with the condition does not exist. All is chemistry and make-believe. In patients who are referred to me with these symptoms I do not hesitate to diagnose duodenal ulcer, and I advise operative treatment. At the operation I am able to demonstrate the ulcer to an onlooker who before may have been sceptical. That, briefly put, is the difference between Dr. Hutchison and myself. We describe the same group of symptoms; he attributes these symptoms to a "functional" disorder; I have been able to demonstrate in over 230 cases that they are due to a structural lesion in the duodenum. And I know that duodenal ulcer can be diagnosed from the symptoms alone with a margin of error that is less than 5 per cent.

I state the position quite frankly, because it seems to me that it is hardly a matter to be settled by debate. There is need rather for patient and unprejudiced observation. If Dr. Hutchison will submit his cases to the surgeon, he will find a duodenal ulcer to be the cause of the symptoms I have described. If he is loth to do this, I shall be happy to give him the opportunity to see and examine my cases before operation, and at the time of operation I will accept his decision as to the existence and the position of any organic lesion. Dr. Hutchison does not tell us what knowledge he possesses as to the condition of the viscera of the patients in the early "stages" he describes. Before he can predict what is or what is not present he must enlarge his experience of the "pathology of the living." Then, and not till then, is he qualified to pose as critic; not until then can he speak with full authority. I shall consider it a privilege to equip him with this necessary experience, and to furnish him with the material upon which alone a sound opinion can be based.—I am, etc.,

Leeds, March 22nd. B. G. A. MOYNIHAN.

SIR,—The diagnostic significance of hunger pain in chronic ulceration of the duodenum is of sufficient importance to render its probable cause worthy of some possible explanation.

As it is practically certain that the pain which is felt in chronic ulcer either of the stomach or the duodenum is associated either with the contact of food with the eroded surface or with the induced muscular contraction, there are a few well-defined physiological reasons why the differentiation of the two seats of disease should be easily made.

In ulcer of the duodenum pain is usually felt from two to four hours after food—that is to say, at that period when the pyloric aperture relaxes. The acid contents of the stomach escape into the duodenum, and the latter undergoes contraction. And here it may be stated that the more indigestible the meal, or the more solid the food taken, the longer are the contents of the stomach retained in that viscus, and, consequently, the greater the interval between ingestion and the appearance of the pain. Patients find that when the pain occurs it is relieved by taking food, hence it has come to be termed "hunger pain." The relief afforded is based on the simple physiological fact that when food is taken into the stomach the pyloric aperture is closed, and for the time being there is a temporary cessation of the passage of the food from the stomach and over the surface of the ulcer, and of any duodenal peristalsis.

Another peculiar and significant feature about pain associated with ulcer in the duodenum is the frequency with which the seizure takes place during the night. Here, again, the probable explanation lies in the physiological fact that when a meal is taken in the evening gastric digestion is delayed during sleep, and it is not for some hours that the stomach ejects its contents through the relaxed pylorus, and the patient is awakened by pain.—I am, etc.,

Glasgow, March 19th. A. ERNEST MAYLARD.

SIR,—Whilst I have read with great interest Dr. Hutchison's letter, I do not think that he makes out his case. The theory which he has adopted of a regular sequence commencing with hyperchlorhydria, followed by continuous hypersecretion with ulcer as the terminal stage, was first enunciated by Robin in his classical treatise upon diseases of the stomach published in 1901. He also coined the term "hypersthenic dyspepsia" to designate this sequence. This hypothesis, never frankly accepted by the majority of those working at diseases of the stomach, has recently been further discredited, as later work has shown:

1. It is quite an open question to what extent hyperchlorhydria really exists as such. It is obvious that the percentage of hydrochloric acid found to be present in the stomach contents after a test meal must be the resultant of three factors—the total bulk of gastric juice secreted, the amount of chyme which has passed out of the stomach, and the acid content of the pure gastric juice. It may thus be quite possible that from some abnormality in the pyloric reflex we may have an absolutely normal gastric juice, although the examination of the test meal will show hypochlorhydria. The converse is, of course, true. With modern methods of estimating the amount of gastric juice secreted, the whole question of the acidity of the gastric juice will need revision.

2. It is practically certain that in the cases which present what we know as symptoms of hyperchlorhydria, there must be some factor in addition to the excess of acid to account for the pain. The theory that the pain is directly due to the excess of acid is absolutely contradicted by known facts. In the first place, we often meet with cases which are absolutely without pain, although the hydrochloric acid content of the stomach contents is very high. Dutton Steele found acidity of over 70 in 3 cases without symptoms; Stockton has repeatedly found acidity of 100 in similar cases; Kauffman in 19 cases free from gastric symptoms found an acidity of over 70 in 10 and of over 100 in 2. Similar observations have been made by Gintls, Schule, Meyer, Brandeburg, and Illoway. In the second place, Soupault, Verhaegen, and Luigi Sansoni have recorded numbers of cases in which the hyperchlorhydria symptoms having been cured by appropriate measures, the hydrochloric acidity of the stomach was found to be just as high as ever. Thirdly, in many cases presenting the hyperchlorhydria syndrome, the hydrochloric acid content of the gastric juice was found to be subnormal.

3. If there is one thing more certain than another it is that when you find more than 50 or 60 c.cm. of hydrochloric acid-containing fluid in the stomach before breakfast, together with food residues, there is practically always an ulcer close to the pylorus or some contraction of the pylorus from adhesions due to old ulceration. If this is associated with rigidity of the rectus muscle, with or without the characteristic tender spot and hyperaesthetic skin area, our confidence in the diagnosis will be correspondingly increased.

I think that, taking all the facts which I have mentioned into consideration, we are justified in concluding that the pain of hyperchlorhydria is not due to the amount of hydrochloric acid in the stomach, but to the presence of some other factor, which may be an ulcer or a hyperaesthetic condition of the stomach or even of the solar plexus. As regards the hunger pain, which is so characteristic of ulcer of the duodenum, Dr. Hutchison evidently confounds it with the hyperchlorhydria pain which I have been discussing. The hunger pain is *sui generis*, and comes on when the stomach is empty, and is probably of purely mechanical origin, due to the dragging of the retracted empty stomach upon the adhesions by which the ulcer is attached to surrounding parts. On introducing food into the stomach the tension is relaxed, and the pain ceases.

In conclusion, whilst cordially agreeing with Dr. Hutchison that the great majority of cases of dyspepsia met with in practice are due to functional disorder and not to organic disease, I think that there is much more chance of hyperchlorhydria being considered functional when the real cause is irritation from a gall stone or a duodenal ulcer than the reverse. It is so fatally easy to be misled by the latent periods which appear characteristic of these affections into deluding oneself that you have cured a functional disorder.—I am, etc.,

London, W., March 21st. GEORGE HERSCHELL.

DIAGNOSIS OF DUODENAL ULCER.

SIR,—Mr. Alexis Thomson, in his paper on the diagnosis of chronic duodenal ulcer, lays stress on the cause of pain as being due to peristalsis and not to the contact of the hyperacid residue of digestion with the ulcers.

It is difficult to reconcile the explanations he puts forward with personal experience.