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To enable statistical evaluation, the following histologic criteria were chosen: presence or absence of cardiomyocyte necrosis and degree of peri- and endomysial myocardial fibrosis; presence or absence of vascular changes in small pulmonary veins; presence or absence of foci of interstitial nephritis; presence or absence of hepatic centrolobular congestion in connection with hepatocyte degeneration.

Results and discussion

Cardiomyocyte necrosis was found on 5.8% of histological slides of control animals, on 10.3% in B animals, and on 15.1% of A animals (C vs. A: $p < 0.01$; B vs. A: $p < 0.05$). When 3 or more out of the 5 slides from each heart showed necrosis, the animal was classified as «necrosis positive» (Np): 9 A, 5 B and no C animals fell into this category. 7 of these 14 animals (4 A and 3 B) presented additionally distinct myocardial fibrosis, a finding not recognized in animals without significant cardiomyocyte necrosis. Sporadic changes in other organs (lung, kidney, liver) were rare and there was no evidence of statistical differences between the three groups ($p > 0.05$).

Biochemical myocardial amino-acid analysis revealed the following amino-acid ratios:

Amino-acid ratios	Controls (N=12)	Confirmed cases of BCMP (N=6)	Steers with fibrosis and necrosis (N=3)
Glu/OH-Pro	27.06 ± 4.37	4.64 ± 0.95*	9.63 ± 2.60*
Lys/OH-Pro	18.56 ± 2.98	2.46 ± 0.59*	5.03 ± 1.22*
His/OH-Pro	4.69 ± 0.76	0.73 ± 0.15*	1.66 ± 0.45*

* $p \pm 0.001$ when values are compared with respective control values

The study population consisted of animals kept in comparable environmental conditions, they only differed in their ancestry. The identified cardiomyocyte necrosis (Np) could represent the first visible lesion of BMCP, because necrosis without fibrosis could be observed, whilst fibrosis without necrosis did not occur. Degeneration of cells attracts macrophages and also induces fibroblast proliferation. This could partially explain the observed increase of collagen fibers. Additionally, loss of contractile units through cardiomyocyte necrosis may result in hypertrophy of remaining cardiomyocytes in order to maintain an adequate heart performance. As the disease progresses, heart failure develops and wall tensions then rise through the increasing ventricular dilatation, so provoking an additional increase in myocardial collagen. The lesions found in liver and lung of advanced cases most probably are secondary changes resulting from congestive heart failure. The result of myocardial amino-acid analysis discloses a possible approach to a biochemical test for detecting early stages of the disease. Further studies are needed to confirm these findings, and to expand biochemical analysis to other domains such as finding markers for the disease in body fluids, with the aim to establish a simple and rapid screening test for this fatal and costly disease.

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BIOLOGY OF THE FELINE IMMUNODEFICIENCY VIRUS

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Feline immunodeficiency virus (FIV), a lentivirus, was first described in 1987 in cats with clinical symptoms of immunodeficiency. Since its discovery, it has been detected all over the world and it has been cloned and sequenced. In its structure it is very similar to the lentiviruses of other species. Major components of the virus are gag proteins with apparent molecular weights of 10 000, 15 000 and 24 000 daltons, and components of the envelope with 43 000 and 130 000 daltons. Immunologically FIV is only distantly related with lentiviruses of other species. An exception is equine infectious anemia virus (EIAV); antibodies to FIV p24 strongly cross-react with EIAV p26.

In nature FIV-infection is mainly transmitted by bites. In most cases kittens born to FIV-infected queens are not infected. FIV-infection is routinely diagnosed by the detection of antibodies, usually by ELISA's or by immunofluorescence assay. Up to 15% of FIV-infected sick cats are antibody-negative. Therefore the frequency and importance of FIV-infection in sick cats may be underestimated.

The overall FIV-infection rate in different countries varies greatly. Countries with a low rate of around 1–3% are Switzerland, Germany and the Netherlands; in many other countries the frequency is higher; the maximum rate being in Japan (over 25%). FIV-infection affects mostly male cats with access to outdoors. The average age of FIV-infected sick cats in the so-called AIDS-phase of the disease was determined to be around 5 years.

The major clinical symptoms found in FIV-infected cats include fever, malaise, weight loss, chronic infections of the oral cavity and the upper respiratory tract, conjunctivitis and bacterial infections of the skin and ears. However, as similar symptoms are also observed in animals with other viral infections, FIV-infection can not reliably be diagnosed by the clinical symptoms alone. At present, there is no etiologic therapy available. Therapy consists of symptomatic treatment of secondary infections.

