and from 74 to 80% of the potassium iodide placed in the jejunum was absorbed, while no more than 31% of the iodide placed in the stomach was absorbed. The difficulty in recovering the contents of the stomach, this author suggested, might have caused a loss which would place the percent absorbed higher than the actual amount absorbed. In support of the absorptive role of the stomach are the findings of Rankin(4), Henning(5) and Bertrand(6). Rankin(4) instilled potassium iodide into the rumen of sheep and detected iodine in the saliva in 4 minutes, concluding that the stomach was a principal site of iodine absorption. Henning(5) and Bertrand(6) found that patients with chronic gastritis and gastric ulcer excreted salivary iodine more rapidly than normal subjects, assuming that potassium iodide was absorbed in the stomach.

The difficulty in determining the exact time of pyloric passage may account for the discrepancy between our results and those of Henning(5) and Bertrand(6). Henning assumed that by placing the subject in the left lateral decubitus position, negligible amounts of gastric contents would enter the duodenum, while Bertrand determined time of passage using an air-fluid contrast technic under fluoroscopic control. In the present study, the use of radio-opaque media permitted more accurate determination of the passage of small quantities of potassium iodide into the small intestine. Also, the time elapsed between passage into the duodenum and salivary appearance of iodide agreed closely with the time required for detection of salivary iodide when potassium iodide was instilled directly into the duodenum.

Summary. Potassium iodide mixed with radio-opaque media was administered orally to human subjects studied under fluoroscopic control. Iodide appeared in saliva of these subjects 2-4 minutes after mixture passed into the duodenum. Iodide instilled directly into the duodenum of 2 subjects was detected in their saliva 3-4 minutes later. 14.9% of the iodide placed in the stomach and 48.9% of the iodide placed in the duodenum of rats was absorbed within one hour. It appears that the small intestine is the principal site of absorption in humans and in rats.

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The Local Tissue Reaction in Rabbits to Gelfoam Implants Containing Desmosterol or Cholesterol.*† (26367)

DAVID M. SPAIN

Department of Pathology, Beth-El Hospital, Brooklyn, N. Y.

A recently developed compound, Triparanol (MER-29), has been shown to inhibit biosynthesis of cholesterol(1). It has been further demonstrated that the biosynthesis of cholesterol is probably inhibited at the step which requires reduction of the side chain double bond of 24 dihydrocholesterol, with a resultant increase in serum levels of desmosterol (precursor to cholesterol)(2). MER-29 is at present being studied in hu-

* The desmosterol used in this study was supplied by Wm. S. Merrell Co., Cincinnati, Ohio.

† MER-29 is trade name of compound produced by Wm. S. Merrell Co.

mans as to its value as a serum cholesterol lowering agent. Since the serum level of desmosterol appears to be increased under these circumstances, it is a matter of importance to know the effects of desmosterol on mammalian tissues. It is known that extracellular deposits of cholesterol or the esters of cholesterol excite an inflammatory reaction with fibrosis and foreign body granuloma in the body tissues. The basic ingredients of this reaction are essentially similar, whether the cholesterol is found in the subintima of blood vessels or elsewhere. The purpose of this paper is to report on the reaction to desmosterol that was introduced into the extracellular tissues experimentally in the rabbit. The technic employed in this study has been described(3).

Materials and methods. Standardized pieces of sterile absorbable gelatin (gelfoam) sponges each weighing approximately 3 mg were placed in 1 cc of ether containing 4.3 mg of desmosterol (the entire amount of desmosterol available for this study). The ether was then allowed to evaporate. The 6 gelfoam sponges were estimated to contain approximately 0.7 mg of desmosterol each. These desmosterol-containing gelatin sponges were implanted in the subcutaneous region through a small surgical incision in the abdominal surface of 6 rabbits. Also implanted simultaneously in each one of the rabbits, at separate sites, were similar size gelfoam sponges saturated with cholesterol and gelfoam sponges that were not impregnated with any material. Each rabbit, therefore, had 3 subcutaneous implants with various gelfoam sponges. The rabbits were males of equivalent weight and age and maintained on stock diets. After 14 days the implants and surrounding tissue were excised from these rabbits, fixed in formalin and sections were prepared that were stained with hematoxylin and eosin. The number of implants was limited because of the small amount of desmosterol available.

Findings. The reaction to the gelfoam-desmosterol implants revealed an intense acute inflammatory response with considerable cellular necrosis. This reaction permeated the entire extent of the sponges of all 6 rabbits. Minimal to moderate granulation tissue surrounded each one of these implants. The histologic reaction to the gelfoam cholesterol implants revealed a similarly intense inflammatory reaction. However, in this situation the macrophages predominated. Granulation tissue in amounts similar to those seen in the gelfoam desmosterol implants was present. The histologic reaction to the control gelfoam sponge implants revealed no inflammatory reaction and only slight fibroblastic proliferation surrounding the sponges.

Summary and conclusions. It would appear from this limited study in rabbits that desmosterol lying free in extra-cellular sites is not innocuous, in that it excites an intense inflammatory reaction in the subcutaneous site of implantation. The fate of desmosterol and its distribution in the body tissues is not yet known. Whether or not desmosterol may appear freely in the extra-cellular tissues of blood vessel walls as is the case with cholesterol is also not known. Because of the fact that desmosterol does incite an intense inflammatory reaction, its fate and distribution in the body should be determined before MER-29 can be considered to be a relatively safe serum cholesterol lowering agent.

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