tory distress after exertion. Auscultation of the respiratory organs revealed no evidence of rales. The pulse was slightly accelerated, and a prominent jugular pulse was observed. On Jan. 13, 1950, a blood sample was drawn and sent to the clinical laboratory for a differential white blood cell count. Results of the count were within normal limits, indicating that a bacterial infection was not involved.

The next four days showed a progressive increase in the edema of the underline with increasing dyspnea. The patient died on Jan. 18, 1950.

The cadaver was removed to the post mortem laboratory where a necropsy examination was performed. A generalized subcutaneous edema was found. The thoracic cavity was completely filled with serous fluid. Ascites was also demonstrated by the presence of a great amount of this fluid in the peritoneal cavity. Extreme diffuse hepatic cirrhosis was observed and widespread hemorrhages were seen on the thoracic and abdominal viscera. Passive congestion was also quite pronounced on the abdominal organs. The right atrium and right ventricle of the heart had undergone extreme hypertrophy. Examination of the right atrioventricular valves revealed that complete closure was impossible because of the distortion caused by the hypertrophied condition of the atrium and ventricle. Passive congestion of the brain was also noted. Cultures taken from the lungs, spleen, liver and heart were negative for bacterial growth.

In order to satisfactorily and logically explain these lesions and the ante-mortem symptoms observed it seems necessary to start with a valvular insufficiency. With the tricuspid valves not closing properely, there resulted an accumulation of blood in the right atrium. This resulted in dilatation and subsequent hypertrophy of the right side of the heart which only helped to exaggerate the valvular insufficiency. With each systole of the right ventricle, some blood was forced in a reverse direction back into the vena cava emptying into the right atrium and caused the juglar pulse so noticeable on ante-mortem examination. This was also the cause of the passive congestion of the brain.

This back pressure in the posterior vena cava caused a passive congestion of the liver. With the engorgement and stagnation of the circulation in this organ, death of the hepatic cells resulted with subsequent proliferation of the connective tissue stroma causing the extreme cirrhotic condition we observed. This cirrhosis of the liver retarded portal circulation and resulted in the passive congestion observed in the abdominal viscera and the hydroperitoneum. Since the hepatic epithelium was displaced by connective tissue and function of the liver was impaired, less albumin was produced for the blood plasma. This decrease in serum albumin lowered the colloidal osmotic pressure of the blood which further aggravated the developing ascites, hydrothorax and anasarca.

The extreme hydrothorax condition mechanically impeded respiration and caused the dyspnea observed in the living animal. In view of the absence of bacterial infection or any other evident etiological factor, and the history stating that two other younger animals in this same herd had shown similar symptoms, it seems logical to conclude that this is a congenital cardiac condition. As growth of the animal progressed the demand on the heart increased to a point which it could no longer meet because of its valvular insufficiency. In order to compensate for this demand, hypertrophy of the heart musculature occurred, only to aggravate the insufficiency and to precipitate the above mentioned pathological changes.

R. M. Hacecky, '50

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4 **Suppurative Nephritis in a Hereford Cow.** A registered 6-year-old Hereford cow entered Stange Memorial Clinic on Dec. 30, 1949 with a history of having been ill since Nov. 21. A tentative diagnosis of traumatic gastritis had been made in the field immediately after her illness was noted. Treatment given at this

time by the attending veterinarian was 1 million O.U. of penicillin. The same dosage of penicillin was repeated on Nov. 22. The cow appeared much improved and her temperature returned to normal.

On Dec. 23, the cow was examined again and the following symptoms noted: discharge from the vulva, temperature of 100°F., poor general condition and little desire to eat. The owner stated that she had not been bred and was having irregular heat periods. Treatment given was 1 million O.U. of penicillin and diethylstilbestrol (dosage not stated). The same treatment was repeated on Dec. 24 and Dec. 26. The cow failed to show any improvement.

Treatment was initiated immediately when the cow entered the clinic. On Dec. 31 she was restrained and a blood sample taken. Hemoglobin content was found to be 46 percent of normal or 5.53 Gm. The erythrocyte count was 6,050,000; the leucocyte count was 6,080 which was broken down as follows: juvenile or younger neutrophiles 800, segmented or older neutrophiles 2,800, monocytes 100 and lymphocoytes 2,500. On Jan. 3 the patient was given 24 Gm. of equal parts powdered gentian, ginger and nux vomica in capsule form per os, and 30 cc. of 1 percent cobalt chloride solution in a capsule per os. On Jan. 4 expiratory dyspnea was noted. Treatment given was the same as for the previous day.

Fig. 2. Phonepritis.

On Jan. 5 it was found that the animal had died during the previous night. Autopsy revealed the following lesions: left kidney greatly enlarged, both kidneys contained numerous large abscesses filled with thin purulo-hemorrhagic exudt, uremia as evidenced by ulcerations in the gastrointestinal tract, fatty degeneration of the liver, diffuse ecchymotic hemorrhages of the liver, subepicardial and subendocardial hemorrhages and marked interstitial emphysema of the lungs.

A diagnosis of suppurative nephritis was made on the basis of these post mortem findings. Bacteriological smears taken from the abscesses of the kidney revealed *Corynebacterium pyogenes.*

The symptoms of suppurative nephritis are similar to chronic vaginitis and metritis, and traumatic gastritis. An examination of the urinary system is important in making a differential diagnosis in these conditions.

Verle Foote, '51

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5 **A Necropsy Report.** On Jan. 31, 1950, a dog was presented for necropsy. The cadaver was that of a borwn Doberman Pinscher bitch, 15 months old, and weighted about 50 lbs. According to the history, the owner noticed the bitch in heat and because he had no place to keep her confined during this period, he desired to have her spayed. Although the clinician informed him of the risk incurred when a bitch