Experimental infection with FIV of specific pathogen free (SPF) cats leads to neutropenia, intermittent fever and lymphadenopathy for a period of several weeks starting 3 to 5 weeks p.i. After this initial phase cats usually recover and remain free of clinical symptoms for months and even years. The AIDS-phase of the disease can only rarely be observed under SPF-conditions although after prolonged infection the immune system of the cats is severely impaired: In a recent study it was shown that after longterm experimental FIV-infection (>26 months) SPF cats exhibited a depressed humoral immune response to a T-dependent, synthetic polypeptide. The cats also exhibited a decrease in the number and percentage of CD4+ lym-

phocytes and a corresponding depression of the CD4+:CD8+ ratio. In contrast, cats with short-term FIV-infection (5–6 months) developed antibody responses to the T-dependent immunogen that did not differ significantly from those of matched control cats. Therefore, the observation that immunosuppressed cats infected with FIV do not show clinical symptoms may be explained by the lack of secondary infectious agents in SPF facilities.

FIV infection has attracted interest as a model for human AIDS. The reasons for this are that FIV is a lentivirus related to HIV, that the natural course of disease is similar to that of human AIDS, that cats are readily available and that the infection can easily be transmitted under experimental conditions.

There is no evidence of FIV being a threat to public health. This was concluded from studies showing that FIV does not replicate in lymphocyte cultures from other species; and that antibodies to FIV could not be detected in veterinarians and animal care personnel with prolonged exposure to sick cats.

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SEARCHING LITERATURE FOR VETERINARY PATHOLOGY. I. HOW TO TACKLE THE AVALANCHE OF PERIODICAL LITERATURE IN THE FIELD OF PATHOLOGY

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An overview is given of journals publishing articles of interest for the (veterinary) pathologist. The sheer number of these journals and the articles they contain forces to apply modern methods of information retrieval. Next to online retrieval, subscribing to SDI-services or using Current Contents (in paper or electronic form), the consultation of databases on CD-ROM and sebsequent downloading of search results offer elegant solutions to conquer seemingly unsurmountable mountains of paper.

In order to reach optimal solutions for local needs a close cooperation between the library and the clinics, laboratories and research departments is essential. A new type of intermediary is evolving: a member of an endusers group being a specialist in a certain branch of (veterinary) medicine and familiar with literature search. The required qualities of this «intermediary/documentalist» are:

– Knowledge of the discipline of the regarding user group.
– Knowledge of the specific needs of the different members of the user group in connection with their diagnostic tasks and their research projects.
– Knowledge of the contents and the structure of databases.
– Knowledge of conversion programmes.

This new type of intermediary can fill the gap between the general information resource manager/librarian and the enduser oriented database searcher.

The impression is that the export of specialized and well selected files into the working and teaching area of academic departments can improve the understanding of bibliographic characteristics of staff-members and students and can promote the rational use of the library resources. As an example an elaboration will be given of a veterinary pathology file, installed in the dissection room of the Department of Veterinary Pathology of Utrecht, as an aid in Diagnostic Pathology (part II, next presentation).

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ACUTE SEROUS PANCREATITIS («EDEMATOUS PANCREATITIS») IN SLAUGHTERED PIGS

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An acute serous pancreatitis has been described in slaughtered pigs since 1977 (8). In this report the histologic and ultrastructural pancreatic changes are characterized and the main pathogenetic factor is demonstrated.

Material and methods

A post mortem examination of 21,719 pigs (average body wt. 164.4 Kg) was carried out in the same abattoir. Before slaughtering the pigs were fasted (only water was available) up to 40 hours (15,678) and up to 60 hours (5,775). 226 pigs were slaughtered after protracted fasting (± 70 hours). Organs fixed in Carson's formalin were: pancreas with edema (82), control pancreas (12), liver and gastric lymphnodes. Paraffin sections were stained with HE, Masson trichrome, and PAS. Ultrathin sections of pancreas fixed in glutaraldeyde were stained with uranyl acetate and lead citrate and examined with a Zeiss EM 109.

Results

Diffuse subcapsular and interlobular edema (fig. 1) was found in the pancreas of pigs from different stock-farms. The incidence but not the severity of the lesions varied greatly in relation to the fasting-period before slaughter (Table 1): very low (0.06%) in 15,678 pigs fasted up to 40 hours, increased (2.3%) in 5,775 pigs fasted up to 60 hours. The incidence reached 36.8% in 266 pigs slaughtered after protracted fasting (± 70 hours). No gross lesions were seen in other organs.

Histologic pancreatic changes consisted in an acute interstitial serous inflammation. The interlobular stroma was distended by an acidophilic and weakly PAS positive exudate with scattered mononuclear cells and neutrophils (fig. 2). Multifocal alteration of single or grouped acinar cells was constantly noted. Affected cells were filled with zymogen granules and contained a single large vacuole, or occasionally, multiple small vacoules. Many acinar vacoulated cells bordered the interlobular edematous interstitium. The large cell vacuole appeared empty or included an irregular corpuscle and few