreign body is located in the caudal nares, gagging and retching may be the predominant signs.

Food insufflation will also lead to a foreign body rhinitis. Conditions which may lead to this include oro-nasal fistulae, pharyngeal inco-ordination and mega-oesophagus.

Extension of dental disease into the nasal chambers.

This is another rare cause of nasal discharge as damage to the maxillary teeth, particularly the carnassial, normally results in a discharging sinus beneath the eye.

Occasionally periapical abscesses of other teeth, in particular the canine and incisors, may cause suppuration directly into the nasal chamber and hence nasal discharge.

Loss or incorrect extraction of a canine tooth can lead to oronasal fistulation and resultant foreign body rhinitis as mentioned previously.

Trauma.

Traumatic damage to the nose can cause intranasal haemorrhage, epistaxis, fractures or any other combination. Blood is an ideal medium for opportunist organisms which may lead to chronic sequelae.

Osteomyelitis can occur. Damage to the turbinate structure or the presence of a sequestrae can result in a chronic discharge. Alternatively depression fractures of the frontal bone have been reported causing a frontal sinus mucocoele which may necessitate fracture repair in some instances.

Sinusitis.

Primary sinus disease is rare although it can develop as an extension of other intranasal conditions. Primary paranasal tumours have been reported.

LITERATURE REVIEW

SECTION II

Nasal Neoplasia and its major Differential Diagnoses.

Breed Incidence

i. Neoplasia.

As a generalisation it is often stated that nasal neoplasia affects medium to large breeds of dogs with long noses, and that branchycephalic dogs are somehow "immune".

Most authors found that medium and large breeds predominated in their series. Morgan *et al* (1972) reported that all 70 dogs in their survey were over 10kg with 50 per cent over 20kg. The results provided by MacEwen *et al* (1977) and Lane (1982) were similar; giant and minature types were seldom affected.

There have been infrequent reports of the disease affecting the giant breeds, namely one Saint Bernard and five Great Danes (Madewell *et al*, 1976; Confer and De Paoli, 1978; Hayes *et al*, 1982). Small and toy breeds although uncommon were encountered particularly in the larger surveys (Delmage, 1973; Madewell *et al*, 1976; Confer and De Paoli, 1978; Harvey *et al*, 1979; Norris, 1979; Hayes *et al*, 1982).

The initial statement that dolicocephalic breeds are at highest risk is not generally supported by the literature. Morgan *et al* (1972) and Norris (1979) both found that breeds with a mesaticephalic skull structure were most commonly affected.

This was in contrast to the findings of Hayes *et al* (1982) which were based on a large survey of 504 dogs. They said that dolichocephalic dogs had 2.5 times the risk of mesaticephalics whilst branchycephalics showed a significantly lower risk. They then went on to say that the risk estimate for dolichocephalic breeds basically represented the prevalence of the Collie and Sheltie breeds. They also commented that within the mesaticephalic breeds there was no association between body size and risk.

Although brachycephalics are not commonly affected they still occurred in small numbers in the larger surveys, particularly the boxer (Morgan *et al*, 1972; Bradley and Harvey, 1973; Confer and De Paoli, 1978; Norris, 1979; Hayes *et al*, 1982).

Brodey (1970) commented that mixed breeds appeared under-represented. This is not supported by other published papers.

In reviewing the different series certain breeds were recorded more frequently, namely: mixed breeds, Collies, German Shepherd dogs, Shetland sheepdogs, Spaniels and Poodles (Sande and Alexander, 1970; Morgan *et al*, 1972; Bradley and Harvey, 1973; Madewell *et al*, 1976; MacEwen *et al*, 1977; Confer and De Paoli, 1978; Harvey *et al*, 1979; Norris, 1979; Hayes *et al*, 1982)

ii. Aspergillosis

As with neoplasia the dolicocephalic and mesaticephalic breeds appeared more susceptible (Lane, 1976; Bedford, 1978). However, there are occasional reports of occurences in brachycephalic dogs. Harvey *et al* (1979) recorded a case of aspergillosis in an English Bulldog. In a personal communication a recent survey carried out by Lane (1982) showed the comparitive distribution of breeds affected by neoplasia and aspergillosis.

Aspergillosis		Neoplasia
14	Labrador/retriever	24
23	Collie	19
9	German Shepherd Dog	8
7	Spaniel	7
4	Old English Sheepdog	2
4	Afghan Hound	1
3	Irish Setter	2
3	Jack Russell Terrier	2
3	Dachshund	2
1	Boxer	5
29	Others	28
100	TOTAL	100

TABLE 1. The comparative distribution of breeds affected by Neoplasia and Aspergillosis.

iii. Other Causes of Chronic Nasal Disease.

These can occur in any breed of dog. Lane (1982) stated that Dachshunds and Whippets appeared particularly prone to chronic hyperplastic rhinitis and that polyps, although uncommon in dogs, arose most often in the German Shepherd Dog.

Age Incidence

i. Neoplasia.

The overall view was that nasal neoplasia affected middle to older aged dogs. Madewell *et al* (1976) calculated that the risk of nasal neoplasia generally increased as the animal aged.

The average age at the time of diagnosis was between eight and ten years (Brodey, 1970; Sande and Alexander, 1970; Bradley and Harvey, 1973; Delmage, 1973; MacEwen *et al*, 1977; Confer and De Paoli, 1978; Norris, 1979; Lane, 1982). However the ages ranged from one to sixteen years. Several authors recorded a small, but significant, number of dogs in the one to five year age bracket (Morgan *et al*, 1972; MacEwen *et al*, 1977; Harvey *et al*, 1979; Lane, 1982)

ii. Aspergillosis.

Lane (1982) wrote that dogs of any age may become infected with *Aspergillus fumigatus*, but that younger patients were more frequently involved. The range was from less than one year old to thirteen years old with a median of two to three years and a mean of about four years.

In contrast Harvey *et al* (1979) in a smaller survey recorded a mean of approximately six years with a similar age range.

iii. Other causes of chronic nasal disease.

The combined surveys of Delmage (1973) and Harvey *et al* (1979) showed a range of four months to eight years with no predilection.

Sex Incidence.

Most authors agreed that the distribution of nasal neoplasia shows no sex predilection (Bradley and Harvey,

1973; Delmage, 1973; Madewell *et al*, 1976; Harvey *et al*, 1979; Norris, 1979; Lane, 1982).

In contrast Brodey (1970) reported a male:female ratio of 3.3:1 and Morgan *et al* (1972) in a large survey also found a higher prevalence in males 1.8:1. The relevance of these results was questioned by MacEwen *et al* (1977) when he commented that these figures had to be assessed with reference to the overall sex ratio of the population seen. Frequently, the results were then not found to be statistically significant.

However, the largest survey, carried out by Hayes *et al* (1982), still concluded that malignancy was more common in males. After adjustment for breed and age their risk was 1.3 times that of females.

Clinical signs.

i. Neoplasia.

All authors concluded that the clinical signs of nasal neoplasia were non-specific and numerous. There was little variation between the reports as to the most common presenting clinical sign; nasal discharge, sneezing or epistaxis.

The majority of workers noted that any discharge was normally initially unilateral, but could become bilateral as the lesion expanded. An exception was cited by Delmage (1973) where the symptoms were bilateral from the onset. Norris (1979) made the unusual observation that seven of the thirty four dogs studied showed central nervous system (C.N.S) signs. Two of these had no other common classical signs and showed behavioural change, salivation, ataxia, dilated pupils and convulsions.

A list of all clinical signs noted by the authors follows:-

a. Common signs.

Nasal discharge
- Unilateral or bilateral
  - Mucoid
  - Purulent
  - Blood tinged

Epistaxis
- Unilateral or bilateral

Sneezing

b. Signs seen in most surveys but far less frequently.

Snorting/gagging
Superficial swelling and/or facial deformity
Nasolacrimal duct obstruction
Ocular proptosis
Ventral deflection/distortion/erosion of the hard palate

c. Signs seen occasionally in fewer surveys.

Stertorous breathing
Mouth breathing/Nasal obstruction
Dyspnoea
CNS disorders including fits

d. Signs rarely seen.

Coughing
Oral mass
Tonsillitis
Loosened/displaced/missing teeth
Dysphagia
Nasal pain

(Brody, 1970; Sande and Alexander, 1970; Morgan *et al*, 1972; Bradley and Harvey, 1973; Delmage, 1973; Bright and Bojrab, 1976; Madewell *et al*, 1976; MacEwen *et al*, 1977; Norris, 1979; Lane, 1982).

Most of the above authors commented that the duration of signs preceding a definitive diagnosis was generally less than one year although exceptional cases had histories of two, three and four years duration. The average was two to three months (Bradley and Harvey, 1973; Madewell *et al*, 1976; MacEwen *et al*, 1977): Although Norris, (1979) reported a longer than average six months. The reports showed that clinical signs could either be sudden or insidious in onset.

ii. Aspergillosis.

Bedford (1978) and Walshaw and Ford (1980) said that nasal discharge could be either unilateral or bilateral. However, Lane *et al* (1974), Gibbs *et al* (1979) and Lane (1982) observed that it was more commonly unilateral, but may progress to be bilateral.

The frequency of epistaxis noted by authors showed some variation. Lane (1982) said that epistaxis was an occasional finding whereas Coulson (1982) said it was more common than in neoplasia.

Pain over the bridge of the nose or facial discomfort was an occasional finding, but facial deformity was rare (Bedford, 1978; Lane, 1982; Coulson, 1982).

iii. Other Conditions.

a. Chronic Hyperplastic Rhinitis.

Madewell *et al* (1976) commented that unilateral ocular and nasal discharge was characteristic of early neoplasia whereas discharge associated with chronic inflammation was usually bilateral. However Gibbs *et al* (1979) showed almost 75 per cent (15/22) of their cases of mucosal hyperplasia were unilateral.

In addition to a nasal discharge, coughing or sneezing sometimes occured, but epistaxis was rare.

Walshaw and Ford (1980) noted, in contrast to Madewell *et al* (1976), that a unilateral ocular discharge could also be associated with chronic rhinitis due to blockage of the nasolachrimal duct.

White *et al* (1984) reported an unusual case of unilateral cystic naso-lachrimal obstruction with adjacent chronic turbinate inflammation causing intermittent ocular discharge as well as nasal stertor, episodic sneezing and unilateral facial swelling.

b. Foreign Bodies.

Sudden onset persistent sneezing, head shaking and nasal frenzy progressing to a chronic unilateral purulent discharge were the classical signs. However, the initial incident could go unobserved making the diagnosis more difficult (Bedford, 1978; Walshaw and Ford, 1980; Lane, 1982).

A more unusual case was referred to by Delmage, (1973) where there was an extension to bilateral involvement.

Tumour Type.

Brodey (1970) in a review of feline and canine

neoplasia said that tumours of the respiratory tract comprised 2.3 per cent (66/2917) of the canine neoplasms seen. One third of these tumours were of nasal origin and about one sixth arose from the paranasal sinuses (frontal and maxillary).

More than thirty different types of neoplasm have been identified in the nasal cavity and paranasal sinuses (Madewell *et al*, 1976).

The two main tumour groups recorded were carcinoma and sarcoma. Some authors said the incidence of carcinomas was about 70 per cent (Brodey, 1970; Bradley and Harvey, 1973; MacEwen *et al*, 1977; Confer and De Paoli, 1978; Norris, 1979), whereas others found only a slightly higher incidence when compared with sarcomas (Morgan *et al*, 1972; Madewell *et al*, 1976; Gibbs *et al*, 1979)

Although nasal tumours were found to be very invasive locally the overall conclusion was that they rarely produced regional or distant metastases and few examples have been described.

MacEwen *et al* (1977) reported one case where an adenocarcinoma metastasised to the lungs. Norris (1979) recorded three cases: one adenocarcinoma which metastasised to the lungs; one carcinoma to the lung, liver and spleen; and one squamous cell carcinoma to the local lymph nodes. Hayes *et al*, (1982) found that approximately 10 per cent of their series had evidence of metastatic spread.

Diagnosis, Treatment and Survival Times.

Brodey, (1970), Morgan *et al*, (1972), Bright and Bojrab, (1976) and Gibbs *et al*, (1979) all commented that one of the major clinical problems was making a diagnosis early enough to effect a possible cure. In many cases the signs were well established at presentation and showed obvious radiological changes. This suggested that the lesion was already too extensive to allow complete surgical removal in most cases. Early cases presented the greatest problem in differential diagnosis and these were under-represented in the surveys. Clinical signs initially may be the same as in other nasal disorders.

Bradley and Harvey, (1973) said that while it may be possible surgically to remove some intra-nasal tumours at an early stage in their development this would require a presumptive diagnosis and immediate exploratory surgery

for every dog showing suspicious clinical signs. However, they also found that about 28 per cent (8/29) of the dogs in their series showing marked radiological signs of neoplasia had a clinical history of one month or less.

Cook (1964) described a case with a clinical history of ten days duration in which the tumour was bilateral having eroded through the nasal septum.

Bradley and Harvey (1973) treated three dogs with a clinical history of one month or less; two were euthanased immediately after surgery and the third after three months. Conversely two other dogs in their series - one receiving no treatment and the other radiotherapy - survived eighteen months and twenty-six months respectively.

A variety of treatment methods have been used as shown in Table 2. None of the methods produced uniformly good long term survival rates although Norris (1979) suggested that radiation alone or combined with surgery merited further investigation.

This was undertaken by Thrall and Harvey in 1983. They treated 21 dogs with radiation therapy, 18 of which also under went prior debulking surgery. The range of survival times was from five to 79 months, with a two year survival rate of 48 per cent.

Radiography.

All the authors reviewed routinely used the lateral and intra-oral dorso-ventral radiographic views on anaesthetised patients. The latter was found most diagnostic. In addition, the majority used a combination of dorso-ventral skull, oblique lateral or skyline views of the frontal sinuses either in every case or where indicated. The intra-oral views were taken using non-screen film for enhanced contrast and detail. (Sande and Alexander, 1970; Morgan *et al*, 1972; Bradley and Harvey, 1973; Delmage, 1973; Bright and Bojrab, 1976; MacEwen *et al*, 1977; Gibbs *et al*, 1979; Harvey *et al*, 1979; Norris, 1979).

Radiological Features of Nasal Neoplasia.

There is some dispute as to the value of radiology in the diagnosis of nasal neoplasia. There are two opposing points of view.

	Bradley (1973)	Delmage (1973)	Madewell (1976a)	Madewell (1976b)	MacEwen (1977)	Norris (1979)	Withrow (1982)	TOTAL
No Treatment	3(9)	-	-	-	5(7)	2(1)	-	3.7(17)
Surgery	1(13)	3(7)	12(4)	4.5(38)	1(2)	5.5(2)	-	4(66)
Surgery & Radiation	9(2)	-	5.3(15)	9(2)	4(1)	7.9(7)	-	6.5(27)
Surgery & Immuno-therapy	-	-	-	-	7(6)	-	-	7(6)
Immuno-therapy	-	-	-	-	3(3)	-	-	3(3)
Radiation	39(2)	-	-	78(2)	-	-	-	58.5(4)
Cryosurgery	-	-	-	-	1(3)	1(1)	3.5(8)	2.6(12)
Cryosurgery & Radiation	-	-	-	-	-	8(1)	5.5(2)	6(3)
Cryosurgery & Immuno-therapy	-	-	-	-	20(1)	-	38(2)	32(3)
Chemo-therapy	-	-	-	-	6(1)	-	-	6(1)

TABLE 2. The methods of treatment and average survival times of 142 dogs with nasal tumours compiled from the available literature.

The survival times are shown in months. The numbers in brackets refer to the number of cases treated.

Many authors felt that the radiological changes were fairly non-specific and that it was seldom possible to differentiate between nasal tumours, rhinitis or foreign bodies. It was useful in establishing the extent of the lesion, degree of turbinate erosion or other bone destruction, but could not be used for diagnosis of neoplasia unless the lesion was very extensive. (Sande and Alaxander, 1970; Delmage, 1973; Madewell *et al*, 1976; MacEwen *et al*, 1977; Harvey *et al*, 1979).

Other authors disagreed with this assessment. Morgan *et al*, (1972), Bright and Bojrab, (1976) and Gibbs *et al*, (1979) all agreed that radiology was the most convenient and reliable method of diagnosing intra-nasal disorders in the dog. Despite this they also said that very early lesions may cause a problem. If changes were equivocal they recommended taking repeat films seven to ten days later.

Gibbs *et al* (1979) also commented that the irrigation of diseased nasal chambers provided no significant improvement in radiographic contrast.

Morgan *et al* (1972) were alone in describing three groups of radiographic changes which they said made it possible to predict the cell type of the malignant disease with some degree of accuracy. Their conclusions appeared very generalised. All three groups caused loss of nasal turbinate pattern. They said chondrosarcomas usually displaced the nasal septum. Their results showed six cases; three with septal deviation and three with septal destruction. This was apparently distinct from carcinomas which were classified as destroying the nasal septum. However, of the thirty three carcinomas, the tabulated results showed thirteen with septal destruction, three with deviation and seventeen were not mentioned - presumably as there was no septal involvement.

Gibbs *et al* (1979) disagreed with these findings. They argued that when the radiographic changes associated with carcinomata and sarcomata were compared the features of these two groups were essentially similar except that intra-nasal mineralisation was observed in chondrosarcomata.

The characteristic changes reported in neoplasia were:-

i. Loss of normal fine trabecular pattern of the turbinates together with an increase in density - it was rarely possible to determine the full extent of the lesion

since obstruction of the airways leads to an accumulation of secretions particularly caudal to the expanding mass.

ii. Frontal sinus opacification was common due to the accumulation of exudate or extension of the mass.

iii. Deviation or erosion of the nasal septum.

iv. Erosion and lysis of adjacent structures such as the maxilla and overlying cortical bone, hard palate, orbit and cribriform plate.

iii and iv were seen in the more progressive lesions.

The last two features were recorded in about 40 - 50 per cent of reported cases (Morgan *et al*, 1972; Bradley and Harvey, 1973; Bright and Bojrab, 1976; MacEwen *et al*, 1977; Gibbs *et al*, 1979)

Bright and Bojrab, (1976) and Lane, (1982) said that intra-nasal neoplasia commonly originated from the ethmoturbinate region. Gibbs *et al* (1979) agreed and that 46 of the 50 cases of neoplasia showed involvement of the caudal portion of the nasal cavity. In 42 cases an increase in radiographic density was accompanied by loss of trabecular pattern indicating that the tumour mass itself was caudally located.

The Nasal Septum.

Gibbs *et al* (1979), considered that the integrity of the nasal septum as shown radiographically was not a reliable indicator of unilateral confinent of destructive processes. On the lateral radiograph it was too thin to produce a definitive image, and in dorso-ventral projection the relatively dense vomer bone ventrally is superimposed on the thinner dorsal portion which may be extensively penetrated without accompanying radiological signs. In their series eight of the twenty four cases with bilateral involvement showed no radiographic evidence of a septal defect. Morgan *et al* (1972) had also reported this finding.

Harvey (1979) radiographed a dog's nose using an intra-oral dorso-ventral view before and after the cartilaginous nasal septum had been removed and concluded that there were no differences in the midline radiopacities previously referred to as nasal septum.

The cartilaginous nasal septum was not visible radiographically and the midline linear radiopacity visible in the normal dogs was the vomer bone.

Radiological changes with reference to other nasal disorders.

The difficulty of identifying the radiological changes in nasal disorders in the early stages has been mentioned previously.

i.Destructive rhinitis due to *Aspergillus fumigatus* infection.

Gibbs *et al* (1979) proposed that radiological signs of destructive rhinitis were definitive and in all cases consisted of loss of the normal intra-nasal trabecular pattern together with increased lucency of the affected chamber or chambers. A mixed density pattern may also be seen. Lesions involved predominantly the middle portion of the nasal cavity and tended to progress rostrally. The caudal segment was affected only in advanced cases.

Lane and Warnock (1977) and Sullivan *et al* (1986) agreed that the signs were characteristic.

Sullivan *et al* (1986) described the changes in more detail:-

i. turbinate loss with punctate lucencies of the supporting bones.

ii. increased radiolucency rostrally

iii. mixed density pattern caudally

iv. frontal sinus opacity

v. thickening and mottling of the frontal bone.

Of the forty five cases studied only six showed mild changes. The remainder were moderate to severe; Fifty eight per cent of the cases were bilateral. This contrasts with Gibbs et al (1979) where unilateral signs predominated - 70 per cent (18/26)

Gibbs *et al* (1979) and Harvey *et al*, had both observed that in advanced cases septal destruction, facial bone destruction, sequestra formation and facial swelling could occur.

ii. Chronic Hyperplastic Rhinitis.

Nasal neoplasia causes an increase in opacification of the nasal chambers. Many authors have therefore described the difficulty in distinguishing it from hyperplastic rhinitis and haemorrhage which also causes an increase in radiopacity (Sande and Alexander, 1972; Morgan *et al*, 1972; Delmage, 1973)

Gibbs *et al* (1979) established that in hyperplastic conditions there is an increase in volume of the nasal mucosa together with hypersecretion which leads to obliteration of the nasal air-spaces. This produced a radiographic appearance of increased density. Since these pathological changes were not invasive; turbinate structures remained intact and were masked, not destroyed.

However, Harvey *et al* (1979) did report on two exceptional cases of chronic rhinitis which apparently showed bony erosion; one of which also showed no increase in opacity.

iii. Miscellaneous conditions.

Radiopaque foreign bodies were easily identified however radiolucent ones did cause a problem. Intra-nasal foreign bodies could result in inflamation and hence increased radiopacity or infection of the nasoturbinates and overlying bone. (Morgan *et al*, 1972; Gibbs *et al*, 1979)

Harvey *et al* (1979) cited one case of osteomyelitis which caused erosion of the vomer bone. Osteomyelitis could also result in sequestra formation.

Positive-Contrast Rhinography.

Goring *et al* (1982, 1984 a,b) described in detail the use of contrast rhinography as a simple non-invasive procedure that may be a useful diagnostic aid in the radiographic evaluation of the nasal cavity, nasopharynx and paranasal sinuses in the dog. Four contrast media were compared: 60 per cent propyl iodine; 30 per cent organically bound iodine; a 60 per cent barium sulphate suspension; and a 30 per cent barium sulphate suspension.

On the basis of the radiographic contrast detail, distribution and degree of evoked tissue reaction a 30 per cent barium sulphate suspension was selected for use. A

detailed description of the procedure used was given. A dose rate of 1ml of contrast per 5kg of body weight was used.

An evaluation of the technique in six dogs with nasal disorders concluded that positive contrast rhinography could be used to obtain diagnostic information that was not provided by survey radiographs.

Additional Diagnostic techniques.

i. Microbiological examination.

The value of microbial culture obtained by swabbing the nasal discharge or nasal flushing was questioned by most authors. The bacteria most commonly isolated were *Coliform spp, Streptococcus spp, Staphylococcus spp* and *Pasteurella spp*. Cultures could be sterile, most commonly found with haemorrhagic nasal discharges, or alternatively produced mixed or pure growths. It was difficult to interpret the relevance of any results as in most instances the organisms were upper-airway commensals which were acting as opportunist invaders.

Examination occasionally yielded *Aspergillus fumigatus* which was thought to be diagnostic. However Aspergillosis was normally diagnosed serologically (Delmage, 1973; Lane, 1976; Bright and Bojrab, 1976; MacEwen *et al*, 1977; Withrow, 1977).

The most accurate method used was the double diffusion test as performed by Murray and Mahgoub (1968), or the more rapid counter-immuno-electrophoresis (C.I.E) described by Richardson *et al* (1982). Cultures and direct smear testing were generally unreliable.

Penicilliosis could also be diagnosed serologically, but was an extremely rare isolate of the nasal cavity (Harvey *et al*, 1981)

ii. Cytology and histopathology.

The use of these tests definitively to diagnose neoplasia or suggest other disease processes were made by a number of workers. Samples were obtained by a variety of methods depending on the site of the lesion:-

a. Nasal flushing either passive or aggressive
b. Direct aspiration with a needle
c. Nasal smears
d. Simple incision biopsy (where the tumour has eroded through the overlying bone)

e. Biopsy using endoscopy.

(Bradley and Harvey, 1973; MacEwen *et al*, 1977; Withrow, 1977; Gibbs *et al*, 1979; Norris, 1979; O'Brien and Harvey, 1983)

MacEwen *et al* (1977) achieved a fifty per cent success rate using a nasal flush technique and cytology to diagnose neoplasia (11/22). However negative results did not preclude it as inflammatory change was frequently noted in cases later confirmed as neoplasia (Bradley and Harvey, 1973; MacEwen *et al*, 1977)

Withrow (1977) described a far more aggressive and vigorous nasal flushing technique to obtain sample material. He claimed that it was relatively non-invasive, simple, rapid and was a hundred per cent successful. However, not all his cases were definitely diagnosed and one animal died as a direct result of the technique whilst another developed meningitis one week later. He also suggested that the technique had a therapeutic benefit in the treatment of rhinitis when using a final flush of 10 per cent povidone-iodine solution.

iii. Rhinoscopy.

Cook (1964) described the examination of the rostral aspect of the nasal turbinate system using a nasal speculum or auroscope in large dogs. He also described the use of a warmed dental mirror with a good light source for examining the naso-pharynx after the soft palate had been pulled forward using Allis tissue forceps.

The introduction of the fibre optic endoscope led to the investigation of its usefulness in this area. MacEwen *et al* (1977) used an endoscope via the pharynx in dogs weighing approximately 10 kg or more. Smaller dogs were reported as being unsuitable as lack of space in the pharynx prevented manipulation of the instrument. Three of the twelve animals assessed had visible tumours and were all biopsied (using a special attachment) confirming malignancy.

Other authors also identified further limitations. Gibbs *et al* (1979) said that endoscopy of a nasal cavity obstructed by neoplastic tissue or containing excessive purulent, mucoid or haemorrhagic secretions was likely to be unrewarding.

O'Brien and Harvey,(1983) also stated that a 4mm diameter endoscope could only be used to examine the nasal

meatus and nasopharynx in large breed dogs and that the middle third of the nasal passage could not be examined directly.

In contrast White *et al* ((1984) described the use of a 4mm endoscope to examine the dorsal meatus and a distended naso-lacrimal canal in a Dachshund.

ANATOMY PHYSIOLOGY AND PATHOPHYSIOLOGY

The following descriptions are based on information obtained from the following veterinary texts: Hare, (1975); Bedford, (1979); Miller, (1979); Lane, (1982); O'Brien & Harvey, (1983).

The nasal cavity forms part of the respiratory tract. It is divided into two symetrical halves by the nasal septum. The septum is a sheet of cartilage which sits on the vomer bone caudally while rostrally it is only ossified at its junction with the palate.

Each chamber, or nasal fossa, is bounded by the septum medially, the cribriform plate caudally, the palate ventrally and the maxilla, frontal and lacrimal bones dorsally and laterally.

Within each fossa is a system of intricate turbinate scrolls which are highly vascular and result in a number of air-conducting passages. The dorsal nasal choncha (nasoturbinates) and ventral nasal choncha (maxilloturbinates) are located rostrally. The ethmoidal choncha (ethmoturbinates) arise from the cribriform plate and lie caudally. Two of these turbinates may extend into the frontal sinuses reducing the effective volume.

Where the caudal edge of the nasal and maxilloturbinates overlap with the rostral edge of the ethmoturbinates there may be an asymmetric area seen on dorsoventral radiographs even in normal dogs.

The paranasal sinuses; the frontal, maxillary and sphenoid, are in close association with the nasal cavity. The function of the paranasal sinuses is as yet unanswered. The maxillary sinus is not a true sinus, but a lateral diverticulum of the nasal chamber.

Each nasal chamber has its related frontal sinus which is subdivided by fine septa into three to five smaller sinus chambers which open via narrow ostia into the ethmoturbinate region. These openings may become obstructed when the turbinates are diseased or invaded by neoplastic tissue.This may then lead to the formation of a sinus retention mucocoele. The mucus is often sterile and should not be confused with primary sinusitis.

The olfactory function in the dog is highly developed. It is the deeper recesses of the ethmoturbinates that are lined by the olfactory neuroepithelium.

The structure of the nasal chambers and frontal sinuses are covered by a secretory lining which is highly vascular. The ciliated epithelium of the mucosa contains goblet cells; the stroma numerous mucous and serous glands. The mucus blanket that coats the ciliated epithelium traps the minute particles and bacteria brought in with the air inspired through the nostrils. The secretions are cleared by ciliary action to the external nares or the pharynx. Much of the fluid produced is lost by evaporation as the air is warmed and humidified almost to complete saturation 'en route' to the lungs.

If the flow is obstructed by any disease process, then a rapid build up of stagnant mucus can occur which is ideal for bacterial multiplication. Often some reverse flow will occur which then appears at the nostrils as a discharge.

The nasal mucosa is extremly sensitive. Two main responses occur when it is irritated. The first is a reflex sneezing which is very common especially in acute nasal disorders. This results from the attempts to dislodge the irritant by forceful air movements. Other signs such as snorting or coughing may also occur. Paroxysms of sneezing may result in epistaxis. The second common finding is an increase in production of mucus in an attempt to wash the irritant away. Sometimes there is an initial serous discharge which may progress to a mucoid discharge or, with a breakdown of cellular elements (leukocytes, histiocytes, epithelial cells and bacteria), to a frankly purulent discharge. If there is epithelial erosion and congestion of the mucous membranes the discharge may become blood-streaked or haemorrhagic. Since dogs are meticulous self-groomers and nose lickers, nasal discharge may not be noted by the owners for some time.

Nasal breathing is normal for the resting animal, but mouth breathing is necessary in a high environmental temperature or with physical activity.

The presence of discharge in the nasopharynx may produce gagging and coughing. Noisy breathing (snuffling sounds), may result from air passing through an obstructed nasal passage. If the obstruction is severe and bilateral

mouth breathing may occur.

The intimate association of the nasolacrimal canal with the nasal cavity should also be noted as it may become involved in nasal disorders with a resultant ocular discharge.

(Figs 1 & 2)

MATERIALS AND METHODS

All the clinical material used in this study was obtained from cases referred to the University of Glasgow Veterinary Hospital.

The investigation consisted of a review of the records of canine nasal neoplasia from August 1973 to September 1985 and the personal examination of clinical cases seen between October 1985 and May 1986.

SECTION I - RETROSPECTIVE STUDY.

The records of dogs diagnosed as having nasal neoplasia were examined with respect to:-

a) Breed

b) Sex

c) Age

d) Owners primary complaint

e) Duration and progression of clinical signs

f) Clinical signs on presentation

g) Radiographic features

h) Diagnosis and treatment (if any)

Any serial follow up radiographs were also examined.(In certain cases the clinical information was incomplete)

The radiographs were evaluated using the parameters described by Sullivan *et al* (1986). The intra-oral view was divided into rostral and caudal regions by a line drawn between the craniomedial roots of the carnassial teeth, and right and left regions by the vomer bone. The changes in radio-density were defined as predominantly increased radiolucency, increased radio-opacity or a mixed pattern when neither predominated.

An assessment was also made of turbinate loss or

masking, bone destruction or erosion, vomer destruction or septal deviation, and soft tissue swelling. Any other interesting or unusual features were also noted.

Additional information on views other than the intra-oral was considered and its usefulness ascertained. This included lateral, oblique, dorso-ventral and rostro-caudal views.

SECTION II - CLINICAL CASES.

The cases were referred to the Department of Surgery, University of Glasgow, by local practitioners during the period October 1985 to May 1986. At the initial examination the full history was recorded.

Emphasis was on the following:-

a) Initial presenting sign

b) Duration of signs

c) Nature of discharge and how it had altered - if relevent.

d) Discharge -
Unilateral
Bilateral
Varied

e) Epistaxis

f) Sneezing

g) Facial deformity

h) History of trauma

i) General clinical signs

A thorough general physical examination was then carried out followed by a more detailed study assessing the following:-

a) Oral cavity including dental arcade

b) Oropharynx

c) Facial deformity / soft tissue mass

d) Pain - frontal, nasal, maxillary bones,

nares

e) Softness of tissue indicating bone lysis

f) Epiphora (nature and distribution)

g) Lymph node enlargement

h) External nares - patency and ulceration (air flow checked with a wisp of cotton wool)

i) Nasal discharge
- unilateral or bilateral
- type of discharge

The animal was then anaesthetised using a cuffed endotracheal tube to minimise the risk of inhalation of any nasal discharge that may be present.

A more detailed examination of the oral cavity was then possible:-

a) Hard and soft palate - direct visualisation of deviations or masses as well as digital palpation

b) Dental crowns

c) Tonsils

d) Oropharynx

The nasopharynx was examined, where possible, by using a 4.8mm (Olympus BF - P10) flexible endoscope. The tip of the endoscope was introduced into the oropharynx and then retroverted. This allowed it to be hooked behind the soft palate to examine the nasopharynx and caudal conchae.

Rostral Rhinoscopy.

The endoscope was introduced into the middle/common nasal meatus via the external nares when the size of dog permitted. As much of the nasal cavity as possible was examined.

Ten normal animals were also examined in order to ensure that lesions could be accurately identified and

compared with the normal appearance of the nasal cavity.

