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SPONTANEOUS AORTIC LESIONS IN MOOSE (ALCES ALCES L)

By

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POUNGSHOMPOO, SOMLAK and CLAES REHBINDER: *Spontaneous aortic lesions in moose (Alces alces L).* Acta vet. scand. 1985, *26*, 533—548. — In 17 out of 22 aortas from moose, 6 months to more than 18 years, aortic lesions were found. Five different types of lesions were observed, fatty streaks were seen in 1, fibrous plaques in 7, fatty streaks with complicated lesions (calcification and acid mucopolysaccharides) in 3, fibrous plaques with complicated lesions in 2 and fibrous plaques with complicated lesions and fat droplets in 4 of the aortas.

Elastic tissue degeneration of the inner two thirds of the tunica media was principally found in the aorta of the animals beyond 4 year of age. There was no statistical evidence for a correlation between age and frequency ($P \backsim 0.10$) but a trend towards age dependence was seen. The percentage of involved surface was found to increase significantly ($0.05 > P > 0.02$) with age. There was no significant difference between affected male and female animals in the different age groups. The frequency of involved surface in different affected regions, comprising all age groups were, in the posterior abdominal portion, 13.0 %, in the anterior abdominal portion, 3.0 %, in the posterior thoracic portion 5.2 %, in the anterior thoracic portion, 0.1 % and in the aortic arch, 0.1 %. Proliferating endothelial cells and the source of smooth muscle cells were discussed.

atherosclerosis; fatty streaks; complicated lesions; fibrous plaques.

Spontaneous atherosclerotic lesions of the aorta have been reported in man and many different animals (*Wilkin et al.* 1959, *McKinney* 1968, *Bhalla & Prasad* 1977 and *Gupta & Rehbinder* 1981). Roe deer in Sweden develops a wide range of spontaneous aortic lesions correlated to age (*Poungshompoo & Rehbinder* 1983). This led us to conduct comparative studies in moose, which is considered to be a long-lived member of the family Cervidae. According to *Gardell* (1958), a moose marked in East Prussia attained an age of 27 years. Moose found in

various parts of Sweden may live more than 18 years (*Markgren* 1969).

Moose live under similar conditions as do roe deer, but are mainly browsers while roe deer are mainly grazers (*Ahlen* 1975).

The objective of this investigation has thus been to study the incidence and morphological patterns of spontaneous aortic lesions and their association with age and sex in moose.

MATERIAL AND METHODS

Aortas form 22 moose, (11 males and 11 females) 6 months to more than 18 years of age, were collected during part of the hunting season, between October 16, 1982 and November 11, 1982. Age was estimated on the basis of dental wear. The material was divided into 4 age groups, yearling (up to 1 year), > 1—4 years, > 4—8 years and > 8—18 years. The aortas were removed, opened longitudinally and fixed in 10 % buffered formalin.

The material was processed according to the methods described by *Poungshompoo & Rehbinder* (1983). In addition, affected surface was measured by using a planimeter (Kontron, Messgeräte GMBH).

Histological sections were examined and the lesions were graded. When more than one lesion was present in a vessel, all lesions were graded by mean of histology. The lesion which occupied the greatest areas of a segment was considered the predominant lesion.

Statistical analysis was performed by means of regression analysis.

RESULTS

Five types of lesions were found in 17 of the 22 examined animals; fatty streaks, fibrous plaques, fatty streaks with complicated lesions (calcification and acid mucopolysaccharides), fibrous plaques with complicated lesions and fibrous plaques with complicated lesions and fat droplets. The distribution is shown in Table 1.

Regression analysis gave no evidence for a correlation between age and frequency ($P \approx 0.10$) but a trend towards age dependence can not be excluded. Concerning the percentage of involved surface a slightly significant ($0.05 > P > 0.02$) increase

Table 1. Number of moose and distribution of different kinds of aortic lesions.

Age in years	Number of animals	Aortic lesions						
		Fatty streaks	Fibrous plaques	Fatty streaks with complicated lesions	Fibrous plaques with complicated lesions	Fibrous plaques with complicated lesions and fat droplets	Total number	% affected
≤1	5	—	2	—	—	—	2	40.0
>1—4	6	—	3	—	1	—	4	66.7
>4—8	6	1	1	2	1	1	6	100.0
>8—18	5	—	1	1	—	3	5	100.0
Total number	22	1	7	3	2	4	17	

correlated to age was found (Table 1, Fig. 1). Furthermore, at the histological investigation the lesions appeared more severe in older animals. There was no significant difference ($P > 0.05$) between male and female animals within each group, but a tendency was seen towards more involved surface, in the female (Fig. 2). The distribution of affected surfaces were wider in females than in males of same age. Besides, the proportion of affected surface showed an increase directly proportional to old age in the female group. The distribution of the lesions starting from the posterior abdominal aorta throughout the aortic arch in each group is shown in Fig. 3. In the yearling group, lesions occurred only in the posterior abdominal region. In the animals of the >1—4 year group as well as in animals of >4—8 year group the lesions occurred in the posterior abdominal, anterior abdominal and posterior thoracic portions. In the group >8—18 years, lesions were found in all regions. The frequency of involved surfaces in affected regions, comprising all age groups were: in the posterior abdominal portion, 13.0 %, in the anterior abdominal portion, 3.0 %; in the posterior thoracic portion, 5.2 %; in the anterior thoracic portion, 0.1 % and in the aortic arch, 0.1 % (Fig. 4).

