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phagosomes consisting of cell debris and/or erythrocytes, as well as electron-dense structures of 20–30 nm diameter resembling the HVD virus particles.

Phagocytosis stimulation normally leads to intravascular macrophage expansion (9), which might be related to the loss of the flattened appearance of these cells in healthy animals (9). Such a loss has also been reported to occur on inoculation of endotoxin (8) and the African swine fever virus (7); in this last case, and because of the cytopathic effect of the virus on the macrophages, where it replicates, they undergo some rounding and show few linking modes, which is consistent with the image of most of the macrophages observed by us, where replication did not seem to occur.

Most authors (2, 7–9) relate the phagocytosis of blood forming elements and cell debris to hydrodynamic pulmonary changes which may arise from the expansion of the macrophages, with the resulting

increase in capillary resistance and contribution to pulmonary hypertension and edema, the last of which is reportedly one of the major features of HVD.

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ELECTRON MICROSCOPIC OBSERVATIONS ON ATRIAL MYOENDOCRINE CARDIAC CELLS IN BOVINE, PIG AND RABBIT HEART TISSUE

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Adult mammalian atrial myocardium contains a cell population characterized by the presence of electron dense membrane-bound granules referred as atrial specific granules.¹

Immunohistochemical studies at light and electron microscopic levels carried out in man and rat, showed that these granules contain bioactive polypeptides, referred as Atrial Natriuretic Peptide (ANP) or Atrial Natriuretic Factor (ANF), involved in water-electrolyte



Fig. 1: Electron micrograph, left auricle, rabbit. Atrial specific granules (arrows) are typically found at the nuclear poles. Bar = 0.40 μ m.

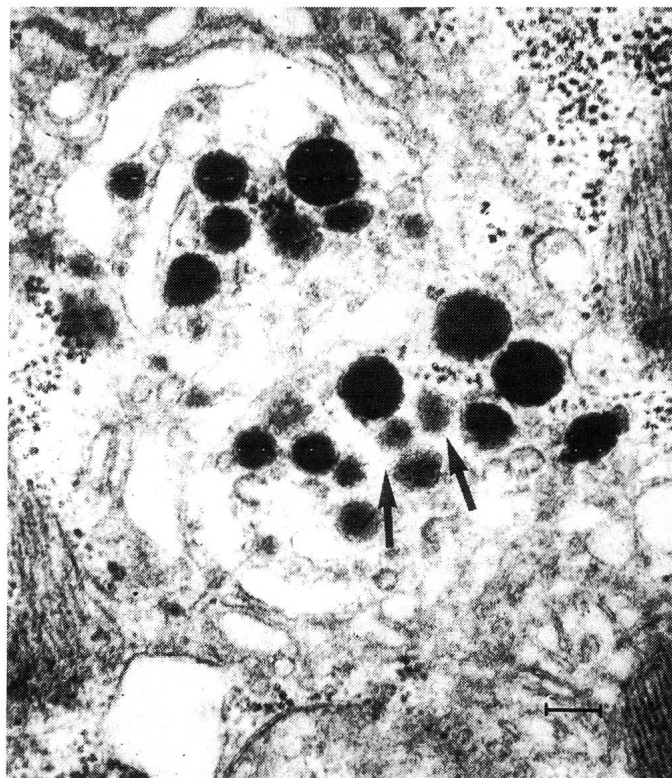


Fig. 2: Electron micrograph, right auricle, cow. Some progranules (arrows) are present in the Golgi cisternae. Other mature granules can also be observed. Bar = 0.15 μ m.

balance by a combination of complementary actions. Although in man and rat the ultrastructural appearance and distribution of atrial specific granules have been well described,² less morphological data are available for other mammalian species. In order to better characterize the ultrastructural features of normal atrial myoendocrine cardiac cells and to obtain morphological data on the morphology and the distribution of atrial specific granules during normal condition in cattle, pig and rabbit, 19 samples (5 from cattle, 8 from pig and 4 from rabbit) were taken from the right and left auricular appendages and fixed in 2.5% glutaraldehyde in 0.1 M cacodylate buffer (pH 7.4). Samples were post-fixed in 1% osmium tetroxide, dehydrated in a graded ethanol series and embedded in an Epon-Araldite mixture. Thin sections were stained with uranyl citrate and lead citrate and photographed with a Zeiss EM 109 with transfiber-optic photography.

