Acta vet. scand. 1983, 24, 153-169.

From the Department of Pathology, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, Uppsala, Sweden.

SPONTANEOUS AORTIC LESIONS IN ROE DEER (CAPREOLUS CAPREOLUS L)

By

Somlak Poungshompoo and Claes Rehbinder

POUNGSHOMPOO, SOMLAK and CLAES REHBINDER: Spontaneous aortic lesions in roe deer (Capreolus capreolus L). Acta vet. scand. 1983, 24, 153—169. — In 34 out of 60 aortas from roe deer, aged from 6 months to more than 8 years, aortic lesions were found. The frequency of affected regions involved were, in the posterior abdominal portion 53.3 %, in the aortic arch 18.3 %, in the anterior abdominal portion 16.7 %, in the posterior thoracic portion 8.33 % and in the anterior thoracic portion 3.3 %. Of the observed lesions, fatty streaks were seen in 3, fatty streaks and fibrous plaques in 3, fatty streaks with complicated lesions (calcification and acid mucopolysaccharides) in 14 and fibrous plaques with complicated lesions in 14 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 years of age. The lesions significantly (P < 0.001) increased in number and severity with age and appeared to be more progressive in pregnant animals. There was, however, no significant difference between affected male and female animals in the different age groups.

roe deer; atherosclerosis; fatty streaks; complicated lesions; fibrous plaque.

Spontaneous atherosclerotic lesions of the aorta have been reported in several species of ruminants i.e. in cattle (Skold et al. 1967), Indian water buffaloes (Gupta & Singh 1978), goats (Prasad et al. 1976), reindeer (Gupta & Rehbinder 1981) and sheep, goat, white tailed deer and reindeer (Wigger et al. 1971).

Of the cervidae only reindeer and white tailed deer have been investigated. Roe deer are free ranging and rapidly growing and aging cervidae. Thus the objective of this investigation has been to study the prevalence of aortic lesions and their association with age and sex in this species.

MATERIAL AND METHODS

Aortas from 23 male and 37 female (of which 9 pregnant) roedeer (6 months to more than 8 years) were collected in connection with other investigations. 42 of these animals died from inanition, the rest were killed by hunters or predators during the period August 16, 1981 to March 25, 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 years. The aortas were removed intact, opened longitudinally, and fixed in 4 % buffered formalin.

The entire aorta from each animal was stained with Sudan IV to delineata fatty lesions. In order to study topographic relationships, the aortas were divided into 5 different anatomical regions. Those were: the aortic arch (including the area of the aortic valve), the anterior thoracic, the posterior thoracic, the anterior abdominal, and the posterior abdominal portion (including the bifurcation and the first 1 centimetre of the external and the internal iliac arteries). The lesions were then graded according to the WHO Study Group on Atherosclerosis (1958).

For histology, longitudinal pieces of tissue were cut from sudanophilic and other macroscopical lesions observed in the aortas. Sections were cut 5 μ and stained with haematoxylin and eosin, van Gieson, von Kossa, alcian blue and Hueck. Frozen sections were stained with Sudan IV for neutral lipid determination. Statistical analysis was performed by means of the χ^2 -test.

RESULTS

Four types of lesions were found in 34 of the 60 examined animals; fatty streaks, fatty streaks and fibrous plaques, fatty streaks with complicated lesions (calcification and acid mucopolysaccharides) and fibrous plaques with complicated lesions. The distribution is shown in Table 1.

The lesions significantly (P < 0.001) increased in number and severity with age (Table 1, Fig. 1). There was no significant difference (P > 0.05) between male and female animals inside the groups but a tendency towards a more frequent appearance in females (Fig. 2). In addition lesions appeared to be more progressive in pregnant roe deer. In pregnant roe deer 8 out of 9 (3 out of 4 pregnant animals of the >1-4 year group, 2 pregnant animals of the >4-8 year group and 3 pregnant animals of the

Age in years	Number of animals	Aortic lesions					
		Fatty streaks	Fatty streaks and fibrous plaques	Fatty streaks with com- plicated lesions	Fibrous plaques with com- plicated lesions	Total number	% Af- fected
1	22		3		2	5	22.7
>1-4	12			2	3	5	41.7
>4—8	10	2		4	3	9	90.0
>8	16	1	—	8	6	15	93.8
Total	60	3	3	14	14	34	

Table 1. Number of roe deer and distribution of different kinds of aortic lesions.