Fatty streaks

Gross staining with Sudan IV revealed yellowish, soft flat or slightly elevated areas being intimal fatty streaks varying in size between 0.1—1.0×0.5—23 cm. However, in some cases the

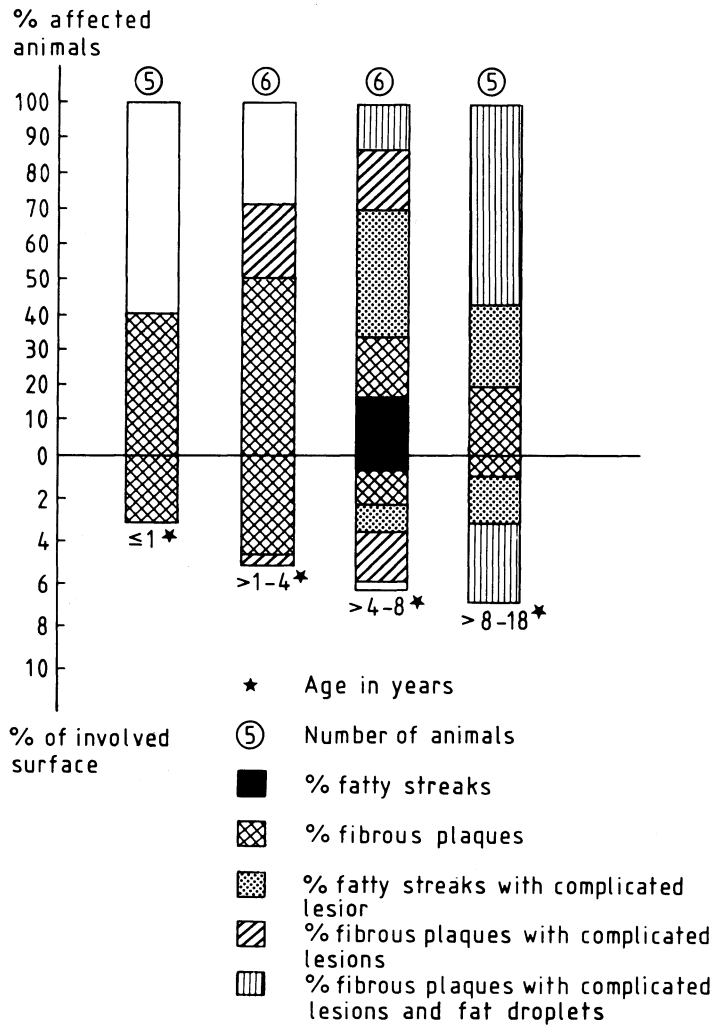

Figure 1. Percentage of affected moose in different age groups and percentage of involved surface covered by different kinds of aortic lesions.

lesions were in the form of irregular streaks or patches covering large areas. In the aortic arch, fatty streaks were found at the remnants of ligamentum arteriosum. In the posterior thoracic portion and anterior abdominal portion, the lesions were usually parallel, linear and longitudinal streaks localized at the central mid-line of the aorta (Fig. 5). In the posterior abdominal portion, fatty streaks appeared as large irregular areas, mostly

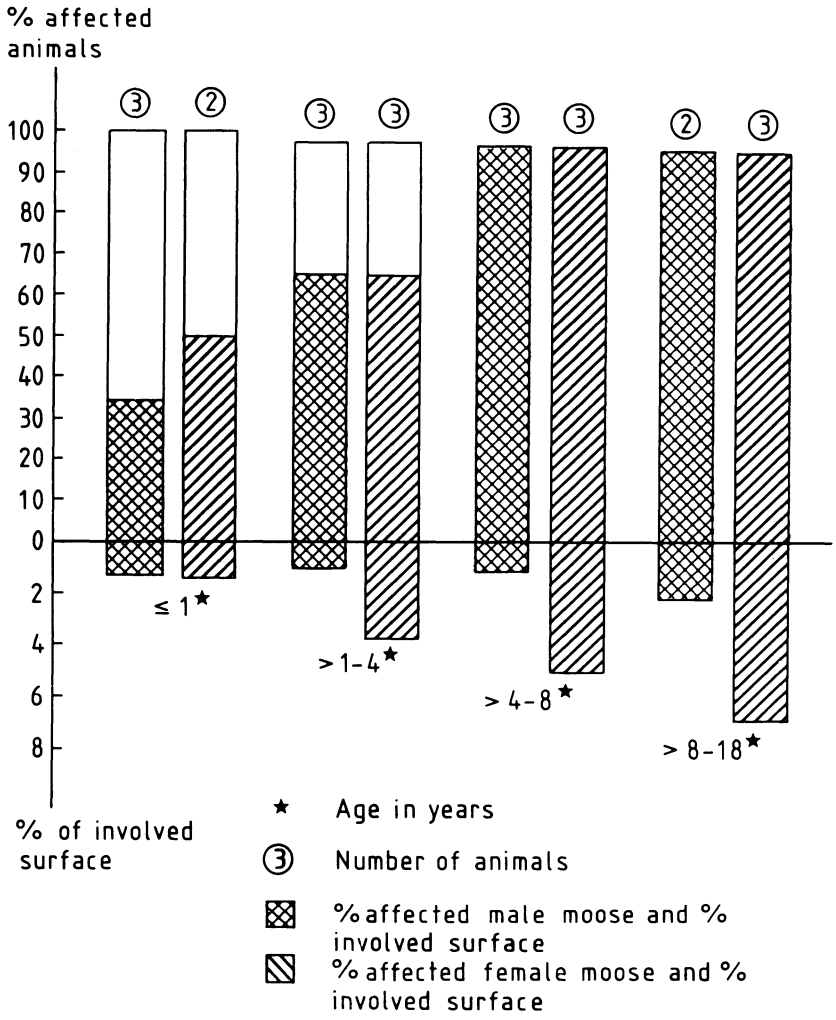

Figure 2. Comparison of percentage affected male and female moose and percentage of involved surface in each group.

located at the mid-line, ventrally or dorsally, close to the iliac bifurcation. In the external and the internal iliac arteries, the fatty changes did not appear as streaks but as stained circular, wavy surfaces.