Bacteriology.

When a nasal discharge was present nasal swabs were taken for bacterial isolation and sensitivity.

Serology.

If Aspergillosis was suspected blood samples were taken, allowed to clot, and spun down to remove the serum. The presence of antibodies in the serum was tested for by using the agar gel double diffusion test (Murray and Maghoub, 1968) and counter-immuno-electrophoresis (Richardson *et al* 1982).

Biopsy.

Under general anaesthesia samples were taken using one or more of the following techniques:-

a) Directly in the case of accessible lesions

b) At rhinotomy

c) Using the endoscope biopsy attachment

d) Using a non specific suction technique. A 50ml syringe was attached to a wide bore male dog catheter. The tip was inserted via the nares until resistence was met in the nasal cavity. Suction was applied followed by a sharp withdrawal action.

Radiography.

The radiographs were taken using a standard rotating anode diagnostic x-ray machine (Siemens Heliophos 45, 125 KV). The x-ray tube was a Mullard Guardian 125 - incorporating a light beam diaphragm controlling the field to be exposed. The machine was used on fine focus (0.6cm) with a film - focal distance of 92cms.

The exposure factors for all views except the intra-oral were 60 -77 KV and 10 - 25 mAs at 80 mA depending on the size of dog. With these exposures, cassettes with regular intensifying screens were used (Kodak X-omatic regular intensifying screens) and a standard film (Fugi; NIF New R-X). A moving Potter-Bucky grid with a ratio of 10:1 was also used to enhance radiographic contrast.

For the intra-oral views non-screen film (Medical x-ray film 3M Type S: 13cm x 18cm) without a grid was used to give improved detail. In this case the exposures were 70 - 77 KV and 32 mAs at 80 mA. A lead plate was used beneath the film to prevent back - scatter.

The use of grids is not essential when taking radiographs of the nasal chambers except in larger dogs for the rostro-caudal view. However this technique has been found to give radiographs a good contrast and definition in the past and so was continued in order to obtain meaningful comparisons of new or follow-up cases.

Routinely radiographs were taken in lateral and intra-oral positions; dorso-ventral and rostro-caudal views were used where indicated.

Occasionally lateral thoracic radiographs were taken if neoplasia was suspected, even though metastases are rare.

Radiographs were labelled with a left or right indicator, the hospital, date, name of owner and patients case number.

All dogs radiographed were anaesthetised so that whenever possible positioning was done entirely with the use of sand bags, ties or foam supports. If assistance from personnel was needed they were protected by lead aprons and lead gloves. All people not directly involved left the room prior to the exposure being made.

An automatic processor (Kodak RP X-OMAT Model 101) was used. This took 110 seconds to process and dry each film with the exception of the intra-oral films which needed additional drying once machine processing had finished.

The films were examined with a standard x-ray viewer. Where a film appeared dark or required a closer examination a spot light was used.

Description of Radiographic Positioning.

The canine skull can cause problems both in radiography and in radiological interpretation. This is further exacerbated by the variation in anatomy between different breeds and even within breeds. It is essential that the radiographs are of good quality with respect to both exposure factors and positioning.

Radiographs showing asymmetry or tilting are not considered acceptable for interpretation. As previously stated all animals were anaesthetised.

i. Dorso-ventral whole skull

The animal was placed in sternal recumbency and supported by sand bags or foam bolsters. The head and neck extended. The sagittal plane (which divides the skull into two symetrical halves) must be vertical, in line with the central ray and at right angles to the film.

In the anaesthetised animal the endotracheal tube was temporarily removed.The beam was centred on the midline between the eyes.

ii. Dorso-ventral intra-oral (Fig.3)

The animal was placed in sternal recumbency. A lead plate was placed under the head to absorb radiation and prevent back scatter. The intra-oral film was placed diagonally into the mouth with one corner as far into the pharynx as possible ensuring it lay horizontally and symetrically. The sagittal plane should be vertically in line with the central ray and at right angles to the film.

The beam was centred between the inner canthus of the eyes in the mid-line.

iii. Lateral (Fig. 4)

The animal was placed in a lateral recumbency with the nasal chambers to be investigated closest to the film. A gag was placed between the canine teeth as this was found to assist in positioning.

Pads were placed under the neck or under the rostral part of the mandible where necessary.

The sagittal plane should be parallel to the film. The beam was centred to the middle of the nasal chambers.

iv. Rostro-caudal

This view was used to highlight the frontal sinuses.The animal was placed in dorsal recumbency supported by sand bags or foam bolsters with the forelegs drawn backwards.

The neck was flexed until the head was at right angles to the film. A tape placed around the bridge of the nose and pulled caudally aided positioning.

The sagittal plane was vertical, in line with the main beam, and centred between the inner canthi of the eyes in the midline.

v. Positive-contrast rhinography (Fig. 5 & 6)

This technique was carried out on anaesthetised dogs with a cuffed endotracheal tube in place.

The patient was placed in lateral recumbancy with the structures for investigation on the dependent side and the nares slightly elevated. Based on the findings of Goring *et al*, (1984 a,b) contrast medium was used at a rate of 1ml per 5kg body weight. A short plastic catheter (either a tom cat catheter or a shortened dog urethral catheter) was inserted along the dorsal/middle meatus caudally until resistance was felt. Two-thirds of the total contrast was administered from an attached syringe. The catheter was then withdrawn and the remainer instilled into the rostral turbinate region.

A dampened cotton wool plug was inserted into the nares to reduce contrast leakage. The animals head was gently rotated from side to side several times to aid constrast dispersal.

Intra-oral and lateral views were taken routinely. Additional views were only used were indicated.

The use of two contrast media was investigated.

a) Sodium metrizoate solution (Triosil, Nyegaard).

b) Barium sulphate suspension (Baritop 100 suspension) at either 30 per cent or 60 per cent concentrations.

During recovery from anaesthesia the pharyngeal area was inspected and any residual contrast removed. Excess contrast could be removed from the nasal cavity via the nares by gravity drainage.

Endoscopy.

The fibreoptic endoscope provided a reliable means of non-invasive exploration of the nasal chambers of most

dogs with direct visualization of the nasal and maxillo-turbinates and in larger dogs the ethmo-turbinates. In small dogs the external diameter (4.8mm) of the endoscope was too large to allow introduction into the nares although in all dogs examination of the oropharynx and nasopharynx was possible.

The endoscope used was an Olympus BF - P10 with the following specifications:-

Insertion tube outer diameter	-	5mm
Total length	-	760mm
Working length	-	550mm
Depth of field	-	3-50mm
Field of view	-	90°
Range of tip bending	-	180° up 100° down

Photographs were taken with an Olympus OM1 camera and an OM Xenon adaptor. (Fig. 5)

Surgical Technique.

After premedication with acetylpromazine general anaesthesia was achieved by induction with thiopentone sodium and maintenance with an oxygen, nitrous oxide, halothane mixture.

i. Rhinotomy.

The pharynx was packed with swabs to prevent blood from entering the lower airway. The animal was placed in sternal recumbency and the operation site routinely prepared.

A mid-line skin incision was made from the most rostral extent of the nasal bones caudally to the level of the medial canthus. The skin was retracted to the side indicated for rhinotomy. The periosteum was elevated and reflected.

A window was then cut in the nasal bone starting at the mid-line using an osteotome and hammer. The extent of the window was dependant on the extent of the lesion and

size of the dog. An average window was 8-10cm long and 1cm wide.

The bone flap was then discarded. The opposite side could be approached through the nasal septum. The turbinates were then removed by blunt dissection using a curette. Samples for histopathology were taken at the outset. It was essential that all the abnormal turbinates were removed or in the case of neoplasia all the tissue in the chamber excised. The septum was carefully examined for evidence of invasion. If the septum was damaged the contralateral side was also curetted.

Haemorrhage was controlled by swabbing. Packing of the nasal cavity was not found to be necessary. The wound was closed using absorbable suture material in the periosteum and subcutaneous tissue and non-absorbant sutures in the skin. Post operative analgesia was provided where necessary. Broad spectrum antibiotics were administered for ten days and then the skin sutures were removed. Some nasal discharge was anticipated for several days. Post operative recovery was normally rapid and uneventful.

ii. Frontal sinus and nasal cavity irrigation.

The animal was prepared as described for a rhinotomy, but the incision was made over the affected frontal sinus. A trephine hole was made using a 6.5mm drill at a site midway between the mid-line and the supraorbital process. The connection with the nasal cavity was bluntly enlarged to allow the passage of a flexible polythene drainage tube to the level of the nasal conchae.

The tube was attached to the skin caudal to the incision to facilitate flushing and the wound closed routinely. Once irrigation was completed the tube was removed and the small wound healed by granulation.

SECTION I - RETROSPECTIVE STUDY.

Breed incidence

Comparative figures were used to highlight the fact that although a certain breed may be seen more commonly in a review series this could simply imply that this breed is more prevalent and so, statistically, more likely to be represented. It does not necessarily mean that there is a breed predilection.

It should be noted that the referral population was reviewed over seven years, but the study over twelve years, so an accurate comparison can not be made.

A wide variety of breeds were recorded (Table 3). Mixed breeds predominated comprising 25 per cent of the total. This was significantly higher than the referral population would have predicted. Labrador Retrievers, German Shepherd Dogs and Rough Collies were the most frequent pure bred dogs seen. Of the breeds comprising 5 per cent or more of the affected dogs, a number occurred more frequently than the referral population expectation, namely the Rough Collie, Border Collie, Irish Setter, Shetland Sheep Dog and West Highland White Terrier.

The majority belonged to the medium to large weight range although a noteable number, 13 per cent (8/60), were small and toy breeds. An unusual finding was the inclusion of three giant breeds; two Great Danes and one Deerhound.

Mesaticephalic skull types were the commonest with only 16 per cent (10/60) being dolicocephalic. One brachycephalic, a boxer, was also featured.

Age incidence

The ages of affected dogs ranged from 16 months to 14 years (Fig 6). The median was 8 years and the mean 9.2 years.

Although middle-age to older dogs predominated 10 per cent (6/60) were five years or under.

Sex incidence

Thirty three males compared with twenty seven females were recorded. Approximately half of the females were

neutered. This resulted in a male to female ratio of 1.2:1.

Breed	Number	Percent.	% in Ref. Popn.*
Cross-bred	15	25	12
Labrador retriever	6	10	11.4
German Shepherd Dog	5	8.3	11.2
Rough Collie	4	6.6	2.6
Border Collie	3	5	2.7
Irish Setter	3	5	1.6
Shetland Sheepdog	3	5	2.5
West Highland White Terrier	3	5	0.3
Afghan Hound	2	3.3	0.6
Doberman Pinscher	2	3.3	1.8
Great Dane	2	3.3	0.9
Golden Retriever	2	3.3	4
Boxer	1	1.6	3
Cairn Terrier	1	1.6	1.9
Cavalier King Charles Spaniel	1	1.6	1.7
Deerhound	1	1.6	0.2
Keeshund	1	1.6	0.1
Old English Sheep Dog	1	1.6	1.3
Poodle	1	1.6	2.7
Scottish Terrier	1	1.6	0.7
Whippet	1	1.6	0.4
Yorkshire Terrier	1	1.6	2.3

* The referal population was taken from the total number of dogs seen at Glasgow University Veterinary Hospital between 1978 and 1984 inclusive. The total number was 15,108 and the number of times each breed was seen is expressed as a percentage of this figure.

TABLE 3. The breed incidence of cases of nasal neoplasia compared to the breed incidence of the referral population.

Duration of clinical signs

The duration of clinical signs preceding a diagnosis of neoplasia varied from one week to twenty four months (Fig 7).

The median was two months and the mean 2.8 months. With the exception of three cases all the animals had a clinical history of seven months or less. The duration of signs in the remainder were 11, 12 and 24 months.

Tumour type

Table 4 shows the histopathological diagnosis of the thirty one nasal tumours for which reports were available. Of these five of the carcinomas were not fully classified. Seven specific neoplasms were identified. The tumours can be more simply grouped into nineteen carcinomas, ten sarcomas and two undifferentiated types.

Histopathological Diagnosis	
Adenocarcinoma	11
Carcinoma	5
Chondrosarcoma	5
Osteosarcoma	3
Respiratory epithelial carcinoma	2
Squamous cell carcinoma	1
Fibrosarcoma	1
Plasma cell sarcoma	1
Poorly differentiated tumour	2
TOTAL	31

TABLE 4. The histopathological diagnosis of thirty one cases of nasal neoplasia.

Radiological features

The results are recorded in Appendix II. These were then collated into small tables to simplify their evaluation.

Considering the cases showing clinical signs of six weeks or less 36 per cent showed unilateral radiographic changes, but 64 per cent showed bilateral changes.

One bilateral case had a history of only two weeks duration (Fig 8). In contrast one case (Fig 9) with an

Table 5 shows that seventeen cases had unilateral clinical signs, but bilateral radiographic changes. Four had bilateral clinical signs, but unilateral radiographic changes.

Clinical signs	Radiological changes	No. of cases
Bilateral	Unilateral	4
Bilateral	Bilateral	21
Unilateral	Bilateral	17
Unilateral	Unilateral	18

TABLE 5. A comparison of the clinical signs and radiological changes in sixty cases of nasal neoplasia.

In this study each side was considered seperately. Therefore of 60 cases, there were 120 sides of which 97 showed radiological evidence of change. Each radiological feature was considered in turn.

Turbinate loss referred to destruction, not masking of the fine trabecular pattern (Fig 9). Table 5 showed that turbinate loss was evident in 96 per cent (95/97) of the affected sides. Of these only three sides showed the more subtle focal destruction. Seventy five per cent showed destructive changes both caudally and rostrally.

Turbinate loss	No. of sides affected
Rostral only	11
Caudal only	10
Rostral and Caudal	74

TABLE 6. The distribution of radiological changes with reference to turbinate loss in sixty cases of nasal neoplasia.

Tables 7 to 9 show the alterations in nasal cavity radiodensity recorded.

From table 7 it can be seen that 88 per cent (85/97) of the affected sides showed an increase in opacification. (Fig. 9). Of these 62 per cent showed changes both rostrally and caudally. Opacification was also seen in association with an increase in lucency or mixed pattern on the same side. Considering the 60 cases, increase in opacification was a feature in 90 per cent (54/60)

Increased opacity	No. of sides affected
Rostral only	7
Caudal only	8
Rostral and Caudal	60
Combined with mixed density pattern	8
Combined with increased radiolucency	2

TABLE 7. The distribution of radiological changes with reference to increased opacity in sixty cases of nasal neoplasia.

Mixed density was recorded when neither an increase in opacity or lucency predominated, but both were present.

A mixed pattern was evident in 19 sides (Table 8), however there were only five cases showing a mixed density alone. In the remaining cases it was associated with opacification on the same or contralateral side. Of these five cases, difficulties in diagnosis could arise in Case numbers 20, 22, 38 and 57 (Figs. 10,11,12 & 13).

Mixed density	No. of sides affected
Rostral only	2
Caudal only	4
Rostral and Caudal	5
Combined with increased opacity	8

TABLE 8. The distribution of radiological changes with reference to mixed density in sixty cases of nasal neoplasia.

Radiolucency was an uncommon finding (Table 9). It occurred in Case numbers 25 and 48. In both cases opacification was also a feature, however the latter case did resemble destructive rhinitis (Fig 14)

Increased radiolucency	No. of sides affected
Rostral only	1
Caudal only	0
Rostral and Caudal	0
Combined with increased opacity	2

TABLE 9. The distribution of radiological changes with reference to increased radiolucency in sixty cases of nasal neoplasia.

Punctate lucencies were defined as small pinhead areas of radiolucency within an area of relative opacity. (Fig 8). Plate destruction was recorded where there was an obvious loss in the radio-dense shadow of the compacta of the facial surface of the splanchnocranium medial to the alveolus (Schebitz and Willkins, 1968)(Figs. 8,15 & 17)

Case destruction was recorded where there was a defined area of radiolucency indicating destruction of any of the bones forming the nasal cavity namely the palatine bone, nasal bone, maxilla and/or frontal bone (Figs. 8,15 & 17) (Table 10)

Intra-oral radiograph	No. of sides affected
Punctate lucencies	20
Plate destruction	34
Case destruction	21

TABLE 10. The distribution of destructive features other than those affecting the turbinates.

There was evidence of septal/vomer involvement in 60 per cent (36/60) of the radiographs (Table 11).

When assessing alterations in the midline opacity this was a feature noted normally in combination with other abnormalities. Thinning was a subjective view, but after studying sufficient numbers of radiographs an impression was gained of the uniformity of density of the vomer/septum. A decrease in density centrally or caudally could sometimes be appreciated without complete absence. However for these results definitive changes only were included. Absence and deviation was also seen in combination (Figs. 16 & 17).

Septal / Vomer bone changes	No. of cases affected
Loss of mid-line opacity	26
Deviation	11
Thinning	7

TABLE 11. The alteration in the mid-line opacity referred to as the nasal septum or vomer bone.

Table 12 shows the findings of the 46 lateral radiographs taken. Of these six showed no abnormalities at all. Lateral views were not taken in every case, because changes were either not anticipated or expected to add to the information already gained from the intra-oral views. Turbinate loss was difficult to fully evaluate due to the superimposition of the two cavities. In fifteen cases bone destruction was seen in association with soft tissue swelling (Fig 18a), but in only six of these had there been evidence of case destruction on the intra-oral view. Frontal sinus opacification was common (Fig 18). It could also be appreciated to some extent on the intra-oral view (Fig 15).

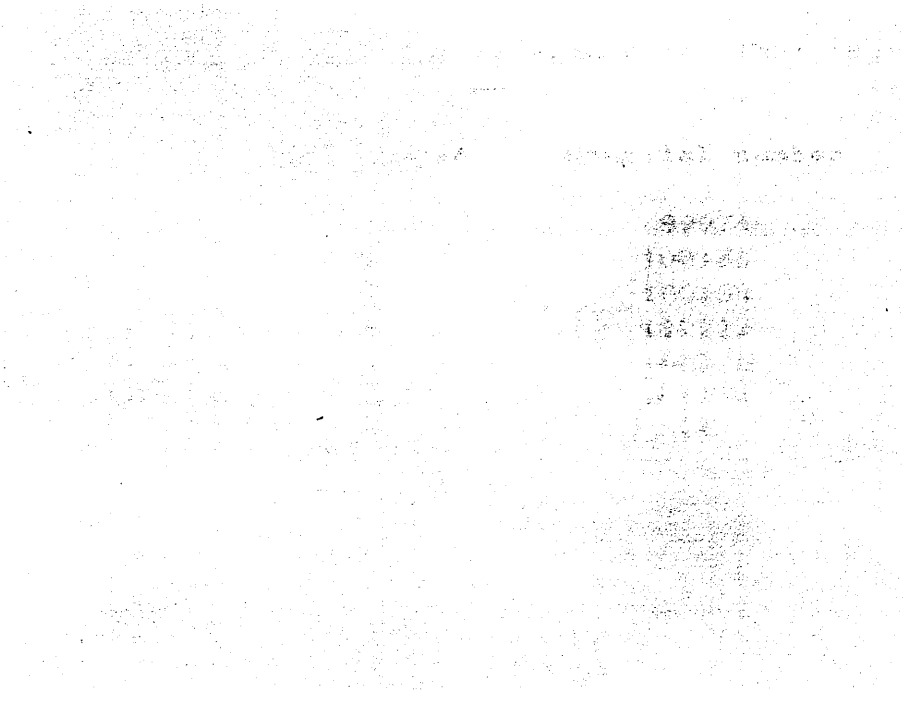
Lateral radiograph	No. of cases affected
Turbinate loss	37
Bone destruction	18
Frontal sinus opacification	38
Extra-nasal soft tissue swelling	18

TABLE 12. The radiological feature recorded on the lateral radiographs.

Mineralisation was seen in seven cases. It varied, either being obviously apparent on both the intra-oral and lateral views (Figs 20 & 21), or more subtle and focal (Fig 22). Of those which were classified, two were chondrosarcomas, two adenocarcinomas and one an osteosarcoma.

The rostro-caudal view to skyline the frontal sinuses was not routinely used in this series. When the possibility of aspergillosis arose, or if there was obvious involvement in that region then it was utilised.

Twenty-two thoracic radiographs were taken to look for possible metastatic spread. No changes were evident.



SECTION II - CLINICAL CASES

This prospective study is presented as a series of twenty clinical cases seen at The University of Glasgow Veterinary Schoolduring the period October 1985 to May 1986. The reports take the form of a synopsis of the investigation undertaken with the results and diagnosis where appropriate.

The cases seen were -

Neoplasia	8 cases
Chronic rhinitis	3 cases
Aspergillosis	2 cases
Rhinarial ulceration	2 cases
Foreign body	1 case
Hard palate defect	1 case
Periapical abscess	1 case
Autoimmune dermatosis	1 case
Undiagnosed	1 case
TOTAL	20

The numbers used for reference in the text are as follows

Text number	Hospital number
1	99974
2	100136
3	100184
4	100227
5	100270
6	100379
7	100442
8	100452
9	100915
10	71414
11	101202
12	101258
13	101445
14	101636
15	101812
16	101993
17	102005
18	102122
19	102119
20	101608

i.

Case number 99974

Diagnosis: Rhinarial Ulceration

Border Collie 7 years Male 27.5kg

History

Three month duration, sudden onset sneezing and nasal irritation. Temporary response to antibiotics. Radiographically unremarkable. More recent nasal discomfort and left haemorrhagic nasal discharge.

Clinical Examination (Fig. 23)

Slight sero-sanginous discharge left nostril. Rhinarial ulceration on the inner lateral and dorsal wall of the left nares.

Endoscopic investigation

Left nares: One centimeter diameter fleshy proliferative lesion with a white margin and a central necrotic plaque. Partial destruction of alar fold at bulbous enlargement allowed easy visualisation of the base of the normal nasal turbinates.

Right nares: NAD

Radiographic investigation

Lateral skull and intra-oral nasal cavity. Loss of soft tissue density at left alar fold resulting in a direct passage from the external opening to the nasal turbinates. Diagnosis: Left nares erosion.

Histopathology

Inflammatory tissue

Outcome

Re-examination four weeks later. Left sided lesion appeared less inflamed, but a small eroding area of ulceration had begun in the identical position on the right nares. Mild bilateral blood tinged mucoid discharge.

Treatment

Three weeks antibiotics. Three to four weeks prednisolone. Decreasing dose according to response.

Outcome

Lost to follow up.

2.

Case number 100136

Diagnosis: Nasal Neoplasia

Poodle 9 years Male 9kg

History

Four month duration intermittent right ocular and nasal discharge. Nasal discharge white and watery. Temporary response to antibiotics. One week duration left mucopurulent nasal discharge, occasionally blood tinged. Increasing snorting and gagging. Nasal irritation. Intermittent pain over head and neck. Reduced hearing 2-3 weeks.

Clinical Examination

No air flow through either nostril. Intra orally, thick mucoid discharge in the nasopharynx and pharyngeal region. On digital palpation there was a loss of the bony structure forming the lateral boundaries of the caudal nares. A small area of ulceration was present medial to the second upper molar on the left hand side.

Endoscopic Investigation

Right: Normal nasal turbinates covered in a thick mucoid discharge. The endoscope was blocked at the start of the ethmoturbinates.

Left: View obscured by discharge.

Radiographic Investigation (figs. 24 & 25)

Intra-oral nasalcavity, lateral skull, dorso-ventral skull and rostro-caudal skull.

Intra-oral nasal cavity: Masking of turbinate detail rostrally caudally at level of PM4; turbinate destruction particularly right and septal thinning; complete destruction of vomer caudally; overall increased opacity involving frontal sinuses; defined areas of radiolucency - case destruction.

Dorso-ventral skull: Bilateral destruction of much of the bony architecture relating to the front of the cranial cavity and frontal sinuses; destruction of the medial walls of the orbit, pterygoid processes and bones surrounding the frontal sinuses; soft tissue mass on the right side at the level of the destroyed frontal sinus.

Lateral skull: Destruction of the frontal sinus ventrally and irregularity of the cranial bones caudal to the orbit; soft tissue mass occupying the orbital region.

Rostro-caudal skull: Frontal sinuses infilled by a

granular soft tissue opacity throughout; overlying frontal bone appears normal.

Diagnosis: Nasal neoplasia.

Outcome

Euthanasia five months later without re-examination.

Pathology Report (Fig.26)

Extensive tumour infiltration filling the nasal cavity and extending into the pharyngeal area. Thinning of the bone overlying the nasal cavity and destruction of the cranial cavity leaving a thin shell of fibrous tissue only. Possible local invasion and infiltration into the cranium.

Histopathology

Nasal adenocarcinoma with extension into the frontal sinuses and through the cribiform plate into the brain.

3.

Case number 100184

Diagnosis: Nasal neoplasia

Collie x 11 years Male 32 kg

History

Intermittent sneezing and slight nasal discharge; duration unknown.
Six weeks ago exophthalmus of left eye noted which had progressed.

Clinical Examination

Thick purulent left nasal discharge. No air flow. Periorbital soft tissue mass displacing eye. Palpable bony destruction of orbit and zygomatic bone. Facial swelling around orbit, predominantly around medial canthus and frontal sinus. No marked pain.

Endoscopic investigation

Left nasal cavity only affected. The endoscope passed easily to the level of the medial canthus. Detailed visualisation of turbinate structure obscured by thick discharge, but disruption was evident particularly caudally.

Radiographic investigation

Lateral skull, intra-oral nasal cavity, dorso-ventral skull, rostro-caudal skull, lateral thorax and rhinography.

Intra-oral nasal cavity: Overall increase in soft tissue density on the left side both rostrally and caudally; marked turbinate destruction from PM3 caudally; maxillary plate destruction PM2 caudally. A more pronounced defined area of radiolucency at the level of PM3/4 indicated case destruction; vomer absent caudally with soft tissue density infilling bilaterally with turbinate loss. No other abnormalities present in the right nasal cavity. Loss of bone forming the medial side of the left orbit and soft tissue swelling laterally.

Rhinography: Contrast outlined the area of radiolucency (level with PM4) and passed lateral to the dental arcade confirming a bony defect of the nasal/maxillary bone; septal defect at M1 allowed contrast to pass to the right side; defect not apparent on the plain film. Circumscribed soft tissue filling defect outlined caudally suggesting a mass lesion.

Dorso-ventral skull: Further bony destruction on the

left side identified including destruction of medial wall of frontal sinus, orbit and rostral caudal vault; the rostral portion of the zygomatic arch almost completely lysed.

Lateral skull: Destruction of the frontal and nasal bone with some periosteal reaction and soft tissue swelling; frontal sinus radiolucency replaced with a soft tissue density which appears irregular and mottled; irregularity of the bones forming the cranial cavity dorsal to the coronoid process.

Rostro-caudal (skyline) skull: Left frontal sinus replaced with a soft tissue density extending outwards beyond the normal contour; right frontal sinus replaced with a soft tissue density; mottled areas of lucency; left frontal bone completely destroyed and right partially destroyed.

Lateral thorax: NAD.

Diagnosis: Nasal neoplasia

Microbiology

Beta-toxin producing staphylococci (pathogenic) and streptococci.

Outcome

Euthanasia

Pathology Report

The tumour originated from the posterior aspect of the ethmoturbinates. It had spread caudally into the orbit displacing the eye and caused considerable destruction to the zygomatic arch and surrounding bony structures. It had also caused some pressure and bone thinning of the cranial cavity.

The mass was fairly well circumscribed and very firm, almost cartilagenous on cross section.

Histopathology

Poorly differentiated chondrosarcoma.

4.

Case number 100227

Diagnosis: Idiopathic rhinarial ulceration

Great Dane 7 years Male 60 kg

History

Intermittent low grade nasal discharge for 18 months. Initially unilateral, now bilateral. Occasional sneezing. Responds to antibiotics then recurs

Clinical Examination

Slight mucoid crusting around right nares. Moderate to severe rhinarial ulceration with longitudinal cracks at the mucocutaneous junction around both nostrils.

Endoscopic investigation

Slight mucoid discharge was present in the right nasal cavity just caudal to the external nares. Caudally right turbinates appeared normal. Turbinates on left normal.

Radiographic investigation

Intra-oral nasal cavity and Lateral skull.

The rostral turbinates had a granular mineralised appearance thought to be a normal variation in older Great Danes. No other abnormalities detected.

Treatment

Three week course of antibiotics. Three week course decreasing dose prednisolone.

Advised on management, and on use of sunblock creams

Etiology

Unknown. No other indications of an auto-immune component. May recur.

5.

Case number 100270

Diagnosis: Autoimmune dermatosis

Wheaten Terrier 8 years N.Female 15 kg

History

Poor history. Possibly bitten on nose ten days prior to presentation. Infection unresponsive to antibiotics

Clinical Examination

Dull, reduced appetite. Painful stifle. Temperature: 105°. Painful, scabby lesion on bridge of nose progressively worsening despite antibiotics. Overlying tissue sloughed leaving a deep ulcerated lesion and dog developed bilateral mucopurulent nasal discharge blocking both nostrils.

Radiographic investigation

Intra-oral nasal cavity and lateral skull.

Intra-oral nasal cavity: Destruction of the trabecular detail evident; septum appears intact; an area approximately 1 cm diameter of increased radiolucency adjacent to the left canine and PM3 spanning the midline, suggestive of overlying tissue destruction.

Lateral skull: Deep soft tissue defect over the dorsal aspect of the nasal bone, possibly involving it; no loss of nasal bone turbinate pattern or increased soft tissue density; slight frontal sinus opacification.

Diagnosis: Soft tissue erosion over nasal bone

Microbiology

Profuse mixed growth of bacteria. No mycosis.

Treatment

The lesion was debrided and cleansed with pevidine. Areas of erosion through the nasal bone into the nasal cavity had occurred. The resultant defect 3cm x 4 cm was left to granulate. Antibiotic medication and topical treatment given.

Outcome

The dog made good progress initially but then regressed. Diagnosis on serology was an auto-immune dermatosis and the dog was euthanased.

6.

Case number 100379

Diagnosis: Chronic Distemper Rhinitis

G.S.D 5 years Female 20 kg

History

Mild ocular and nasal discharge progressing to thick mucopurulent discharge, four weeks duration. Sudden onset neurolgical abnormalities: staggering, circling, head tilt and inco-ordination one week later.

Clinical Examination

Recumbant, mouth breathing. Intermittent extensor rigidity of forelimbs with paddling. Brown mucopurulent discharge blocking both nostrils.

Radiographic investigation (Fig.27)

Intra-oral nasal cavity: Right dependent side, an overall increase in opacity rostrally and a mixed pattern caudally. No evidence of frontal sinus opacity. Appearance consistent with a rhinitis with unilateral changes caused by the dogs recumbancy.

Treatment

Euthanasia

Histopathology

Distemper encephalitis resulting from a recent infection.

7.

Case Number 100442

Shetland Sheepdog 10 years Male 27.5 kg

Diagnosis: Chronic rhinitis.

History

Intermittent left nasal discharge for ten months with periods of remission of up to two months. Sneezes mucus when excited. Nasal neoplasia diagnosed radiologically by referring veterinary surgeon when first seen.

Clinical Examination

No visible discharge. No bony or soft tissue distortion. No air flow from either nostril, tendancy to mouth breath.

Endoscopic investigation

Right: No discharge visible and normal rostral turbinates. Endoscope could not pass beyond rostral ethmoturbinates.

Left: Endoscope passed easily to caudal aspect of the ethmoturbinates. Visualisation poor. No normal turbinates. No obvious discharge. Suggestive of increased soft tissue, but no solid mass appreciated.

Radiographic investigation

Lateral skull and Intra-oral nasal cavity (Fig 28). Repeated three months later. No evidence of progression.

Intra-oral nasal cavity: Unilateral increase soft tissue opacity with turbinate destruction rostrally and caudally; no evidence of plate or case destruction; slight irregularity and thining of nasal septum (PM3 level); frontal sinus opacification

Lateral skull: NAD

Radiological diagnosis: Neoplasia

Microbiology

A mixed growth, nothing significant.

Treatment

Left rhinotomy and turbinectomy was performed. The cavity was comprised of diffuse oedematous soft tissue and remnants of turbinates which were removed by currettage. A right turbinectomy was also performed as a small septal defect was present. The right nasal turbinates appeared normal.

The dog was discharged on a ten day course of antibiotics.

Outcome

The dog made an uneventful recovery. Six weeks later the dog was euthanased for an unrelated cause.

Clinical examination prior to euthanasia. Bilateral airflow, but snuffly breathing and a thick mucopurulent discharge was present.

Endoscopic investigation

Bilaterally rostrally no turbinates present. Caudally, particularly on the right, remnants of ethmoturbinates visible indicating that the turbinectomy had not been complete. No evidence of a soft tissue mass, but a copious discharge was present.

Radiographic investigation

Intra-oral nasal cavity: Increased radiolucency on the left rostrally and bilaterally caudally although some ethmoturbinate structure visible on the right; most of the midline opacity absent; increased opacity with punctate lucencies on the right rostrally - may represent discharge on dependent side.