>8 year group) developed aortic lesions. The distribution of the affected regions involved was wider in pregnant animals than in barren animals of the same age. Most of the affected pregnant animals developed fatty streaks at the insertions of the aortic valves whereas this was seldom seen in the affected non pregnant animals. Furthermore, at the histological investigation the lesions of pregnant animals appeared to be more severe and were usually found to be fatty streaks or fibrous plaques with complicated lesions. In the yearling group, lesions occurred only in the posterior abdominal region. In animals of the >1-4 year group as well as in animals of the >4-8 year group, the lesions occurred in almost all regions except for the anterior thoracic portion. In the group >8 years, lesions were found in all regions (Fig. 3) and distributed as seen in Fig. 4.

Gross staining with Sudan IV sharply delineated intimal fatty streaks varying between $0.1-0.3 \times 0.5-10.0$ cm. In the aortic arch, fatty streaks were frequently noted at the insertions of the aortic valves. In the thoracic portion the lesions appeared as parallel, linear and longitudinal streaks localized at the ventral mid-line, between the orifices of the intercostal vessels. In contrast, in the abdominal portion, fatty streaks occurred as large irregular areas, mostly located at the mid-line, ventrally or dorsally, close to the iliac bifurcation. In the external as well as the internal iliac arteries the fatty changes did not appear as streaks but as stained circular wavy surfaces.

The histopathological investigations revealed that, in fatty



Figure 1. Percentage of affected roe deer which were showing different kinds of aortic lesions in each group.

streaks, the intima was 2—5 times thicker than a normal intima and in connection with intimal changes the media was also regularly $1\frac{1}{3}$ —2 times thickened. The thickened part of the tunica intima was composed by fibroblasts, fibrocytes, elastic fibres, a few mononuclear cells and a few smooth muscle cells. Both intracellular and extracellular vacuoles were observed. Intimal smooth muscle cells were found to be mainly oriented parallel to the long axis. The internal elastic lamina had lost its normal un-



🔲 : % affected male roe deer.

🔀 : % affected female roe deer.

There was no significant difference between affected male and female roe deer in each group.

Chi-square test: P > 0.05, df = 1.

Figure 2. Comparison of percentage affected male and female roe deer in each group.

dulation and at places also undergone fragmentation or was partly dissolved (Plate 1). In the tunica media varying degrees of oedema, elastolysis, vacuolation and disorientation of smooth muscle cells were seen. Smooth muscle cells in the inner thirds of the media were often seen to be perpendicular to the long axis of the aorta, their polar facing a gap in the disrupted internal elastic lamina (Plate 1).

Frozen sections showed varying degrees of lipid droplet deposition appearing either intracellularly or extracellularly or both in the inner half of the intima and sparsely in the $\frac{1}{3}$ to $\frac{2}{3}$ of the media (Plate 1 and 2). In contrast, calcification was found as fine or coarse granules deposited either intracellularly or extracellularly in the $\frac{1}{3}$ to $\frac{2}{3}$ of the media but also sparsely in



Figure 3. Distribution of aortic lesions in 5 different anatomic regions.



Figure 4. Percentage of different affected regions.

SPONTANEOUS AORTIC LESIONS IN ROE DEER (CAPREOLUS CAPREOLUS L)

By Somlak Poungshompoo and Claes Rehbinder



Plate 1. Abdominal aorta showing dense lipid accumulation of the inner half of the tunica intima near the internal elastic lamina. Note smooth muscle cells with their polars facing the disrupted internal elastic lamina. 8 years, female roe deer (Sudan IV \times 280).



Plate 2. Section through abdominal aorta illustrating lipid accumulation in the tunica intima of a 4 years, male roe deer (Sudan IV \times 110).



Plate 3. Abdominal aorta showing nodular fibrous plaques consisting mainly of smooth muscle cells. Note the medial smooth muscle cells disoriented, oblique and vertical with their polar facing the internal elastic lamina and the intimal smooth muscle cells also appearing irregular in their orientation to the long axis of the aorta. 5 years, male roe deer (H & E \times 280).