The histological investigation revealed that the intima of fatty streaks or spots was 2—10 times thicker than normal. In connection with intimal changes the media was also regularly 1½—2 times thickened. The thickened part of the tunica intima

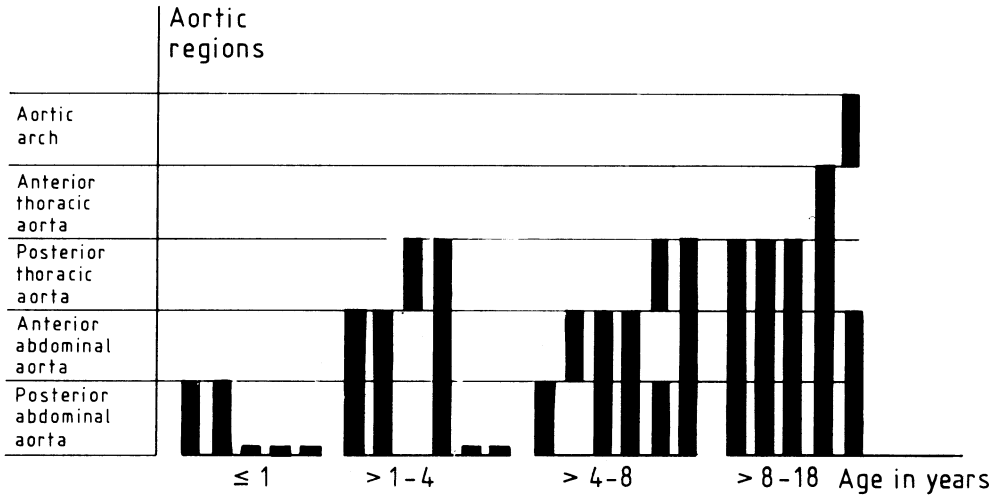

Figure 3. Distribution of aortic lesions of individual animal in each group in 5 different anatomical regions.

comprised a single layer of endothelial cells, fibroblast, fibrocytes, elastic fibres, collagen fibres, a few mononuclear cells and a few smooth muscle cells. The components were separated by oedema fluid. Collagen fibres were occasionally seen in the the interstitial space of the fatty streaks. The nuclei of the endothelium ap-

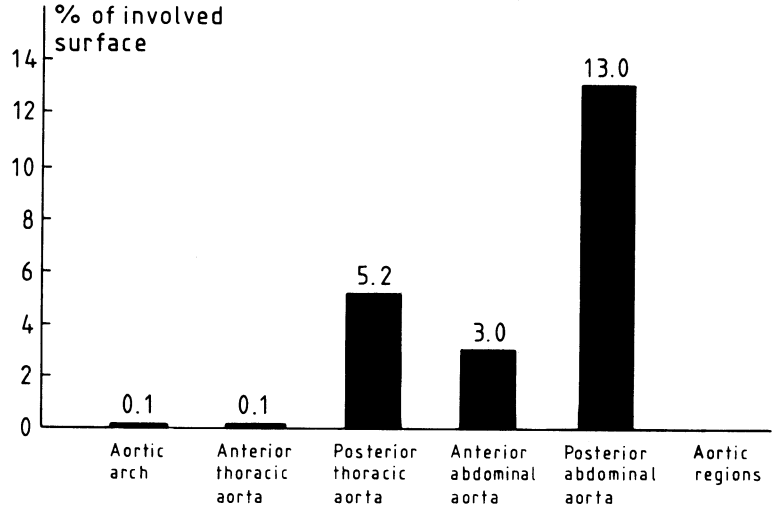

Figure 4. Percentage of involved surfaces in different affected regions, comprising all age groups.

peared either flat or as big dense bodies. In the latter case an increase of cytoplasmic granules were observed. In the subendothelial layer, adjacent to the endothelium, scattered mononuclear cells were seen, most of them with cytoplasmic vacuoles. In contrast, the mononuclear cells found near the internal elastic lamina appeared bigger and all of them contained vacuoles of various size. Smooth muscle cells were found to proliferate near the internal elastic lamina. They appeared large, fusiform or strap-shaped. Smaller and less typical smooth muscle cells were present close to the endothelium. They varied in shape but tended to be stellate with short, thick extensions, in contrast to the thin and long processes of the fibroblasts. Most intimal smooth muscle cells were oriented parallel to the long axis but some of them appeared to be without definite orientation. The internal elastic lamina revealed a combination of changes like oedema, fragmentation, undulation, dissolution and reduplication. In the tunica media, the lesions were characterized by varying degree of edema, elastolysis, vacuolation and disorientation of smooth muscle cells. In connection with areas of diffuse vacuolation, the continuity of the medial smooth muscle cell-layer was interrupted. These cells had indistinct cell membrane, cytoplasmic vacuoles, and eccentric pyknotic nuclei. An increase in acid mucopolysaccharides around the area of degeneration was observed.

Smooth muscle cells in the inner third if the media were often oblique or perpendicular to the aortic canal. Clusters of disoriented medial smooth muscle cells were regularly found facing a gap of the disrupted internal elastic lamina.

Frozen sections from fatty streaks revealed lesions in which cells of the endothelial and subendothelial layers had varying amounts of fat droplets in their cytoplasm. In addition a varying amount of fine lipid droplets were found in the extracellular space. These areas of lipid deposition either intracellular or extracellular or both, were regularly found in the inner half of the intima including the internal elastic lamina. In the inner ⅓ to ⅔ of the tunica media extracellular and intracellular lipid droplets were sparsely found.

Fatty streaks with complicated lesions

Fatty streaks with complicated lesions, (characterized by calcification and depositions of acid mucopolysaccharides) were

found predominantly in the inner ⅓ to ⅔ of the media rather than in the tunica intima and in the internal elastic lamina. Calcification appeared as fine or coarse granules deposited both extracellularly and intracellularly. An increase in acid mucopolysaccharides in the ground substance was evident especially around lesions with calcium and lipid depositions.

Fibrous plaques

Fibrous plaques were found as non-sudanophilic firm, gray-white, round or elongated lesions in the aortic intima varying in size from 0.1—1.5×0.1—20 cm. Lesions with calcifications were considered as fibrous plaques with complicated lesions. Lesions with a central portion of soft-like material containing fat were classified as fibrous plaques with complicated lesions and fat droplets. In the aortic arch, fibrous plaques were occasionally seen at the site of ligamentum arteriosum. In the posterior thoracic aorta and the anterior abdominal aorta, fibrous plaques were usually seen as two parallel elevated ridges running cranially from the orifices of celiac and anterior mesenteric arteries, and often surrounding areas revealed also fatty streaks. In some cases fatty streaks occurred between the two raised ridges (Fig. 5).