Histopathology

Biopsy sample from rhinotomy; oedema of the connective tissue and cystic hyperplasia of the mucosal glands with infiltration by neutrophils. There was no evidence of tumour cells. The changes were consistent with chronic inflammation.

8.

Case number 100452

Diagnosis: Neoplasia

Shetland Sheepdog 10 years Male 6.5 kg

History

Four weeks ago dog developed right purulent nasal discharge. Progressed to coughing, snoring, snorting and mouth breathing. One week ago discharge became blood tinged, nasal swelling developed and dog became dull.

Clinical Examination

Depressed. Thin. Right-sided haemorrhagic nasal discharge. Ventral depression of hard palate and discharging sinus medial to the right carnassial

Endoscopic investigation

It was not possible to introduce the endoscope through the small nares.

Radiographic investigation

Intra-oral nasal cavity, lateral skull and rhinography.

Intra-oral nasal cavity:

Right: Overall increase in soft tissue density rostrally and centrally; caudally trabecular pattern still present; area of relative radiolucency around the medial root of the right carnassial with loss of the lamina dura; possible case destruction; septum appearred thin rostrally.

Rhinography: No evidence of septal erosion, contrast present overlying the medial root of the carnassial suggesting a bony defect (a defect in the hard palate confirmed on the lateral view). Large filling defect around the carnassial suggests a soft tissue mass.

Left: NAD

Lateral skull and rhinography: Filling defect outlined above the carnassial. Normal surrounding turbinate structures outlined. Granular soft tissue density present ventral to the hard palate.

Diagnosis: Nasal neoplasia

Outcome

Euthanasia

Histopathology

Malignant melanoma. The origin of the growth was not ascertained. (Fig 29).

9.

Case number 100915

Diagnosis: Nasal neoplasia

Rough Collie 12 years Female 21 kg

History

Six months ago dog became head shy, cried out if touched. That regressed as a small swelling appeared over the right frontal sinus which progressively enlarged causing epiphora and proptosis. Occasional snorting with serous discharge. No pronounced nasal discharge or sneezing.

Clinical Examination

Irregular prominent soft tissue swelling over the frontal sinus region, predominantly on right side. On palpation bone destruction was appreciated. Clear right-sided epiphora. Mild proptosis and partial protusion of the nictitating membrane. Air flow normal.

Endoscopic investigation

Left: Normal maxilloturbinates. No discharge. Endoscope stopped at rostral ethmoturbinates.

Right: Normal ventral turbinates caudally. Endoscope passed to the level of the inner canthus. Ethmoturbinates poorly defined and difficult to visualise due to haemorrhage.

Radiographic investigation

Lateral and Dorso-ventral skull (Intra-oral nasal cavity should have been included)

Dorso ventral skull:

Right: Increase in soft tissue density with turbinate destruction; central area of lucency indicating case destruction; caudally septal destruction and frontal sinus opacity; lysis of the medial wall of the orbit and replacement with an opacity and irregular areas of mineralised periosted reaction.

Left: NAD

Lateral skull: Marked soft tissue swelling over the dorsal aspect of the frontal sinus; destruction of the frontal bones and dorsal ethmoturbinates; spicules of calcified periosteal reaction passing into the soft tissue mass.

Diagnosis: Frontal sinus tumour

Treatment

Wedge biopsy over swelling. There was bony

destruction allowing direct access into the frontal sinus through the biopsy site.

Histopathology

Nasal carcinoma arising from respiratory epitheliam

Outcome

Advised euthanasia when the dog's condition deteriorated.

10.

Case number 71414

Diagnosis: Unconfirmed chronic hyperplastic rhinitis

West Highland White Terrier 8 years Female 9 kg

History

Six months ago developed tracheitis with sneezing and snuffling. Cough cleared, but sneezing with mucus approximately five times daily continued. Responded to antibiotics then recurred. No nasal discharge between sneezes

Clinical Examination

Very moist nose. No other pertinent findings.

Endoscopic investigation

Endoscope introduced via nares with difficulty. Normal turbinates covered with glistening clear mucus. Attemps to take a biopsy unsuccessful due to lack of manouverability within the small nasal cavity

Radiographic investigation

Intra-oral nasal cavity, lateral skull and rhinography

Intra-oral nasal cavity: No evidence of turbinate destruction or over all increase in opacification; the rostral turbinates reduced clarity; caudally the trabecular pattern asymmetric with irregular areas of turbinate masking.

Rhinography. 2ml of sodium metrizoate solution ("Triosil") introduced into each nostril seperately. Poor penetration of the contrast through the nasal turbinates which appeared thicker than normal particularly on the right. Caudally the contrast produced of flocular appearance and did not outline the ethmoturbinates.

Lateral skull: NAD.

Diagnosis: Hyperplastic rhinitis

Microbiology

A Beta-haemolytic streptococcus and a possible actinomycete isolated.

Treatment

Four weeks potentiated sulphonanides and bisolvin

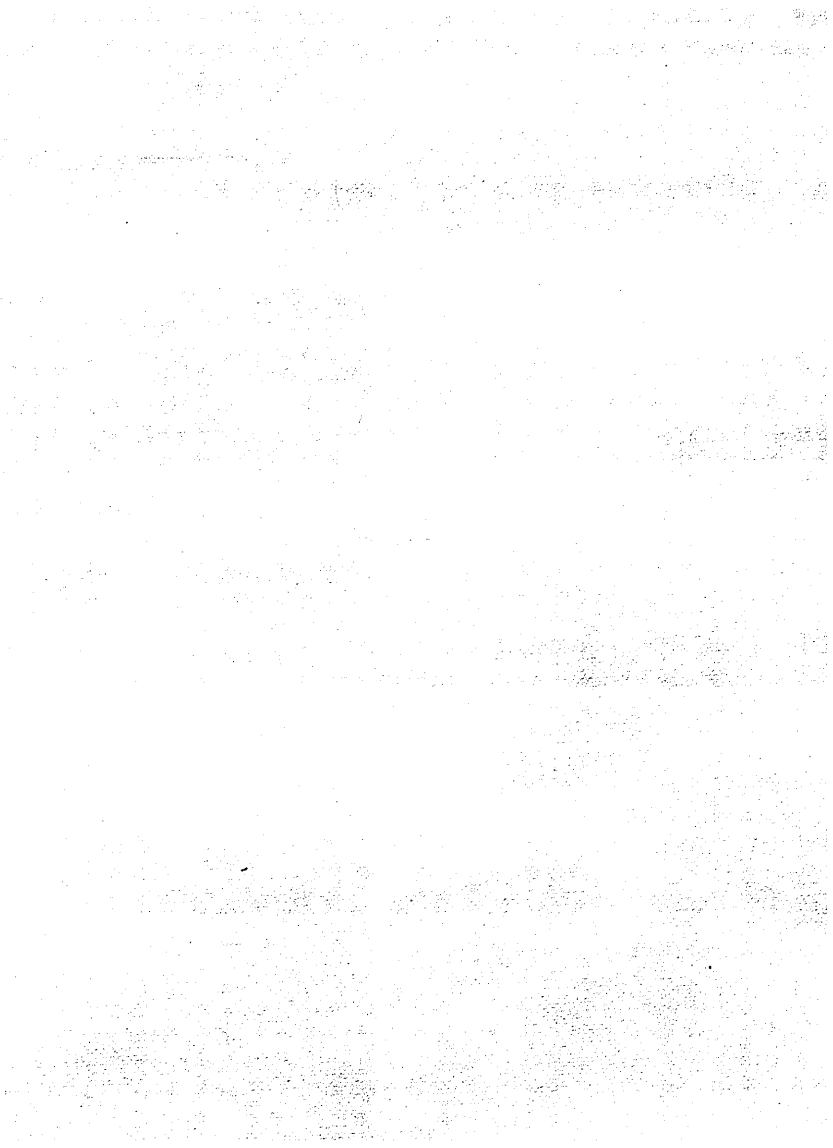
Outcome

Re-presented six weeks later. Responded well to

treatment. Recurrence of sneezing etc 2-3 days after the treatment had stopped.

Repeat endoscopy and radiography showed no change

Advised use antibiotics intermittently if discharge became mucopurulent. Continue bisolvin. Rhinotomy only if condition deteriorates.



11.

Case number 101202

Diagnosis: Nasal neoplasia

Labrador 7 years Male 36.5 kg

History

Six months of right nasal irritation. Sudden onset right nasal epistaxis two months ago with a small swelling medial to the right eye which then regressed. Episodes of choking. Radiographed by referral veterinary surgeon and congestion diagnosed.

Clinical Examination

No nasal discharge, pain or deformity. Normal air flow.

Endoscopic investigation

Left: NAD

Right: Nasal cavity more open and cavernous due to destruction of turbinates rostrally and centrally. Remnants of turbinates visible with irregular edges. More normal turbinates visible caudally. No fungal hyphae or foreign body.

Radiographic investigation (Fig 30)

Intra-oral nasal cavity, lateral skull, oblique skull, dorso-ventral skull, rostro-caudal skull and rhinography.

Intra-oral nasal cavity and rhinography.

Left: NAD

Right: Mixed density pattern mid-cavity and rostrally with turbinate destruction; turbinates intact caudally; frontal sinus opacification; irregular pattern of rostral nasal turbinates outlined by contrast; pooling of contrast laterally suggesting a loss of tissue in the rostral ethmoturbinate region.

Lateral/oblique skull and rhinography: Irregularity and small areas of lysis at the junction between the frontal and nasal bone. Increase soft tissue density delineated by the contrast in the dorsal ethmoturbinates and slight overlying swelling.

Rostro-caudal skull: Opacification of right frontal sinus with no mottling or irregularity of the frontal bone.

Diagnosis: Early tumour or atypical destructive rhinitis

Treatment

Right rhinotomy and turbinectomy. Dorsal nasal turbinates normal, but remainder were abnormal with a reduced volume of tissue. Retained mucus with a grey jelly-like consistency. No tumour mass identified. Ten days antibiotics prescribed.

Outcome

Re-presented two months later. Continued epistaxis, nasal discharge, noisy breathing, snorting and gagging. Bright. One week previously bilateral swelling around eyes and muzzle regressed with treatment, but recurred around the right eye. Severe epistaxis. Euthanasia was performed.

Clinical examination

Firm painful soft tissue mass over nasal bone at right inner canthus. Muzzle and face swollen. Continual low grade epistaxis. Reduced air flow.

Endoscopic investigation

Left: Normal structures, but appeared inflammed

Right: Impossible to visualise anything due to copious thick haemorhagic discharge. No resistance to passage of endoscope.

Examination repeated after euthanasia: Irregular nodular soft tissue mass caudally which was difficult to visualize. Generally more tissue present than normal post rhinotomy. Three punch biopsies taken. Biopsy taken using suction technique.

Radiographic investigation

Intra-oral nasal cavity and lateral skull.

Intra-oral nasal cavity:

Right: Post rhinotomy there is normally an extensive area of lucency. In this case, an area of lucency rostrally with an overall increase in soft tissue opacity. Punctate and focal lucencies with a larger irregular lucency (level PM4) suggestive of case destruction. No evidence of turbinate pattern. Frontal sinus opacification. No evidence of septal destruction or deviation.

Left: NAD

Lateral skull: Destruction of frontal bone and soft tissue swelling.

Diagosis: Nasal neoplasia (some opacification may be due to copious thick discharge.)

Pathology and Histopathology

Initial rhinotomy biopsy report: Rhinitis with a mixed cellular infiltrate, mucosal congestion and oedema

and granular hyperplasia. Cartilaginous proliferation rather than neoplasia.

Final post mortem, suction and endoscopic biopsy report. Nasal carcinoma. The lesion was centred on the right frontal sinus with extensive local destruction. The carcinoma was in places so poorly differentiated that sheets of cells superficially resembled lymphosarcoma.

12.

Case number 101258

Diagnosis: Nasal neoplasia (unconfirmed)

Corgi 11 years Male 13.5 kg

History

Two episodes of right epistaxis during previous four weeks. Two weeks ago a 2 cm diameter swelling developed adjacent to the right medial canthus. Snorting and gagging for several weeks.

Clinical Examination

Soft tissue swelling with obvious facial bone destruction. No pain, discomfort or discharge. Sneezed a small amount of blood tinged mucus; gagging; reduced air flow right nostril.

Endoscopic investigation

Left: Difficulty in entering nasal cavity. NAD

Right: Passed endoscope easily to level of swelling. Rostrally normal turbinate pattern with haemorrhagic inflammed streaking and copious clear mucus. Caudally provoked haemorhage immediately which obscured the view.

Radiographic investigation

Intra-oral nasal cavity, lateral skull, rostro-caudal skull and rhinography.

Intra-oral nasal cavity: Turbinate destruction causing an increase in radiolucency of the palatine fissure predominantly on the right. Irregularity of the bone and periosteal reaction on the labial edge of right PM2/3 alveolus. Adjacent to PM3 a large area of radiolucency - case destruction.

Caudally an overall increased opacification and partial turbinate destruction; frontal sinus opacity and absence of the vomer. Centrally mottling of the septum/vomer with adjacent turbinate destruction on the left. Defect confirmed using contrast, but very poor contrast penetration and distribution particularly caudally.

Lateral skull: Circumscribed area of lysis of the maxillary bone.

Rostro-caudal skull: Slight patchy opacification of right frontal sinus; left sinus NAD.

Diagnosis: Nasal neoplasia

Treatment

Wedge biopsy of soft tissue swelling resulted in a direct opening into the nasal cavity

Histopathology

Inconclusive. Normal muscle, connective tissue and vessels. No evidence of neoplasia. This result probably reflects an inadequate biopsy technique.

Outcome

Euthanasia recommended when dog's condition deteriorated. Lost to follow up.

13.

Case number 101445

Diagnosis: Nasal foreign body

Springer spaniel 5 years Female 19 kg

History

Three months previously the subject went to retrieve a bird and returned with blood on the left nostril. The dog became dull with intermittent snuffling, sneezing, left-sided epistaxis and discharge. The referring veterinarian found no abnormality on radiographic and auroscopic investigastion of the nasal cavity.

Clinical Examination

No nasal discharge. Necrotic odour. Pain 3 cm from nose tip.

Endoscopic investigation (Fig 31)

Obstruction to passage of endoscope just caudal to alar fold. Mucoid material between turbinates. Yellow solid foreign body observed in middle meatus.

Radiographic investigation

Intra-oral nasal cavity and Lateral skull.

Intra-oral nasal cavity: caudally and right rostral NAD.

Left rostral: Thin radiolucent line adjacent to the nasal septum resulting in an apparent extension of the palatine fissure to the level of PM4. No other evidence of turbinate masking or destruction.

Lateral skull: NAD

Diagnosis: Early destructive rhinitis. Possibly radiolucent foreign body. Inconclusive.

Microbiology (Fig 32)

A mixed growth, nothing significant.

Treatment

A twig, 5-6 cm long, removed using crocodile forceps under endoscopic control. Residual turbinates ulcerated. Ten days antibiotics prescibed.

Outcome

The dog made an uneventful recovery.

14.

Case number 101636

Diagnosis: Nasal aspergillosis

X-breed 6 years Male 7.25 kg

History

Bilateral, predominantly left sided, nasal discharge of three months duration. Sudden onset, thought to be associated with trauma. Frequent sneezing and head shaking. Thick purulent discharge, intermittently blood flecked. Ocular discharge. Snoring. Dull.

Clinical Examination

Very dull, thin, poor condition. Thick purulent discharge. Foul odour. Slight swelling over bridge of nose and mild deviation to left-hand side. Three centimeter diameter area of ulceration through hard palate. No air flow left nostril, reduced right nostril.

Endoscopic investigation

Bilaterally easy passage of endoscope to level of inner canthus. Discharge prevented thorough inspection. Septal destruction evident allowing endoscope to pass to the contra-lateral side.

Radiographic investigation (Fig 33)

Intra-oral nasal cavity and lateral skull:

Intra-oral nasal cavity: Nostril slightly deviated to the left. One left incisor absent. Discharge around nostril and partially occluding the alar channel. Increased lucency bilaterally rostrally with bilateral destruction of the trabecular turbinate pattern.

Small area of septal destruction rostral to the canine with a larger defect caudal to the canine; no deviation; punctate lucencies especially rostrally. Caudally partial destruction of the ethmoturbinates with patchy areas of opacity - possibly discharge. Caudal to carnassial, trabecular pattern and septum still intact. Slight frontal sinus opacification. No evidence of case destruction.

Lateral skull: Destruction of the trabecular pattern. Slight frontal sinus opacification, but no irregularity of the frontal bone.

Diagnosis : Nasal Aspergillosis - atypical, very destructive.

Microbiology

A profuse mixed growth of bacteria isolated.

Treatment

Euthanasia was performed on humane grounds.

Pathology and Histopathology (Fig 34)

The nasal septum and rostral conchae were virtually completely destroyed. In the ethmoidal region was a large fluffy white fungal mass 5 mm in diameter. The white and grey patches extended caudally particularly on the right side with lysis of the bone. The fungal mass was comprised of *A.fumigatus* hyphae.

15.

Case number 101812

Diagnosis: Not diagnosed

Lurcher 9 months Female 16.5 kg

History

Three month duration sneezing and slight epistaxis at the onset of exercise. As the dog sneezed it hit its nose on the ground and the owner considers this resulted in the mild haemorrhage. No significant epistaxis. No nasal discharge or sneezing at any other time. No reduced exercise tolerance.

Clinical Examination

NAD

Endoscopic investigation

Visualisation to base of ethmoturbinates bilaterally. No discharge or abnormality seen.

Radiographic investigation

NAD

Microbiology, serology, haematology

NAD

Treatment

None recomended. Re-examination suggested if epistaxis continued or condition deteriorated.

16.

Case number 101993

Diagnosis: Periapical abscess of the left canine.

X-bred 13 years Male 10kg

History

Three weeks ago episode of sneezing, head shaking, blood and mucus from left nostril. Since then sneezing with occasional epistaxis. Pain over affected rostral nasal cavity. Increased halitosis.

Clinical Examination

Left nasal discharge. No reduction in air flow. Enlarged submandibular lymph nodes. No distortion of bones. Facial pain evident.

Endoscopic investigation

A focal area of clear muco-pus observed in left common meatus at level of the canine tooth. Beyond this turbinates normal.

Radiographic investigation

Lateral skull and Intra-oral nasal cavity.

Intra-oral cavity: Small area of increased opacification with turbinate masking medial to left canine. No apical halo evident. Right side and caudal left NAD.

Lateral skull: NAD

Diagnosis : Inconclusive.

Treatment

Left canine tooth extracted easily due to absence of the medial periodontal ligament; inspissated pus around the amelocemental junction; altered pale grey tissue attached medial to tooth root and within the socket. No fluid appeared at nostril when socket flushed.

Histopathology

The biopsy consisted of fragments of bone and degenerating squamous epithelum with focal accumulations of polymorphonuclear leucocytes and plasma cells. These were consistent with an immuno-inflammatory reaction.

17.

Case number 102005

Diagnosis: Unconfirmed nasal neoplasia

Labrador 9 years Female 32kg

History

Three months previously sudden onset left nasal discharge and epistaxis with sneezing. Now reduced intermittent left nasal discharge and epistaxis. Breathing increasingly noisy, snorting and snuffling particularly at rest or eating.

Clinical Examination

Slight left nasal discharge. No facial distortion or pain. Snorting and snuffling. Bilateral reduced airflow. Hard palate convex.

Endoscopic investigation

Right hand side: Normal nasalturbinates, but septum pushed to obstruct common meatus.

Left hand side: Absence of turbinates, replaced by homogenous tissue. Epistaxis induced caudally.

Radiographic investigation

Lateral skull and intra-oral nasal cavity.

Intra-oral nasal cavity:

Left: Overall increased opacity; turbinate destruction; area of lucency suggestive of case destruction (level PM3). Rostrally, absence of nasal septum/vomer. Caudally nasal septum deviated to right. Left frontal sinus opacification.

Right: Caudally NAD. Adjacent to septal defect some increased opacification and turbinate destruction.

Lateral skull: Increased opacification caudal nasal turbinates and rostral ethmoturbinates; loss of turbinate clarity; small lucent areas suggested defects in maxillae or nasal bones. Frontal sinus opacification. No soft tissue swelling.

Diagnosis : Nasal neoplasia

Treatment

Euthanasia advised when the dog's condition deteriorated.

18.

Case number 102122

Diagnosis: Acquired hard palate defect.

Miniature Pinscher 9 months Male 2.25kg

History

Vague. Presented with bilateral purulent nasal discharge of unknown duration with a possible earlier traumatic incident.

Clinical Examination

Bilateral nasal discharge, predominatly left-sided. Two defects approximately 2cm long visible orally, one in the hard palate to the left of the midline and the second at the junction between the hard and soft palates.

Endoscopic investigation

Endoscope too large to enter nares.

Radiographic investigation

Intra-oral nasal cavity: slight masking of the turbinate pattern bilaterally. Caudally on the left a 1cm radiolucent defect visible within an area of opacity indicating possible case destruction and a small area of turbinate destruction.
Diagnosis: Bony defect level with the caudal molars. Possible hard palate defect.

Treatment

The two lesions were repaired surgically.

Outcome

The dog clinically improved although following a wound breakdown part of the oro-nasal fistula recurred.

Lateral and Rostro-caudal skull: Minimal frontal sinus opacification; no frontal bone irregularity.

Diagnosis : Destructive rhinitis (aspergillosis)

Serology and microbiology

A. Fumigatus confirmed. The fungus was identified from a plaque removed from the nasal cavity, but not from a nasal swab.

Treatment

An indwelling plastic tubing catheter (0.5cm diameter) placed through a trephine hole into the right frontal sinus and the mid-nasal cavity region. 1.25ml (diluted in 5ml of water) of enilconazole ("Imaverol") instilled into the cavity twice daily for five days. Antibiotic treatment continued for 10 days. Thiabendazole treatment reinstituted (500mg BID) for four weeks (Sharp and Sullivan, 1986)

Re-examination

The dog was re-examined two weeks following the completion of the course of treatment. Following the enilconazole the dog had had no further significant discharge and appeared to the owners clinically normal. Endoscopically there was no evidence of fungal plaques but regenerating, truncated turbinates were observed.

20.

Case number 101608

Diagnosis: Nasal neoplasia

Samoyed 6 years Male 29 kg

History

Three months previously sudden onset soft, non painful swelling between the eyes. Responded to lancing and antibiotics.

Two weeks ago soft swelling over bridge of nose. Pain and difficulty opening mouth. White purulent left nasal discharge.

Clinical Examination

Left nasal discharge and reduced air flow. No air flow right nostril. Snorting/snuffling and mouth breathing. Unable to open mouth more than two inches even under anaesthesia. Soft fluid swelling over frontal bone.

Endoscopic investigation

Unable to pass the base of the ventral turbinates. NAD.

Radiographic investigation

Lateral skull, intra-oral nasal cavity, dorso-ventral skull, rostro-caudal skull, thorax.

Intra-oral nasal cavity and dorso-ventral skull: Marked bilateral and central increase in soft tissue density; turbinate destruction and masking extending rostrally; destruction of vomer caudally; opacification of the frontal sinuses (also appreciated on rostro caudal view).

Lateral skull: Bone lysis of nasal bone, rostral frontal bone and base of frontal sinus with spicules of new bone formation radiating into an overlying soft tissue swelling. Frontal sinus opacification.

Rostro-caudal skull: Bilateral frontal sinus opacification. No frontal bone destruction.

Lateral thorax: NAD

Diagnosis: Neoplasia

Treatment

Biopsy of mass over frontal sinus. Disintegrating frontal bone overlying a retention mucocoele. Euthanasia performed.

Histopathology

Osteosarcoma. A well defined firm tumour, two centimeters in diameter, in the posterior ethmoturbinates. The frontal sinuses were filled with thick mucopus.

DISCUSSION

Nasal neoplasia has been recorded in the literature, and in this study, affecting a wide variety of breeds. Any review which seeks to identify susceptibility in particular breeds will therefore have to contain significant numbers. Comparisons should also be made with the general population to assess the expected level of risk for each breed. As details of the general population are not readily available, the hospital referral population has been used instead. It was considered that significant errors would not result.

Even this survey of sixty cases is still not sufficiently large to draw conclusions about the incidence in less common breeds. This is exemplified by the case of the Great Dane breed. The referral population prediction was 0.9%, however two cases were seen in this series which, while significant in percentage terms, in reality has little meaning. Medium to large breeds predominated. Giant breeds were rarely affected and the incidence in small and toy breeds was only occasional.

Evaluation of the risk of breeds with particular skull structures was complicated by the fact that no workers have defined which breeds they have allocated to which skull type be it brachycephalic, mesaticephalic or dolichocephalic. Possibly the prevalence of Shelties and Collies in some series reflects a common genetic background and so a breed susceptibility rather than a skull type as such.

Nasal neoplasia may be related to environmental carcinogens. It could therefore be hypothesised that brachycephalics may be spared as many of them are predominantly mouth breathers. This simplistic view is undermined by the survey of Madewell *et al* (1976) which included such breeds as the Boston Terrier, Pekinese and Bulldog! However, it is interesting to note that Boxers, the brachycephalic breed in which nasal tumours are most commonly recorded, tend to be nose breathers. A further indication that environmental factors may play a part is that mixed breeds, often spared from other tumours, are as susceptable as purebred animals.

The age range and mean age of affected animals in this series agreed with the results of other authors. As with many tumours they predominantly affect the middle to older aged animal. However younger dogs were also

represented, the youngest being sixteen months old. This was unusual, but had been noticed in earlier larger surveys. (Morgan *et al*, 1972; Madewell *et al*, 1976). The age of an animal can be used as a diagnostic indicator in nasal disorders, but the results show that neoplasia should be considered even in the young adult.

There was no sex predisposition found in this review. The marginally higher incidence in males was not statistically significant. These findings agree with many authors including the larger survey of Madewell *et al* (1976).

Small case numbers can obviously distort the apparent distribution considerably; Confer and De Paoli (1978) recorded 4 males to 6 females. Brodey 1970 and Morgan *et al* (1972) also reported a higher incidence in males, but neither compared this with their expected sex risk based on the hospital or general populations and thus its relevance is questionable. Hayes *et al* (1982) did conclude after statistical adjustment that males were slightly more at risk.

The median, mean and range of duration of clinical signs in this study are in agreement with several of the series reviewed (Bradley and Harvey, 1973; Madewell *et al*, 1976; MacEwen *et al*, 1977).

Owners may ignore a nasal discharge for a while, while assuming the dog to have a "cold", before deciding to have the problem investigated. Dogs exhibiting epistaxis however may be presented more promptly because of the social inconvenience and a realisation that this is abnormal.

When the signs are less aggressive and more insidious in onset many owners are unsure of their exact duration. The dog, being a nose licker, may well clear minor discharges away from the nares so that the owner is aware of them only when they become profuse. In addition if sneezing and/or nasal discharge are intermittent the fact that many dogs are unobserved for large parts of the day again reduces the likelihood of owners noticing these clinical signs.

If the tumour originates in the caudal part of the nasal cavity abnormal discharges will pass into the pharynx and be swallowed. Only when the quantities become considerable will the dog exhibit gagging and choking or a discharge at the nares.

It can therefore be concluded that the duration of clinical signs is a poor indicator of the length of time the disease has been in progress. Furthermore, in many cases neoplasia will be radiologically evident by the time clinical signs are significant enough to warrant the owner seeking veterinary attention. Even dogs with a recent onset of clinical signs may show radiographic changes, as demonstrated by case 49 (Appendix II).

In this series, as in the literature, animals presenting which had exhibited signs over a long period of time were reported infrequently (Bradley and Harvey, 1973; Madewell *et al*, 1976; Gibbs *et al*, 1976)

The clinical presentations in this series were similar to those observed by other workers.

Twenty-one cases exhibited an extra-nasal soft tissue swelling indicating that the tumour was destructive and well advanced. This feature was highly suggestive of neoplasia, but has occasionally being recorded in other conditions. Gibbs *et al* (1979) noted that in two out of twenty six cases of destructive rhinitis in which the lesion was very advanced, facial bone destruction and swelling did occur. It could also occur in osteomyelitis.

Seven cases of neoplasia exhibited pain over the nasal cavity, behind the ears and between the eyes. This sign was infrequently noted by other authors.

Central nervous system (C.N.S) abnormalities were rarely seen in this series or reported by other authors. Since nasal tumours are locally invasive and destructive it is possible that if allowed to progress they may invade the cribriform plate and directly affect the cranial cavity and C.N.S or cause complications by pressure. However, most dogs are euthanased on humanitarian grounds before the more distressing signs become evident.

No dogs, in this series were presented with C.N.S signs alone and apart from Norris (1979) no other authors have recorded this feature.

In the early stages of tumour growth clinical abnormalities may not be apparent particularly if it originates in the ethmoturbinate region. Firstly, when the neoplasm is small there will be little obstruction to the natural secretory drainage and any increase in secretions will initially pass back into the nasopharynx. Secondly, infection may not occur until the lesion progresses, thus the initial discharge may appear serous or mucoid rather than purulent.

Finally, as previously discussed, minor clinical signs may go unnoticed since the dog has a great capacity to compensate for abnormalities without complaining.

A higher incidence of carcinomata than sarcomata was recorded which was in agreement with the findings of several other authors (Brodey, 1970; Bradley and Harvey, 1973; MacEwen *et al*, 1977; Confer and De Paoli, 1978; Norris, 1979).

A wide variety of tumour types are recorded originating in the nasal cavity although no specific sequence of pathological changes have been identified. In this series adenocarcinoma and chondrosarcoma were predominant.

In a small number of cases the clinical signs did not correlate with the radiological changes when considering unilateral or bilateral involvement. Either of these discrepancies could be due to poor owner observation. Where the radiological changes were bilateral and the clinical signs unilateral this would support the theory that clinical signs are not seen in the early stages of the disease.

The second and infrequent observation that clinical signs were bilateral with unilateral radiological changes, would contradict this. It may be that in some cases of neoplasia clinical signs are evident very early in the course of the disease. Alternatively, and more possibly, there may be defects in the septum, not radiologically apparent, allowing discharge to pass out of both nares.

One of the most important factors when trying to differentiate between various nasal disorders was to establish whether or not there was turbinate destruction or masking on the intra-oral view. In conjunction with this there must also be an assessment of any overall or focal alteration in density within the nasal cavity.

The tabulated results show clearly that an increase in radiopacity associated with turbinate destruction is the predominant features in nasal neoplasia. An increase in radiolucency was an unusual finding and a mixed density pattern an occasional finding.

Many authors did not make the distinction between turbinate loss and masking. They encountered difficulties in distinguishing between, for example, hyperplastic rhinitis - where the turbinate structure stays intact, but

the mucosal hyperplasia and hypersecretion results in an overall increase in radiopacity - and neoplasia.

As the tumour mass expands it causes turbinate destruction, but this is generally replaced with more radiopaque tumour tissue. A mixed pattern may result from an increase and accumulation of secretions and tissue mass intermixed with areas of turbinate and/or bony destruction.

Very occasionally tumours appear more destructive and less proliferative in nature resulting in very little increase radiopacity. This can also produce a mixed pattern or even areas of lucency which may prove very difficult to distinguish from a destructive rhinitis.

Radiographically destructive rhinitis shows predominantly turbinate destruction and therefore an increase in radiolucency. This is most frequently seen centrally then progressing rostrally. Where a mixed density is present caudally it may be due to a build up of debris and secretions before the destruction is sufficiently advanced to allow free drainage.

A mixed density pattern should not be confused with sizeable areas of lucency within an area of radiopacity. These may represent defects in the bony structures - maxillae, nasal, frontal or palatine bones - referred to as case destruction. In this review it was seen in approximately thirty per cent of the cases and is highly suggestive of neoplasia. Sizeable bony defects are unlikely to result from other nasal disorders. However, there are rare reports of advanced cases of destructive rhinitis causing facial bone destruction (Gibbs *et al*, 1979; Harvey *et al*, 1979).

Punctate lucencies were noted in some cases. These are also a feature of destructive rhinitis and so were not considered diagnostic. Sullivan *et al* (1986) suggested that this appearance is due to pitting or focal lysis of the bone caused by *Aspergillus fumigatus* itself or its toxins. This may also result in the "moth eaten" appearance of the frontal bone with or without an increase in bony thickness due to periosteal reaction. These changes in the frontal bone are diagnostic of destructive rhinitis and are not recorded as a feature of neoplasia either in this study or the literature reviewed. The punctate lucencies in neoplasia may also be a result of pitting erosions in the supporting bones or to entrapment of air within the tumour mass (Gibbs *et al*, 1979).

Plate destruction was a feature in over half the cases indicative of local bone lysis which was often severe. However, this feature can be difficult to evaluate fully, particularly if there is any rotation of the nasal cavity and is not considered a useful diagnostic parameter.

Alteration of the mid-line opacity on the intra-oral radiographs was a noteable finding. In contrast to the views of Harvey (1979), it did appear that the septal cartilage contributed to the midline density. Although Harvey (1979) concluded that it was only the vomer that was visible, he did comment that assessing the radiograph in which the septum had been removed "a minor decrease in the radiopacity of the middle nasal septal area was visible on close inspection". Even from the published illustrations the difference was evident particularly over the middle third. The cartilaginous and bony nasal septum when removed measured about 2 cm deep in some areas. It is very unlikely that this would not be visible radiographically to some extent.