Plate 4. Left external iliac artery showing fibrous plaques consisting of dense elastic fibres. Note the medial smooth muscle cells disoriented oblique and vertical with their polar facing the internal elastic lamina. Older than 8 years, male roe deer (van Gieson × 280).



Plate 5. Left external iliac artery. Acid mucopolysaccharides in the ground substance of the inner two thirds of the media. Clusters of smooth muscle cells can be seen in the disrupted internal elastic lamina. 5 years, female roe deer (Alcian blue \times 280).



Plate 6. Patchy calcification, in the inner third of the tunica media and multifocal calcification of the tunica intima found at the bifurcation. 8 years, female roe deer (van Kossa \times 280).

multiple foci in the intima (Plate 6). Both lipid droplet deposition and calcification were also seen in the internal elastic lamina. These lesions were usually found in combination with an increase of acid mucopolysaccharides in the ground substance.

Macroscopically, the fibrous plaques appeared as non sudanophilic, raised, pearly glistening, firm and nodular lesions varying in size from 0.3 to 1.5 cm in diameter and usually situated at the site of the ligamentum arteriosum and in the posterior abdominal portion.

Microscopically, fibroplasia was found which was confined to the subendothelial portion of the intima. The thickness of the intima was markedly increased usually 10-40 times that of the normal and was more pronounced than that of the media (Plate 3). The plaques varied in their composition. Some plaques appeared as a cap of fibrous tissue with fibroblasts, fibrocytes, some smooth muscle cells, a few mononuclear cells and in complicated cases small deposits of calcium and acid mucopolysaccharides. Some plaques contained more components, i.e. elastic fibres, collagen fibres, proliferating endothelial cells and proliferating smooth muscle cells and in complicated cases deposits of calcium and acid mucopolysaccharides. The internal elastic lamina showed varying degrees of reduplications but also oedema, fragmentation and focal elastolysis. The most significant changes of the media were disoriented smooth muscle cells and degenerative changes. The smooth muscle cells were disoriented with an oblique and vertical position with their polars facing the internal elastic lamina (Plate 4). Clusters of smooth muscle cells could be seen in the disrupted internal elastic lamina and noted was also an increase of acid mucopolysaccharides in the ground substance of the intima and the media (Plate 5).

The inner $\frac{1}{3}$ to $\frac{2}{3}$ of the media, showed varying degrees of degeneration, i.e. hydropic changes, vacuolation and hyalinization of smooth muscle cells and elastic lamellae. In some cases numerous smooth muscle cells were found as round cells with intracytoplasmic vacuoles and excentric pyknotic nuclei. Calcification appeared as fine or coarse granules deposited either intracellular or extracellular in the $\frac{1}{3}$ to $\frac{2}{3}$ of the media and also sparsely in multiple foci in the intima. Necrotic cores of calcification could occasionally be seen in tissue adjacent to the internal elastic lamina in the inner thirds of the media (Plate 6). The internal elastic lamina was mostly also affected by multifocal calcification. Focal proliferation of vessels were occasionally observed in the inner $\frac{2}{3}$ of the media.

In the yearling group, fibrous plaques appeared as a cap of fibrous tissue with some smooth muscle cells and in complicated cases small deposits of calcium and acid mucopolysaccharides. In the groups of animals more than 4 years, the fibrous plaques contained more somponents, i.e. elastic fibres, collagen fibres, fibroblasts, fibrocytes, and some few mononuclear cells but also proliferating smooth muscle cells and in complicated cases deposits of calcium and acid mucopolysaccharides. Medial elastic tissue degeneration characterized by oedema, fragmentation and focal elastolysis of the $\frac{1}{3}$ to $\frac{2}{3}$ was mostly found in the groups of animals more than 4 years. In 3 cases lipid droplets could be found in the deeper part of the fibrous plaques. Only 1 case of fibrous plaques with complicated lesions appeared iron positive in the inner third of the media.

DISCUSSION

The fatty streaks, fibrous plaques and complicated lesions found in the aortas of roe deer resembled those found in man (WHO Study Group on Atherosclerosis 1958, Holman et al. 1958) and other wild or domestic ruminants (Skold et al. 1967, Wigger et al. 1971).