The microscopic structures observed in fibrous plaque were more variable than those of the fatty streaks. The intima was much thicker than that of fatty streaks, usually 10—15 times that of the normal intima and more pronounced than that of the media. The uncomplicated plaques varied in shape, i.e. flattened and dome shaped and in their composition (Figs. 6 and 7). Occasionally fibrous plaques were covered by proliferating endothelial cells forming a cap. The polarity of the intimal cells, below the cap, appeared different (Figs. 8 and 9). In severe advanced plaques, endothelial cells were fragmented, necrotized or occasionally totally absent.

Fibrous plaques with complicated lesions

Fibrous plaques with complicated lesions were characterized by calcification and depositions of acid mucopolysaccharides. Calcification generally appeared as fine or coarse granules deposited either intracellularly or extracellularly in the inner ⅓ to ⅔ of the media but also sparsely in multiple foci in the intima.

In extensive cases of calcification, lesions were composed of a core of extracellular calcified debris, representing a dystrophic calcification of the inner third of the tunica media (Fig. 10). This was usually found in combination with an increase of acid mucopolysaccharides in the ground substance. Positive alcian blue material was faintly accumulated in the superficial and basal regions of the intima. Large amounts were noted as a thick band along the fibrous cap and within the interlamellar space of the media in advanced plaques.

Fibrous plaques with complicated lesions and fat droplets

In fibrous plaques with complicated lesions and fat droplets, the amount of lipid comprising the atheromatous core varied from small to massive accumulations relative to the volume of the lesions (Fig. 12). Foam cells could be numerous in the fibrocellular cap or around the atheroma but could also be entirely absent. Vascularization was a common change in fibrous plaques. A varying number of capillaries were seen in the periphery of atheromas and in the inner thirds of the tunica media. These capillaries appeared to arise from the vasa vasorum of the adventitia.

DISCUSSION

The fatty streaks, fibrous plaques and complicated lesions found in the aortas of moose resembled those found in man (*Prior & Jone* 1952, *Holman et al.* 1958, *WHO Study Group on Atherosclerosis* 1958) and in other wild and domestic ruminants (*Prasad et al.* 1973, *Gupta & Rehbinder* 1981, *Poungshompoo & Rehbinder* 1983).

In moose lesions did not significantly increase in number and severity with age as found in roe deer (*Poungshompoo & Rehbinder* 1983). This may be due to the fact that it was hard to get a sufficient number of specimens. But the area involved, the development of lesions and the distribution in the aorta in 5 different anatomical regions indicated a development coupled to age (Fig. 1, 3). In the yearling group, the aortic lesions appeared as fibrous plaques confined to the posterior abdominal part of the aorta. In the groups of older animals other lesions like fibrous plaques with complicated lesions, fatty streaks with complicated lesions and fibrous plaques with complicated lesions

and fat droplets occurred in different anatomical regions. The prevalence of fibrous plaques was higher than that of fatty streaks. In man, fatty streaks is shown to gradually convert into fibrous plaques and other complicated lesions (*Holman et al.* 1958) with increasing age. In roe deer fatty streaks and fibrous plaques occurred in the same specimen in the group of animals younger than 1 year. The number in fibrous plaques and other complicated lesions increased with age indicating the conversion of fatty streaks into fibrous plaques (*Poungshompoo & Rehbinder* 1983). The increasing incidence of fibrous plaques in aging moose may indicate that fatty streaks also in this species may change into fibrous plaques.

With time the uncomplicated fibrous plaques may undergo a variety of morphological changes varying from one lesion to another. These changes generally are referred to as complications and the resulting lesion as a complicated fibrous plaque, i.e. calcification, hemorrhage, acid mucopolysaccharides and thrombosis (*Geer & Haust* 1972). The same findings were also found in moose and in roe deer (*Poungshompoo & Rehbinder* 1983). *Webster et al.* (1974) showed that several naturally occurring fibrous intimal thickenings in aortas of rabbit had greater permeability for horseradish peroxidase (HRP) and ferritin than did adjacent normal intima. In addition, the atherosclerotic lesions in rabbits evolved from an immunologically induced fibromuscular intimal lesion which later preferentially accumulated lipid in the presence of hypercholesterolemia (*Hardin et al.* 1973). The prevalence of fat droplets in older fibrous plaques and complicated lesions indicates an greater permeability of fibrous plaques than of the adjacent normal intima also in moose.

The prevalence of affected animals 77.3 % (17/22) has to be considered quite high since the natural food of moose (*Ahlen* 1975) has no characteristics of experimental atherogenic diets (*Buck* 1958, *Toda et al.* 1981). The percentage of affected moose was relatively higher than in roe deer (56.7 %). This may be due to the fact that moose live longer than roe deer but also may suffer from shortage of food and cold weather (*Oldemeyer et al.* 1977, *Bishop & Rausch* 1974). Starvation (*Borensztajn et al.* 1970) and exposure to cold weather (*Radomski* 1966) can induce a marked increase in lipoprotein lipase activity in experimental rats, resulting in hyperlipidemia. Chronic hyperlipidemia may

