THE EXPERIMENTAL PRODUCTION OF PEPTIC HÆMORRHAGIC ŒSOPHAGITIS*

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NUMEROUS pathologists have described the occurrence of erosions covered with chocolate-coloured fluid in the lower third of the œsophagus. The condition which has recently been reviewed by Hamperl[1] and Neubürger[2] has been termed "peptic hæmorrhagic œsophagitis". Occasionally these lesions perforate into the pleural cavities,[3] and, although these perforations cause obvious clinical symptoms of dyspnœa and cyanosis, some investigators considered them to be a post-mortem occurrence.[4, 5, 6]

While most investigators[7, 8] who have studied this condition agree that intravital digestion of the epithelium by stomach juice plays an important part in the pathogenesis of the condition, it has been claimed by many workers that this in itself cannot explain it, since the normal œsophagus is resistant to peptic enzymes. Thus Brosch[9] claimed that he was unable to produce softening of the œsophagus post mortem by treatment with stomach juice except in cases in which the patient had suffered from cerebral lesions. Neubürger[2] and Bartels,[8] who observed 82 cases, also emphasized the frequent combination of cerebral diseases with peptic œsophagitis. They claimed that the normal œsophagus is protected against the digestive effect of stomach juice, and assumed that some other stimulus must first weaken the wall of the œsophagus before it will yield to the peptic enzymes. Hamperl[1] also spoke of a decrease in the resistance of the œsophageal wall which plays an important rôle in the production of peptic œsophagitis.

In order to determine whether regurgitation of stomach juice could produce this condition in the normal œsophagus we ligated the pylorus in 6 female rats, 4 months old. Twelve hours after this operation one of the animals died, and autopsy revealed that the stomach was distended with fluid, part of which had regurgitated into the œsophagus. The œsophagus itself showed large brown patches just above the diaphragm, and in the middle of this area perforation into the pleura occurred. Both pleural cavities were filled with stomach juice. The other five animals of this series were killed 17 hours after the operation, at which time all of them showed marked hæmorrhagic erosions in the lower thoracic part of the œsophagus. Histological examination of this area showed it to be devoid of epithelium and infiltrated by leucocytes and lymphocytes. It also contained numerous small hæmorrhages.

It seems of particular interest that in none of these animals were there any erosions in that part of the œsophagus which lies below the diaphragm or in the portion situated in the upper thoracic or cervical region. The proventriculus of the rat, which is lined by a squamous epithelium similar to that of the œsophagus, was likewise free of peptic lesions. While it is quite possible that the stomach juice never ascended high enough to reach the upper portions of the œsophagus, it obviously had to get in touch with the proventriculus and the lowest part of the œsophagus. It is difficult, therefore, to explain why these regions remained free of any peptic lesions while the supradiaphragmatic portion was so readily attacked by the gastric juice. It is evident from our experiments that mere regurgitation of gastric juice suffices to cause peptic hæmorrhagic lesions in the œsophagus of an otherwise healthy organism, but it is also clear that, at least in the rat, the lowest portions of the œsophagus and the proventriculus are much more resistant against this peptic action than the more cranially situated parts (see Figs. 1 and 2).

In order to ascertain that the lesion was actually due to the local action of the stomach juice and not simply to the operative injury as such we performed a second experiment in which one ligature was placed on the pylorus, just as in the previous series, and another was tied around the cardia. In six rats, 4 months old females, operated on in this manner, no œsophageal lesion was observed 17 hours following the operation when autopsy was performed.

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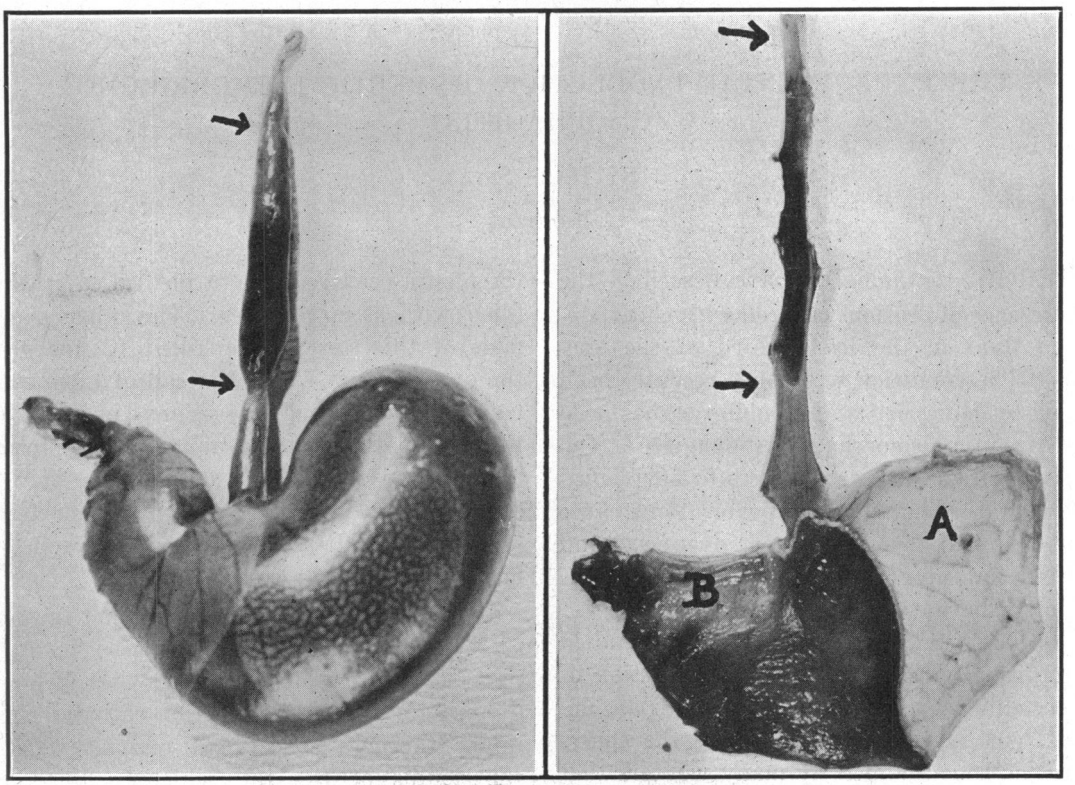

Fig. 1.—Stomach and œsophagus of a rat 24 hours following ligature of the pylorus. The area between the arrows shows the marked development of peptic hæmorrhagic œsophagitis.

Fig. 2.—The same preparation as that shown in Fig. 1 after removing the anterior wall of the stomach and slitting the œsophagus open, in order to expose the inner surface of these organs. A—Proventriculus lined by squamous epithelium similar to that of the œsophagus. B—Ventriculus lined by typical gastric mucosa.

SUMMARY

A condition resembling peptic hæmorrhagic œsophagitis has been produced in the rat by placing a ligature around the pyloric end of the stomach and thus causing a marked accumulation of gastric juice with regurgitation into the œsophagus. It seems obvious, therefore, that the prolonged contact of gastric contents with the wall of the normal œsophagus may lead to digestion of this organ, especially in its thoracic portion which for some reason seems to be less resistant. The fact that in human pathological states the condition is usually encountered in debilitating and terminal states does not necessarily mean, as previous authors believed, that weakening of the resistance of the œsophagus is a necessary prerequisite for the formation of peptic lesions. It seems more likely that such conditions favour the development of these lesions because they facilitate the regurgitation of peptic juice and its stagnation in the œsophagus.

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