In the yearling group, the aortic lesions appeared as fatty streaks and fibrous plaques in 3 cases. The number and degree of the lesions obviously increased with age, i.e. number of fatty streaks with complicated lesions (calcification and acid mucopolysaccharides) and fibrous plaques with complicated lesions. Fatty streaks were recognized to be the prelesion stage of atherosclerosis in spontaneous cases in man (Holman et al. 1958) and in experimental pig (Gerrity et al. 1979, Gerrity 1981). In man, Holman et al. (1958) reported that simple fatty streaks were found in children as young as 9 months of age and that every case beyond the age of 7 years had at least minimal fatty streaks, but after the age of 30 years fibrous plaques were predominantly found. In addition by the age of 40 years, about 20 % of the surface involved by fatty streaks had been converted into fibrous plaques. He pointed out that it required about 15 years for this conversion and that other complicated lesions, i.e. haemorrhage and thrombosis, were rarely seen except an increase in acid mucopolysaccharides. In roe deer with an average of about 3.1 years (Andersen 1953) and a maximum age of about 13 years the development of aortic lesions are more rapid but otherwise quite similar to those of man. The increase in fibrous plaques in aging roe deer indicates that fatty streaks also in this species may change into fibrous plaques.

In roe deer aortic lesions including an increase in acid mucopolysaccharides, collagen fibres, fragmentation of elastic tissue and degeneration of smooth muscle cells were predominantly found in the group of animals more than 4 years. De Faria (1965) found that in the human aorta the volume of muscle cells and elastic tissue declined with age while the amounts of collagen and acid mucopolysaccharides increased. The severity of these changes were proportional to the degree of intimal thickening. He suggested that an increase in acid mucopolysaccharides and collagen will alter the permeability of the vascular wall and may operate as an initiating feature in atherosclerosis. Decrease in muscle and elastic tissue will thus eventually lead to change in the blood pressure. Hemodynamic pressure will then accelerate the deposition of calcium in the arterial elastin as found in elastic tissue in pulmonary arteries (Lansing 1951). The arterial elastic lamina normally acts as a barrier to the outward diffusion of lipoproteins and cholesterol (Wilens 1951b) but in old animals, with an age induced decrease of elastic tissue this barrier is not maintained. Westake et al. (1963) found that plaques in old dogs became infiltrated with lipid when fed an atherogenic diet containing cholesterol, saturated fat and thiouracil. Age changes in the aortic tissue components, characterized by a decrease of elastic and muscular tissue and an increase of collagen and acid mucopolysaccharides, will alter the permeability of the vascular wall and thus also operate as an initiating feature in atherosclerosis. The findings in this investigation indicate a similar relationship in roe deer.

In roe deer, mononuclear cells were usually present in the superficial layer of fatty streaks and fibrous plaques. Some smooth muscle cells appeared as rounded cells with pyknotic nuclei in the deeper part of the lesions. In experimental pigs, *Gerrity* (1981) stated that monocytes were the major foam cells in the prelesion stage of fatty streaks. They were generally seen only in the sub-endothelial layer. In the plaques, lipid laden smooth muscle cells and necrotic foam cells were the predominantly cell types. In addition, *Gaton & Wolman* (1977) demonstrated that monocyte derived macrophages, rich in acid esterase, can phagocytize and metabolize lipid better than smooth muscle cells which act as lipid containing cells in a diet induced atheroma in rabbit. They interpreted that smooth muscle cells are an important factor in the formation of atheroma and monocytes may play a major role in healing of atheroma. Thus mononuclear cells found in aortic lesions of roe deer may originate from monocyte derived macrophages and may thus also play an important role in healing processes. The rounded cells with intracytoplasmic lipid vacuoles and pyknotic nuclei were interpreted as smooth muscle cells.