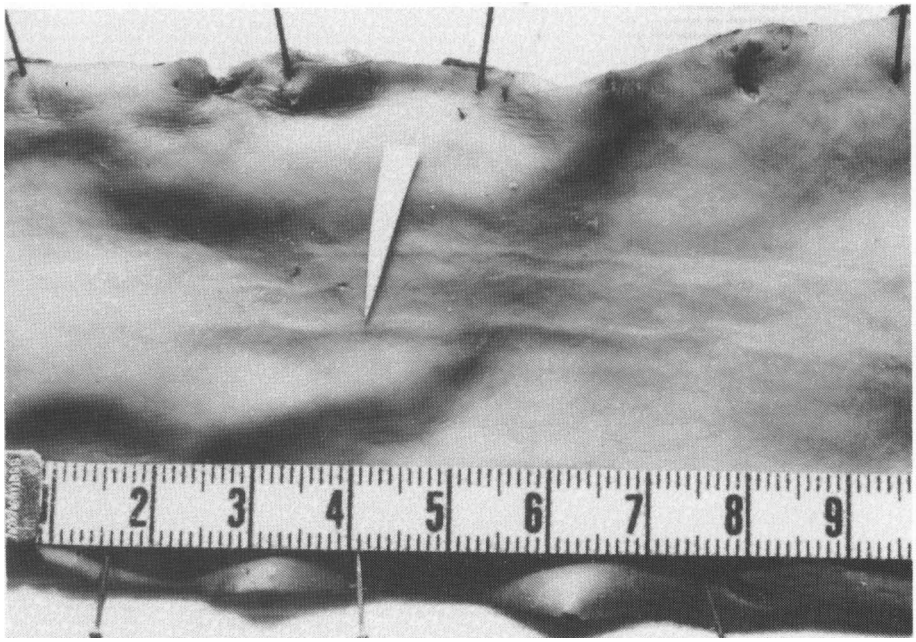

Figure 5. A slight elevated fibrous plaques measuring 1.2 x 7 cm located between two intimal ridges and extending ventrolongitudinally along the posterior thoracic aorta and anterior abdominal aorta. There is a slight discoloration in the intervening intima due to finely dispersed lipid in the superficial layer. 6-year-old moose (Sudan IV).

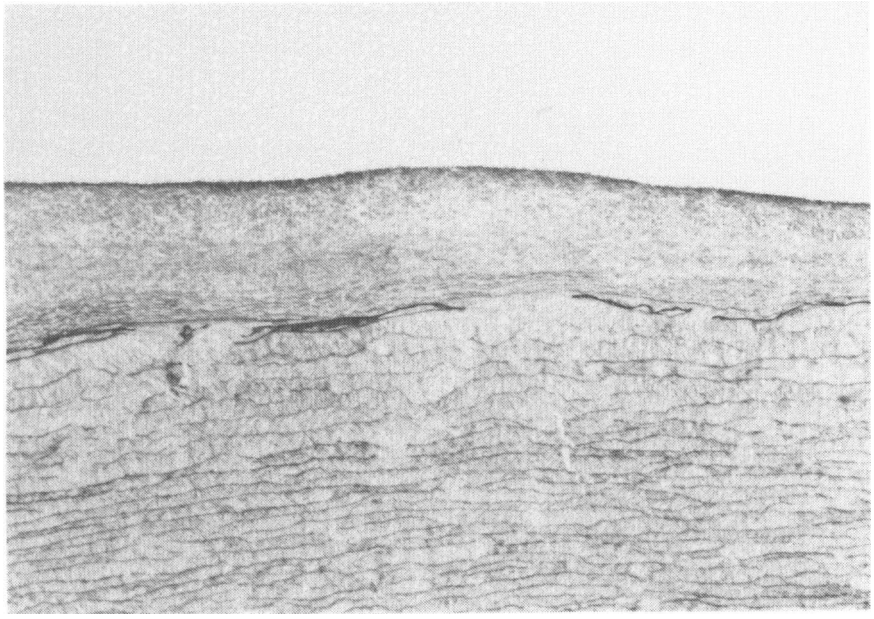

Figure 6. Section through a fibrous plaque in the anterior abdominal aorta (see Fig. 5), showing a flat fibrous plaque consisting predominantly of smooth muscle cells in the outer half and dense elastic tissue in the inner half of the tunica intima. Note the markedly stretched, disrupted and dissolved appearance of the internal elastic lamina and the elastic tissue degeneration accompanying the disoriented of the smooth muscle cells in the inner third of the tunica media. 6-year-old male moose (Verhoeff x 18).

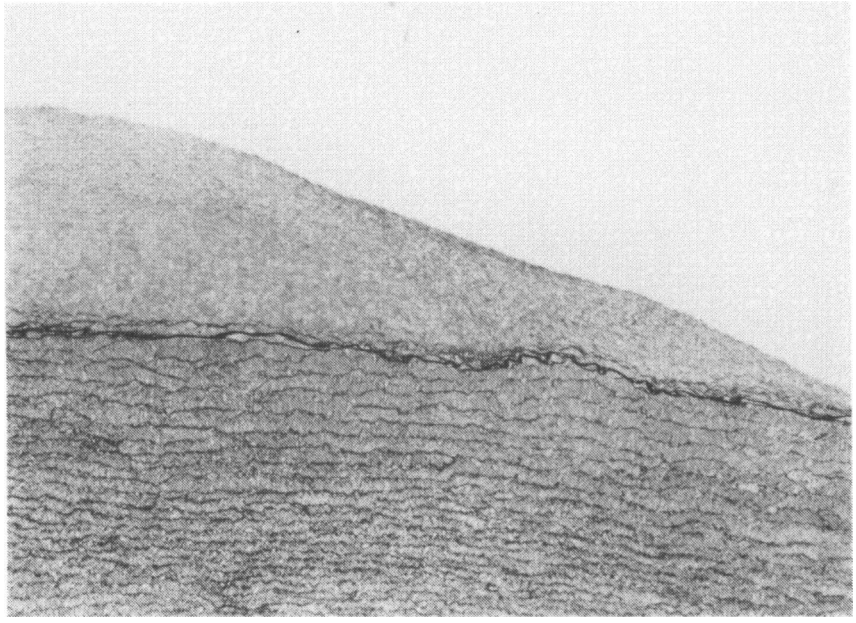

Figure 7. A dome shaped fibrous plaque at the posterior abdominal aorta. Beneath the plaque varying degree of reduplication of the internal elastic lamina is evident. Disorientation of smooth muscle cells and elastolysis (see Fig. 6 and 8) are apparent. 10-year-old female moose (Verhoeff x 44.8).

