inexperienced. If, however, I.U. of patients considered cured by transurethral resection should reveal that no satisfactory decompression of their back-pressure kidneys has taken place, then it can be predicted that sooner or later suprapubic or perineal prostatectomy will hold its leading position in the radical surgical treatment of enlarged adenomatous prostate.

Commenting on hemiprostatectomy as a safer method, the same principle has to be applied. Should the hemiprostatectomy decompress one of two damaged kidneys its value will still be in doubt. If it can be proved by I.U. that only the kidney on the side of the adenoma was damaged and that it could be decompressed by hemiprostatectomy, this conservative method will not only be sufficient but will also be preferable to a more radical procedure.

The surgeon who performs a two-stage suprapubic prostatectomy, even if the interval between cystostomy and prostatectomy is only ten days—as in the two cases quoted by Mr. W. Sampson Handley—runs fewer risks than the operator who trusts his satisfactory functional tests of the kidneys, which really give him no indication of a dormant urinary infection and/or a grossly impaired peristalsis of the ureters. These two latter factors, which can be evaluated only by I.U., are the main reason why prostatectomy still seems such a hazardous operation. With the help of I.U., cases with back-pressure kidneys can be diagnosed and excluded from too early prostatectomy.

I.U. has made it possible to demonstrate that adenoma of the prostate is often a growth which follows a focal infection of the prostate or the periprostatic tissue. With the help of this photographic method it was possible to recognize that the enlarged prostate is not only an obstacle to micturition, an obstacle preventing the evacuation of the bladder, but—and what hitherto has only been suspected but could not be proved—an obstacle in the way of the normal peristalsis and tonus of the ureter.

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Medical Memoranda

Enamel Hypoplasia in Idiopathic Epilepsy

Interest in the finding of associated lesions in hereditary and developmental diseases has greatly increased during the last decade. Little attention has, however, been paid in this connexion to dental malformations, though the pathognomonic significance of Hutchinson's teeth and the incidence of dental defects in cretinism is well recognized.

The following report is the result of dental examination of 65 epileptics who were in-patients in the Mental Hospital, Wuhlgarten-Berlin, and who had either a full set of teeth or, at least, several groups of teeth belonging to the same developmental period. So far as could be ascertained by the history and by clinical and serological findings none of these patients was suffering from congenital syphilis.

In 33 cases defects of the enamel in the permanent teeth were found. These were of the same character in every case, differing only in size or distribution, the buccal or labial side being occasionally more affected than the lingual or palatal; sometimes defects were seen round the whole circumference of the tooth. It was especially striking that the changes in the enamel involved teeth belonging to the same embryonic developmental period and that they were identical in locality, form, and intensity in the fellow teeth. On the other hand the teeth of different groups—e.g., incisors and canines or bicuspid—were affected in different parts according to the time of their em-

bryonal development; but in all these cases the lesions were bilateral (see Figs.). The locality, too, differed between the upper and lower jaws, owing to the germinal layer of the teeth in the mandible. The frequent presence of hypoplastic enamel formation in these cases seems most remarkable. In contrast to other conditions—e.g., congenital syphilis, nutritional deficiencies, or disturbances of calcium-phosphorus metabolism—the enamel was the only dental tissue affected and the enamel hypoplasia was not associated with evident lesions in any other part of the body.

I have had no opportunity of examining another series of cases, and these findings are published in order to stimulate further investigation. As to the relation between the enamel hypoplasia and epilepsy, it seems worth mentioning that the enamel organ originates from the same germinal layer—i.e., the

FIGS. 1 and 2.—Showing the teeth of two patients with epilepsy.

ectoderm—as the nervous tissue. It therefore seems possible that some lesion of or damage to the ectodermal layer is responsible for both conditions. The hypothesis that the enamel hypoplasia is conditioned genetically or by some morbid process of the embryo finds its analogy in the alteration of the permanent teeth in congenital syphilis, said to be due to the presence of *Treponema pallidum* in the tooth follicle. Thus the stigmata of a morbid process which affected the individual prenatally remain uninfluenced by the post-natal life.

The fact that a number of these patients had very small teeth (microdontism) (Fig. 1) further supports the theory of a defective embryonic epithelium or hereditary taint preventing the development of the normal type of tooth. It is not likely that the enamel hypoplasia originates from the time of calcification, which takes place from birth up to the fifth year of childhood. Apposition of calcium and phosphorus is quite normal in the unaffected part of the teeth *before* as well as *after* the manifestation of hypoplasia, since the enamel once formed is beyond the reach of dietetic influence. Enamel hypoplasia appears to be the result of a disturbance of the enamel-forming cells—the ameloblasts—which seem to be missing, or their work must have been suddenly interrupted at a certain stage of their differentiation.

Many factors may be involved in the aetiology of the structural changes in the teeth. As 50% of the cases examined showed enamel hypoplasia a fortuitous association between the two conditions seems unlikely; but the question remains whether they are only associated disorders or whether a more intimate interrelation exists in their pathogenesis. Genuine epilepsy, according to our present knowledge, is due to hereditary influences or to noxae toxic to the germ. These causes may lead to changes or lesions of the ectoderm, resulting in pathological enamel.

We know the period of ectodermal differentiation at which the primitive layer of the permanent tooth and its enamel are formed. We may thus be helped in establishing the time of the morbid process responsible for this particular type of epilepsy, if due to dystrophic changes of the ectoderm.

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A. L. Barach, M. Eckman, and N. Molomut (*Amer. J. med. Sci.*, 1941, **202**, 336) conclude that carbon monoxide poisoning from inhaling tobacco smoke may, in certain circumstances, impair resistance to anoxia, especially in pilots flying without oxygen at altitudes of 10,000 to 12,000 feet. In eighteen smokers who inhaled they found the average CO saturation of arterial blood to be 5.7% after smoking twenty cigarettes from 9 a.m. to 4 p.m.