The most significant change found in the tunica media of roe deer was the position of smooth muscle cells. They appeared disoriented with an oblique and vertical position towards the internal elastic lamina, at places indicating an entry into the intima through the rupture of the internal elastic lamina. This phenomenon was suggested to be influenced by an endothelial cell injury by Ross & Glomset (1976). In roe deer, endothelial cell injury and proliferation was evident in some plaques. Factors as hyperlipidemia, hormone dysfunction and hemodynamic stress may injure the endothelium and alter the nature of the endothelial barrier allowing the passage of blood constituents into the arterial wall. These factors may thus alter the endothelial cell-cell or the endothelial cell-connective tissue relation or both, permitting hemodynamic force to elevate and possibly to detach endothelial cells from endothelial wall. Focal desquamation of the endothelium caused in this way would leave the underlying subendothelial connective tissue exposed to platelets and other elements in the circulation (Ross & Glomset 1976). The platelets adhere to subendothelial collagen, aggregate and release the content of their granules (Mustard & Pacham 1970). The massive infiltration of platelet factors, plasma lipoproteins and possibly other plasma constituents such as hormones at sites of endothelial injury may lead to focal proliferation of arterial smooth muscle cells and to formation of large amounts of connective tissue matrix by these cells and in addition a desposition of lipid both within the cells and in the surrounding connective tissue matrix. According to this hypothesis, the lesion will regress if both the endothelial injury and tissue response are limited. However, if further proliferation or migration of medial smooth muscle cells through openings of the

elastic lamina takes place and later, if these cells actively multiply within the intima, this may result in an accumulation of connective tissue and lipids if the injury to the endothelial is continuous or repeated (*Ross & Glomset* 1976). Endothelial cells injury may thus have induced the medial smooth muscle cell migration and proliferation also in the intima of roe deer.

In roe deer, fibrous plaques varied in their composition, i.e. mononuclear cells, calcium, acid mucopolysaccharides, new vascular channels in the inner third of the media, fibroblasts, fibrocytes and collagen fibres. According to *Wissler & Vesselinovitch* (1977) in the later stages of the plaques, there may often be seen variable quantities of calcium deposits in the edge of the necrotic core of the plaques. In addition monocytes and capillaries were often present in the advanced plaques. The fibrous plaques with complicated lesions (calcification and acid mucopolysaccharides) in which new vascular channels and mononuclear cells were observed ought to be considered as advanced plaques in the later stages also in roe deer.

Elastic tissue degeneration was often found in the inner $\frac{1}{3}$ up to $\frac{2}{3}$ of the tunica media and rarely found in the outer $\frac{1}{3}$ of this part. This may be due to the fact that aortic lesions such as the thickened fibrous plaques and the complicated lesions may alter permeability of blood supply from lumen to the inner part. In addition, in roe deer blood vessels were normally observed in the outer third of media, but it was evident that vascular tracts from vasa vasorum were penetrating to the outer part of the media in connection with plaques and complicated lesions. In man the outer $\frac{1}{3}$ of the aorta is supplied by the vasa vasorum of the tunica adventitia while the inner $\frac{2}{3}$ of the aorta is nourished by direct permeation from the lumen of the principle vessel (Paterson et al. 1957, Clarke 1964 & 1965a&b). In the roe deer aorta, calcification and focal necrosis of elastic tissue were seen in this part even though in some plaques vascularization was detected. Elastic tissue degeneration and necrosis may probably also partly have been caused by insufficient blood supply.

Lesions were found predominantly in the abdominal portion as is reported in man (Holman et al. 1958) and other animals Wigger et al. 1971, Valtonen & Oksanen 1972).

Glagov (1965) observed that certain areas of the arterial vasculature were more prone to develop plaques than other. That was supported by *Texton et al.* (1965) who observed that

atherosclerosis frequently took place at ostia of branches and Jönsson (1979), who also found musculo-elastic thickening both in the tunica intima and tunica media at the ostia of aortic branches, curvatures and bifurcations. Glagov (1965) suggested that mechanical force, associated with blood flow, could be related to atherosclerosis i.e. wall tension, velocity and turbulence. Increased wall tension could result in a selective atherosclerotic localization by altering the vessel wall permeability (Esterley & Glagov 1963). Results obtained by Sako (1962) in experiments on dogs strongly suggested that an increase in the blood flow led to an increased atherosclerotic plaque formation. At branch points and bifurcations turbulence may occur at low velocities and helical pattern may occur as a result of modifications in the velocity profile. This helical pattern of turbulence may be the cause of the circular wavy surfaces found in the iliac arteries of roe deer. Hemodynamic force may thus play a role in the distribution of atherosclerotic lesions also in roe deer.