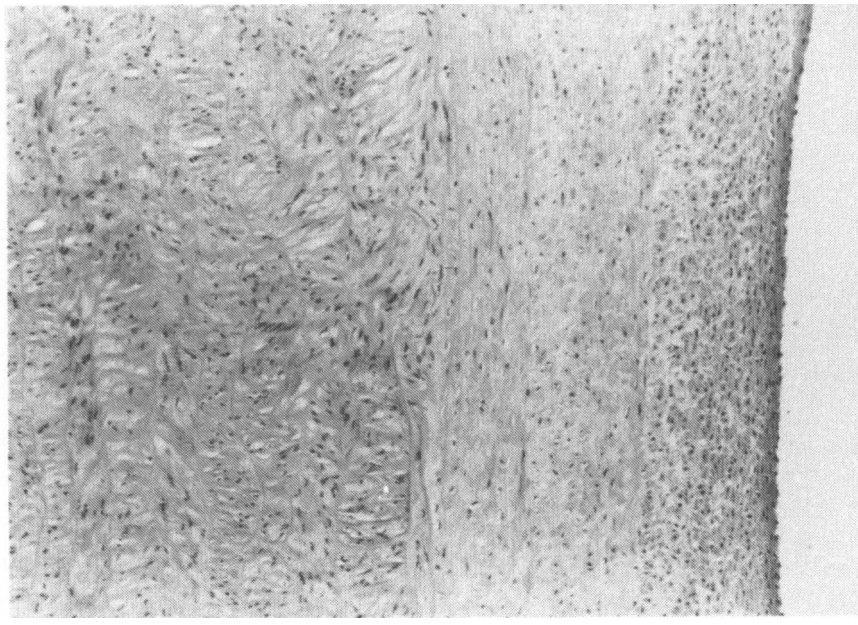

Figure 8. A fibrous plaque at the bifurcation, exhibiting proliferating endothelial cells forming a cap, ground substance, smooth muscle cells, fibroblasts, fibrocytes and elastic tissue. The polarity of the cells of the intima below the cap divide the intima into three different layers. The cells in the outer third of the intima are rounded, lacking polarities. The cells of the middle third are disoriented and the cells of the inner third are generally oriented with their long axis poles horizontal to the lumen of the aorta. Clusters of smooth muscle cells in the disrupted internal elastic lamina as well as disoriented medial smooth muscle cells in the inner half of the tunica media are evident. 6-month-old male moose (H & E x 112).

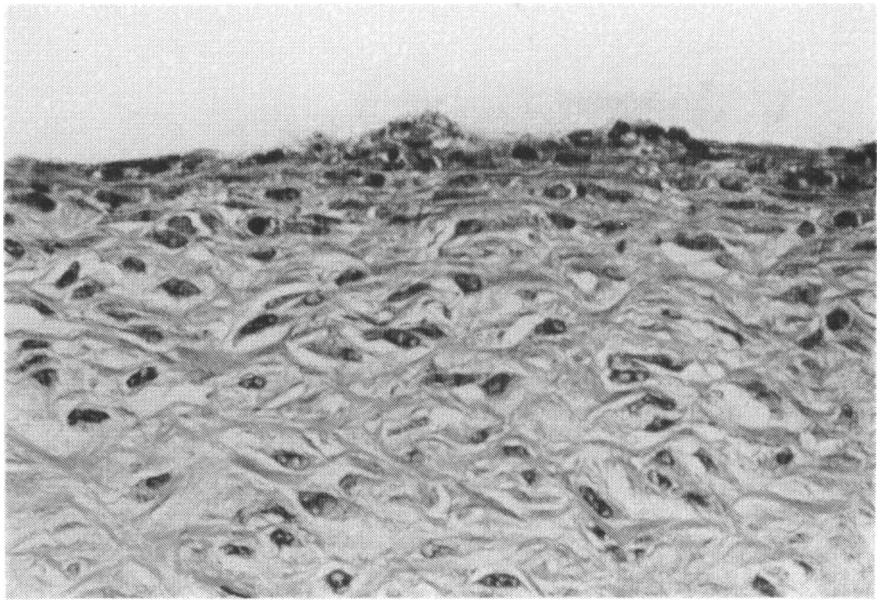

Figure 9. Higher magnification of the same lesion (see Fig. 8) exhibiting 4-5 layers of proliferating endothelial cells. They are flat and elongated and have basophilic cytoplasm. Note that the cells in the fibrous plaque beneath the endothelial cell layers are vacuolated. Mononuclear cells are also noticable (Verhoeff x 448).

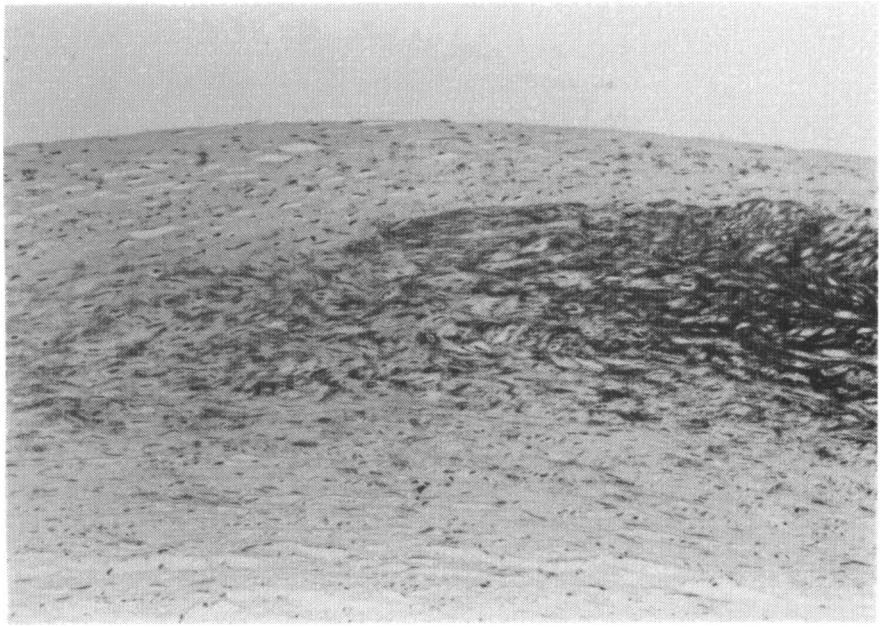

Figure 10. Extensive calcification of the necrotic core of the inner third of the tunica media and multifocal calcification of the tunica intima found at the bifurcation. 10-year-old female moose (von Kossa x 112).