Where there was absence of the midline density the vomer must have been destroyed indicating there may be significant involvement of the contralateral side.

Deviation was recorded where the midline opacity was markedly displaced. The vomer is bone and has an anatomical make-up which would make significant deviation impossible, therefore the deviation observed must be of the septum. This reaffirms the impression that the nasal septum does produce a radiographic density in its own right. Many other authors also commented on septal deviation as a feature of nasal neoplasia (Morgan *et al*, 1972; Delmage, 1973; Madewell *et al*, 1976; MacEwen *et al*, 1977; Norris, 1979)

Significant changes in the nasal septum are not normally associated with nasal disorders other than neoplasia. However, Gibbs *et al* (1979) and Harvey *et al* (1979) both recorded cases of advanced destructive rhinitis where septal erosion occured. This was rare. Sullivan *et al* (1986) noted vomer changes in sixteen out of forty-five cases in which it was either roughened or ill-defined never deviated or absent. Harvey *et al* 1979 also recorded one case of osteomyelitis with septal involvement.

In conclusion erosion of the cartilaginous nasal septum may occur without any significant radiological changes. Where absence or deviation of the midline

opacity is present it is highly suggestive of neoplasia. Only in exceptional cases will erosion be caused by destructive rhinitis or osteomyelitis. Deviation has only been seen in cases of neoplasia.

Mineralisation was seen in association with three different tumour types and so was not type specific. This is in contrast to the findings of Gibbs *et al* (1979) who only observed it in chondrosarcomata. No other radiological patterns were identified to assist in classification even in the broadest sense.

Radiographic changes seen on lateral views are often harder to interpret because of the superimposition of one nasal cavity on the other. Where turbinate loss is seen, its full extent cannot be appreciated. The lateral view alone was rarely diagnostic.

Bone destruction was identifiable in eighteen lateral radiographs and if this was combined with the intra-oral view a more precise picture was gained. A similar number of dogs showed soft tissue nasal swelling on the lateral radiograph. Many showed both bone destruction and soft tissue swelling.

Soft tissue swelling normally accompanied destruction of the nasal, maxilla or frontal bones, but it is only appreciated on a lateral view if it is 'skylined' eg. the nasal or frontal bones. If the swelling is over the maxilla then the superimposition of the nasal structures prevents it being visualised. Oblique views may be used in these cases, although it is not normally necessary. The reverse also occurs when soft tissue swelling is skylined, but the destruction is minimal or obscured.

Invasion by the tumour into the cranial cavity or orbit is also difficult to evaluate radiologically because of the problem of clearly identifying the structures in this region. Destruction of the cribriform plate may be appreciated occasionally.

Opacity of the frontal sinus was present in sixty three per cent of the cases and was most readily appreciated on the lateral view. It may also also be seen to some extent on the intra-oral view indicating either unilateral or bilateral involvement. In most cases the opacity is thought to be due to the tumour mass obstructing the normal drainage resulting in a retention mucocoele. In others there was obvious invasion of the frontal sinuses by the neoplasm. Rostro-caudal views are useful in these instances to see the full extent of the lesion.

Metastatic spread was not identified on any of the thoracic radiographs, nor was it reported in any of the dogs that underwent a full post-mortem examination. This suggested a very low incidence of metastatic spread. However, it may occur more frequently than these and earlier reports show, but fails to be recognised if a full histopathological investigation is not undertaken. Hayes *et al* (1982), recorded a metastatic rate of approximately ten per cent. The low rate may be due either to the fact that many of these tumours grow so rapidly that metastatic spread has little chance to occur or that neoplasia in the nasal chamber, irrespective of its type, produces its effects predominantly by local invasion and expansion.

The prospective study (Section II) comprised of twenty dogs with chronic nasal disease. The distribution of the conditions was in agreement with a larger clinical survey of the case records at Glasgow University Veterinary Hospital (Appendix I). The predominant instigator of a chronic nasal discharge was neoplasia. Other causes included chronic hyperplastic rhinitis, aspergillosis and rhinarial ulceration.

The findings relating to the eight cases of nasal neoplasia were similar with respect to age, duration of clinical signs and breeds affected to those of the retrospective study and literature review. Differences in the male to female ratio highlight the variance which may occur in small surveys.

Nasal discharge was a non-specific clinical sign, but in combination with certain other features could suggest neoplasia. These included evidence of a soft tissue swelling and/or bony destruction, snorting and gagging. A blood-tinged discharge or epistaxis was also a frequent finding.

Just under half (3/8) of the dogs with neoplasia exhibited pain at or before the time of examination. Pain has been more commonly associated with aspergillosis. Sharp *et al* (1984) reported that four out of seven cases of aspergillosis showed facial pain on examination. To obtain information on episodes of pain in the early stages of the disease often necessitated careful, specific questioning of the owner as it was often transistory in nature.

This was exemplified by case 9 which exhibited pain as an initial feature, but once a swelling appeared over the frontal sinus again the pain regressed implying that

the pressure or the initial bone destruction caused by the tumour resulted in the discomfort.

Neurological signs were only seen in one dog (case 2) in the form of reduced hearing for several weeks prior to presentation. This dog also exhibited bouts of acute head and neck pain. This was presumably the result of the extensive nature of the tumour and its infiltration into the cranial cavity.

Three of the six classified tumours were carcinomas. An osteosarcoma and a chondrosarcoma were also identified. An unusual finding was a malignant melanoma (Case 8), the origin of which was not ascertained. The dog had only a four week history of a nasal discharge although the lesion was extensive at the time of presentation.

Malignant melanomas are more usually associated with the oral cavity than the nasal cavity. With the involvement of the hard palate the tumour may have originated in the oral mucosa and progressed dorsally. However all the clinical signs related to the nasal lesion and there was no evidence of oral ulceration or local lymphatic involvement.

The use of various biopsy techniques can provide simple, relatively non-invasive methods of enabling histopathological confirmation of the diagnosis to be made.

The results of biopsies in this study, particularly initially, were variable and to an extent related to the inexperience of the operator. There may be problems in obtaining a representative sample from suction biopsy, forceps biopsy under endoscopic control or following wedge biopsy of a soft tissue swelling.

The suction biopsy technique may provide a cheap simple non-invasive method for general use. It appeared more successful when the catheter impinged onto soft tissue prior to suction. If this was not possible the tendency was to aspirate intra-nasal discharge which usually proved unrewarding histologically. When haemorhage was induced it was minimal and soon ceased without any control measures being taken.

Biopsy under endoscopic visualization did not appear any more reliable than the previous technique although it was expected to be superior. Identification of the tissue for biopsy proved difficult due to the presence of

discharge, the inability to manouvre the tip of the endoscope freely in the small nasal cavity and the inaccessibility of some regions particularly caudally. Once a biopsy had been taken, or attempted, the induced haemorhage obscured further examination. Even when a tissue mass was identified it could still result in inconclusive results. Where the tumour was located caudally, the catheter technique was superior due to its small diameter. Tumours located in the frontal sinus region appeared particularly difficult to biopsy even at rhinotomy.

A wedge biopsy of a soft tissue swelling was diagnostic in case 9, but resulted in only normal tissue in case 12. In the frontal sinus region swelling may result from a retention mucocoele (case 20), as an indirect result of tumour growth. This can result in aberrant biopsies. Also wedge biopsies may only reflect the more superficial layers and so a section of deep tissue, namely turbinates if bone lysis has occurred, should be included.

The evidence of neoplasia on histopathology was diagnostic, but its absence was not and could be confusing. This was exemplified case 11. The biopsy was taken at rhinotomy and was thought to be representative of the lesion which clinically and radiographically was suggestive of neoplasia. In this particular instance, the radiological features were consistent with an early lesion, but as such were not definite. A prognosis based on the histopathology would have proved misleading. Had the dog not undergone a rhinotomy repeat radiographs in four to six weeks would probably have enabled a more conclusive radiographic diagnosis to be made. Two months later both a suction and endoscopic forcep biopsies showed evidence of malignancy.

Cystic hyperplasia was diagnosed from a biopsy obtained at rhinotomy in case 7. This did not correlate with the radiological features which demonstrated all the characteristic features of nasal neoplasia; namely an overall unilateral increase in opacification with marked turbinate destruction both rostrally and caudally.

The histopathology of case 7 and case 11 were broadly similar which questions the validity of the results. If case 7 was, as the radiological features suggested, a tumour, it presented an unusual clinical picture. There had been a long history of clinical signs (10 months) with periods of up to two months remission and no evidence of radiological progression over a three month period.

Conversely, if it was a case of hyperplastic rhinitis there had been extensive turbinate destruction not a feature previously seen here or recorded in the literature (Gibbs *et al*, 1979).

The long term survival of the clinical cases with neoplasia was very poor. Despite the relatively short duration of clinical signs most of the tumours were advanced at the time of examination and euthanasia was generally recommended.

Treatment in the form of rhinotomy and turbinectomy was undertaken in case 11 as this lesion did not appear radiologically too extensive. The two month survival of this dog was not encouraging.

This form of treatment was also used in a case of chronic hyperplastic rhinitis (case 7). However post-rhinotomy the dog suffered from a persistent bilateral nasal discharge, snorting and gagging. At post-mortem residual turbinates were evident which may have acted as a focus for persistent infection. Complete turbinectomy caudally was hard to achieve and, in the case of neoplasia, this increases the likelihood of recurrence.

A number of earlier reports indicated that the response to most forms of treatment was very poor (Table 2). A more recent report by Thrall and Harvey (1983) on the use of radiation therapy following surgical debulking produced encouraging results and merits further investigation.

Two cases of aspergillosis were seen and confirmed serogically and mycologically. Case 19 was characteristic both clinically and radiologically, whereas case 14 showed more unusual signs. This dog was systemically ill, had a bilateral nasal discharge and a large area of ulceration through the hard palate. Clinically these signs were more suggestive of neoplasia. Radiologically there was extensive destructive rhinitis involving the nasal septum. This was one of the most advanced cases of aspergillosis recorded at Glasgow University and given the general state of the dog it is probable that the condition was present for much longer than the reported three months.

The response of case 19 to five days treatment with topical enilconazole was dramatic. Virtually no nasal discharge or sneezing was evident by the fifth day. The thiabendazole was continued although the dog had failed to respond to this alone. To what extent this contributed to the treatment was difficult to evaluate. This result was

consistent with the findings of Sharp and Sullivan (1986) and supports the view that enilconazole may provide an effective treatment regime in aspergillosis.

Chronic hyperplastic rhinitis can result from a number of initiating factors - viral rhinotracheitis (case 10), distemper, (case 6) and oronasal defects (case 16 and 18). The radiological appearance was that described by Gibbs *et al* (1979) with increased opacification causing turbinate masking, but without turbinate destruction.

Radiologically, a foreign body such as a twig (case 13) may show very subtle changes necessitating a careful, detailed examination. Even if these changes are not appreciated the elimination of other disorders such as neoplasia and aspergillosis is in itself informative. In case 13 the clinical history was also highly suggestive of a foreign body having a history of sudden onset, unilateral discharge with pronounced pain on pressure over the rostral nose.

Radiologically seven of the eight cases of neoplasia were well advanced at the time of examination. The characteristic features included turbinate destruction increased opacification, case destruction and soft tissue swelling. Cases 2, 3 and 9 were extremely destructive. Additional views including the dorso-ventral skull were utilised to demonstrate the full extent of the bone lysis, yet no pronounced CNS signs were noted.

Interpretation of the mixed density pattern radiologically presented some difficulties in case 11. In the main review this appearance was seen in a minority of cases. It was thought to be associated with early neoplastic change particularly when epistaxis was a presenting feature, as seen in this case. Repeat radiographs or exploratory surgery should be undertaken if the diagnosis is uncertain. Alternatively, additional radiographs may be of value. Case 11 exhibited bone lysis on the lateral view, highly suggestive of neoplasia.

The results of rhinography were generally disappointing. The comparison between sodium metrizoate solution and 30 per cent barium solution on normal canine noses showed the former contrast agent to be superior providing far better distribution and turbinate outlining. Very little contrast adhered to the rostral ethmoturbinates using either medium.

Transistory sneezing was the only side effect noted following either contrast medium.

In order to appreciate abnormal changes a reasonably constant distribution of contrast medium is necessary in the normal dog. This was difficult to achieve. When the changes were unilateral the contralateral side could be used for comparison. It was possible to identify minor changes not identified on the plain radiographs. Examples were septal/vomer defects - where contrast passed into both cavities after instillation into one nostril- or case destruction - where contrast was seen outside the nasal cavity.

The outlining of soft tissue tumour masses added little additional information. Where gross turbinate destruction had occurred the contrast tended to pool in areas due to lack of tissue to adhere to. If manipulation of the head was carried out to improve distribution the contrast had a tendency to leak out via the nares or pass caudally into the pharynx. Rhinography as a proceedure did not appear as useful as Goring *et al* (1982 & 1984) indicated.

Endoscopic examination was undertaken in eighteen of the dogs. Two of these, weighing 6.5kg and 2.25kg, were too small to allow the passage of the instrument through the nares. A few of the other dogs also required careful introduction of the endoscope, but examination was possible in four small dogs with weight ranges from 9 to 13.5kg.

In all dogs (except case 18) examination of the nasopharynx via the oral cavity was possible, but the findings were generally unremarkable or merely demonstrated discharge passing caudally into the pharynx.

Passage of the endoscope beyond the rostral ethmoturbinates was related to head size and shape and the extent of normal turbinate disruption by disease processes. In larger dogs visualisation to the level of the inner canthus was possible where the architecture was normal or more caudally with certain abnormalities.

The endoscopic examination of tumours was limited by the presence of nasal discharge and the replacement of the turbinates with proliferating tissue which readily haemorrhaged. The insufflation of air via the endoscope was used to help counteract this.

Where the predominant feature was turbinate destruction the endoscopic examination proved more useful and in case 19, aspergillosis was reliably diagnosed. It

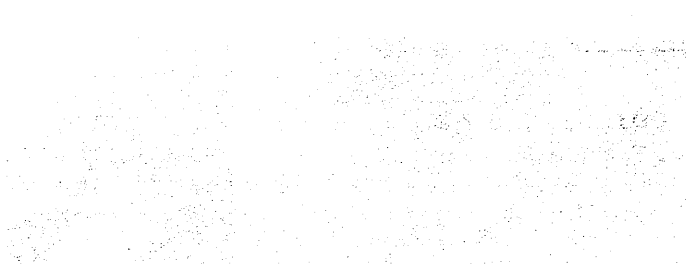
also provided a simple method for monitoring the progress of treatment.

The identification and subsequent removal of the foreign body in case 13 was equally possible using a wide bore auroscope as twigs lodge in the rostral meatus making them easily accesible. Auroscopic inspection can also be used in cases of destructive rhinitis as the absence of turbinates allows introduction of the instrument.

Microbiological swabs were taken both from clinical cases and from dogs with no known history of nasal disease. These provided no useful clinical information. Mixed cultures were obtained in every case including Beta-haemolytic Streptococcus, Staphylococcus spp and gram negative rods all of which had expected antibiotic sensitivities. A pathogenic strain of Staphylococci was even isolated from a normal control dog.

Fungal plaques removed directly from case 19 were cultured confirming *Aspergillus fumigatus* infection, however this was not identified on the routine nasal swabs. The conclusion of these findings agrees with those of other authors that nasal microbiological swabs are of little diagnostic value.

In conclusion, the three main causes of chronic nasal disease in the dog are neoplasia, chronic hyperplastic rhinitis and aspergillosis. Radiography provides the simplest, quickest and most reliable method of differential diagnosis.



APPENDIX I

Nasal disease in the dog - a breakdown of the range of conditions seen at The University of Glasgow Veterinary Hospital in a series of 100 cases during the period 1983 - 1985.

Neoplasia	37
Chronic hyperplastic rhinitis	25
Aspergillosis	13
Acute allergic rhinitis	6
Rhinarial ulceration	5
Epistaxis of unknown origin	4
Foreign body	4
Destructive rhinitis	2
Oro-nasal fistula	2
Inflammatory polyp	1
Non specific inflammation of the nasal septum	1
	100

APPENDIX II

The tabulated results of the retrospective review of 6 cases of nasal neoplasia seen as referral cases at the Department of Surgery University of Glasgow.

Age	- Age at presentation.
Durn.	- Duration of clinical signs reported by the owner.
Uni/Bil Clin	- Unilaterally or bilaterally involved on clinical examination.
Uni/Bil Rad	- Unilateral or bilateral involvement radiologically.
I/oral Rostral	- The radiological features observed in the rostral portion of the nasal cavity on the right and left sides respectively (R/L).
I/oral Caudal	- The radiological features observed in the caudal portion of the nasal cavity on the right and left sides respectively (R/L).
Turb.Loss	- Radiological evidence of turbinate destruction.
Luc.(L)	- Areas of increased radiolucency seen radiologically.
Opac.(O)	- Areas of increased radiopacity seen radiologically.
Mxd.(M)	- Areas of mixed density and seen radiologically.
Punctate	- Presence of punctate lucencies.
Pl.destn.	- Plate destruction.
Ca.destn.	- Case destruction.
Vomer	- Deviation or destruction of the vomer.
Lateral	- Radiological changes observed on the lateral view of the skull.
B.Destn.	- Bone destruction.
Fr.Sin.Op.	- Opacification of the frontal sinus.
Ex.N.S.T.	- Extra-nasal soft tissue mass.
( )	- Focal changes.

APPENDIX IIa

Number	1	2	3	4	5	6
Age	11.5	8	11	8	2	7.5
Sex	M	M	M	M	M	F
Durn.	4	12	2	2	1	2
Uni/Bil						
Clin	U	U	B	B	B	U
Rad	U	B	B	U	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	-/+	+/+	+/-	-/+	+/+	-/+
Luc. (L)						
Opac.(O)	-/0	0/0	-/0	0/0		
Mxd. (M)	M/-	-/M				
I/Oral Caudal						
Turb Loss	-/+	+/+	+/+	-/(+)	+/-	+/+
Luc. (L)						
Opac.(O)	-/0	0/0	0/-	-/0	0/-	
Mxd. (M)	-/M	M/M				
Punctate	+/-					
Pl.destn.	-/+	+/+	-/+	+/-	-/+	
Ca.destn.	+/+					
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+			
B. Destn.	+	+				
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+					

APPENDIX IIb

Number	7	8	9	10	11	12
Age	10	8.5	5	10	5	10
Sex	M	M	M	F	F	M
Durn.	11	1	.75	7	6	24
Uni/Bil						
Clin	U	B	U	U	U	U
Rad	U	B	U	U	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	-/+	+/+	-/+	(+)/-	+/(+)	+/+
Luc. (L)						
Opac.(O)	-/0	0/0	-/0	0/-	0/-	
Mxd. (M)						
I/Oral Caudal						
Turb Loss	-/+	+/+	-/+	+/-	+/-	+/+
Luc. (L)						
Opac.(O)	-/0	0/0	0/-	-/0	0/-	
Mxd. (M)	M/-	-/M				
Punctate	+/-	+/-				
Pl.destn.	-/+	+/-				
Ca.destn.	+/-					
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+			
B. Destn.	+	+				
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+					

APPENDIX IIc

Number	13	14	15	16	17	18
Age	11	11	8	11	6	8
Sex	F	F	M	M	F	F
Durn.	1.5	5	1.5	1.5	1	3
Uni/Bil						
Clin	U	U	B	U	B	B
Rad	B	B	B	U	B	U
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	+/+	+/+	-/+	(+)/+	-/+
Luc. (L)						
Opac.(O)	0/0	0/0	0/0	-/0	-/0	-/0
Mxd.(M)	M/-					
I/Oral Caudal						
Turb Loss	+/+	+/(+)	+/-	-/+	-/+	-/+
Luc. (L)						
Opac.(O)	0/0	0/-	0/-	-/0	-/0	-/0
Mxd. (M)	-/M					
Punctate	+/-					
Pl.destn.	-/+	+/+	-/+	-/+	-/+	
Ca.destn.	-/+	+/-	-/+			
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+	+	+	
B. Destn.	+	+	+	+		
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+	+	+			

APPENDIX IId

Number	19	20	21	22	23	24
Age	14	13	13	5	10	7
Sex	F	M	M	F	M	F
Durn.	4	1.5	2	6	1.5	0.75
Uni/Bil						
Clin	B	U	U	B	U	B
Rad	B	U	U	B	U	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	+/-	+/+	+/-	+/(+)	
Luc. (L)						
Opac.(O)	0/0	0/-	0/-	0/(0)		
Mid.(M)	M/M					
I/Oral Caudal						
Turb Loss	+/+	+/-	+/-	+/-	+/-	+/-
Luc. (L)						
Opac.(O)	0/0	0/-	0/-	0/-		
Mid. (M	M/-	M/-				
Punctate	+/-					
Pl.destn.	+/-					
Ca.destn.						
Vomer	+	+	+			
Lateral						
Turb Loss	+	+	+	+		
B. Destn.	+					
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+					

APPENDIX IIe

Number	25	26	27	28	29	30
Age	9	12	12	8	9.5	13
Sex	M	M	F	M	M	F
Durn.	1	2	1	2.5	1	2
Uni/Bil						
Clin	B	U	B	U	B	U
Rad	B	B	B	B	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	+/-	+/+	-/+	-/+	
Luc. (L)	-/L					
Opac.(O)	O/-	O/-	O/O	-/O	-/O	
Mxd.(M)						
I/Oral Caudal						
Turb Loss	+/-	+/-	+/+	+/+	+/+	+/+
Luc. (L)						
Opac.(O)	O/-	O/(O)	O/O	O/O	O/O	O/(O)
Mxd. (M)						
Punctate	+/-	+/+				
Pl.destn.	+/-	-/+	+/-			
Ca.destn.	+/-	-/+				
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+	+		
B. Destn.	+					
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+	+	+			

APPENDIX IIf

Number	31	32	33	34	35	36
Age	12	4	12.5	12	9	13
Sex	M	F	M	F	F	F
Durn.	.75	1	.75	2	4	2
Uni/Bil						
Clin	B	B	B	B	B	U
Rad	B	U	B	B	B	U
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	-/+	+/+	+/+	+/+	+/-
Luc. (L)						
Opac.(O)	0/0	-/0	0/0	0/0	-/0	0/-
Mxd.(M)	M/-					
I/Oral Caudal						
Turb Loss	+/+	+/+	+/+	+/+	+/-	
Luc. (L)						
Opac.(O)	0/0	0/0	0/0	-/0	0/-	
Mxd. (M)	M/-					
Punctate	+/-					
Pl.destn.	+/+	-/+	+/-	-/+	+/-	
Ca.destn.	+/+	-/+	+/-	+/+		
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+			
B. Destn.	+	+				
Fr.Sin.Op	+	+	+			
Ex.N.S.T.	+	+	+			

APPENDIX IIq

Number	37	38	39	40	41	42
Age	9	8	8	13	8	9
Sex	F	M	M	M	F	M
Durn.	1	2	.25	1	6	4
Uni/Bil						
Clin	B	U	B	U	B	B
Rad	B	U	U	U	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	-/+	+/-	+/+		
Luc. (L)						
Opac.(O)	O/O	-/O	O/-	O/O		
Mxd.(M)						
I/Oral Caudal						
Turb Loss	+/+	-/+	-/+	+/-	+/+	+/+
Luc. (L)						
Opac.(O)	O/O	-/O	O/-	O/O	O/O	
Mxd. (M)	-/M					
Punctate	+/-					
Pl.destn.	-/+	+/+				
Ca.destn.	+/+					
Vomer	+	+	+			
Lateral						
Turb Loss	+	+				
B. Destn.	+					
Fr.Sin.Op	+	+	+			
Ex.N.S.T.	+					

APPENDIX IIh

Number	43	44	45	46	47	48
Age	10	8	8	6	13	9
Sex	F	F	M	M	M	F
Durn.	2	1.5	1.5	1	3	4
Uni/Bil						
Clin	U	B	B	U	U	U
Rad	B	B	B	U	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/+	+/+	+/+	+/-	+/+	+/+
Luc. (L)	L/L					
Opac.(O)	(O)/O	O/O	O/-	O/O		
Mxd.(M)	M/M					
I/Oral Caudal						
Turb Loss	-/+	(+)/-	+/+	+/-	+/+	+/+
Luc. (L)						
Opac.(O)	-/O	O/-	O/O	O/-	O/O	O/O
Mxd. (M						
Punctate	+/+					
Pl.destn.	-/+	+/-	+/-	+/-		
Ca.destn.	-/+	+/-				
Vomer	+	+	+	+		
Lateral						
Turb Loss	+	+	+	+		
B. Destn.	+	+				
Fr.Sin.Op	+	+	+	+		
Ex.N.S.T.	+	+				

APPENDIX III

Number	49	50	51	52	53	54
Age	13	10	12	1.3	8	10
Sex	F	F	F	M	F	M
Durn.	.5	2.5	1.5	.25	3	2
Uni/Bil						
Clin	U	U	U	U	U	U
Rad	B	U	B	U	U	U
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/-	+/-	+/+	+/-	+/-	
Luc. (L)						
Opac.(O)	O/(O)	O/-	-/O	O/-	O/-	
Mxd.(M)	M/-					
I/Oral Caudal						
Turb Loss	+/-	+/-	+/+	+/-	+/-	+/-
Luc. (L)						
Opac.(O)	O/-	O/-	O/O	O/-	O/-	O/-
Mxd. (M)						
Punctate	+/-	+/-	+/+			
Pl.destn.	+/-					
Ca.destn.	+/-					
Vomer	+	+	+			
Lateral						
Turb Loss	+	+	+	+	+	
B. Destn.	+					
Fr.Sin.Op	+	+	+	+	+	
Ex.N.S.T.	+	+				

APPENDIX IIj

Number	55	56	57	58	59	60
Age	9	9	8	11	11	12
Sex	F	F	M	M	M	M
Durn.	2	3	3	2	2	4
Uni/Bil						
Clin	U	U	U	B	U	U
Rad	U	B	U	B	B	B
I/Oral Rostral	R/L	R/L	R/L	R/L	R/L	R/L
Turb Loss	+/-	+/+	-/+	+/+	+/-	+/+
Luc. (L)						
Opac.(O)	O/-	O/O	O/-	O/O		
Mxd. (M)	-/M	M/M	-/(M)			
I/Oral Caudal						
Turb Loss	+/-	+/+	-/+	+/+	+/-	-/+
Luc. (L)						
Opac.(O)	O/-	O/O	O/O	O/-	-/O	
Mxd. (M)	-/M	-/(M)				
Punctate	+/-	+/+	+/-			
Pl.destn.	+/-	+/-				
Ca.destn.	+/-	-/+				
Vomer	+	+	+			
Lateral						
Turb Loss	+	+	+	+		
B. Destn.	+	+				
Fr.Sin.Op	+	+	+			
Ex.N.S.T.	+					

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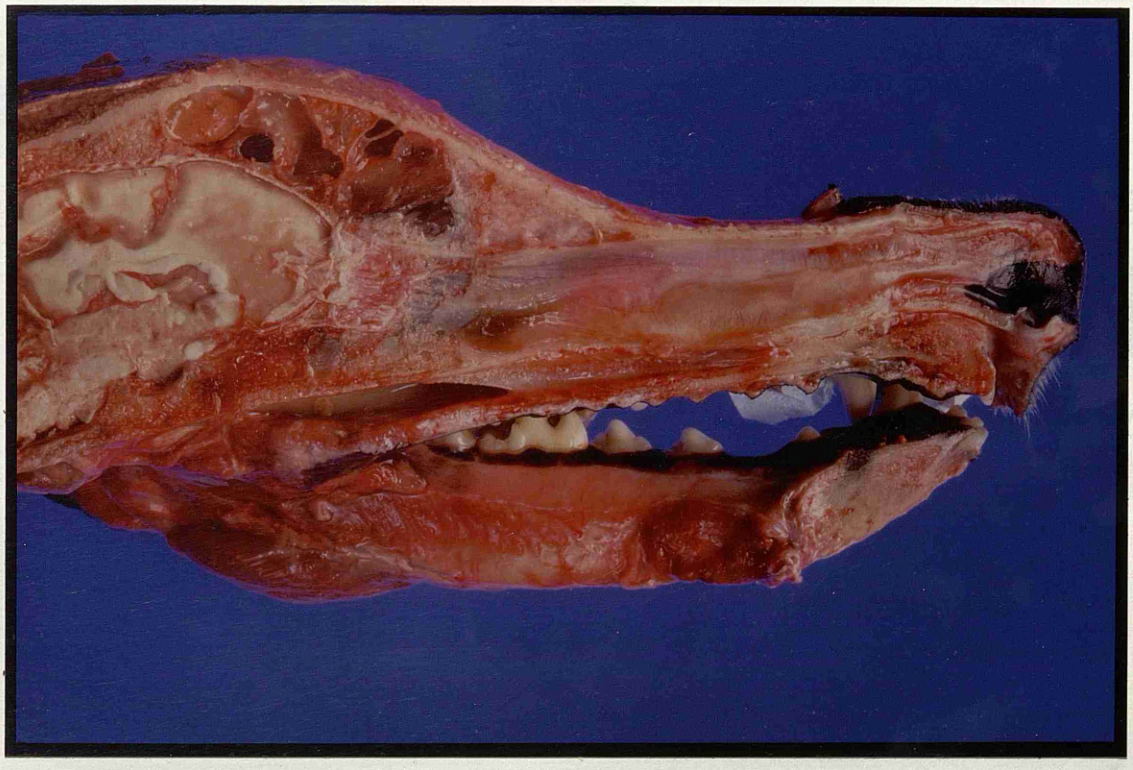

Fig. 1a Sagittal section of a canine skull with the septum and vomer present.

