

RUPTURE OF THE STOMACH.¹

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HAVING read an interesting communication in the *American Veterinary Review* for April, by Dr Johnson, on rupture of the stomach in the horse, and after an experience of ten cases in a period of three years' practice, I determined that the next opportunity I had I would make a more careful examination with a view of finding a solution to the question, viz.: Why is it that the rupture in the external coat is of greater extent than in the internal or mucous coat?

On the morning of April 23 I was called to see a mare suffering from what the owner supposed to be colic. When I arrived at the farm, only two miles distant, I found the animal lying rather quietly. On getting a history of the case, I found that she had been at her usual labour the day previous, but on returning to the stable that evening she refused food, and manifested a slight degree of pain. She was allowed to stand for a short time before anything was done, thinking perhaps she might get over her trouble.

Later the animal received the usual "farmers" dose, soda bicarb. $\bar{3}$ i to iii water q. s. This not seeming to give the desired effect, about two hours later the animal was given a second dose, using the owner's expression, composed of three tablespoonfuls of saleratus and a teacup of vinegar. Immediately after receiving this dose the animal manifested symptoms of great pain. The owner then gave 1 oz. of tn. opii which seemed to quiet her. She was left for the night. In the morning they found her rolling and acting very uneasy, also perspiring slightly; it was at that moment that my services were sought. Upon examination found the pulse 75 and irregular, temperature 103° F., respiration 40. Gave opium and cannabis indica, also hot injection, which resulted in a free evacuation. Left three doses of opium and colocynth, to be given between then and noon, with an occasional enema.

On returning, found that she had lain quiet since my first visit, bowels had moved and micturition occurred. Pulse 80, very feeble, temperature 104° F., respiration 60. We got the animal upon her feet, which occasioned very distressing symptoms; there was a discharge from both nostrils (a white froth), under lip pendulous, mouth partially open, tongue protruding. Frequently the animal would cramp the head against her breast, and would back around in her box. These symptoms pointed to rupture of the stomach, of which I apprised the owner. Left full doses of morphia, to be given hypodermically every hour, and would return in the evening. At that time I found every symptom of rupture augmented, so positive that I wanted to destroy the animal as an act of mercy.

The owner would not listen to this, as she was the family pet. She died during the night; I made a *post-mortem* in the morning, and found the stomach ruptured, in the manner that Dr Johnson speaks of, viz.: That the peritoneal coat was torn about $1\frac{1}{2}$ inches at each end of the orifice more than that of the mucous coat, and that the torn edges through the centre of the aperture were more

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gangrenous than at the ends, while the torn edges of the inner coat presented the same appearance all through. The lesions were situated about one-third the distance from the greater curvature and at right angles with the posterior gastric arteries through the villous portion. This animal had shown indications of pain four days prior to her last sickness, and I have no doubt but that the indurated condition through the centre of the external aperture was due to pre-existing rupture. I can only account for the pre-existing rupture of the external coat, and that it is of greater extent than that of the mucous coat in this way: that the external coat is a serous membrane and practically inelastic, while that of the internal is a mucous membrane and quite elastic, and would, therefore, allow of greater dilation. On examining the stomach of the horse we find this: that the mucous coat is soft and velvety, and lies in small folds or rugæ. When we consider the structure, and the distension that this organ can assume, one can easily understand how an inordinate distension could rupture the external coat, before allowing any such lesion to occur in the internal coat. When the stomach is fully dilated normally, the mucous membrane is not strained in the least, chymification is uninterrupted; whereas, on the other hand, were the mucous membrane so dilated as to be tense, it would interfere with the secretion of the glands, and chymification would cease. Nature has been most beneficent in this, in allowing the animal to eat to the greatest desire, without materially interfering with digestion.

Abstracts and Reports.

TUBERCULOSIS IN SHEEP AND GOATS.

IN the *Zeitschrift für Fleisch- und Milchhygiene* (October 1891) the editor quotes a case of tuberculosis in a goat recorded by Sluys and Korevaar. This animal, which had been born of a healthy mother, but fed with cow's milk, began to cough, gradually lost flesh, and was killed at the age of 15 months. It was then found to be the subject of general tuberculosis, which had had its starting point in connection with the alimentary tract, the lesions in the intestine, mesenteric glands, and liver being apparently of older date than those in the other organs. Infection was ascribed to the cow's milk upon which the goat had been reared.

In citing this case, Dr Ostertag takes the opportunity to combat the widespread but erroneous idea that goats never suffer from tuberculosis. Reports from various slaughter-houses prove that when goats are housed they readily contract this disease from tuberculous cattle or human beings. During his official connection with the Berlin Abattoir Dr Ostertag discovered various cases of tuberculosis in sheep, the diagnosis being verified by recognising the bacilli, and in all these cases the lesions showed that the alimentary canal had been primarily affected.