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INFORMATION

Hokkaido University conferred the degree of Bachelor of Veterinary Medicine to the following 39 graduates of the School of Veterinary Medicine on March 25, 2002.

The summaries of their theses are as follows :

Innervation of the horse pericardium revealed by immunohistochemistry for protein gene product 9.5, a neuron-specific protein

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The pericardium is generally believed to play important roles in fixation, protection, and lubrication of the heart. Some studies, however, suggest that the pericardium functions as a sensory apparatus, because of the rich existence of nerve fibers. The present study deals with entire innervation by using whole mount preparations of the horse pericardium, that has been not reported in detail yet. In this study I used an antibody against protein gene product 9.5 (PGP 9.5) that is the most reliable marker of neurons.

The pericardium consists of three layers : mesothelium on cardiac side, fibrous layer, and mesothelium on thoracic side. PGP 9.5-immunoreactive nerves were distributed in two different layers beneath the mesothelium of cardiac and thoracic sides. Electron microscopically, nerves on cardiac side were localized in connective tissues just under the mesothelium, sometimes contacting the mesothelium, and terminated as free nerve endings. Observation of whole mount preparations demonstrated entire extension of the two nerve plexuses. The highest density of the innervation on each side was found close to the upper part of the heart, especially over the atrioventricular groove. Nerve fibers on

cardiac side repeatedly branched up and overlapped each other to form a characteristic meshwork. While, on the thoracic side, nerve fascicles ran along blood vessels and branched thin nerves that formed a comparatively loose network. No nerve fibers were found in fibrous layer.

Some electrophysiological studies have demonstrated that electrical stimulations onto the pericardium are transmitted to the brain with afferent fibers in the phrenic nerve. Therefore, the rich distribution of nerves at upper region of the heart may be explained by the fact that left and right phrenic nerves directly invade the pericardium in this region. On the other hand, I can interpret that nerves exist densely at the region where physical stimulation and stress are high in magnitude. Kinds of stimulations that pericardial nerves accept remain unknown. Dense distribution of substance P/ CGRP-containing nerves in the pericardium of the rat and mouse suggests that they receive signals released from cardiac and pericardial cells damaged by massive dilation, and inflammatory factors released into the pericardial space. In the present study, I could not observe special nerve endings classified mechanoreceptors like Pac-

inian and Meissner corpuscles in horse pericardium, as in other animals. It is possible to consider, however, that the pericardial nerves

receive and transmit, as whole, physical stimulations, such as pressure and extension, by regularly arranged meshwork of nerves.

Histological study on mucosal lesions of gastric ulcer in horses

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Gastric ulcers have been identified in over 80% of racing Thoroughbred horses. Ulcers in horses differ from those in humans, in that almost all lesions exist at non-glandular gastric mucosa covered with stratified squamous epithelium in horses. The purpose of this study is to understand the pathogenesis of the racehorse-specific illness from a morphological point of view. Six horses (1 to 17 years old) were used in the present study. Ulcerative erosions were detected in three horses at stratified squamous epithelium of the margo plicatus. When gastric ulceration was highly severe, mucosal erosions were also found at pyloric part of the stomach. The horse gastric ulcer was characterized by the invasion of papillary lamina propria with prominent cellular infiltration into stratified squamous epithelium. Electron microscopy (TEM) demonstrated that the infiltrating cells were largely neutrophils, and many neutrophils directly contacted epidermal keratinocytes, some invading into epithelial tissue.

These neutrophils were closely related to necrosis and destruction of epithelial cells. Observation of glandular and non-glandular mucosa injected with indian ink demonstrated less developed vascular systems in non-glandular parts. Although I cannot exclude the possibility of bacterial infection as a cause of gastric ulcer, ischemic necrosis with invasion of neutrophils are more important. It is known that myofibroblasts appear in the repair process of gastric ulcer in humans and laboratory animals. Immunostaining with α -smooth muscle actin antibody demonstrated accumulation of positive cells upon lamina muscularis mucosae, in which the epithelium was lost. Such a myofibroblastic layer was not formed in the part where stratified squamous epithelium still survived. Even though tissue destruction and inflammation expanded to larger areas, erosion did not extend to the submucosal layer and muscular layer. In this point, it might not be proper to use the term "ulcer".