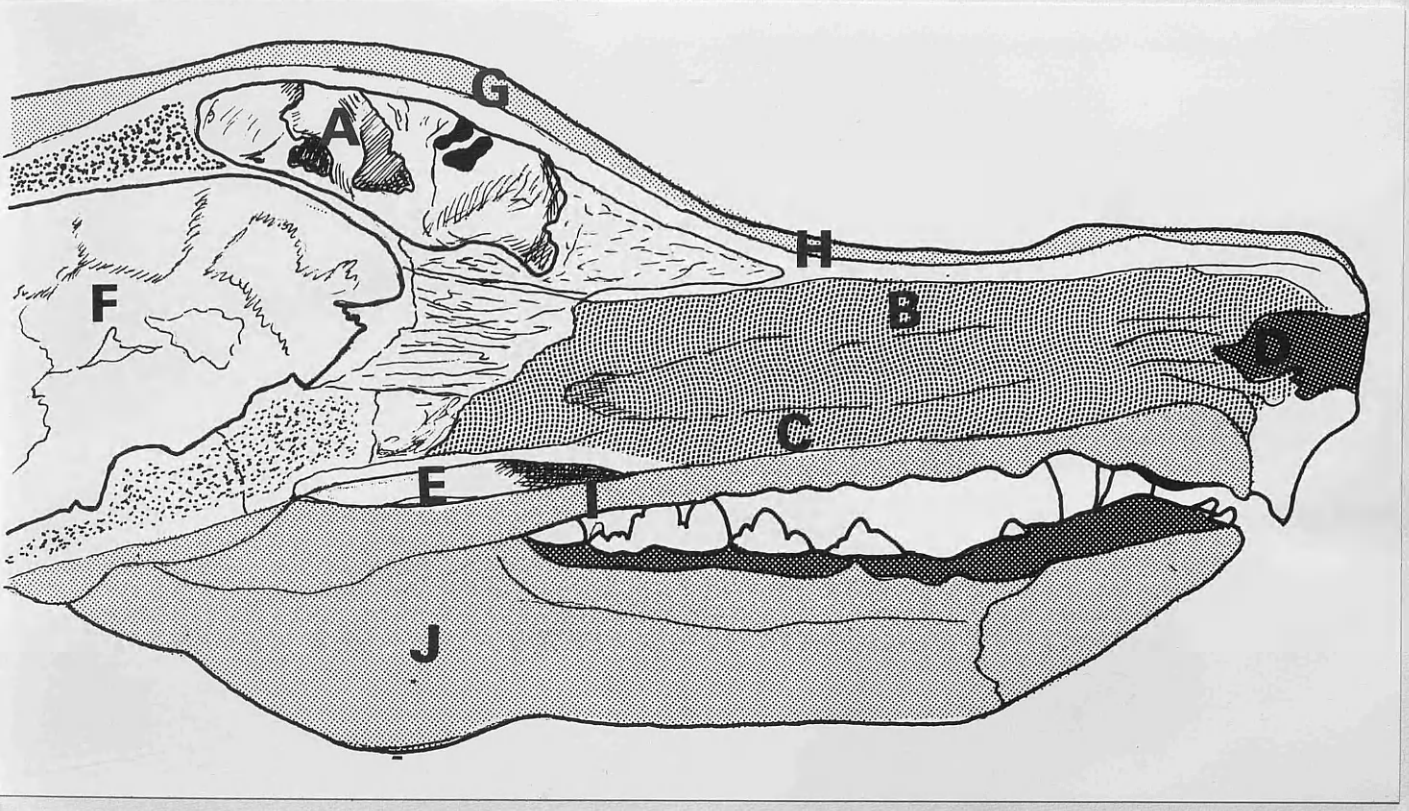

Fig. 1b Labelled diagram of Fig. 1a

A – Frontal sinus
B – Nasal septum
C – Vomer bone
D – Nares
E – Nasopharynx
F – Cranial cavity
G – Frontal bone
H – Nasal bone
I – Hard palate
J – Mandible

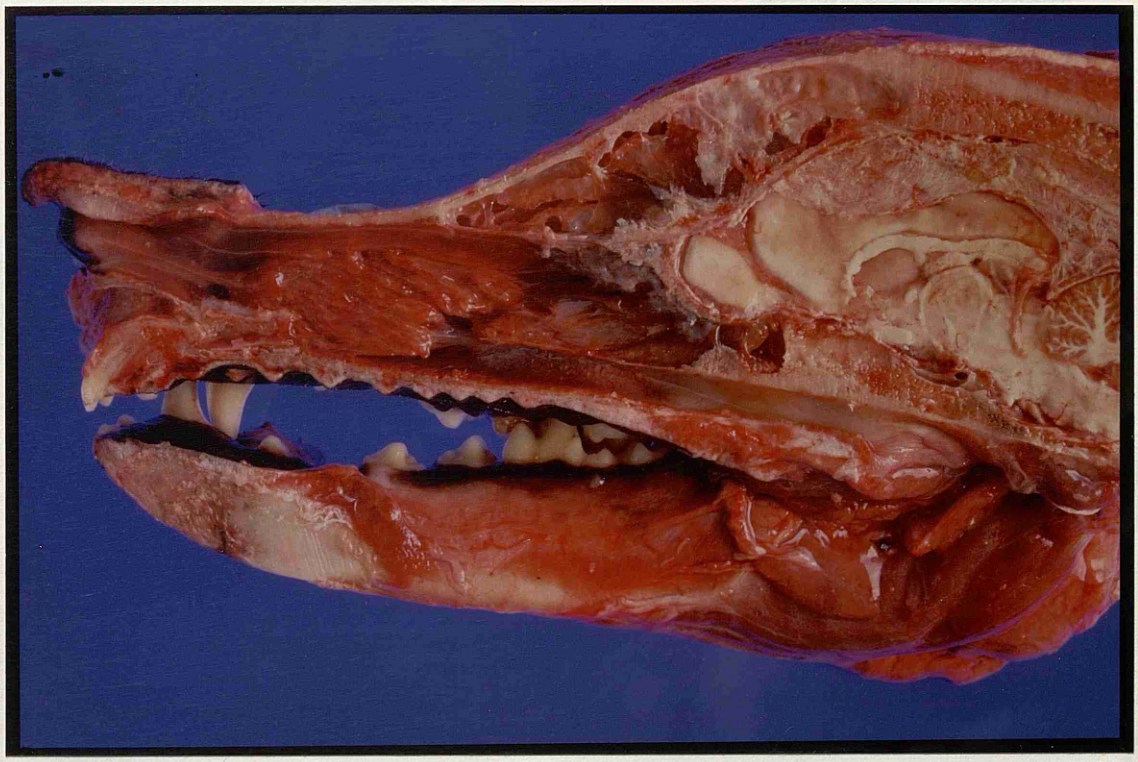

Fig. 2a Sagittal section of a canine skull with the septum and vomer removed.

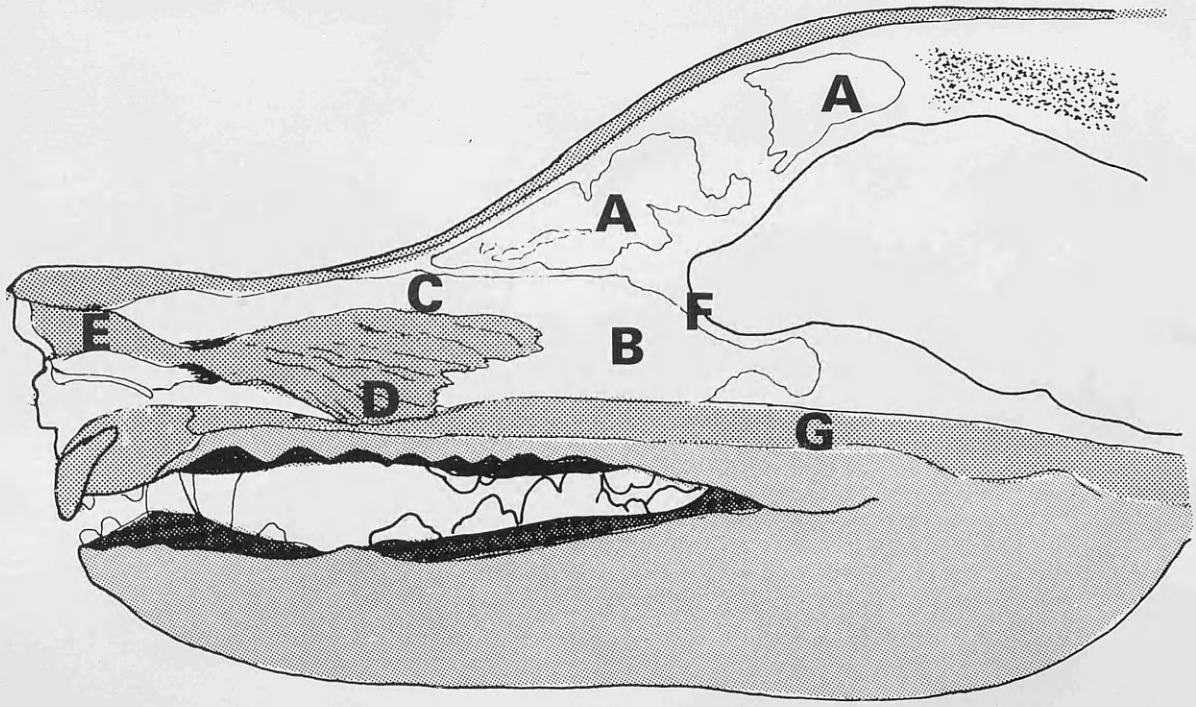

Fig.2b Labelled diagram

A - Frontal sinus
B - Ethmoidal concha
C - Dorsal nasal concha
D - Ventral nasal concha
E - Nares
F - Cribriform plate
G - Nasopharynx

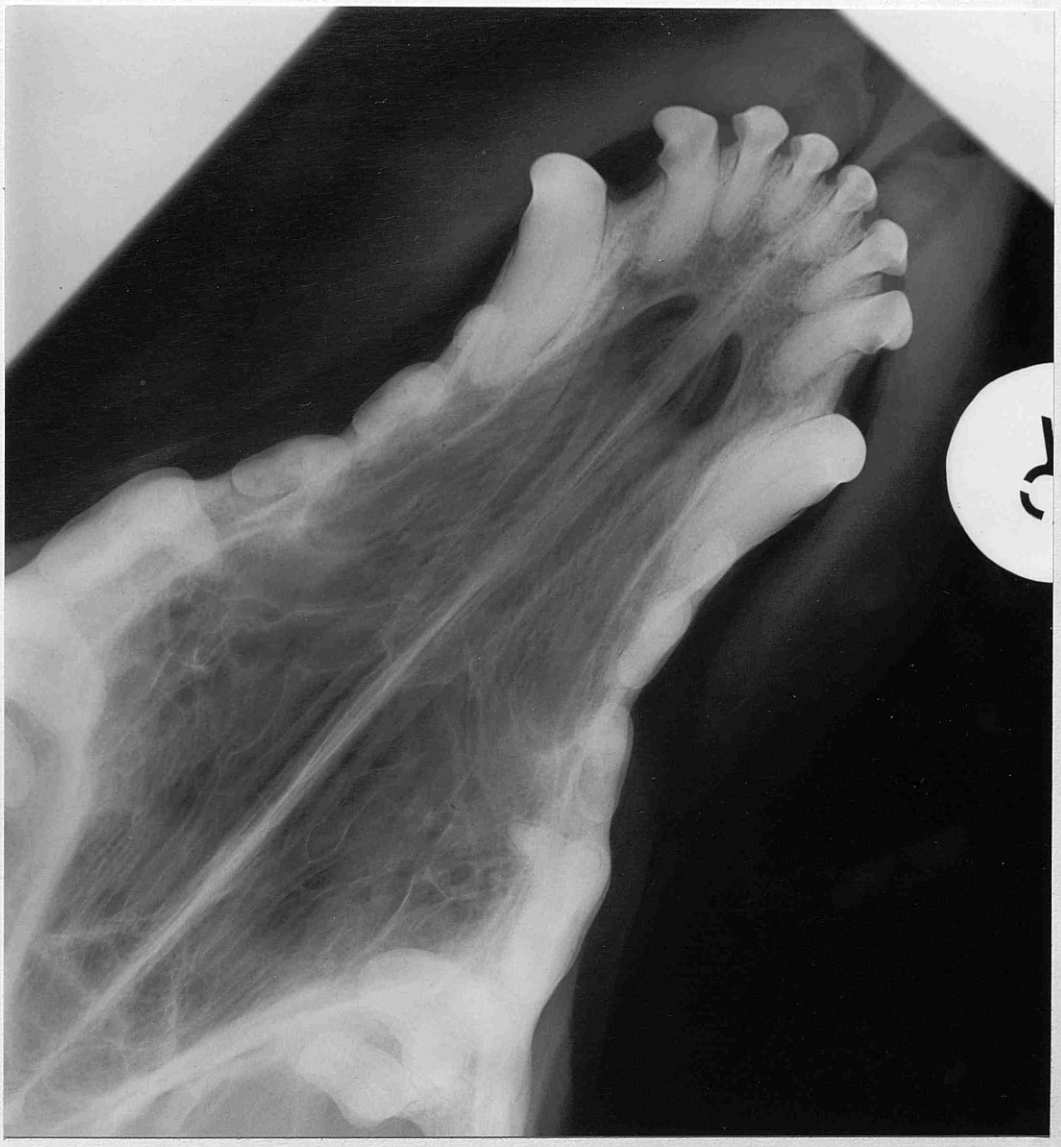

Fig. 3a Dorso-ventral intra-oral radiograph of a canine nasal cavity.

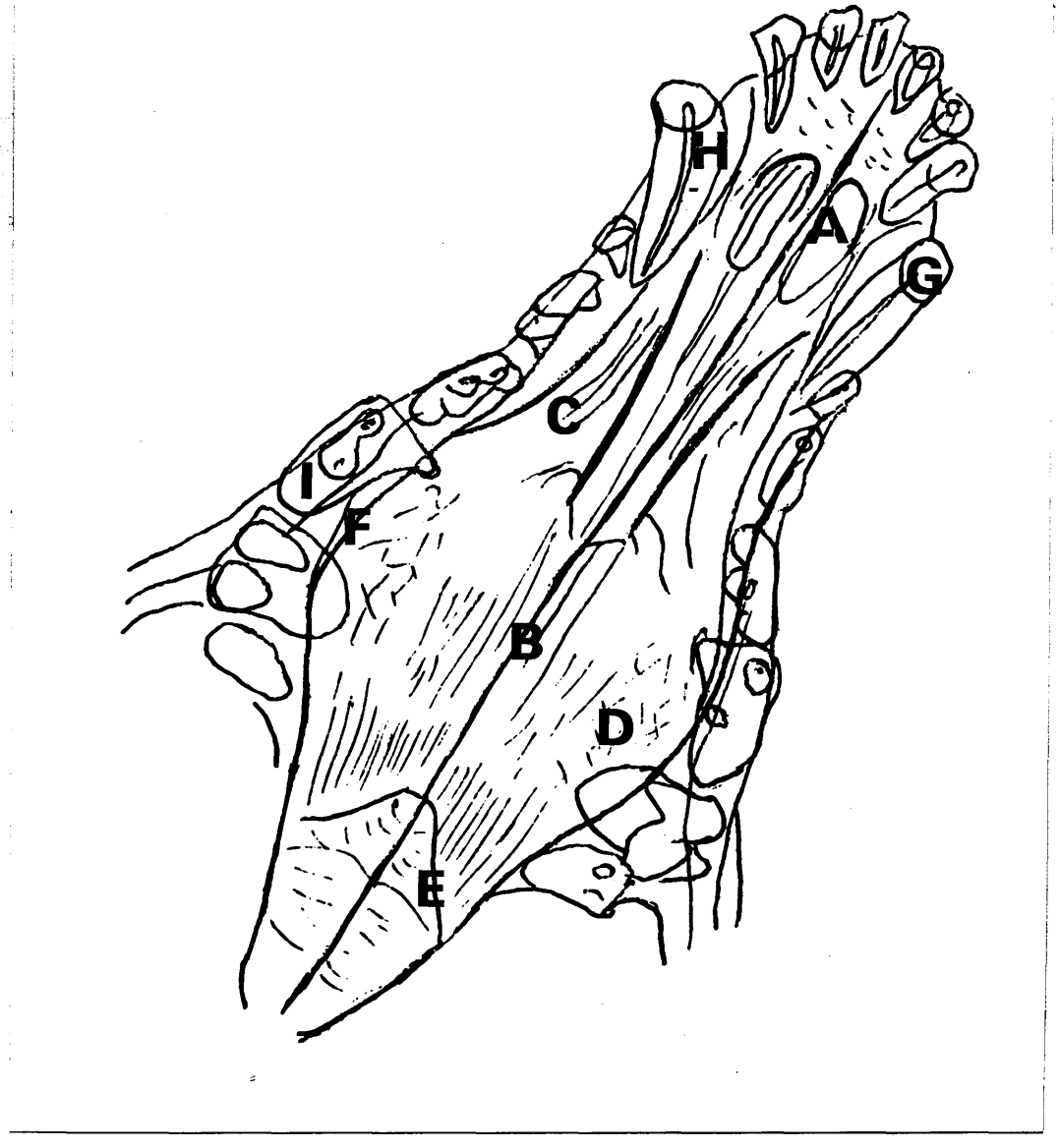

Fig. 3b Labelled diagram

A – Palatine Fissure
B – Nasal septum/Vomer bone
C – Nasal concha
D – Ethmoidal concha
E – Frontal sinus
F – Lat. wall nasal cavity
G – Incisor tooth
H – Canine tooth
I – Forth premolar

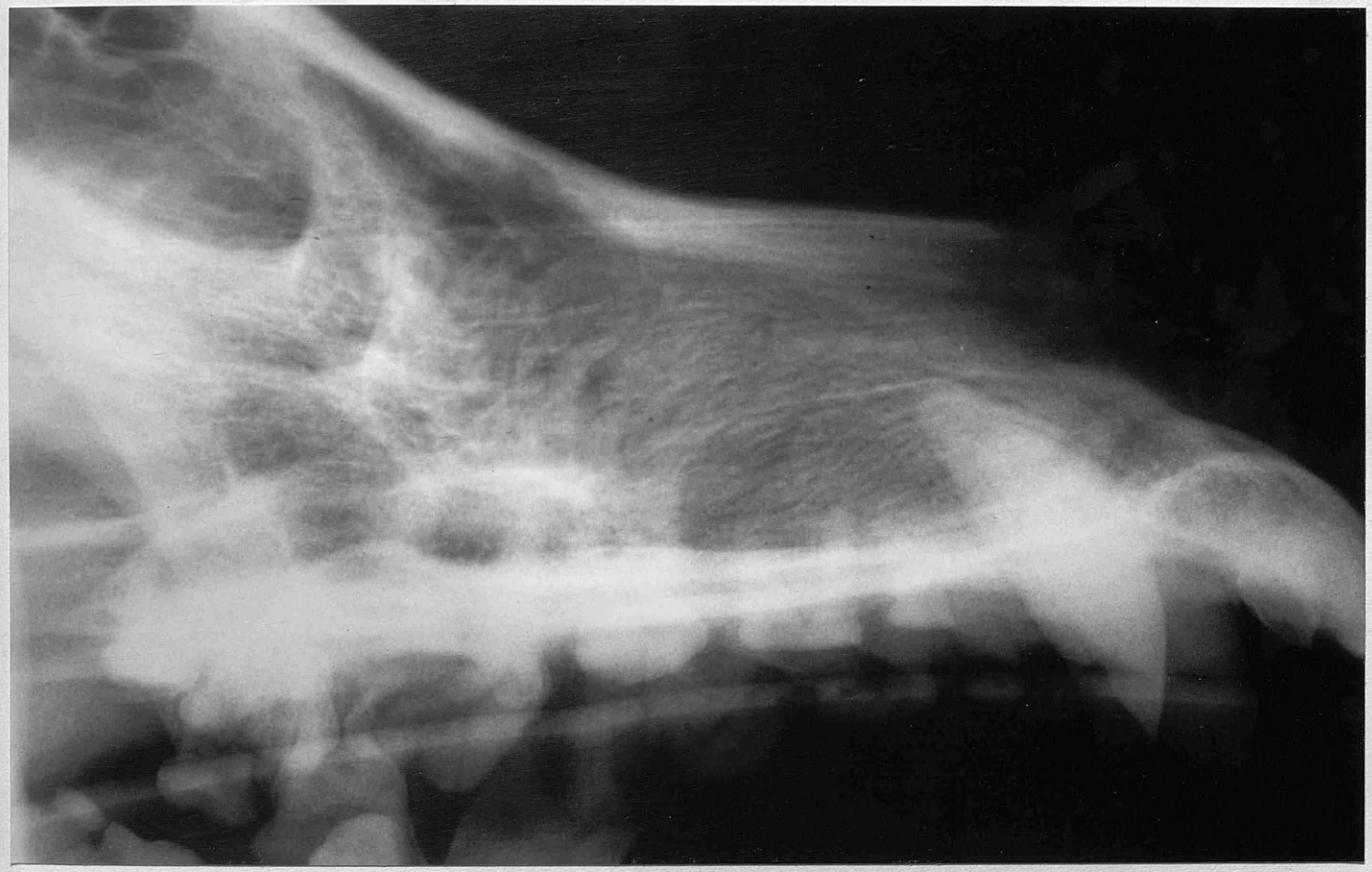

Fig. 4a Lateral radiograph of a canine nasal cavity

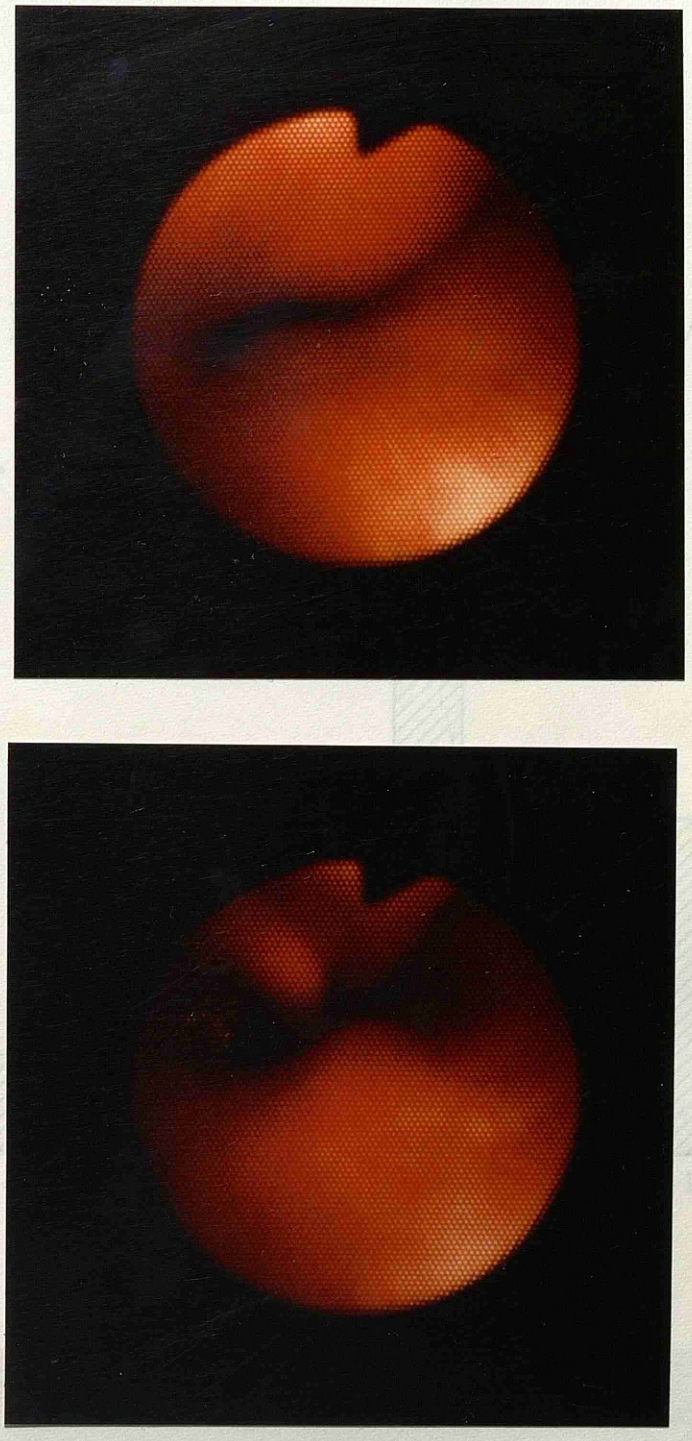

Fig. 5 Normal endoscopic appearance of the nasal concha

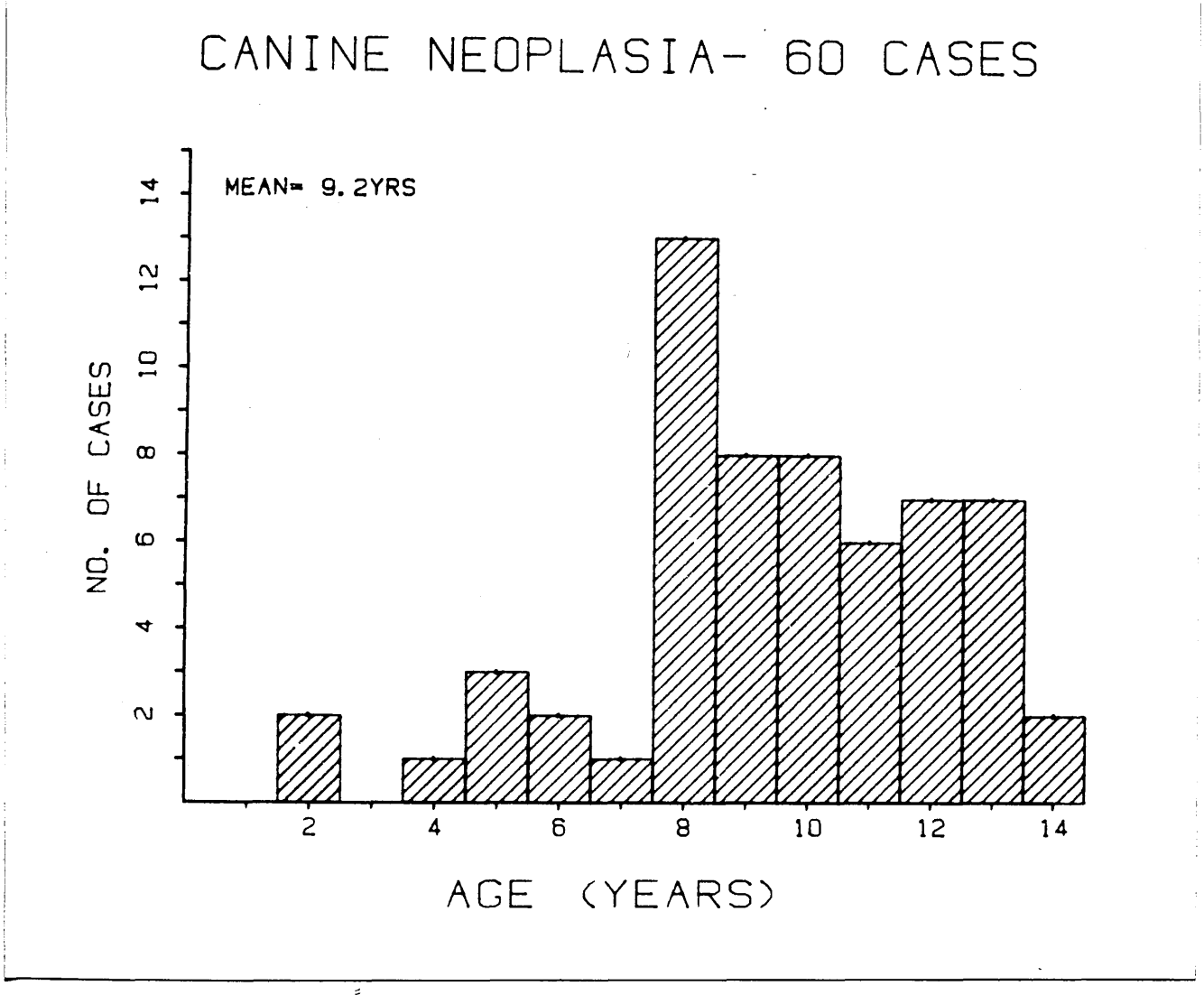

Fig. 6 Histogram showing the age distribution of sixty cases of nasal neoplasia seen at Glasgow University Veterinary Hospital between August 1973 and September 1985.

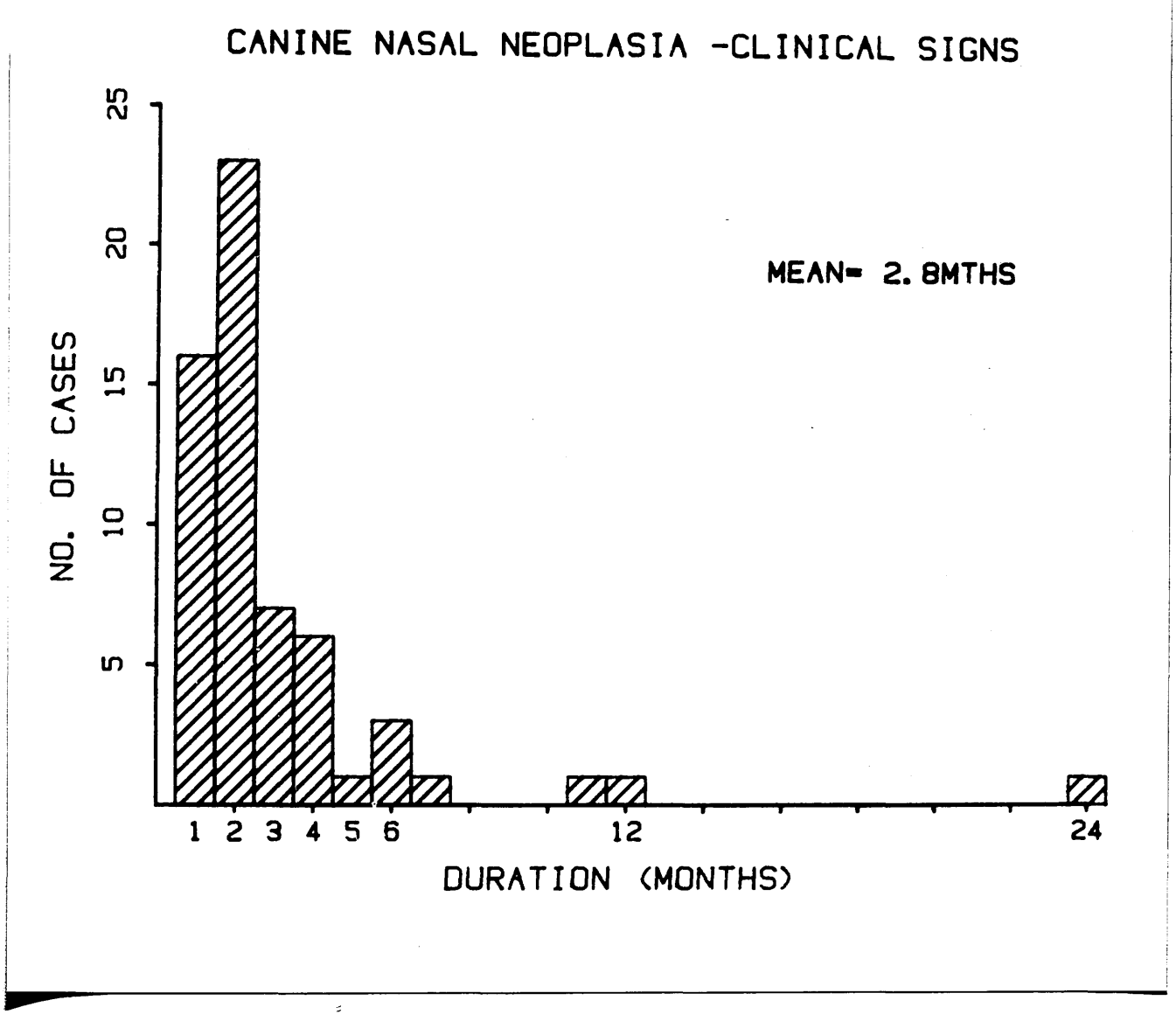

Fig. 7 Histogram showing the duration of clinical signs of sixty cases of nasal neoplasia seen at Glasgow University Veterinary Hospital between August 1973 and September 1985.

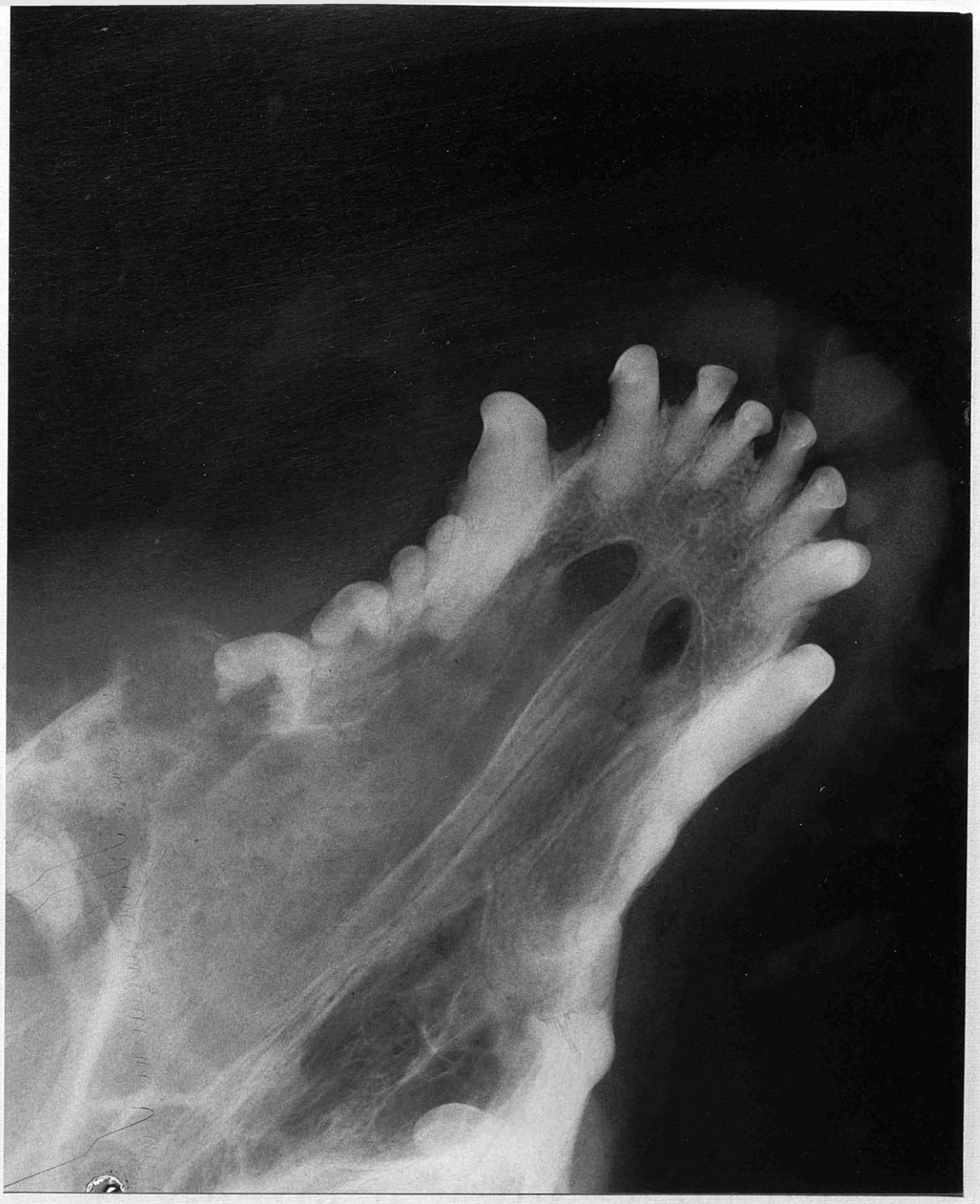

Fig. 8 D.V. intra-oral radiograph of case 49 (Appendix II Bilateral nasal neoplasia) showing increased opacification, turbinate destruction, plate destruction, case destruction, punctate lucencies and areas of vomer absence. A soft tissue swelling can also be appreciated laterally.