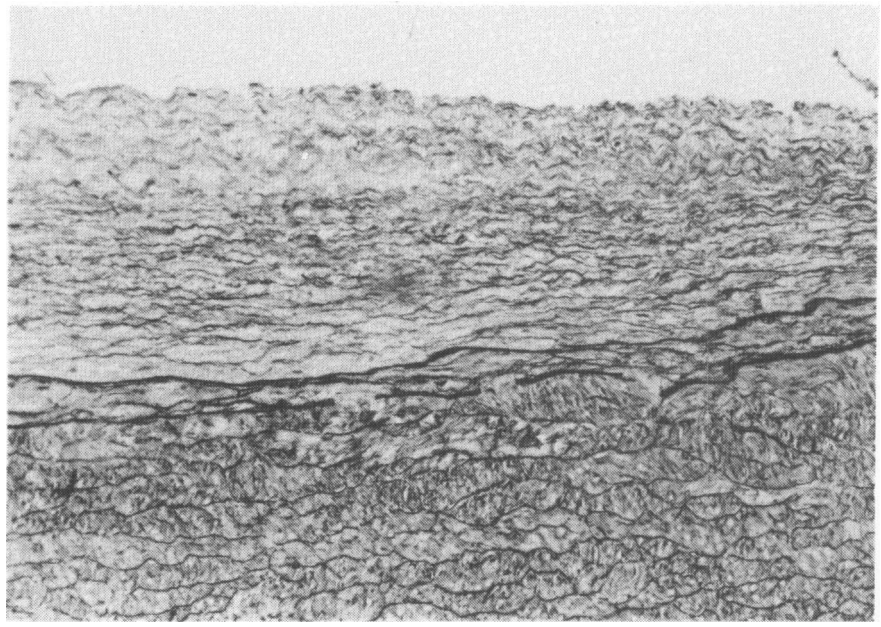

Figure 11. A fibrous plaque in the anterior thoracic aorta, showing dense elastic fibers and increasing numbers of collagen fibers in the inner of the tunica intima. The internal elastic lamina appears stretched, duplicated, fragmented and disrupted in some places. Disorientation of the medial smooth muscle cells associated with elastolysis in the inner half of the tunica media, was observed. 18-year-old female moose (Verhoeff x 112).

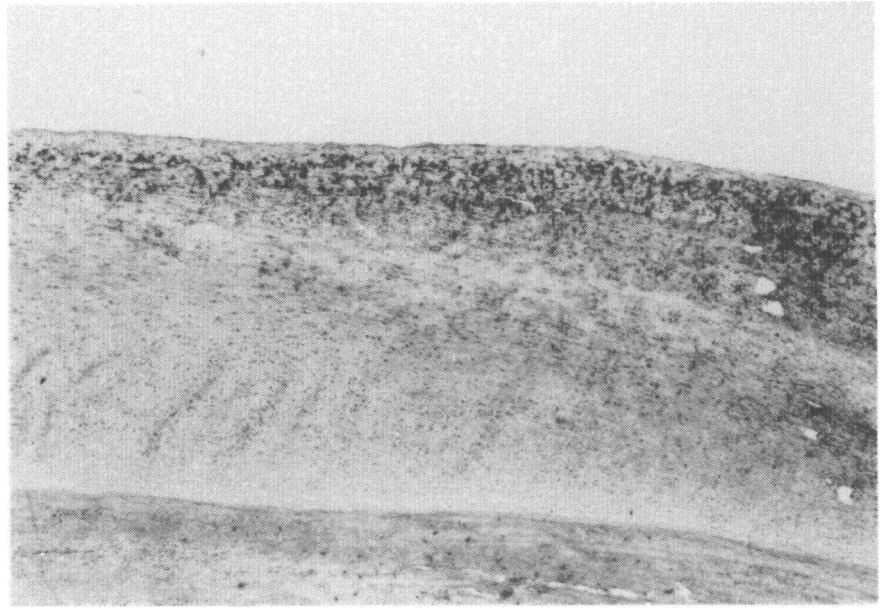

Figure 12. Section through fibrous plaque adjacent to the lesion shown in Fig. 11, containing a dense lipid accumulation in the outer half of the tunica intima and sparse deposition in the inner half of the tunica intima as well as the inner third of the tunica media. 18-year-old female moose (Sudan IV x 18).

result not only in an accumulation in atheromatous lesions but will also produce endothelial injury which is accompanied by platelet consumption and atherosclerosis (*Ross & Harker* 1976). Thus starvation or shortage of food, whereby the animals have to utilize their fat reserves and exposure to cold during winter time may be a contributory factor in the inducement of atherosclerotic lesions in moose.

There was no significant difference between male and female animals within each group but a tendency is seen towards more involved surface in females (Fig. 2) as found in roe deer (*Poungshompoo & Rehbinder* 1983). A comparative study of cholesterolemia between male and female rabbits (*Fillios & Mann* 1956) revealed that the female rabbit was more hypercholesterolemic after cholesterol feeding than the male. Experiments using female rabbits exhibited atherosclerotic lesions within coronary arteries after hypercholesterolemic induction, after a period as short as 24 h (*Parker* 1960). In man more surface involvement of fatty streaks was found in females than in males at the same age (*Holman et al.* 1958). Sex may possibly be a factor in the development of atherosclerotic aortic lesions in moose.