The prevalence of affected animals 56.7 % (34/60) has to be considered quite high though the natural food of roe deer (*Drozdz* & Osiecki 1973, Rehbinder & Ciszuk 1983) has no characteristics of experimental atherogenic diets (*Gerrity et al.* 1979, Okawa et al. 1980). Most of the examined roe deer had died from inanition which is a common cause of death in roe deer during winter time (Borg 1970). Aortic lesions have been found in salmon in connection with incomplete inanition during their swimming up river, prior to spawning (Robertson et al. 1961).

In well fed animals some of the plasma triglyceride fatty acids are taken up by muscle and oxidized. But when those fatty acids are in excess of the immediate caloric needs, they are removed and stored in adipose tissue. This process is taken care of by the lipoprotein lipase, found in adipose tissue, heart, skeletal muscle and capillary walls (Korn & Quigley 1955, Korn 1955a, Schnatz et al. 1963, Moskowitz & Moskowitz 1965). But during fasting state, the plasma triglyceride fatty acids, stored in tissues other than adipose tissues are removed to supplement the energy derived from the oxidation of the fatty acids mobilized directly from adipose tissue (Carlson et al. 1973a and b).

Experimental studies in the dog and in man indicate that lipid in the form of chylomicron triglycerides, are rapidly eliminated from the blood during starvation (*Halberg* 1965). From this point of view the arterial wall would be largely protected against hyperlipaemia during malnutrition. *Wilens* (1951) in addition, observed that the lipid were resorbed from aortic plaques of terminally malnutrition patients, but he found little evidence that calcium and collagen were removed from the lesion.

Roe deer during winter time does not only suffer shortage of food but also from water (*Rehbinder & Ciszuk* 1983). In addition, *Radomski & Orme* (1971) and *Roger & Robinson* (1974) showed that lipoprotein lipase activity will increase in experimental rates exposed to cold weather. Thus in the winter time during cold weather condition, when roe deer need more energy the stored fat will be removed to supply their energy need. During adipose tissue metabolization the plasma lipids may appear as hyperlipaemia in the fasting animals. Later the amounts of plasma lipid will decrease due to the uptake by the muscles for energy consumption or penetrate the arterial wall developing arterial lesions. Some animals may die before reaching the state of lipid resorption from the atherosclerotic lesions. This could explain why lesions were seen in many varying forms.

There was no significant difference between males and females but a tendency towards a more frequent appearance in females (Fig. 2). Holman et al. (1958) observed that the development of the aortic lesions in Negro females was similar to those in the Negro males, except between the age of 15 and 30 years when the lesions, fatty streaks actually involved a larger surface in females than in males of the same age. Similar lesions appeared to be more progressive in pregnant roe deer. In 8 out of 9 pregnant females, aortic lesions were found. The distribution of the affected regions involved was wider and more severe than in the affected barren animals of the same age. This may be due to the fact that pregnant animals require more energy supply (Drozdz & Osiecki 1973). They thus suffered from a more pronounced starvation than the non pregnant animals of the same age. Pregnancy is probably a predisposing cause in the development of aortic lesions.

CONCLUSIONS

Inanition, hemodynamic stress and age dependent changes of the tissue components of the arterial wall are probably involved in the development of aortic lesions in roe deer. In roe deer fatty streaks can apparently gradually change into fibrous plaques. Besides, endothelial cell injury, possibly caused by hyperlipidaemia and hemodynamic stress can initiate medial smooth muscle cells migration and proliferation through openings or fragmentation of the internal elastic lamina, leading to development of atheroma or plaques. Furthermore, mononuclear cells originating from monocyte derived macrophages may play a role in the healing of atheroma. In this investigation, the aortic lseions had a tendency to occur more frequently and to be more severe in females than in males.

The predominant localization of the lesions was in the abdominal aorta and complications were characterized by calcification and deposition of acid mucopolysaccharides resembling the findings in man.

ACKNOWLEDGEMENTS

The skillful technical help by Gunilla Eriksson is gratefully acknowledged.