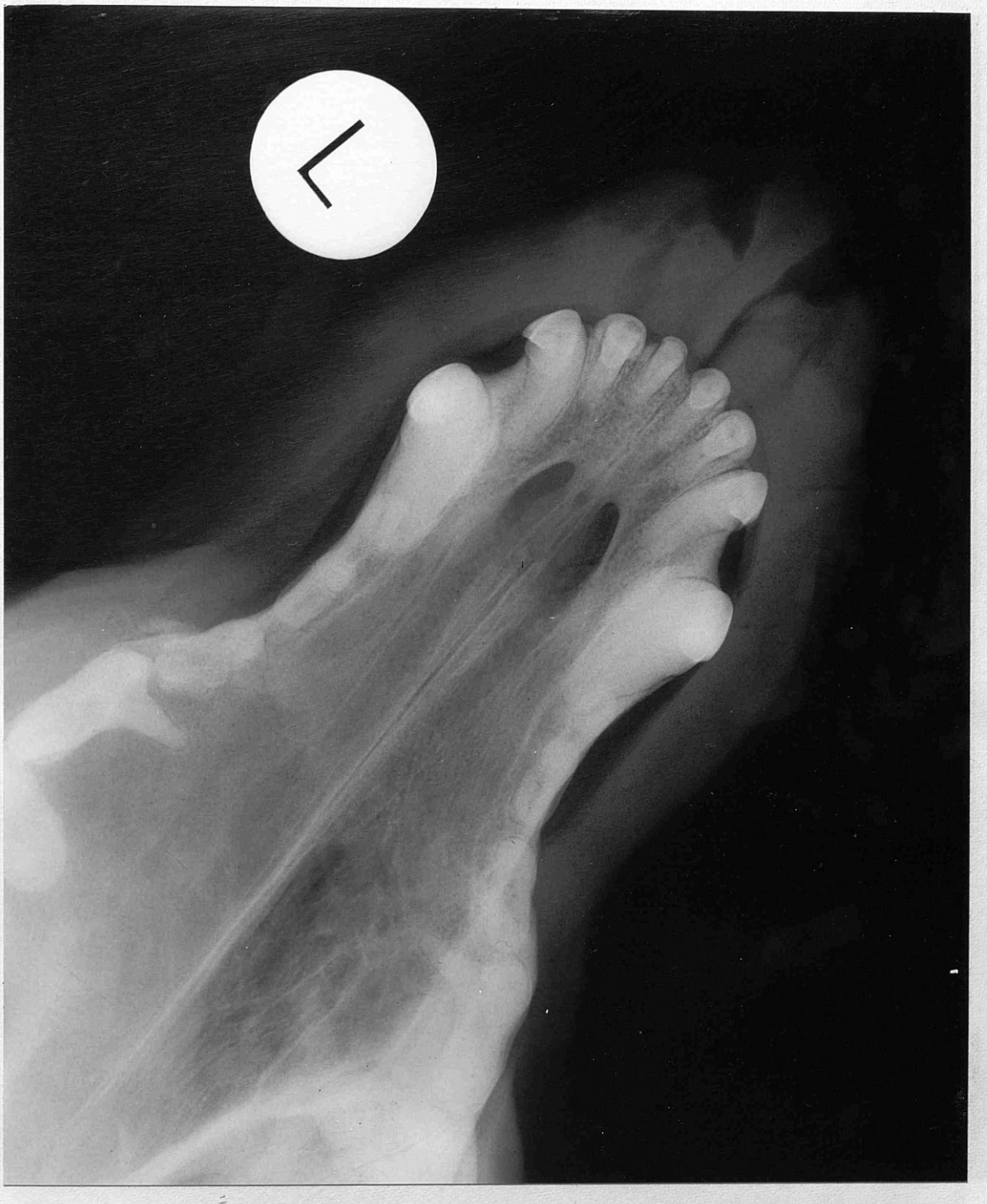

Fig. 9 D.V. intra-oral radiograph of case 7 (appendix II - Unilateral nasal neoplasia) showing increased opacification, turbinate destruction and absence of the vomer caudally.

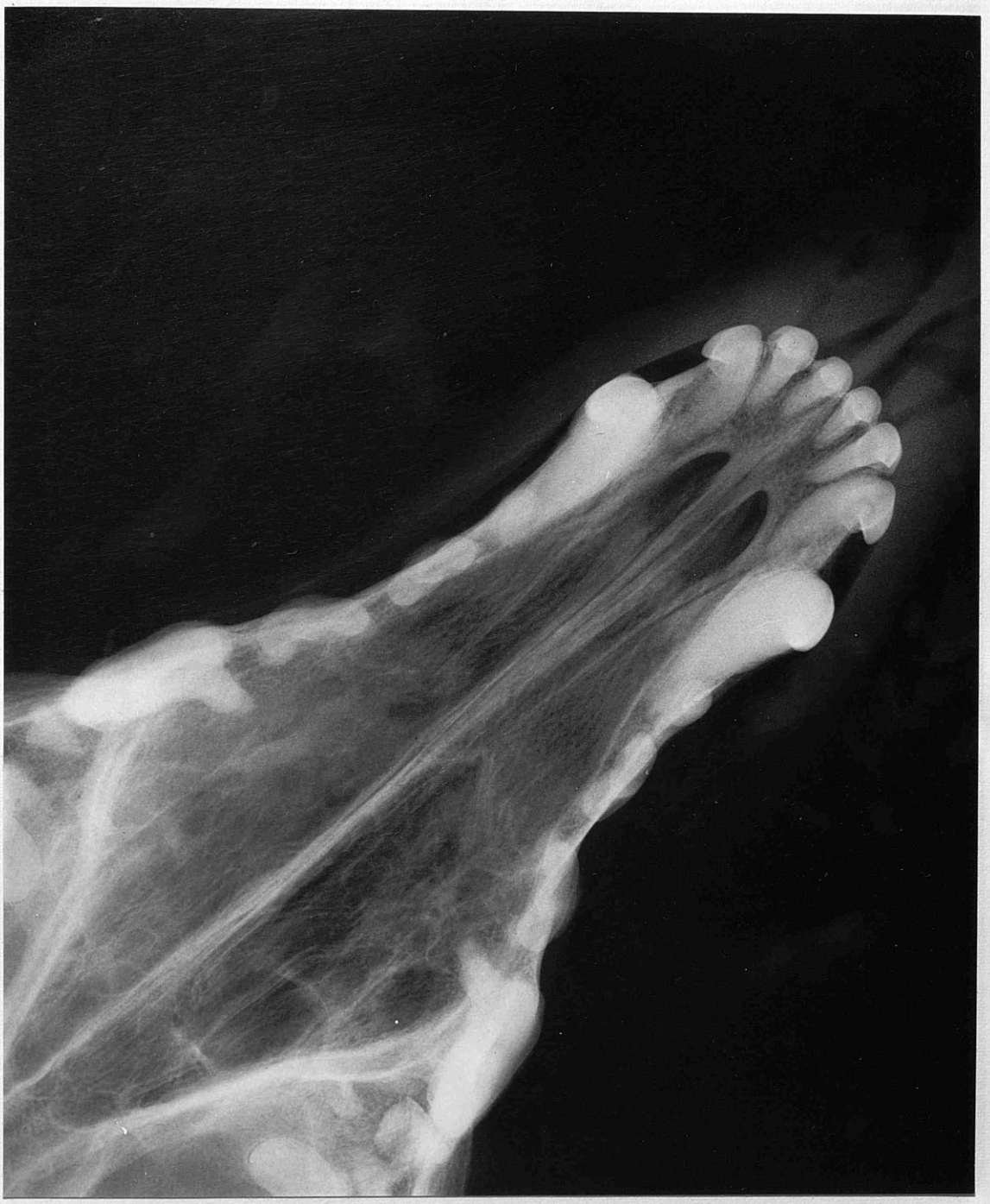

Fig. 10 D.V. intra-oral radiograph of case 20 (Appendix II - Unilateral nasal neoplasia) showing minimal changes. There is focal turbinate destruction and a mixed density pattern caudally.

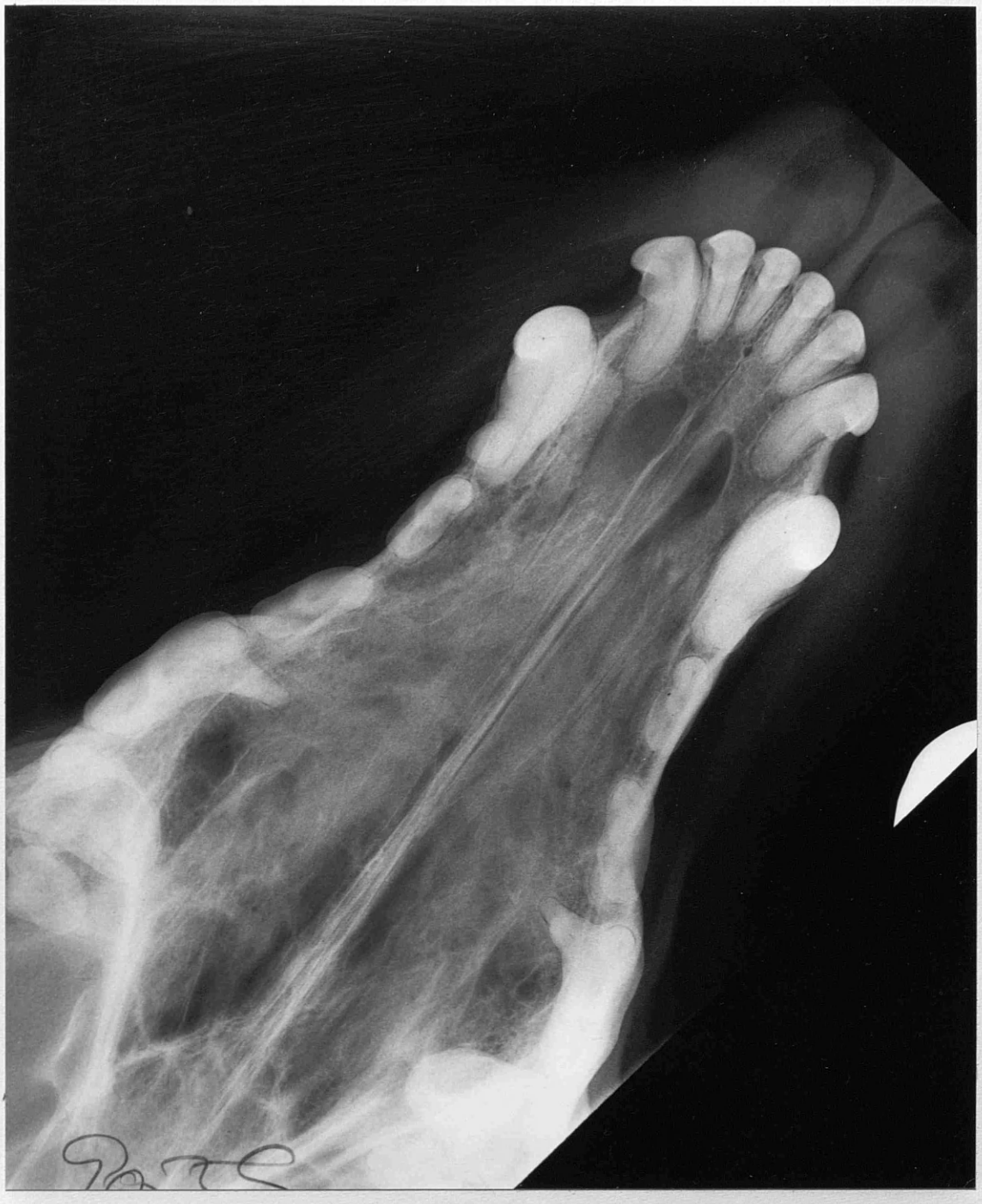

Fig. 11 D.V. intra-oral radiograph of case 22 (Appendix II - Bilateral nasal neoplasia) showing a mixed density pattern and turbinate destruction rostrally and caudally.

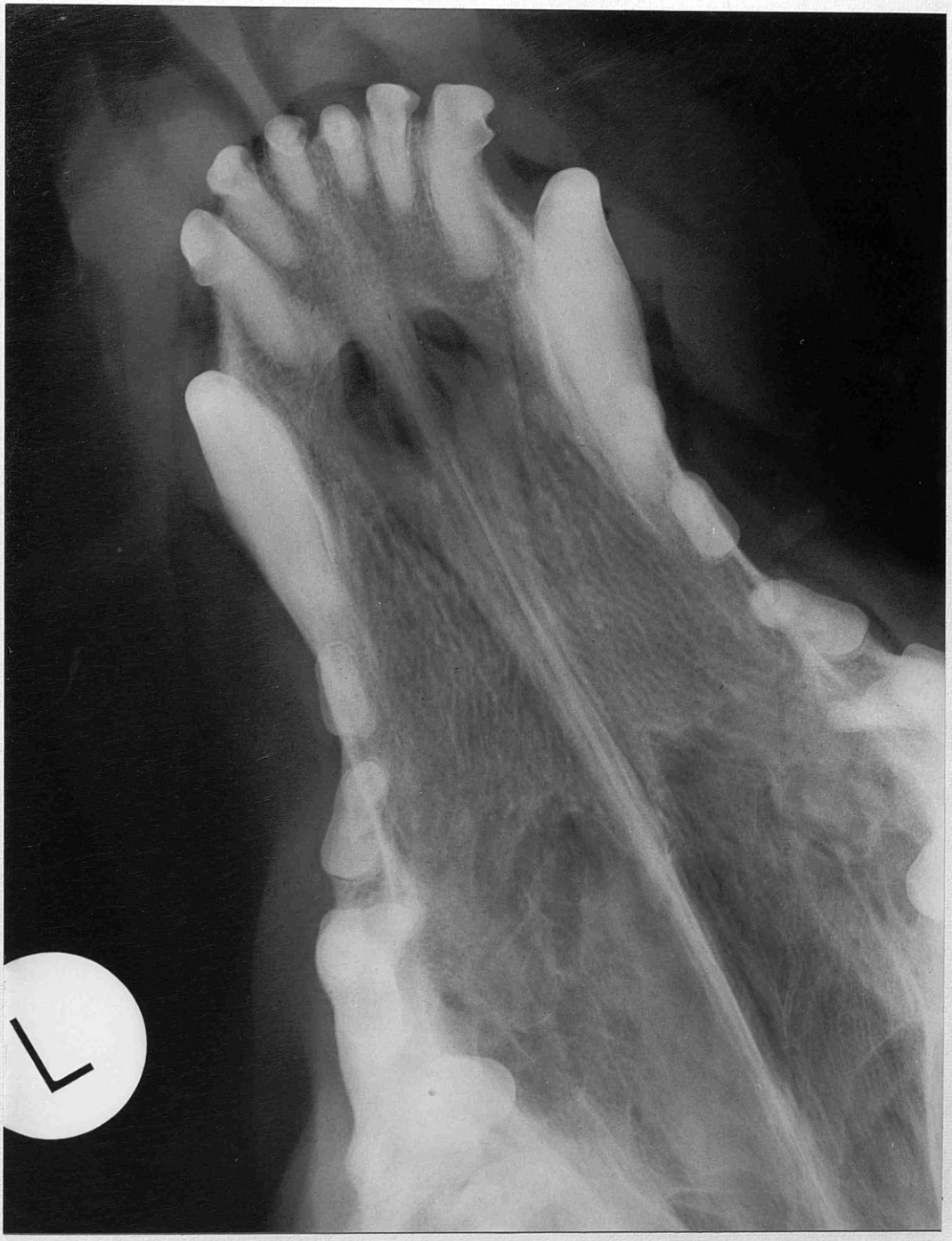

Fig. 12a D.V. intra-oral radiograph of case 38 (Appendix II - unilateral nasal neoplasia) showing minimal early changes. Focal turbinate loss and a mixed density caudally.

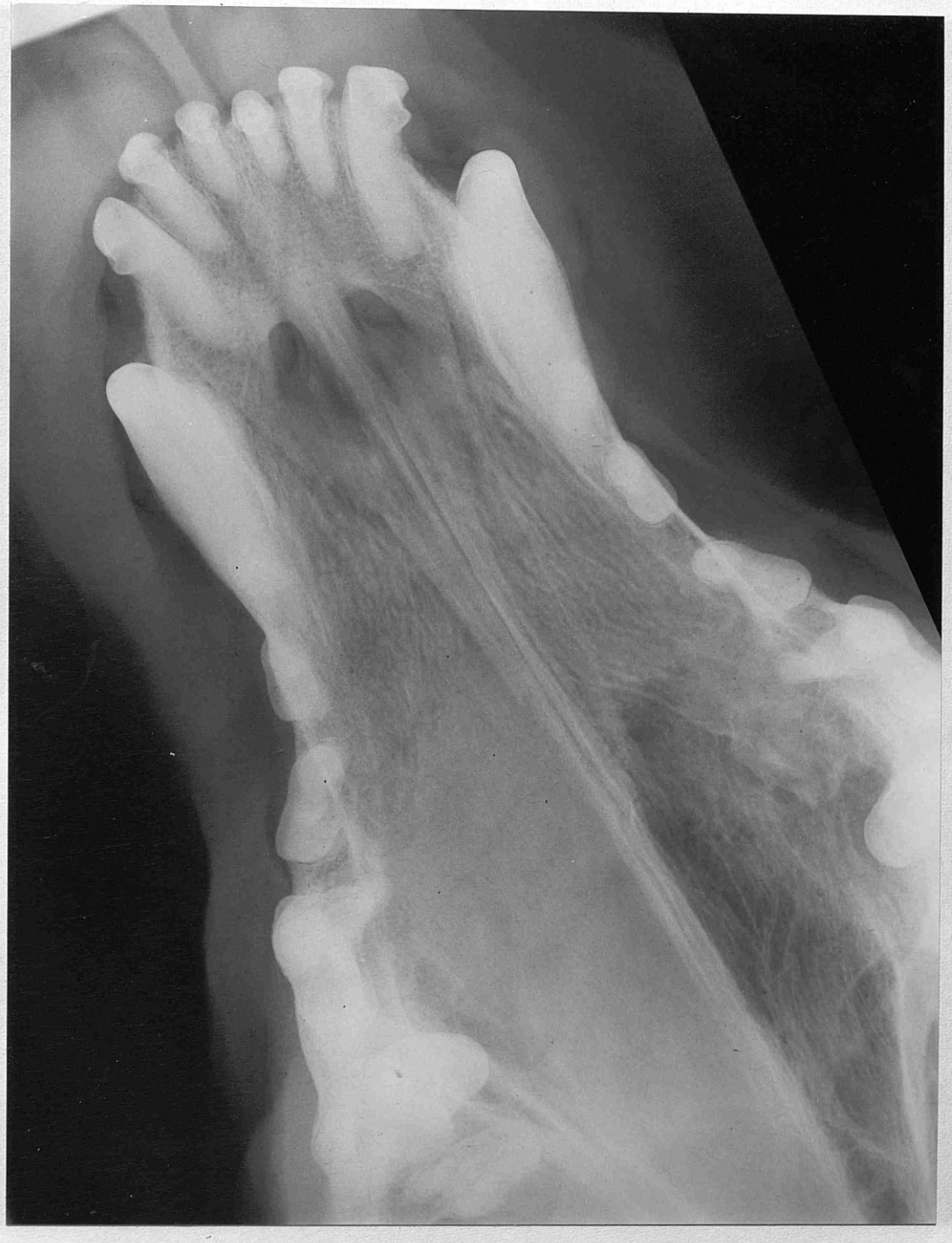

Fig. 12b The same case six weeks later demonstrating the prgression of the radiological changes. There is a marked unilateral caudal opacification with turbinate destruction.

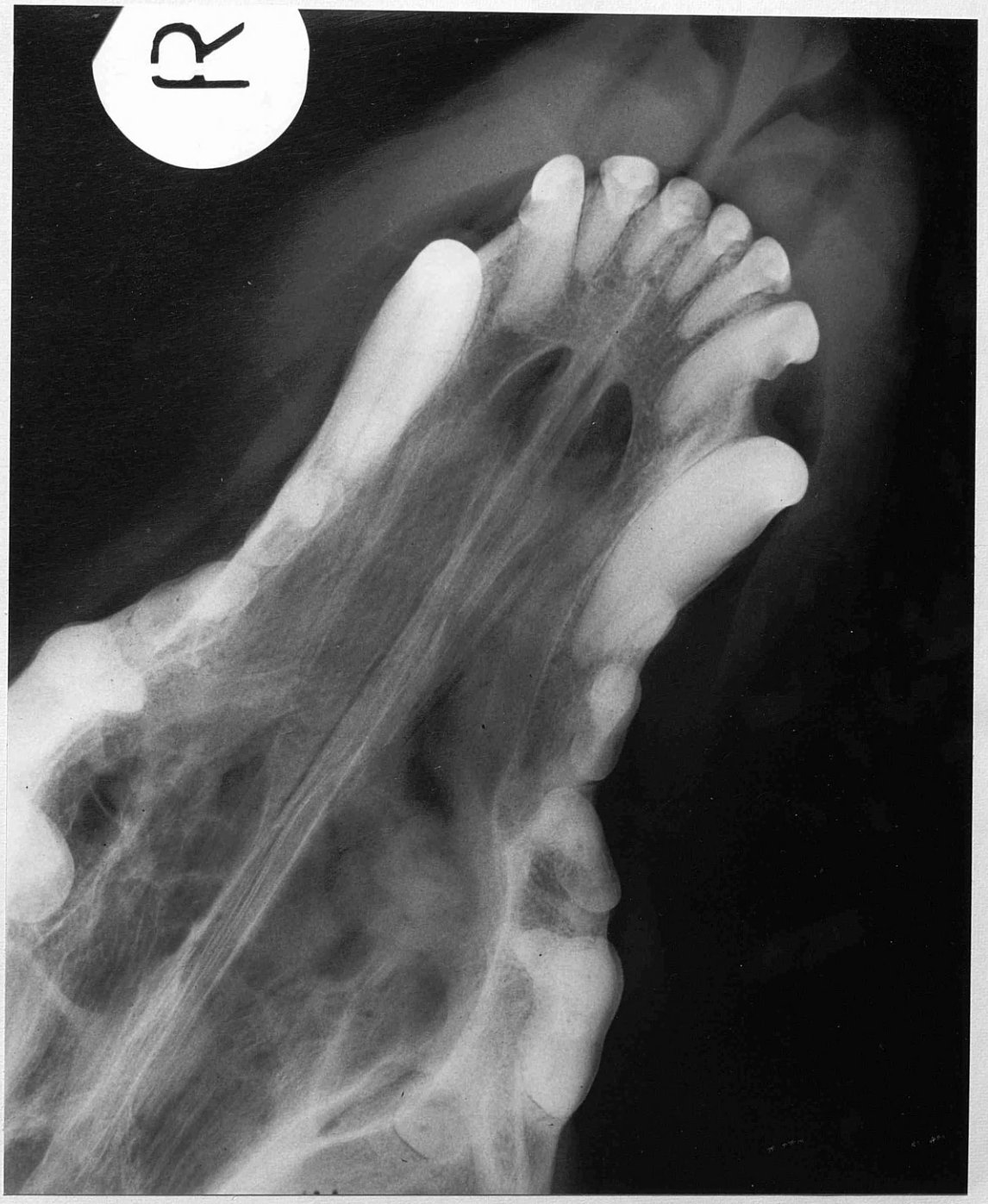

Fig. 13 D.V intra-oral radiograph of case 57 (Appendix II - Unilateral nasal neoplasia) showing a mixed density pattern and turbinate loss both rostrally and caudally.

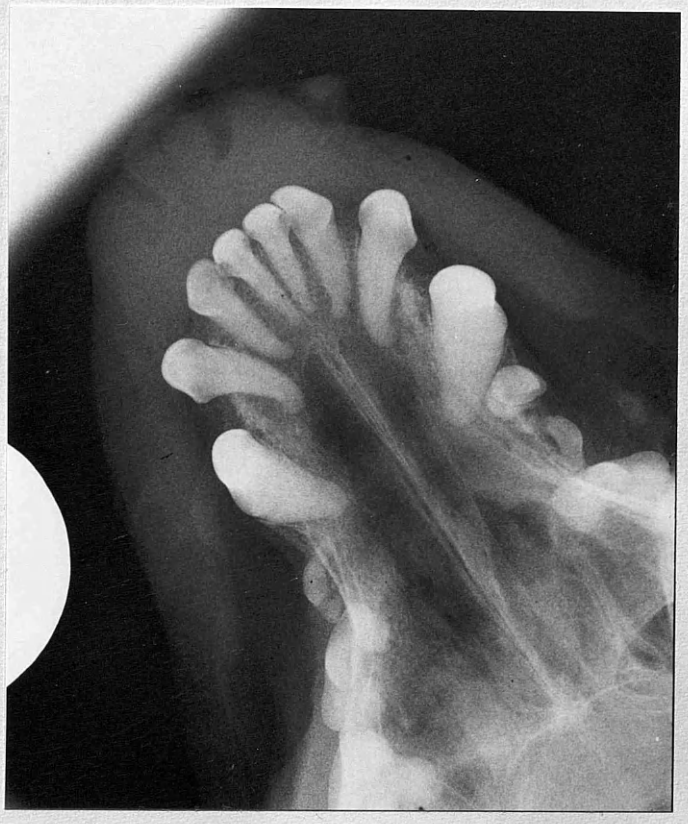

Fig. 14 D.V intra-oral radiograph of case 48 (Appendix II) - Bilateral nasal neoplasia) There is an overall increase in radiolucency particularly rostrally with turbinate destruction and only focal areas of opacification.An appearance which resembles destructive rhinitis.

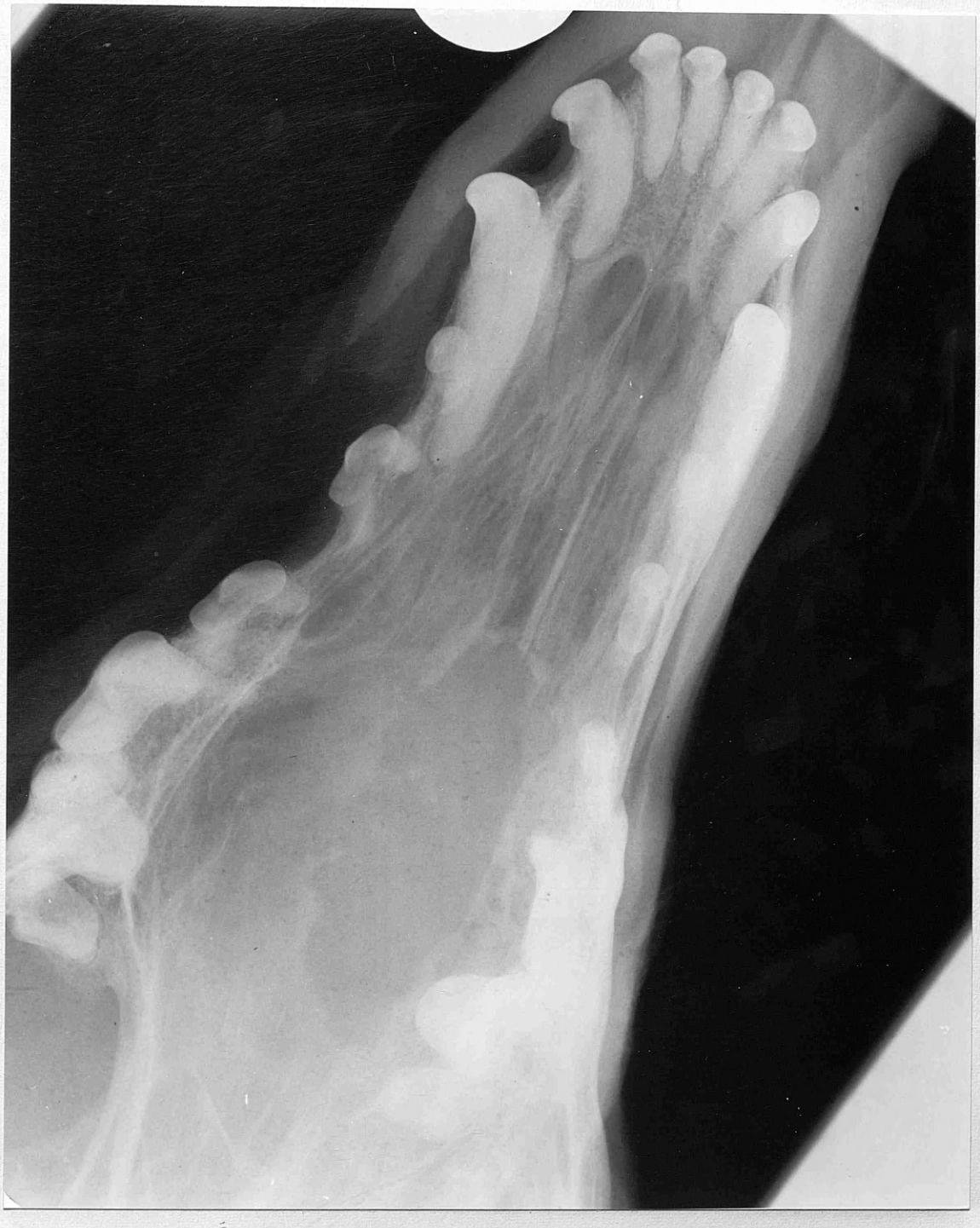

Fig. 15 D.V intra-oral radiograph of case 35 (Appendix II - Bilateral nasal neoplasia) showing a large area of case destruction caudally involving the vomer within an area of opacification. There is also plate destruction and frontal sinus opacification.

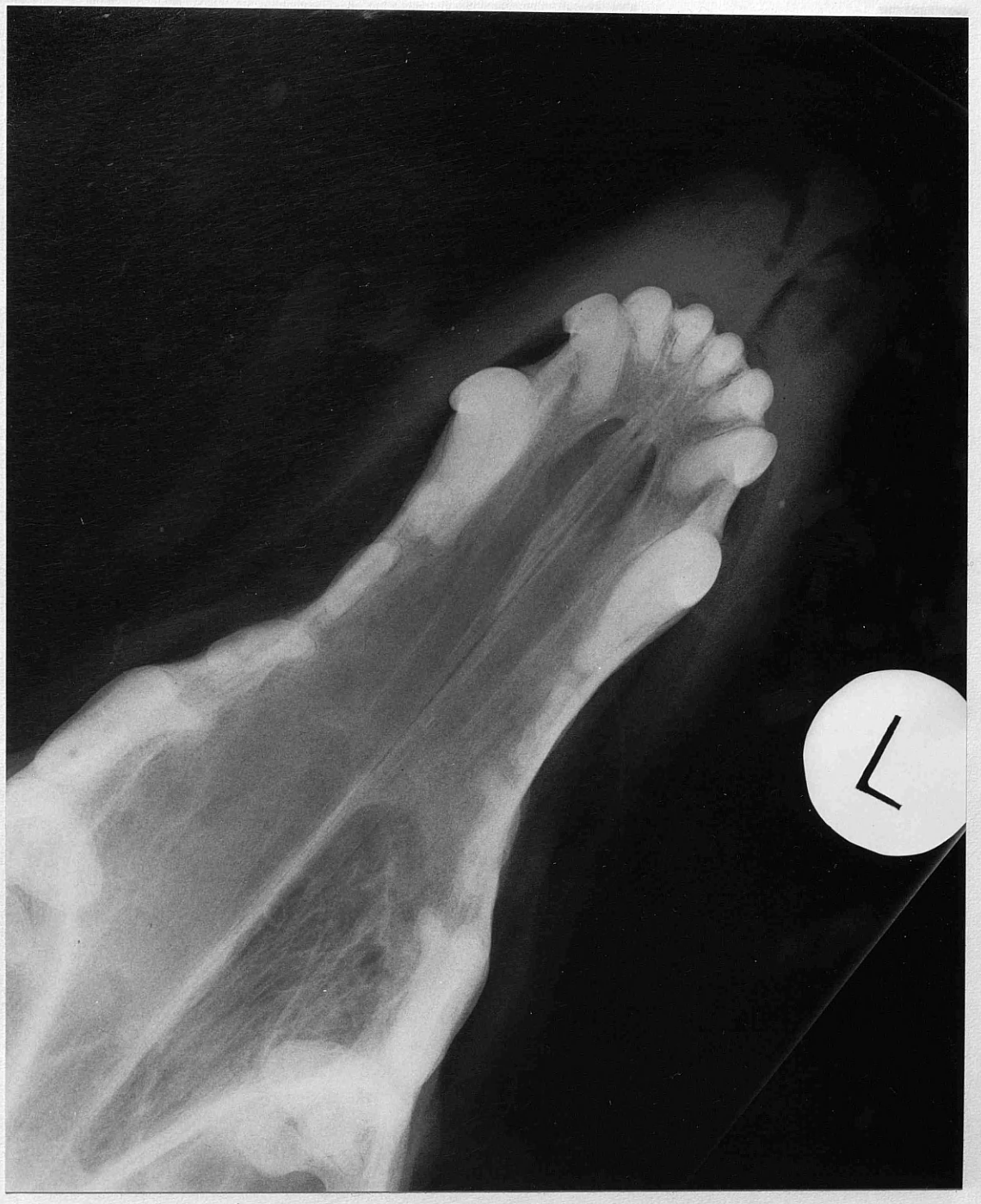

Fig. 16 D.V intra-oral radiograph of case 15 (Appendix II - Bilateral nasal neoplasia) showing increased opacification and turbinate destruction. Absence of the vomer is evident rostrally.