In moose, it was obvious that the aortic lesions started in the posterior abdominal aorta in the young animals. The lesions then gradually spread throughout the aortic arch the older the animals became. In addition, the percentage of involved surface in the different affected regions, comprising all age groups, was larger in the posterior abdominal aorta. The same finding has been reported in roe deer (*Poungshompoo & Rehbinder* 1983). In other ruminants, although without mentioning the percentage of involved surface, aortic lesions were reported to be frequently found in the abdominal aorta (*Prasad & Bhalla* 1977, *Prasad et al.* 1973). In man *Prior & Jones* (1952) observed that intimal fibrous plaques in children tended to be located primarily in the posterior part of the aorta. The morphology of these plaques corresponded closely to that of atherosclerosis in adults. These intimal thickenings were thus considered to represent the earliest phase in the development of larger and more advanced stages in older individuals. In man, *Glagov et al.* (1961) reported atherosclerosis to usually increase with age, and to be more severe in the abdominal than in the thoracic aorta. In addition hemodynamic and other mechanical factors are shown to influence

the localisation of atherosclerotic plaques (*Glagov* 1965). *Texton et al.* (1965) identified vascular dynamics as the primary cause of atherosclerosis while other atherogenic factors such as age, sex and diet should be considered as secondary contributing factors. They pointed out that an increased blood velocity, if other factors remain unchanged, produced severe atherosclerosis. In addition, they found that atherosclerotic lesions whether occurring naturally or induced by altering the hemodynamics, were consistently found or produced at the sites of diminished lateral pressure, such as at sites of bifurcation, branching and tapering. The aortic lesions found in moose thus correspond concerning the predilection sites with the statements by *Texton et al.* (1965). Hemodynamic force may thus also play a role in the distribution of atherosclerotic lesions in moose.

In moose proliferating endothelial cells were occasionally found over fibrous plaques. Endothelial cells have been reported to devide both in vivo and in vitro (*Poole et al.* 1958, *Wright* 1972, *Lewis et al.* 1973). New endothelial cells are formed by mitotic division from preexisting endothelial cells (*Poole et al.* 1958). They usually consist of a single layer of flattened cells but sometimes appear in double layers (*Fishman et al.* 1975). Endothelial mitosis at a localized injury of the aorta shows maximal activity within 24—48 h after damage but the mitotic activity is then slow and of limited extent (*Wright* 1972). In rat endothelial cell mitosis could be seen soon after an injury in the aorta. The initial activity was, however, not maintained, since repair of a superficial wound in the endothelial surface, originally about 1.5—2 cm long, was apparently incomplete as long as 232 days after injury (*Poole et al.* 1958). *Schwartz et al.* (1975) studied the repair process of aortic lining after mechanical denudation. They found that during the first week after injury, a temporary false endothelial lining covered the lesion. This lining consisted of modified intimal smooth muscle cells and did not constitute a continuous cell layer. They acted similar to true endothelial cells in that platelets did not adhere. Even though a continuous layer of endothelial cells was formed within two months after injury abnormalities persisted in the form of incompletely formed intercellular junctions. The same authors pointed out that persistent defects of the endothelial layer could result in a proliferation of the underlying smooth muscle cells, that could delay the healing process of the overlying endo-

thelium. In our investigation the endothelial lining of the fatty streaks and fibrous plaques was observed in forms of discontinuity.

Geer & Haust (1972) stated that with any type of vascular wall injury, the smooth muscle cells react with a change in their structure and undoubtedly a change in their function. The smooth muscle cells change morphologically into what had been described as "modified smooth muscle cells". These cells are smaller than normal smooth muscle cells and have an ovoid or stellate rather than a fusiform shape and are situated near the endothelium. Electron microscopy revealed that modified smooth muscle cells had less typical myofilaments in their cytoplasm (*Poole et al.* 1971). *Takebayashi et al.* (1972) observed and pointed out that the modified smooth muscle cell in the atheriosclerotic lesions was understood as an accelerated state of protein synthesis mostly the synthesis of collagen fibres and elastic fibres. In moose an increase in elastic tissue and two types of intimal smooth muscle cells were also observed.

The origin of smooth muscle cells in the areas of a vascular tissue in atherosclerosis is uncertain. There are three possible sources suggested, endothelium (*Crawford & Levene* 1952, *Movat et al.* 1959, *Haust et al.* 1960), an undifferentiated cell from the blood (*Still* 1966, *Still et al.* 1967, *Ghani & Tibb* 1969) and medial smooth muscle cell (*Lee et al.* 1970, *Poole et al.* 1971). The incidence of medial smooth muscle cells entering the disrupted internal elastic lamina through the tunica intima in moose and in roe deer (*Poungshompoo & Rehbinder* 1983) seems to support the last view that modified smooth muscle cells are derived from medial smooth muscle cells.

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SAMMANFATTNING

Spontana aortaskador hos älg (Alces alces L).

Hos 17 av 22 älgar, 6 månader — äldre än 18 år, förelåg aortaskador. Fem olika typer av skador påvisades; stråk av fett i 1, fibrösa knutor i 1, stråk av fett med komplicerande skador (förkalkning och sura mucosaccharider) i 3, fibrösa knutor med komplicerande skador i 2 och fibrösa knutor med komplicerande skador och fettdroppar i 4 aortor. Degeneration av elastisk vävnad, i de inre två tredjedelarna av tunica media, sågs huvudsakligen hos djur äldre än 4 år.

Det förelåg inget statistiskt säkerställt förhållande mellan ålder och frekvens ($P \sim 0{,}10$) men däremot en tendens till åldersbetingelse. Procenten förändrad yta befanns öka signifikant ($0{,}05 > P > 0{,}02$) med ålder. Signifikanta skillnader mellan könen i olika åldersgrupper förelåg ej.

Frekvensen inbegripen yta i olika skadade delar, omfattande alla åldersgrupper var; bakre abdominala delen 13,0 %, bakre thorakala delen 5,2 %, främre thorakala delen 0,1 % och aortabågen 0,1 %.

Prolifererande endotheliala celler och ursprunget för glatta muskelceller diskuteras.

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