REFERENCES

- Andersen, J.: Analysis of a Danish roe deer population. In: Analysis of Danish review of game biology. Ed. by Spärck, R. and H. M. J. H. Thamrup. Vol. II. Schulz Publisher, Copenhagen 1953, pp. 131-153.
- Borg, K.: On mortality and reproduction of roe deer in Sweden during the period 1948—1969. Swedish Wildlife 1970, 7, 121—149.
- Carlson, L. A., I. Erikson & G. Walldius: A case of massive hypertriglyceridaemia and impair fatty acid incorporation into adipose tissue glycerides (FIAT) both corrected by nicotinic acid. Acta med. scand. 1973a, 194, 363—369.
- Carlson, L. A., G. Walldius & A. G. Olsson: Evidence for a defect in fatty acid uptake by adipose tissue of patient with hypertriglyceridaemia. J. clin. Path. 26. suppl. 1973b, 5, 48-52.
- Clarke, J. A.: An X-ray microscopic study of the vasa vasorum of normal human coronary arteries. J. Anat. (Lond.) 1964, 98, 539-543.
- Clarke, J. A.: An X-ray microscopic study of the postnatal development of the vasa vasorum in the human aorta. J. Anat. (Lond.) 1965a, 99, 887-889.
- Clarke, J. A.: The vasa vasorum or normal human lower limb arteries. Acta anat. (Basel). 1965b, 61, 481-487.
- De Faria, J. L.: Role of medial change in the pathogenesis of the intimal thickening and in atherosclerosis. J. Atheroscler. Res. 1965, 5, 509-515.

166

- Drozdz, A. & A. Osiecki: Intake and digestibility of natural feed by roe deer. Acta theriol. 1973, 18, 81-91.
- Esterley, J. A. & S. Glagov: Altered permeability of the renal of the hypertension rat. An electron microscopy study. Amer. J. Path. 1963, 43, 619-631.
- Gaton, E. & M. Wolman: The role of smooth muscle cells and hematogenous mocrophage in atheroma. J. Path. 1977, 123, 123-125.
- Gerrity, R. G., H. K. Natio, M. Richardson & C. J. Schwartz: Dietary induced atherogenesis in swine. Amer. J. Path. 1979, 95, 775– 785.
- Gerrity, R. G.: The role of the monocyte in atherogenesis. Amer. J. Path. 1981, 103, 181-200.
- Glagov, S.: Hemodynamic factors in localization of atherosclerosis. Acta cardiol. Suppl. 11. 1965, 11, 311-337.
- Gupta, P. P. & B. Singh: Natural occurring atherosclerotic lesions and aging change in the aorta of Indian water buffaloes (Bubalus bubalus). Zbl. Vet.-Med. A. 1978, 25, 231-240.
- Gupta, P. P. & C. Rehbinder: Spontaneous aortic lesions in reindeer. Acta vet. scand. 1981, 22, 60-66.
- Halberg, D.: Elimination of exogenous lipid from the blood stream. An experimental methodological and clinical study in dog and man. Acta physiol. scand. 65. suppl. 1965, 254, 1-23.
- Holman, R. L., H. C. McGill, J. F. Strong & J. C. Geer: The natural history of atherosclerosis (The early aortic lesions as seen in New Orleans in the middle of the 20th century). Amer. J. Path. 1958, 34, 209-235.
- Jönsson, L.: Coronary lesions and myocardial infarct in the dog. Acta vet. scand. Suppl. 1972, 38, 1-80.
- Korn, E. D.: Clearing factor, a heparin activated lipoprotein lipase. I. Isolation and characterization of the enzyme from normal rat heart. J. biol. Chem. 1955, 215, 1—14.
- Korn, E. D. & J. W. Quigley: Studies on lipoprotein lipase of rat heart and adipose tissue. Biochim. biophys. Acta (Amst.). 1955, 18, 143-145.
- Lansing, A. I., E. Roberts, G. B. Ramasama, T. B. Rosenthal & M. Alex: Change with age in amino acid composition of arterial elastin. Proc. Soc. exp. Biol. Med. (N.Y.). 1951, 76, 714-717.
- Moskowitz, M. S. & A. A. Moskowitz: Lipase localization in adipose tissue. Science 1965, 149, 72-73.
- Mustard, J. F. & M. A. Packham: Factor influencing function adhesion, release, and aggregation. Pharmac. Rev. 1970, 22, 97–187.
- Okawa, H., K. Doi, A. Yasoshima, T. Fugita & A. Okaniwa: Pathology of experimental atherosclerosis, change of acute phase in rats loaded with vitamin D₂ and cholesterole. Jap. J. vet. Sci. 1980, 42, 623-633.