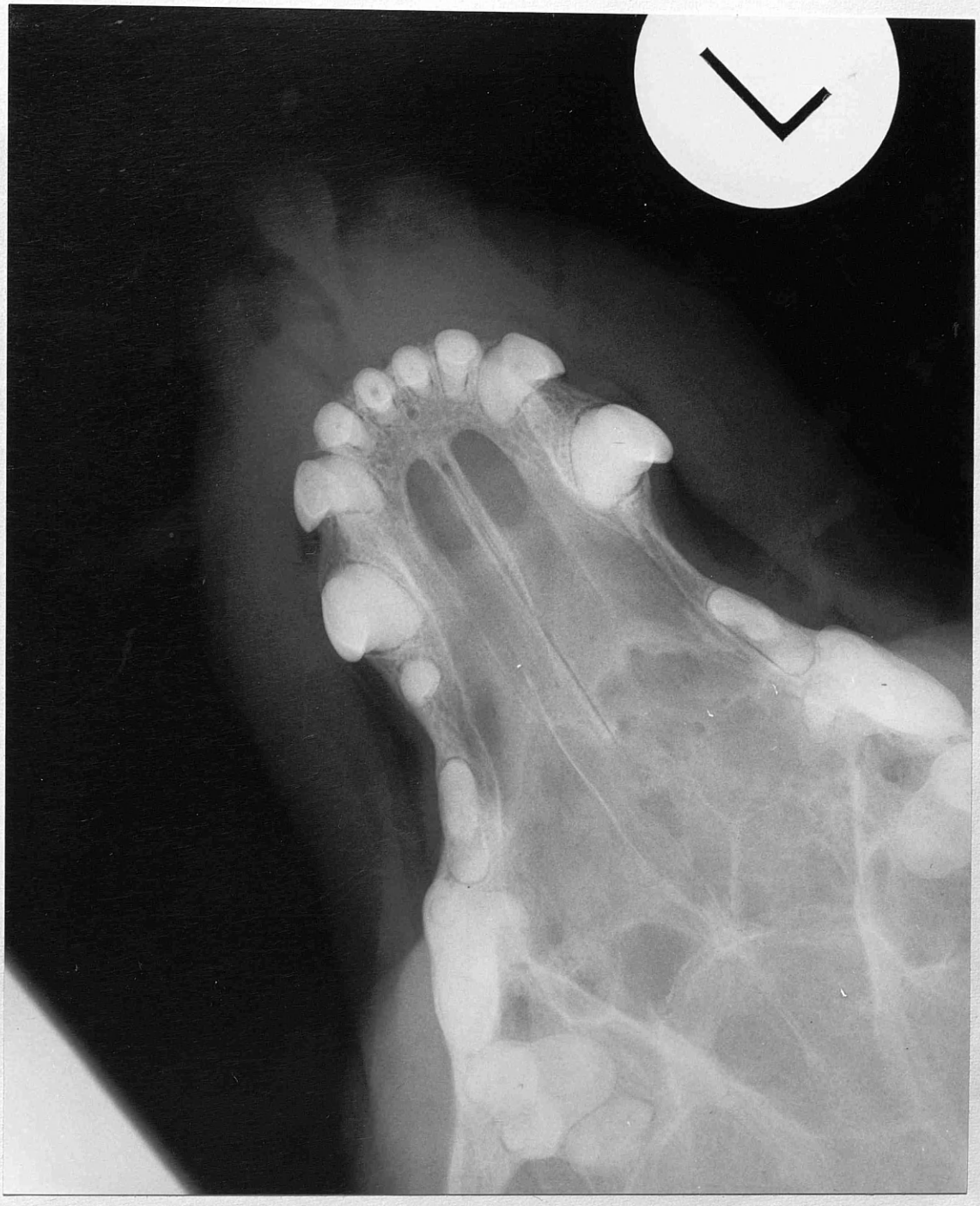

Fig. 17 D.V. intra-oral radiograph of case 41 (Appendix II - Bilateral nasal neoplasia) showing extensive destruction changes namely turbinate, plate and case destruction. Vomer erosion and septal deviation are evident.

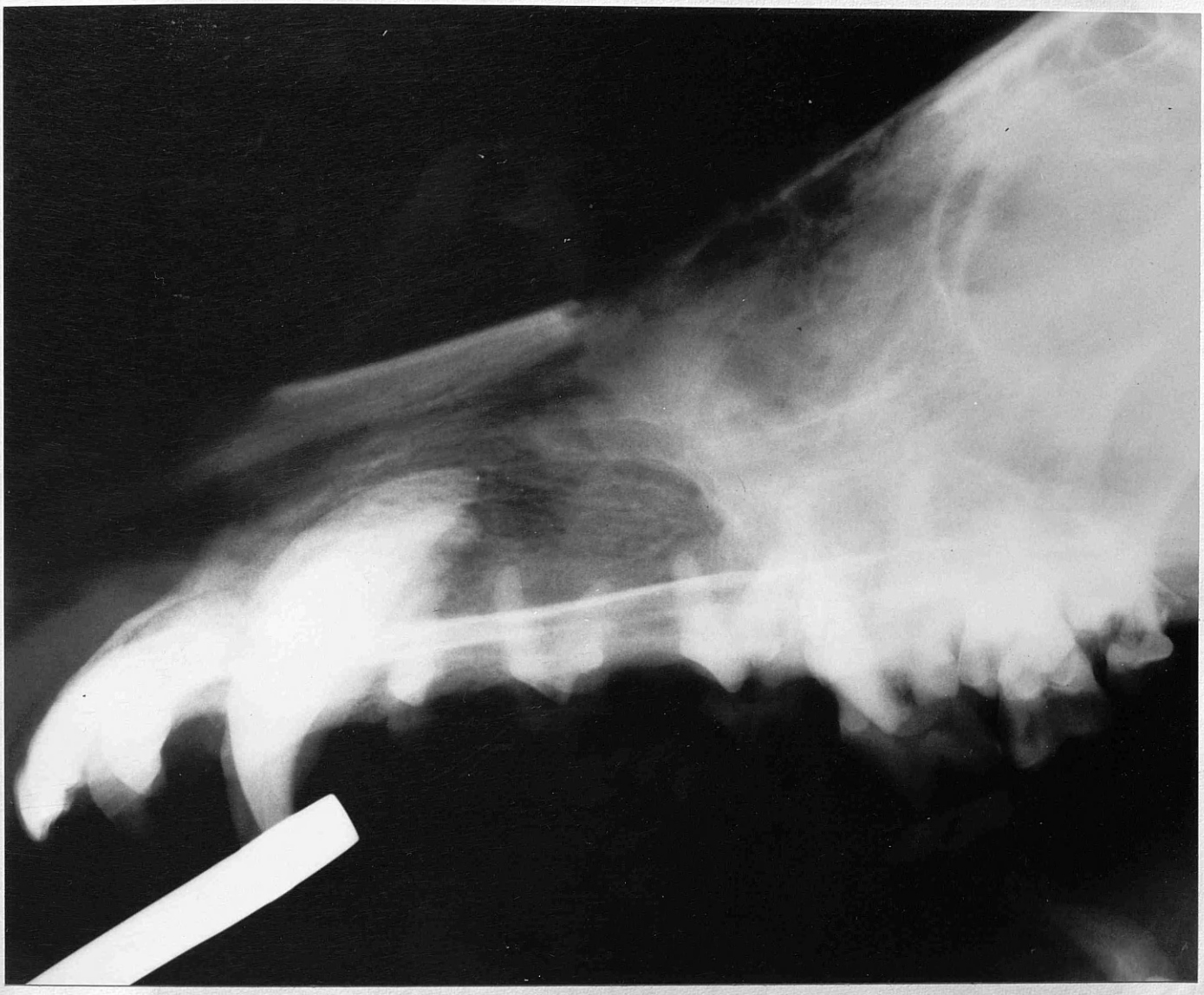

Fig. 18 Lateral radiograph of case 35 (Appendix II - nasal neoplasia) showing nasal and frontal bone destruction. There is also increased opacification of the nasal cavity, turbinate destruction and frontal sinus opacification.

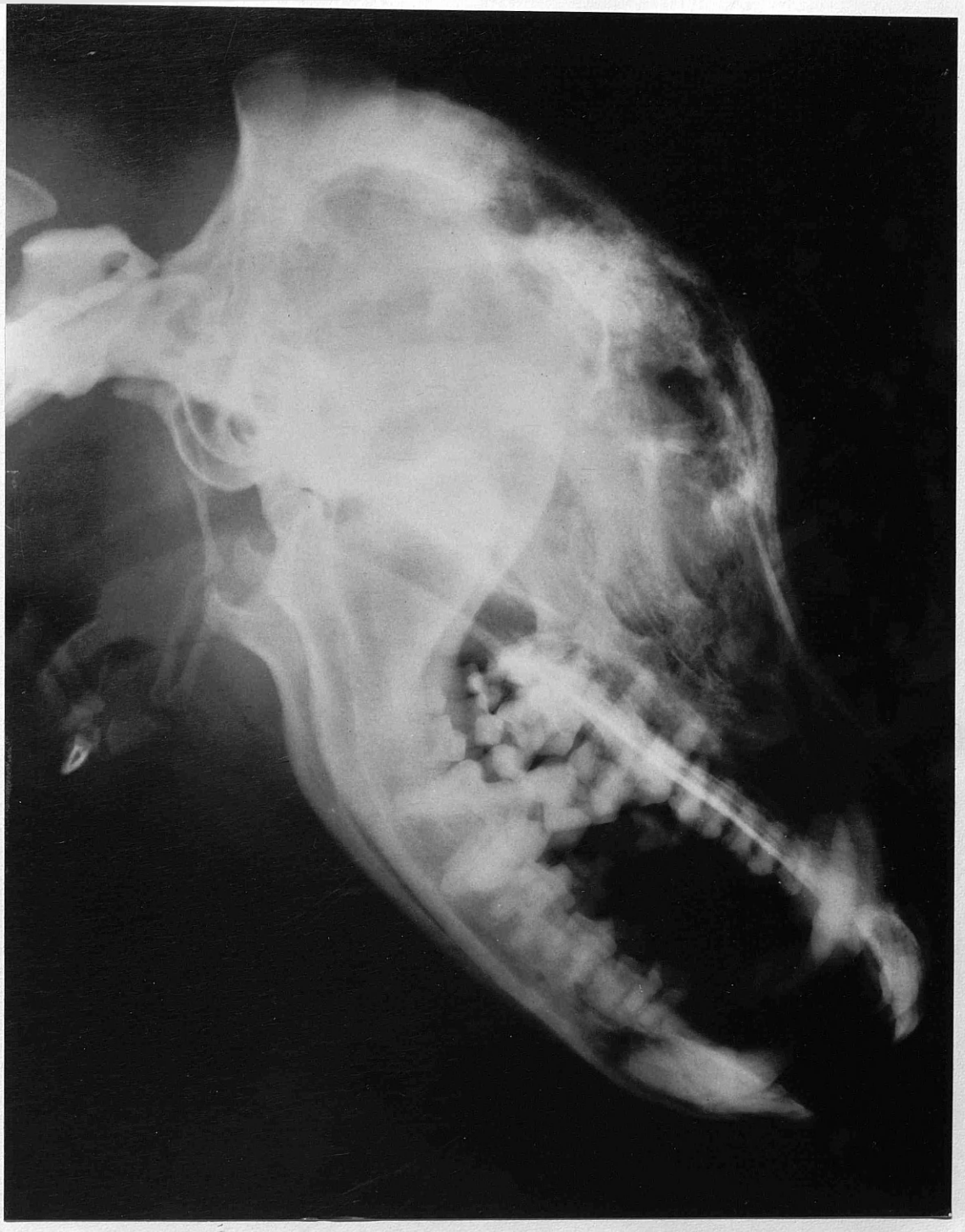

Fig. 19 Lateral radiograph of case 3 (Appendix II - nasal neoplasia) showing frontal bone destruction and diffuse mineralisation. There was also overlying soft tissue swelling, but this cannot be appreciated on the photograph.

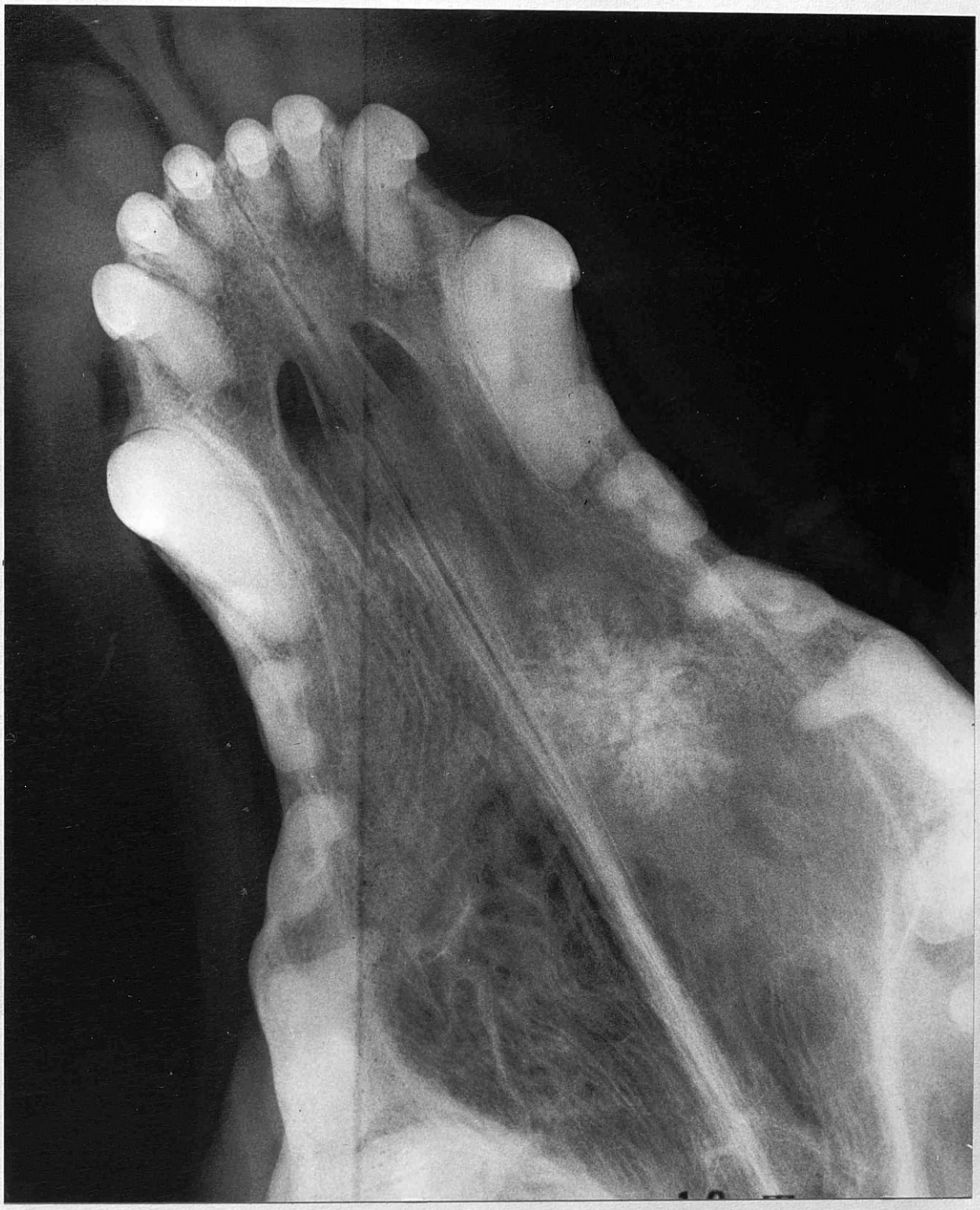

Fig. 20 D.V. intra-oral radiograph of case 23 (Appendix II - Unilateral nasal neoplasia) showing mineralisation, increased opacification and turbinate destruction.

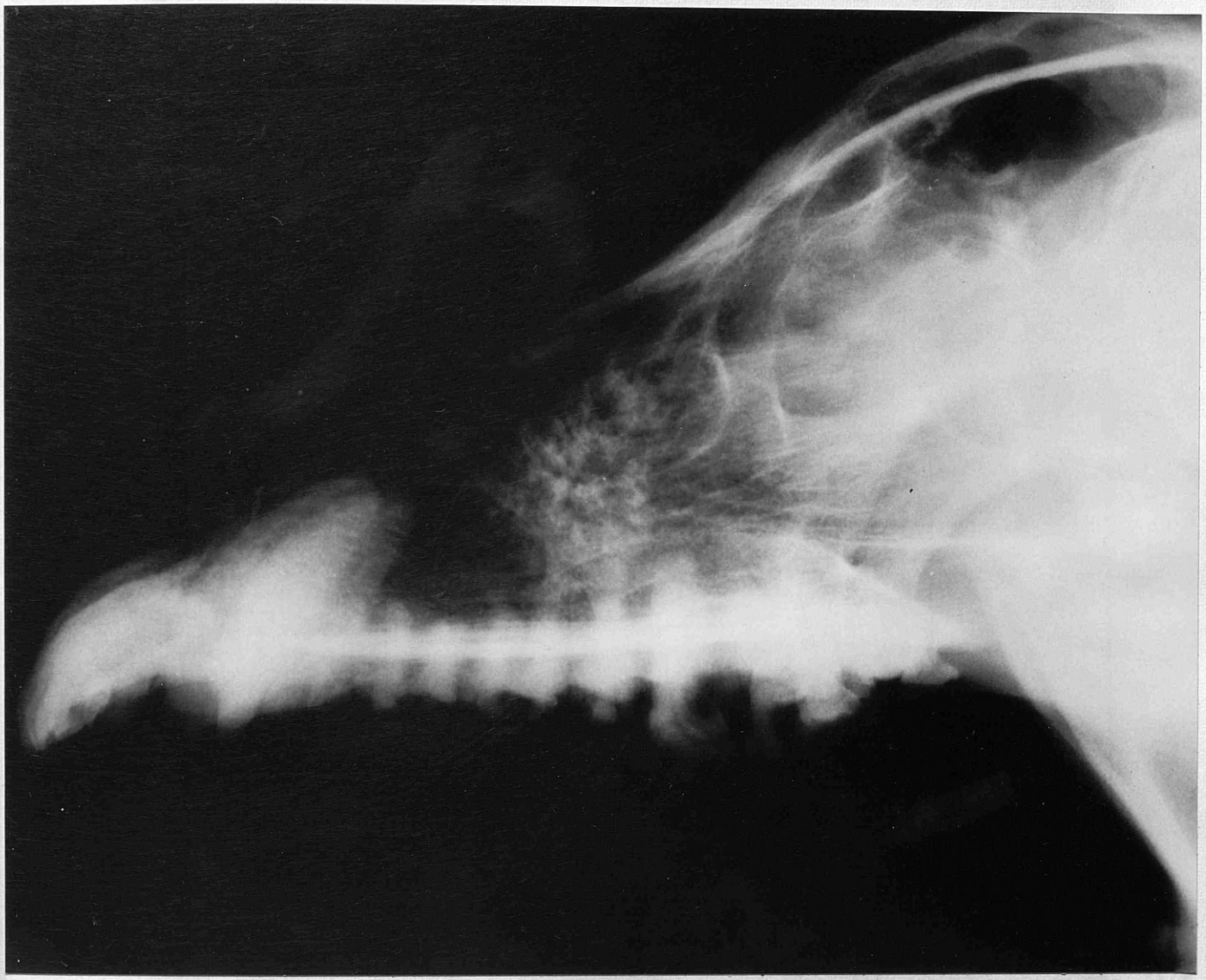

Fig. 21 Lateral radiograph of case 23 (Appendix II). Mineralisation is also appreciated on this view, but its unilateral involvement can not be determined.

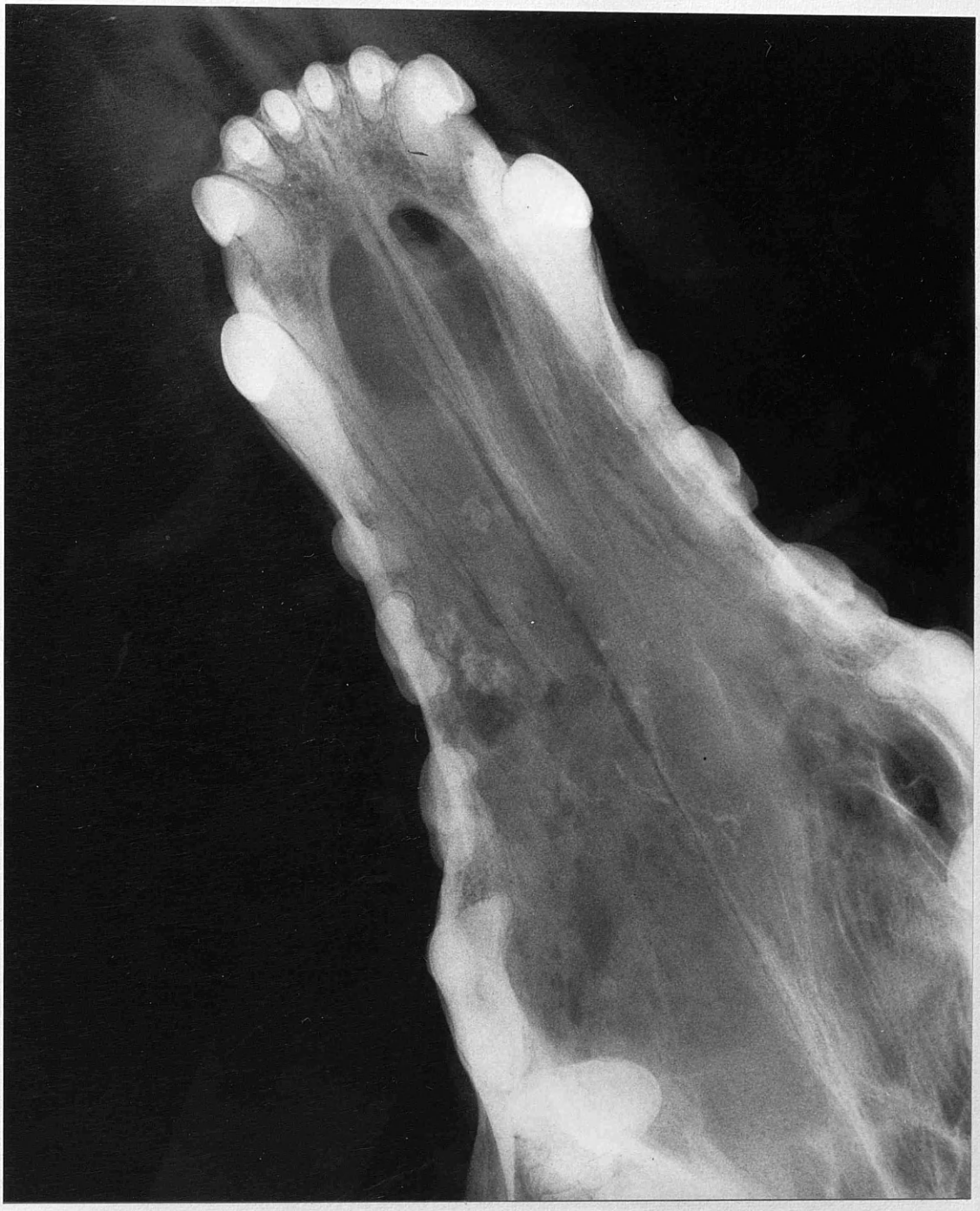

Fig. 22 D.V. intra-oral radiograph of case 14 (Appendix II - Bilateral nasal neoplasia) showing turbinate, plate, case and vomer destruction. There are also focal areas of mineralisation.

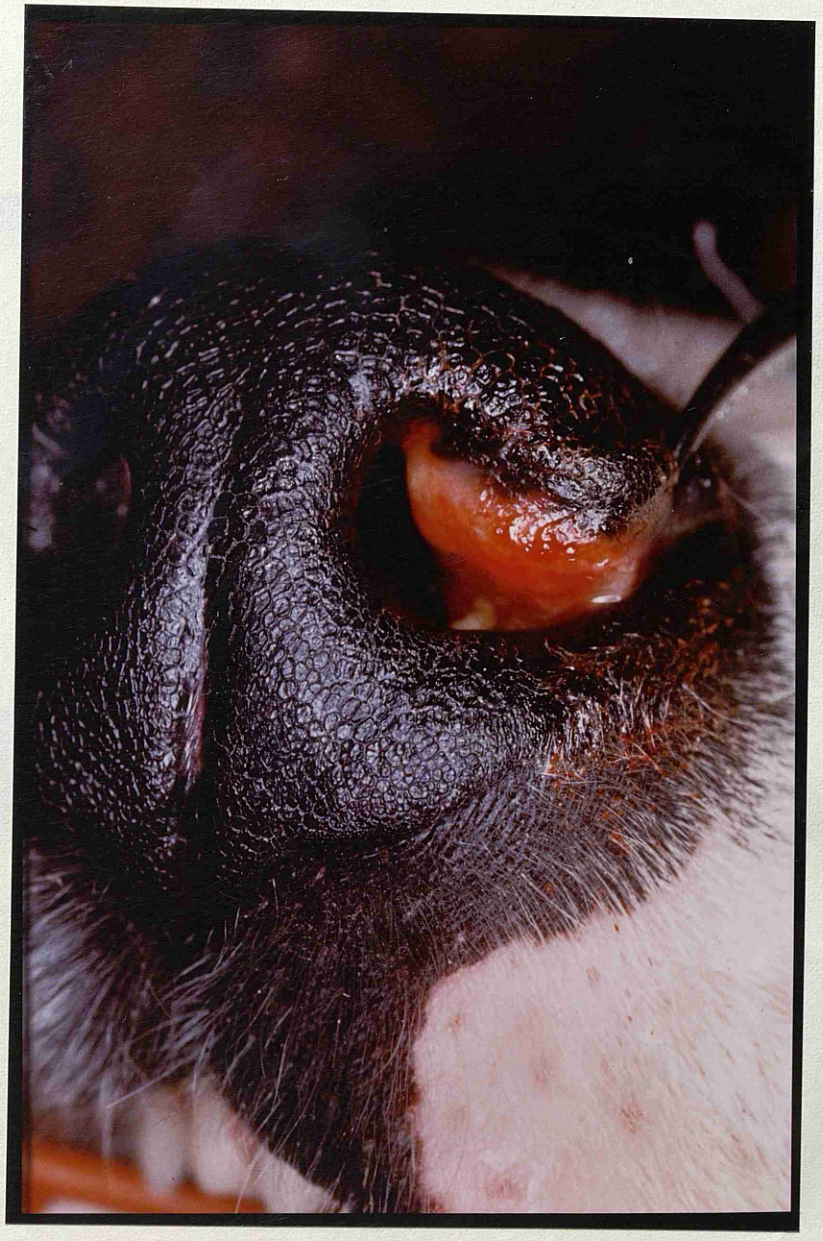

Fig. 23 Photograph of the nares of case 1 (Section II) showing rhinarial ulceration.

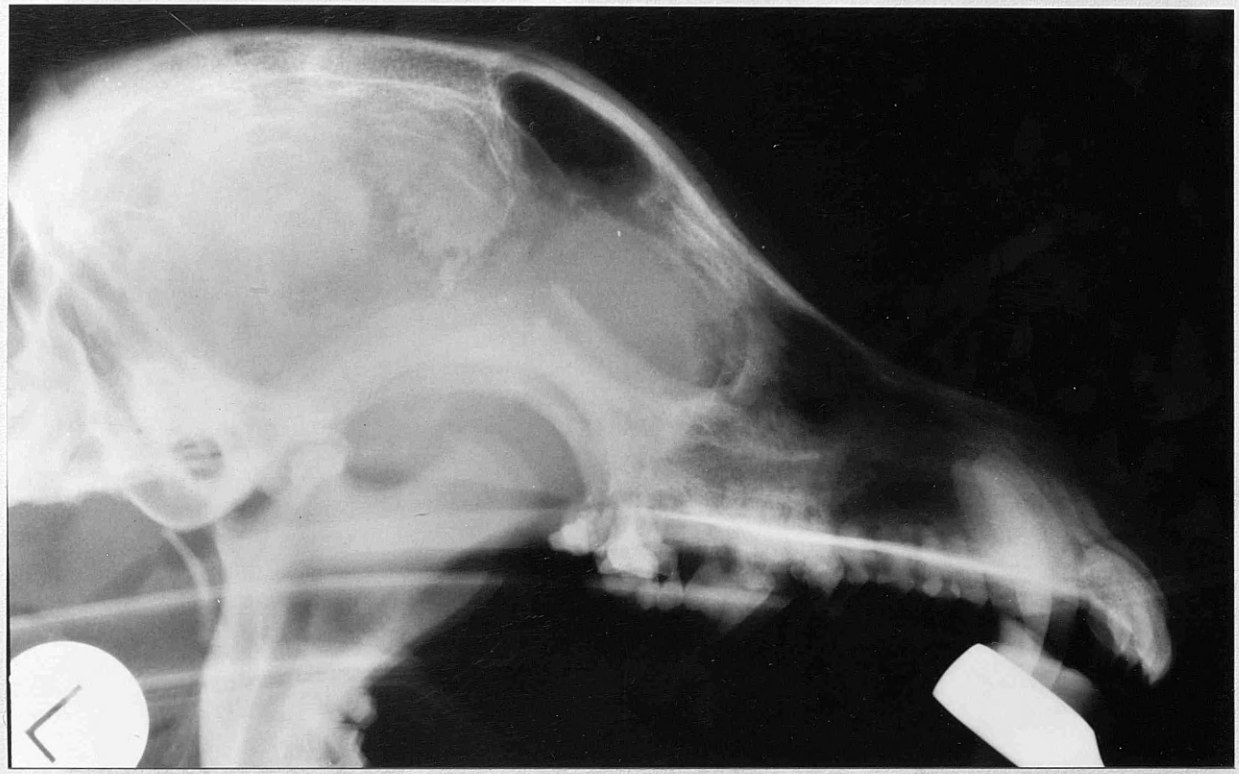

Fig. 24 Lateral radiograph of the skull of case 2 (Section II) showing destruction of the frontal bone ventrally and irregularity of the cranial bones. There is a soft tissue mass in the orbital region.

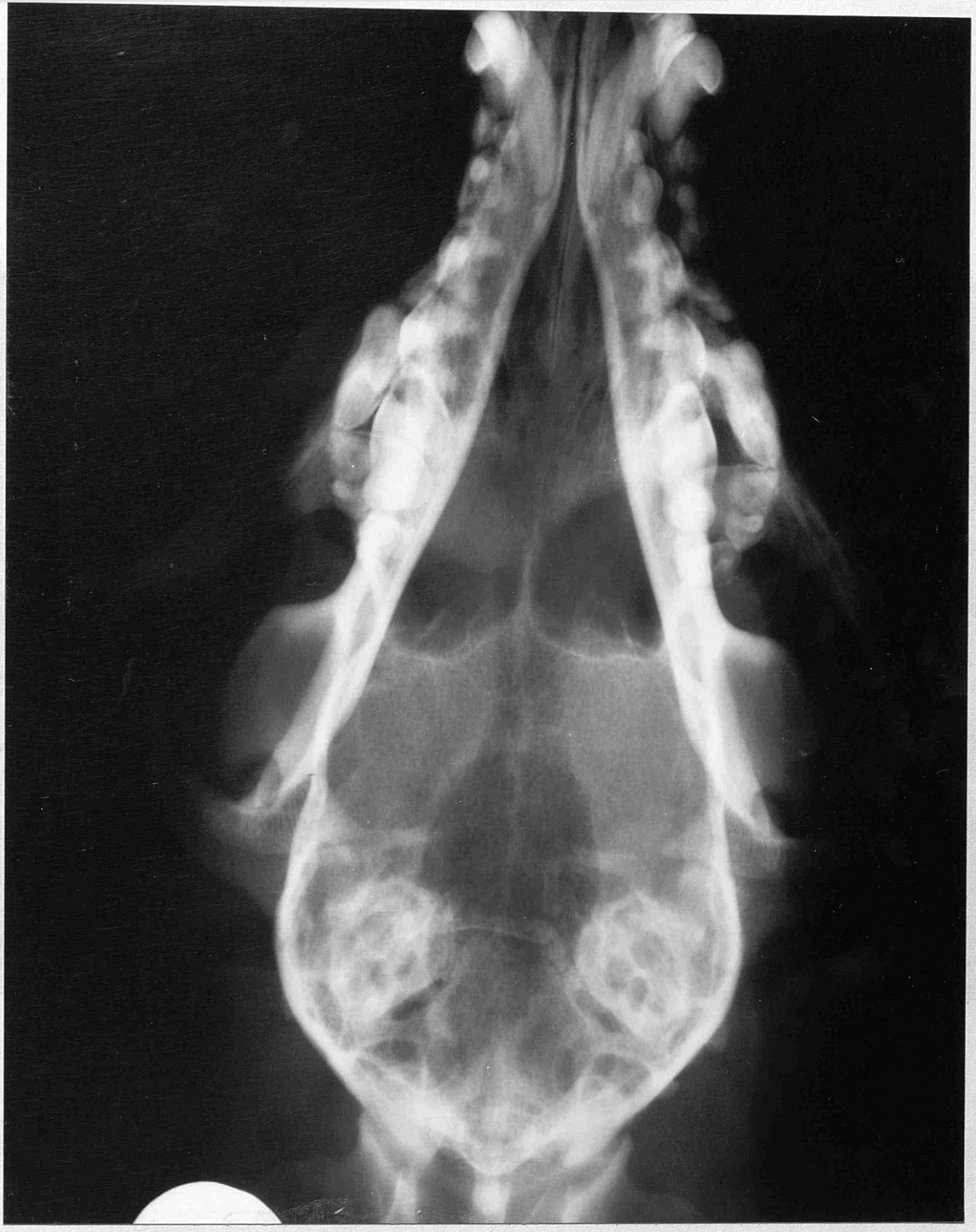

Fig. 25 Dorso-ventral radiograph of the skull of case 2 (Section II) showing bilateral destruction of much of the bony architecture relating to the front of the cranial cavity and frontal sinuses. There is a soft tissue mass on the right hand side at the level of the frontal sinus.