The general morphology of atrial myoendocrine cells did not differ from that of ventricular myocardium. However, atrial specific granules were readily found in almost all auricular myoendocrine cells. Electron microscopic observation revealed that the peculiar location of atrial specific granules in all samples was the sarcoplasmic cone adjacent to the nuclear poles (Fig. 1). However, not infrequently they were scattered between myofibrils in close relationship with mit-

ochondria and in proximity to sarcolemma and the T-tubule system. Mature granules, also referred als A-granules, contained a highly osmiophilic and electron-dense material surrounded by a membrane (Fig. 2). Frequently, between the dense core and the membrane a fine electron-lucent halo could be shown. The diameter of mature granules ranged from 200 to 300 nm in all species tested.

In some cells smaller progranules could be observed within cisternae of Golgi apparatus in close relationship with mature granules (Fig. 2).

No noticeable differences between the three species tested were detected.

The ultrastructural features of atrial specific granules in cattle, pig and rabbit described in this paper will be useful to compare the morphological picture of several pathological states of the endocrine heart.

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LECTIN HISTOCHEMISTRY OF NORMAL AND NEOPLASTIC CANINE TESTICLE

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In male reproductive system of different mammalian species, lectin histochemistry has been used to localize specific domains on spermatogenic cells during differentiation and maturation, and on Sertoli cells^{1,2}. Moreover, in human neoplastic pathology, lectins have been employed to investigate the nature, the origin of several germ cell tumors³, and to distinguish different types of seminoma^{4,5}. In order to characterize saccharide components of complex glycoconjugates in different tumours of the canine testicle and in an attempt to follow the fate of different cell types after neoplastic transformation, we have undertaken a study by means of 12 different biotinylated lectins (PNA, Con-A, DBA, SBA, GS-I, LCA, LEA, PWM, RCA-I, RCA-II, WGA and UEA-I) and the avidin-biotin-peroxidase method (ABC) on four normal canine testicles, five seminomas, five Sertoli cell- and five Leydig cell-tumours. All samples were selected from the files of our department. PNA, DBA and SBA were also incubated after neuraminidase treatment (PNA-N, DBA-N, SBA-N).

In normal canine testis, biotinylated lectins were divided into four groups according to their binding pattern with spermatogenic cells: group 1 (Con-A, LCA, LEA, RCA-II and WGA), reacting with all spermatogenic cells; group 2 (PNA-N and RCA-I), binding to spermatocytes, spermatids, and spermatozoa; group 3 (GS-I, PNA, PWM, SBA and SBA-N), staining spermatids and spermatozoa; group 4 (DBA, DBA-N and UEA-I) which did not stain any spermatogenic cells.

Sertoli and Leydig cells were stained only with six out of the 12 lectins used (Con-A, WGA, LEA, RCA-II, LCA and RCA-I). All the

positive lectins belong to the first and the second group. Eight lectins bound to the tumours tested (Table 1).

Six lectins (Con-A, WGA, LEA, RCA-I, PNA-N and SBA-N) bound to neoplastic cells like in normal conditions. While Con-A, WGA, LEA and RCA-I bound to all neoplasms tested, PNA-N and SBA-N stained only the neoplasms arising from the spermatogenic line. It is noteworthy that none of the lectins which selectively reacted with

Table 1. Distribution of lectin binding sites on normal and neoplastic spermatogenic, Sertoli and Leydig cells.

Lectin used	Spermatogenic Cells		Sertoli Cells		Leydig Cells	
	A*	B	A	B	A	B
Con-A	1	5/5	4/4	5/5	4/4	5/5
WGA	1	5/5	4/4	5/5	4/4	5/5
LEA	1	5/5	4/4	5/5	4/4	5/5
RCA-I	2	5/5	4/4	5/5	4/4	5/5
RCA-II	1	5/5	4/4	0/5	4/4	5/5
LCA	1	5/5	4/4	0/5	4/4	5/5
PNA-N	2	5/5	0/4	0/5	0/4	0/5
SBA-N	3	2/5	0/4	0/5	0/4	0/5

A = normal; B = neoplastic

* The number is referred to the group according to the binding pattern with spermatogenic cells.

Lectins which do not stain any type of tumor are omitted.