- Paterson, J. C., J. Mille & T. Moffat: Vascularization of early atherosclerotic plaques. Archs. Path. 1957, 64, 129-136.
- Prasad, M. C., B. S. Rajya & G. C. Mohanty: Study on lipid pattern and protein content of caprine aortic atherosclerosis. Indian J. vet. Sci. 1976, 46, 179-183.
- Radomski, M. W. & T. Orme: Response of lipoprotein lipase in various tissue to cold exposure. Amer. J. Physiol. 1971, 220, 1852–1856.
- Rehbinder, C. & P. Ciszuk: Supplementary feeding of roe deer (Capreolus capreolus) with late harvest hay. A pilot study. J. Wildl. Manage. 1983, in press.
- Robertson, O. H., B. C. Wexler & F. B. Miller: Deneration changes in the endovascular system of Pacific salmon (Oncornynchus tshawytscha). Circulation Res. 1961, 9, 826-834.
- Roger, M. P. & D. S. Robinson: Effect of cold exposure on heart clearing factor lipase and triglyceride utilization in the rat. J. Lipid Res. 1974, 15, 263-272.
- Ross, R. & J. A. Glomset: Medical progress. The pathogenesis of atherosclerosis. New Engl. J. Med. 1976, 395, 369-377, 420-425.
- Sako, Y.: Effect of turbulent flow and hypertension on experimental atherosclerosis. J. Amer. med. Ass. 1962, 179, 36-40.
- Schnatz, J. D., J. W. Ormsby & R. H. Williams: Lipoprotein lipase activity in human heart. Amer. J. Physiol. 1963, 205, 401-400.
- Skold, B. H., N. L. Jacobson & R. Getty: Spontaneous atherosclerosis of bovine. J. Dairy Sci. 1967, 50, 1712.
- Texton, M., A. M. Imparato & M. Helpern: The role of vascular dynamic in the development of atherosclerosis. J. Amer. med. Ass. 1965, 149, 168—172.
- Valtonen, M. H. & A. Oksanen: Cardiovascular disease and nephritis in dog. J. small Anim. Pract. 1972, 13, 687-697.
- Westake, G. E., S. M. Grundy & R. M. O'Neal: The effect of an atherogenic diet on preexisting aorta intimal thickening in old dog. Exp. Molecul. Pathol. 2. Suppl. 1963, 1, 1-8.
- Wilens, S. L.: The resorption of arterial atheromatous deposits in wasting disease. Amer. J. Path. 1951a, 23, 793-804.
- Wilens, S. L.: Experimental of lipid deposition in existed arteries. Science 1951b, 114, 389-393.
- Wigger, K. D., N. L. Jacobson & R. Getty: Atherosclerosis in ruminants. J. Anim. Sci. 1971, 32, 1037-1041.
- Wissler, R. W. & D. Vesselinovitch: Atherosclerosis in non human primates. Adv. Vet. Sci. Comp. Med. 1977, 21, 351-361.
- WHO Study Group on Atherosclerosis: Classification of atherosclerotic lesions. Tech. Rep. Ser. Wld. Hlth. Org. No. 143, WHO, Geneva 1958, p. 1—22.

SAMMANFATTNING

Spontana aortaskador hos rådjur (Capreolus capreolus L).

Hos 34 av 60 undersökta rådjur, 6 månader — äldre än 8 år, förelåg aortaskador. Frekvensen skadade regioner var: bakre abdominala delen 53,3 %, aortabågen 18,3 %, främre abdominala delen 16,7 %, bakre torakala den 8,3 % och främre torakala delen 3,3 %. Stråk av fett förelåg hos 3, stråk av fett och fibrösa knutor hos 3, stråk av fett med komplicerade skador (förkalkningar och sura mucopolysaccarider) hos 14 och fibrösa knutor med komplicerade skador hos 14. Degeneration av elastisk vävnad i de inre $\frac{4}{3}$ av tunica media förelåg huvudsakligen i aortor hos djur äldre än 4 år. Skadorna ökade signifikant i antal och svårighetsgrad med ålder med en tendens till mer utbredda skador hos dräktiga djur. Det förelåg emellertid ingen signifikant skillnad mellan hon- och handjur i olika åldersgrupper.

(Received March 7, 1983).

Reprints may be requested from: C. Rehbinder, the Department of Pathology, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, S-750 07 Uppsala, Sweden.