

geal sphincter and gastric acid secretion of young normal people and of patients with severe gastro-oesophageal reflux. The shape of the dose response curve and the percentage increase of tone of the incompetent sphincter were the same as for the normal sphincter but twice as much pentagastrin was required to reach the peak response. Similarly, the gastric acid response curve for those with an incompetent sphincter was the same as for the normal people except that for any given dose of pentagastrin only half the normal amount of acid was secreted, and they all had a reduced secretory capacity. The best explanation the authors can offer for both the response of the lower oesophageal sphincter and the gastric acid response is that there is a decreased stimulus by endogenous gastrin.

Cohen and Harris challenge the rationale of surgical repair of hiatus hernia as a means of stopping reflux, and they cast doubt on the usual explanations given for the success of a repair—namely, the production and maintenance of an acute angle of entry of the oesophagus into the stomach and the provision of an intra-abdominal segment of oesophagus, because they find that lower oesophageal sphincter strength is not affected by surrounding pressure. Nevertheless they do not deny that some patients are improved by surgical repair of their hiatus hernias, and it seems reasonable to continue to attempt operations which appear to achieve some success until we have something better to replace them.

They believe that the goal of therapy for reflux should be to increase the strength of the lower oesophageal sphincter, and their findings may well be relevant to some aspects of medical treatment. For example, they and others^{2,3} have evidence that atropine and similar anticholinergic drugs decrease the intrinsic tone of the sphincter and reduce its capacity to contract in response to a stimulus. It would be sensible therefore to eschew all such preparations in the treatment of reflux symptoms. The presence of acid in the stomach inhibits the production of endogenous gastrin, and evidence is accumulating that ingesting antacid may increase gastrin production and so tighten up the sphincter, reducing the amount of reflux.⁴⁻⁶ Because reflux is a constant hazard, the boosting of gastrin production would need to be maintained by frequent small doses of alkali or food. Intravenous metoclopramide appears to double the basal tone of the sphincter⁷ and may prove to be useful when given by mouth if it does not also increase intragastric pressure.

There are many attractive features in Cohen and Harris's hypotheses. But it may still be wise to separate the pharmacological response of the sphincter to gastrin, and its perhaps reflex mechanical response to intragastric pressure, from the idea that the sphincter is the major if not the only component of the antireflux mechanism, until we know more about the true incidence of reflux in patients without a hiatus hernia and the true incidence of hiatus hernia without reflux.

¹ Cohen, S., and Harris, L. D., *New England Journal of Medicine*, 1971, **284**, 1053.

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³ Lind, J. F., Crispin, J. S., and McIver, D. K., *Canadian Journal of Physiology and Pharmacology*, 1958, **46**, 233.

⁴ Castell, D. O., and Harris, L. D., *New England Journal of Medicine*, 1970, **282**, 886.

⁵ Giles, G. R., Mason, M. C., Humphries, C., and Clark, C. G., *Gut*, 1969, **10**, 730.

⁶ Castell, D. O., and Levine, S. M., *Annals of Internal Medicine*, 1971, **74**, 223.

⁷ Heitman, P., and Moller, N., *Scandinavian Journal of Gastroenterology*, 1970, **5**, 621.

Masking of Syphilis

Treponema pallidum is very sensitive to penicillin, and when this antibiotic was first used for the routine treatment of gonorrhoea there was considerable anxiety lest it should mask the development of an incubating syphilitic infection. The disastrous effects of under-treatment of early syphilis with the arsenicals, with subsequent clinical relapse, were only too well known, and doubts about the effect of small doses of penicillin on the behaviour of syphilis were justified.

H. Eagle and his colleagues^{1,2} investigated the effect of penicillin on experimental syphilis in the rabbit when given during the incubation period of the disease. They found that the abortive dose, as shown by the failure to develop lesions and negative lymph node transfer experiments, varied with the infecting dose and the time after infection at which penicillin was given. It remained constant for the first four days but had risen seven-fold by the end of the second week. Asymptomatic infections were rare when inadequate amounts of penicillin were given during the incubation period but became common when treatment was delayed until the primary lesions had appeared. They concluded that a single injection of 15,000 to 50,000 units of penicillin in peanut oil and beeswax might suffice to abort some early infections in man if given within four days of exposure. If the infection was not aborted, the incubation period might be prolonged and the resulting lesions insignificant in nature. Clinical and serological observation for at least four months after treatment of gonorrhoea was recommended and became the accepted practice in clinics.

The dosage of penicillin for gonorrhoea has been steadily increased owing to the emergence of strains of gonococci which are relatively insensitive to the antibiotic. Doses of 1.2 to 2.4 megaunits are now commonly given. These might well have an effect on incubating early syphilis, and K. R. Woodcock³ has analysed the material from a large clinic with this possibility in view. Of 2,992 males with gonorrhoea seen in 1968 and mainly treated with 1.2 megaunits of procaine penicillin 38 were found to have early or early latent syphilis. Thirty were diagnosed when they first came under observation, two developed early syphilis within three months and six within three to six months after treatment of gonorrhoea. Two hundred and eighty one patients treated for early syphilis over an 18-month period were reviewed, and 54 were found to have a previous history of treatment for gonorrhoea. In 41 cases the two infections were concurrent, but the numbers of cases of early syphilis diagnosed in successive three-month periods after previous treatment for gonorrhoea were 4, 10, 19, and 21. Woodcock is rightly hesitant to draw firm conclusions from the small numbers making up this uneven distribution. He inclines to the interpretation that early syphilis may be "accidentally" cured by the treatment for gonorrhoea rather than that it is being masked by prolongation of the incubation period.

The value of routinely performed serological tests for syphilis as part of the initial investigation of a patient with gonorrhoea is emphasized by the numbers of early latent cases, potentially still infectious, thus brought to treatment, in addition to those with clinically evident disease.

¹ Eagle, H., Magnuson, H. J., and Fleischman, R., *Journal of Experimental Medicine*, 1947, **85**, 423.

² Magnuson, H. J., and Eagle, H., *American Journal of Syphilis, Gonorrhoea and Venereal Disease*, 1945, **29**, 587.

³ Woodcock, K. R., *British Journal of Venereal Diseases*, 1971, **47**, 95.