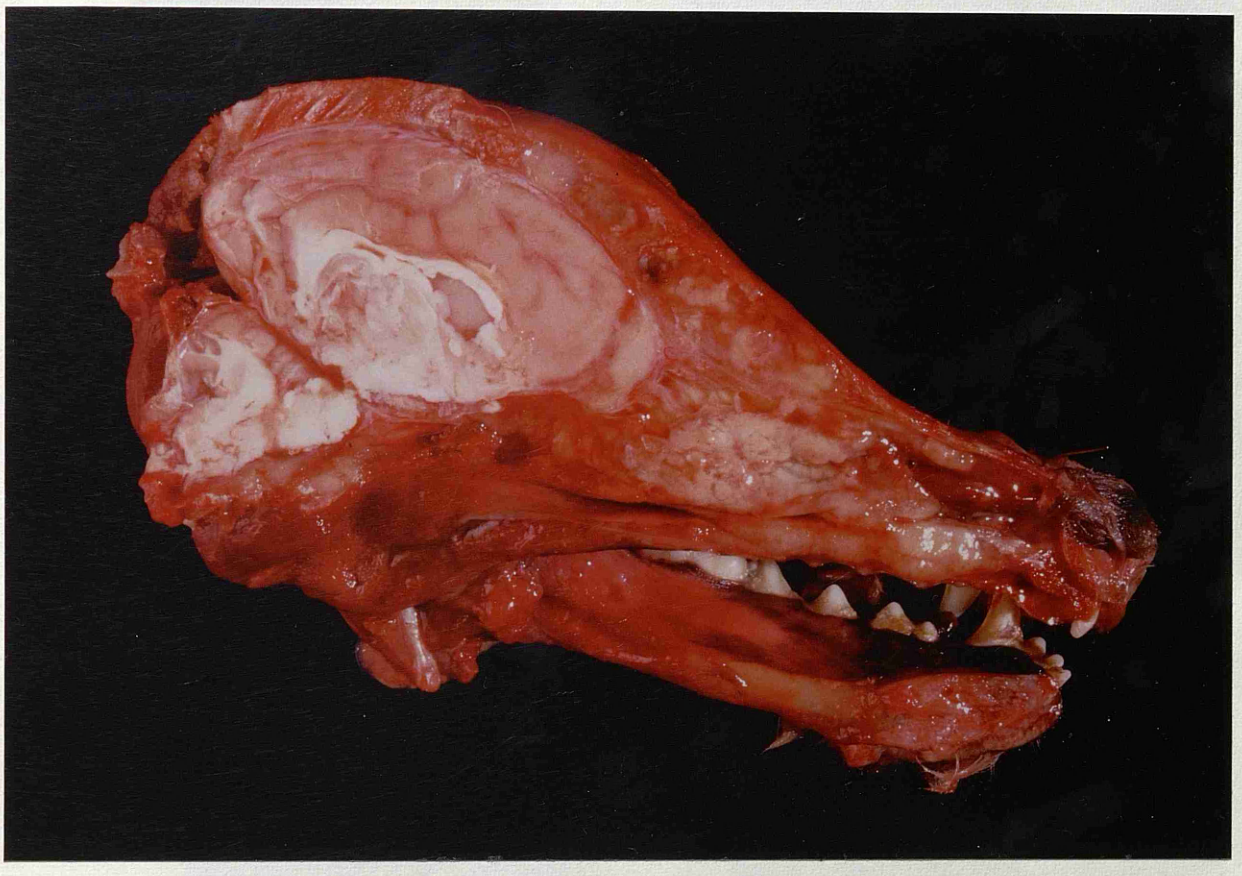

Fig. 26 Case 2 (section II) A sagittal section of the nasal cavity showing extensive infiltration of the adenocarcinoma. Destruction of all, except the most rostral turbinate, has occurred.

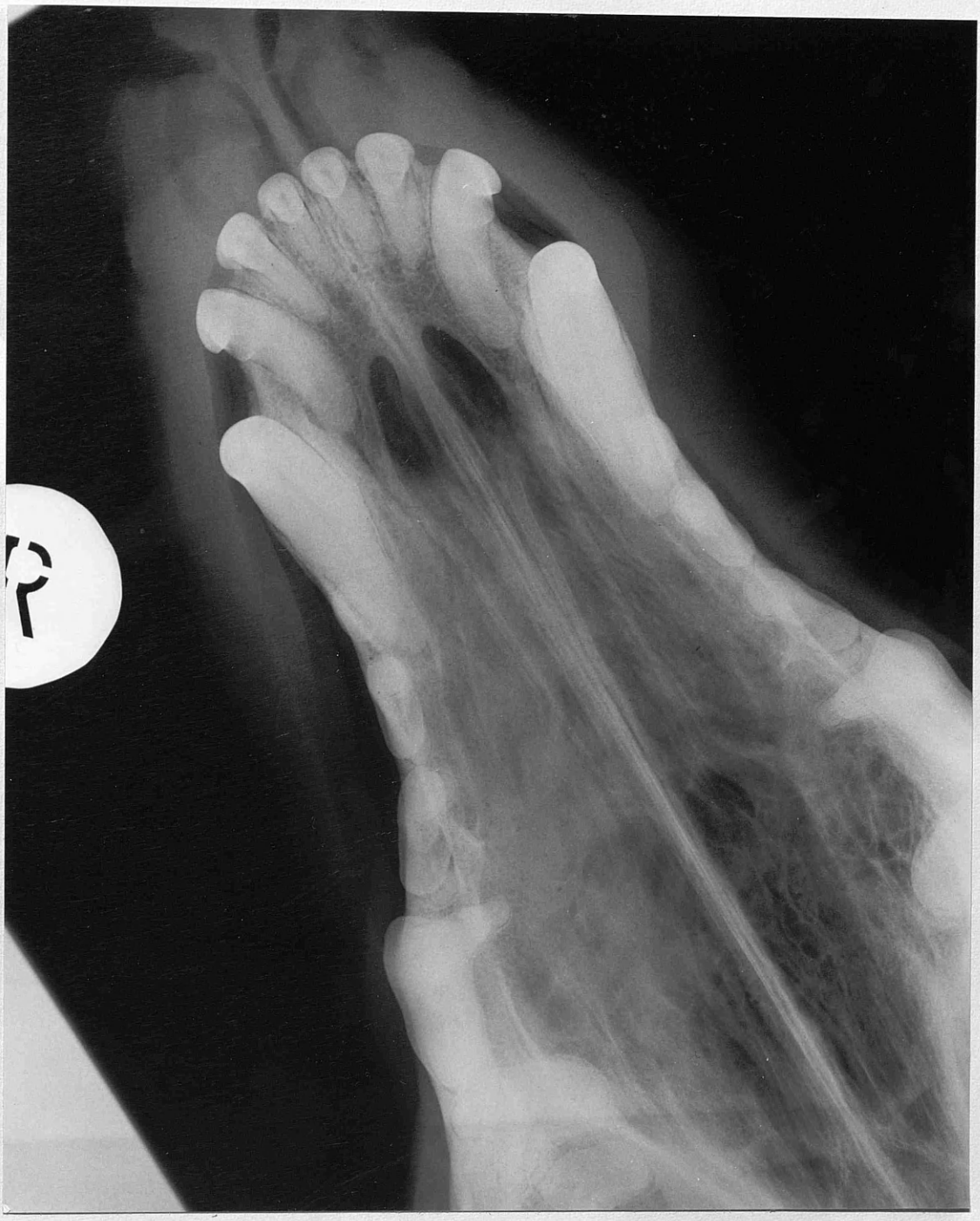

Fig. 27 D.V. intra-oral radiograph of case 6 (Distemper rhinitis) showing unilateral increased opacification rostrally, focal opacification caudally with turbinate masking, not destruction.

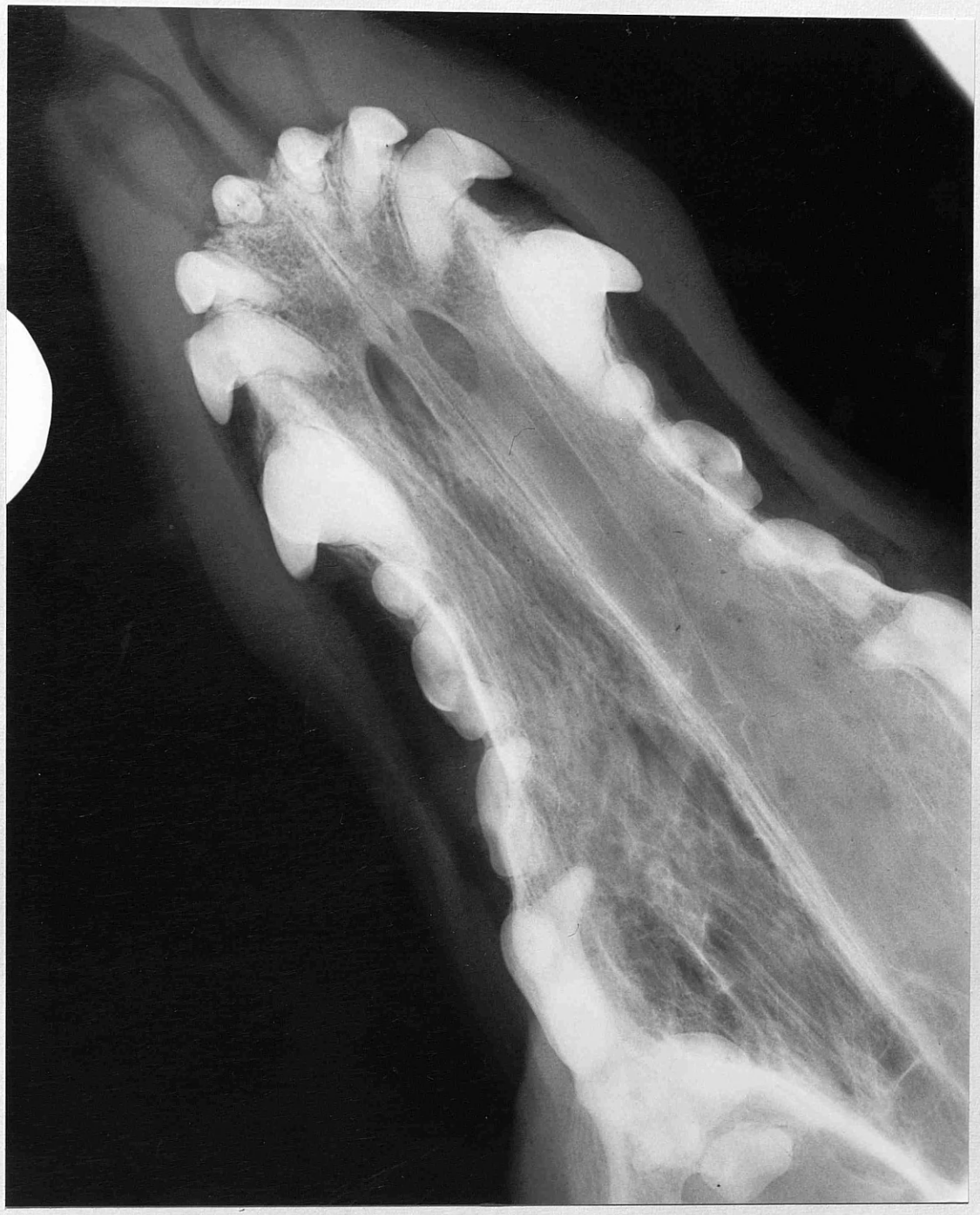

Fig. 28 D.V. intra-oral radiograph of case 7 (Section II - Chronic Hyperplastic rhinitis) Unilateral increase in opacification Unilateral turbinate destruction, not masking.

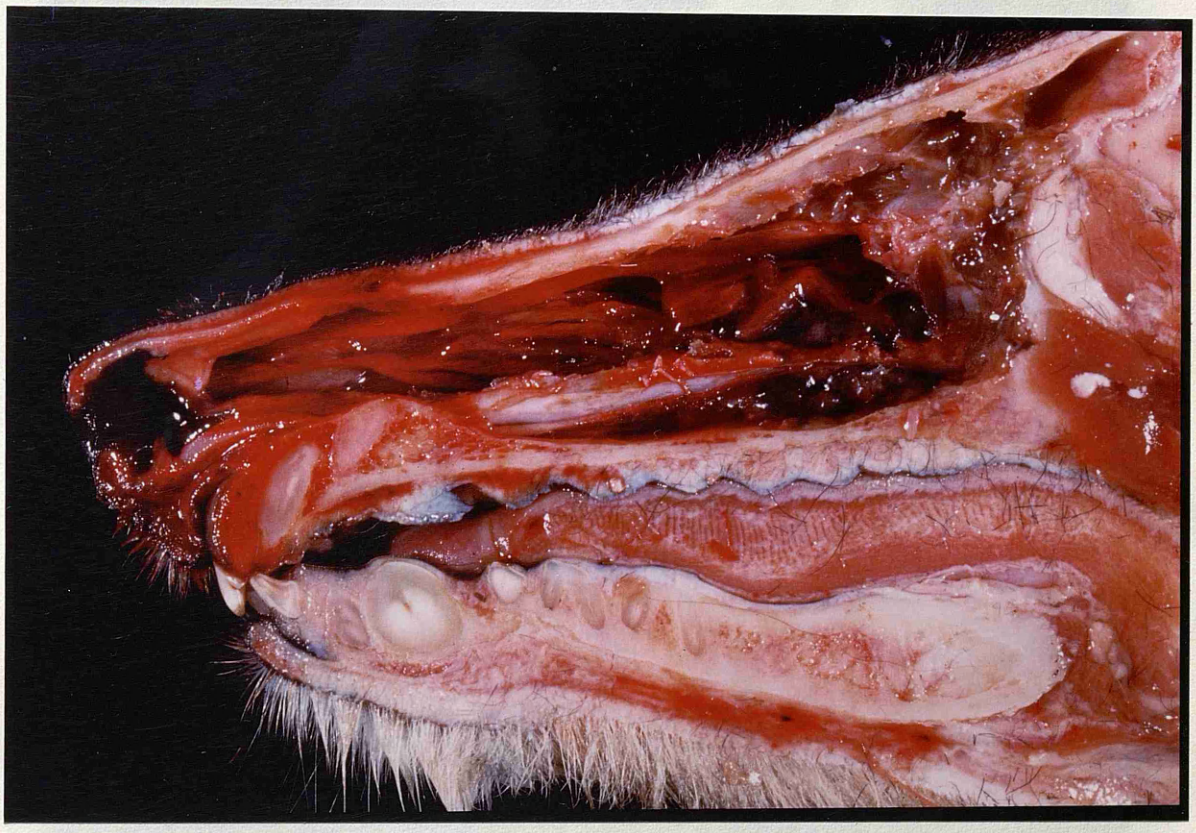

Fig. 29 Case 8 (Section II - nasal neoplasia) Sagittal section of the nasal cavity showing the malignant melanoma ocuppying the caudal nasal chamber resulting in ethmoturbinate destruction.

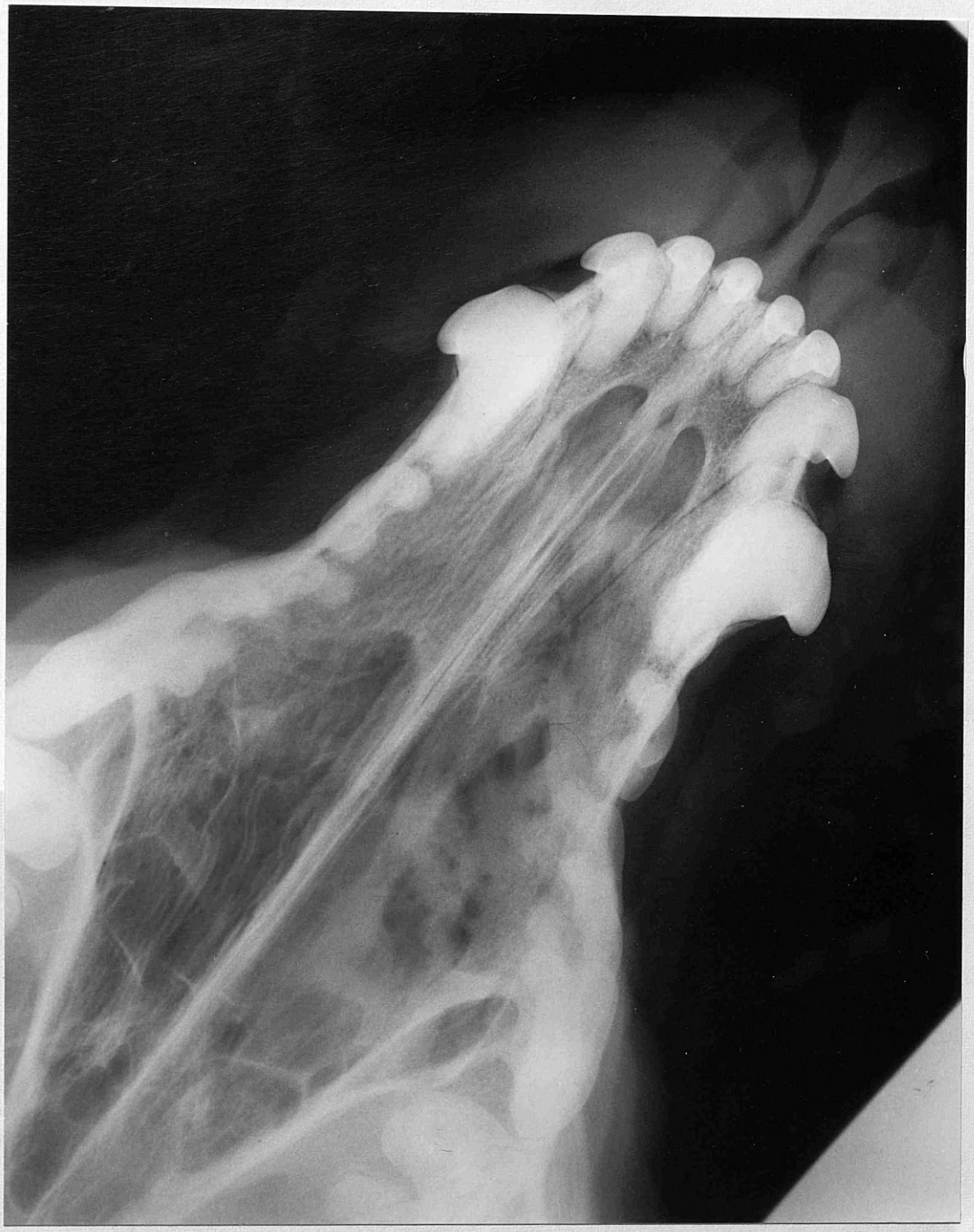

Fig. 30 D.V. intra-oral radiograph of case 11 (Section II) Unilateral mixed density pattern with turbinate destruction and frontal sinus opacification. This is an unusual appearance of nasal neoplasia and may represent an early lesion.

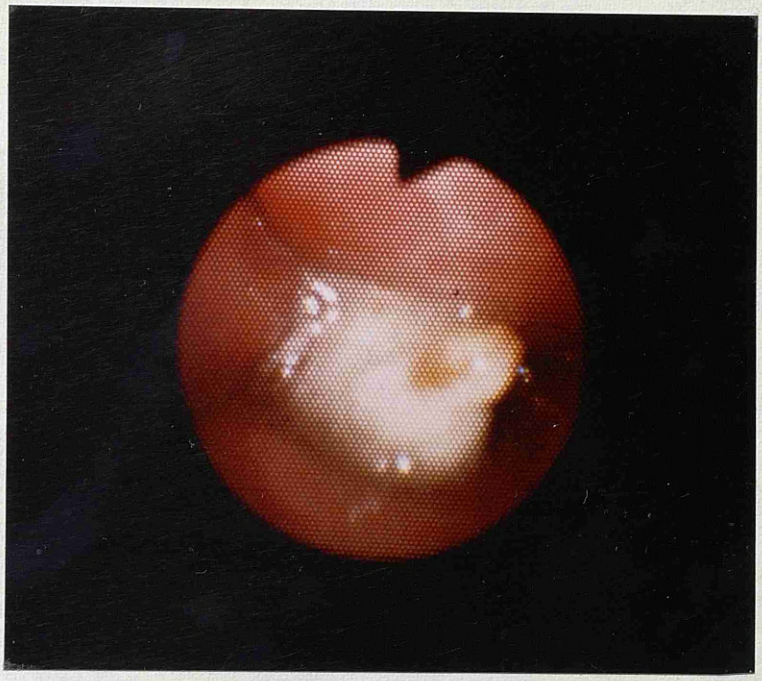

Fig. 31 Endoscopic photograph of case 13 (Section II - nasal foreign body) demonstrating the appearance of a twig lodged in the middle meatus

Fig. 32 Case 13 (Section II) An intra-nasal foreign body - twig.

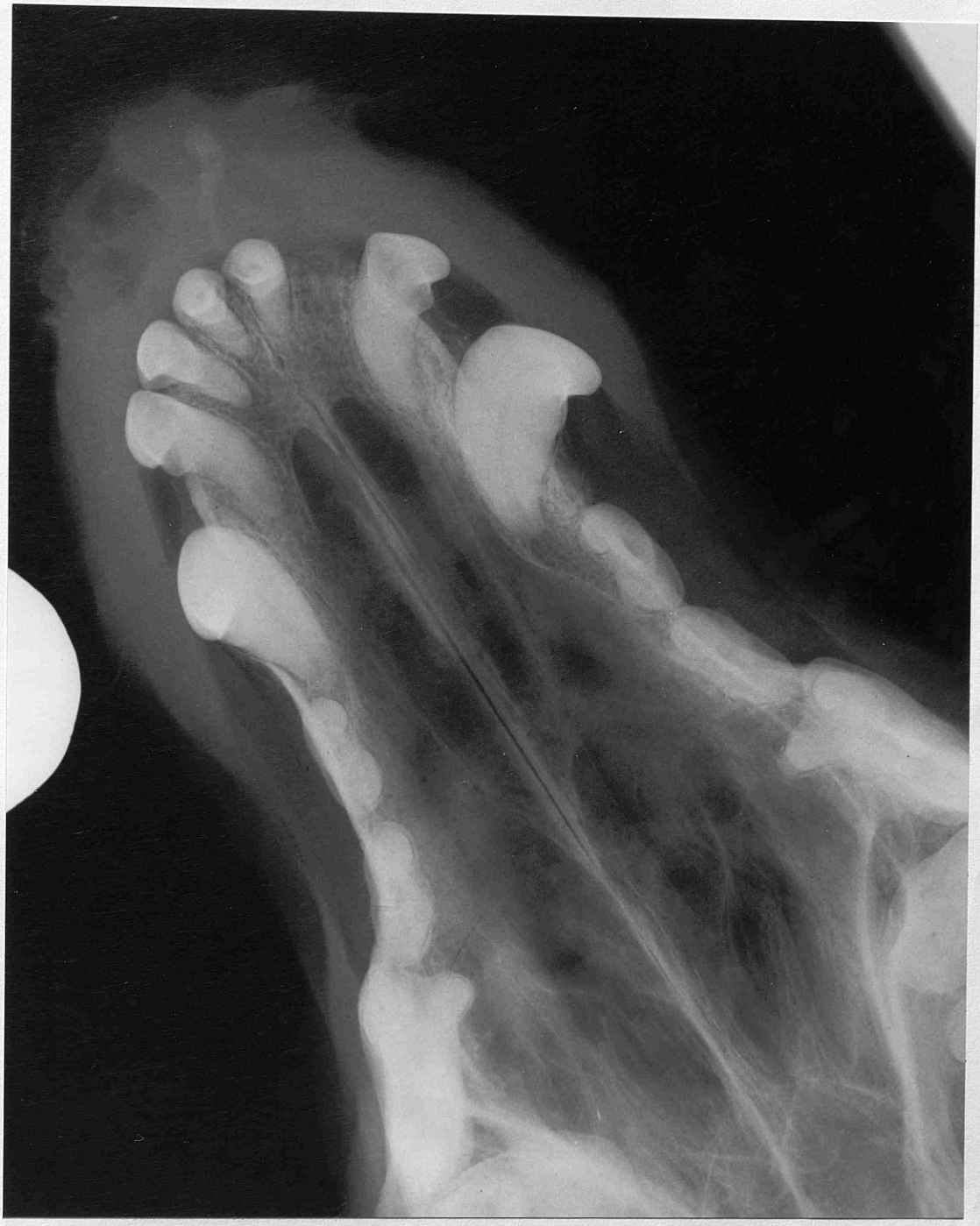

Fig. 33 D.V. intra-oral radiograph of case 14 (Section II) Intra-oral nasal cavity. Bilateral increase in radiolucency and turbinate destruction. Centrally vomer erosion is evident. The diagnosis was atypical aspergillosis with extensive destruction

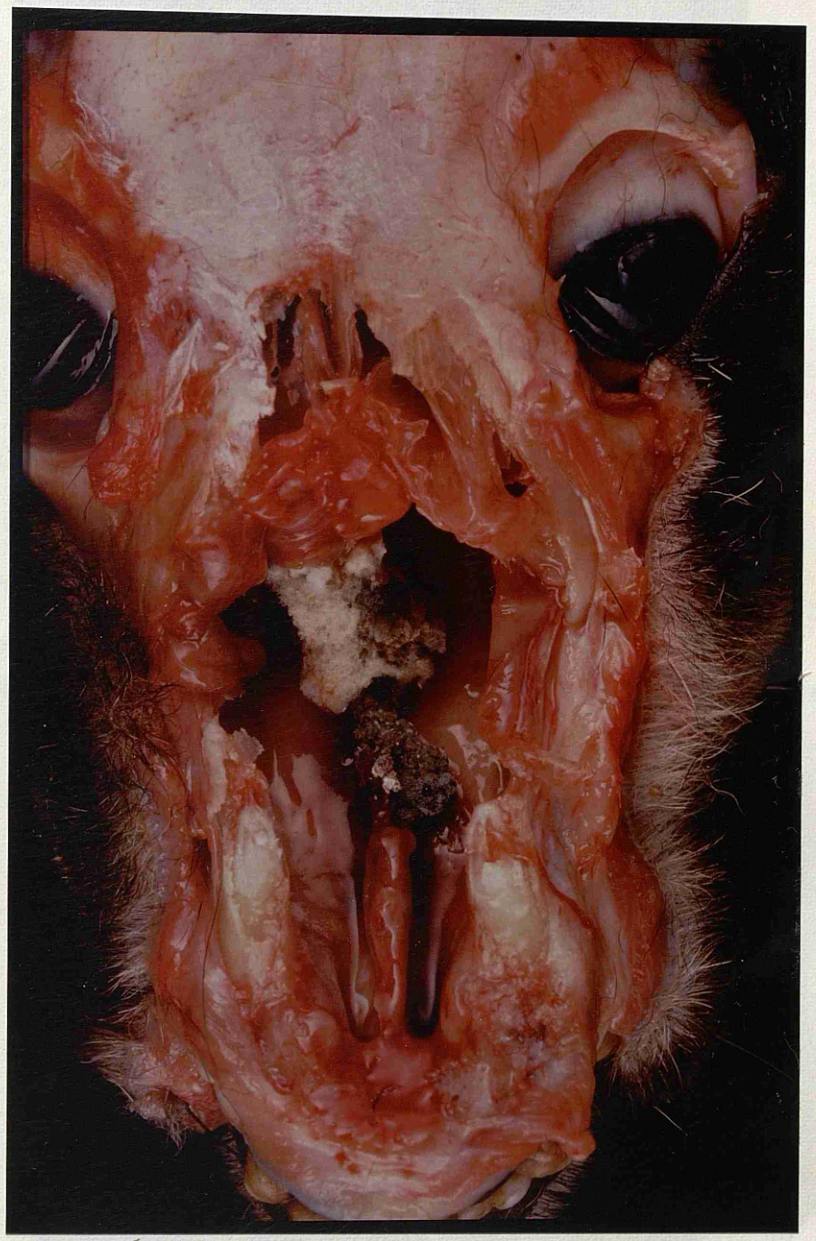

Fig. 34 Case 14 (Section II)
Dorso-ventral view of the nasal cavity with the nasal, maxillary and part of frontal bones removed. A fungal granuloma is present. There is virtual absence of all turbinate structures and the vomer centrally.

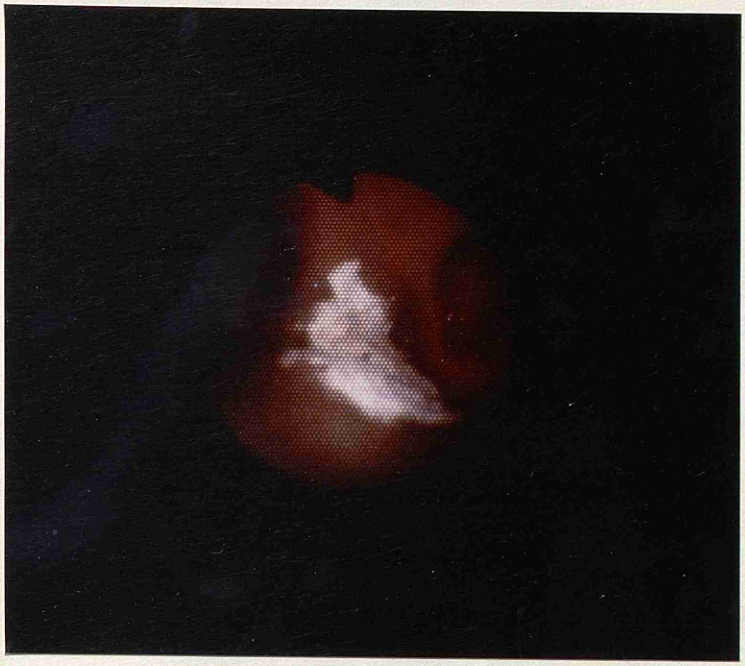

Fig. 35a Case 9 (Section II)
The endoscopic appearance of *Aspergillosis fumigatus* within the nasal cavity of a dog.

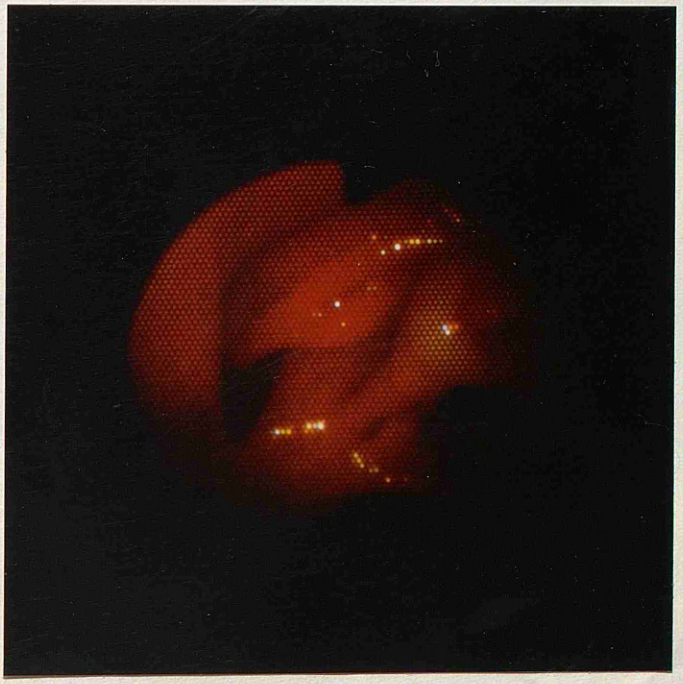

Fig. 35b Case 9 (Section II) The endoscopic appearance following successful treatment with Enilconazole. Truncated ethmoturbinates are seen. There is no evidence of fungal plaques.