the medical student, and Professor Starling had said that from the very beginning of his medical curriculum every scientific subject which a student studied should be considered only in its bearings on his future work as a medical man. Dr. Barron considered that the systematic lecture held too high a place in the curriculum; that while it had its uses in the way of providing a summary of work done or in shaping a course to be followed, in these days of excellent books a systematic course of 100 to 150 lectures on any subject seemed hardly necessary. Above everything, clinical work in the ward and in the hospital laboratories was of paramount importance. Professor Wilson, F.R.S., Professor of Anatomy in the University of Sydney, in moving a vote of thanks for the address, entirely disagreed with the view that from the earliest days students should be constantly led to approach every subject from the point of view of its future utility in practice. The object of the earlier training was to put students in the position of possible clinical observers. The material on which they would feed during the earlier years would have to be used again, but they should observe towards it a disinterested attitude. Knowledge of function should be acquired without any ulterior object, even that for which they were no doubt acquiring the knowledge. It had been said that the kind of young man commercial men wanted was not the one who had studied banking and so on, but the one who had a good sound intelligence. Preliminary training should aim at producing men who would be in a position to turn a sound well-trained mind and judgement to the subjects presented. That did not imply that there should be no science in the schools. It was a paltry school training which neglected the training of the observation, and it was not the acquiring of the facts of chemistry and so on which was important. If further room had to be made for clinical study it must be at the far end of the course. With thirty-three residents' posts waiting for their graduates to give them opportunity for clinical study there was not much to complain about.

COMPULSORY NOTIFICATION OF PULMONARY CONSUMPTION.

Compulsory notification of pulmonary consumption in the City of Sydney was introduced under by-laws of the City Corporation Act at the end of 1904, and in 1905 there were 196 notifications, in 1906 168, in 1907 154, and in 1908 187. When a case is reported in the city, the house is visited and the conditions under which the consumptive patient is living are noted. He is given advice as to what to do—how to treat his sputum, and generally how to avoid the transference of infection to other persons. If the house is dirty, it is cleansed and disinfected. Subsequent visits are paid as often as is considered desirable. When a patient dies or leaves a house, the premises are immediately disinfected. Dr. W. G. Armstrong, the city health officer, reports that the compulsory notification has worked well throughout all the period it has been in force, and that no friction has arisen between medical men and their patients and the department. The Government is now considering the question of making consumption a notifiable disease throughout the State. It is not yet decided whether the powers of local governing bodies should be extended to enable them to deal with consumptives or whether direct legislation dealing with the State as a whole should be passed. Without fresh legislation it is not thought practicable to extend to the whole of the State the law which has hitherto worked so beneficially in the City of Sydney.

OUTBREAK OF SMALL-POX.

On the arrival of the Orient R.M.S. *Otway* at Fremantle last month a suspicious case of illness in a boy in the third class proved to be small-pox. Although the patient had been isolated on the discovery of the illness a few days after leaving Colombo, the infection had already spread to some of the other passengers, and several cases have occurred among the passengers quarantined at Fremantle, Melbourne, and Sydney. The infection has apparently been rather virulent, as some of the cases have been of the haemorrhagic type, and death has resulted in a few hours. On the arrival of the steamer at Sydney she was at once placed in strict quarantine, and over 600 persons were detained at the quarantine station at the North Head. A few days ago one of the seamen, who had been released from quarantine after the ship had been thoroughly disinfected, presented himself at the Sydney Hospital, and he was at once detained as a suspicious case and removed to the quarantine station. The chief quarantine officer, Dr. Ashburton Thompson, states that the suspected case was certainly one of small-pox, but in an exceedingly mild and even abortive form. Immediately upon the detection of the case every precaution was taken, as though it were the disease in a serious form. All the doctors, attendants, and all who came in contact with the patient have had to submit to vaccination. So far no further cases have been recorded. It is now stated that when the *Otway* reached Fremantle on March 22nd, flying the yellow flag, the port medical officer, Dr. Williams, was informed that a case of tropical fever had developed during the voyage from Colombo, but an examination showed the patient to be suffering from small-pox. The authorities, it is said, have decided to institute proceedings against those of the *Otway's* officers who were responsible for the alleged misrepresentation made to Dr. Williams. Summonses will be served on the defendants, and the case is to be heard when the *Otway* reaches Fremantle on April 26th.

Correspondence.

GASTROPTOSIS.

SIR,—In a letter which appeared in the JOURNAL of May 14th, under the title given above, Dr. Scanes-Spicer discusses certain problems relating to visceroptosis, and to the mechanism of respiration. He has been good enough to cite certain inferences drawn by me some seven years ago relating to the cause of visceroptosis. Further observations and the progress of our knowledge has led me to materially modify the statements quoted by Dr. Scanes-Spicer—modifications which I embodied in the article on visceroptosis contributed to the last edition of Allbutt and Rolleston's *System of Medicine*. In true visceroptosis the diaphragm occupies the position of extreme inspiration; its muscular fibres are shortened and contracted, while the muscular fibres of the abdominal wall are elongated and relaxed. In marked cases the diaphragm has reached the limit of its inspiratory descent. There can be no doubt I was in error in inferring that the low position of the abdominal viscera was due to a vitiated action of the respiratory muscles. I therefore agree with Dr. Scanes-Spicer when he states that the condition of visceroptosis is "primarily postural," and not "respiratory." The prolapsed condition of the viscera is due not to a disturbance of the respiratory action of the muscles of the body wall, but to the vitiation of an equally important function—namely, the maintenance and regulation of the intra-abdominal pressure. This, which may be termed the "visceral function" of the muscular parietes, not only plays an important part in regulating the circulatory mechanism, as Dr. Leonard Hill has demonstrated, but serves, especially in orthograde animals like man, to maintain the viscera in position.

By applying the term "postural" to the condition of visceroptosis we are assisted only a little way to a full understanding of its nature and cause. We require a much fuller knowledge of the "visceral function" of the parietal musculature—especially of that complex nerve mechanism which co-ordinates the degree of tone and contraction of the abdominal musculature in relationship to the needs of the abdominal viscera. That the evolution of the orthograde posture was attended by a further elaboration in the reflex mechanism which exists between the viscera and muscles of the body wall in mammals is certain, but as to the anatomical substratum of that mechanism we know almost nothing at present. It is highly probable that the Paccinian corpuscles, which are found so richly distributed in the root of the mesentery and in the subperitoneal tissue in other parts and by the

sides of veins and venous plexuses, are end organs of this mechanism. The cause and probably the cure of visceroptosis will become apparent when this mechanism is better known.

In the investigation of all cases of visceroptosis, and especially of gastroptosis, a determination of the level of the diaphragm is absolutely essential to separate the condition of "true" visceroptosis from a condition of "false" visceroptosis which is due to artificial or pathological deformation of the liver and stomach with dislocation of the kidney. For the estimation of the level of the diaphragm I proposed a line drawn transversely on the body at the notch at the lower end of the sternum. My friend, Professor Wenckebach of Groningen, informs me that he finds the spinal parts of the tenth pair of ribs afford a better standard for estimating the degree of diaphragmatic dislocation than the line proposed by me.—I am, etc.,

Royal College of Surgeons of England, May 20th. ARTHUR KEITH.

DOES GASTROSTAXIS EXIST AS AN INDEPENDENT DISEASE?

SIR,—Dr. C. Bolton's admirable paper in your issue of May 21st should provoke an interesting discussion. He will, however, I hope, forgive me for pointing out that the title he has chosen is a little unfortunate, in that his use of the word "gastrostaxis" is not quite accurate, and rather tends to create unnecessary confusion.

Gastrostaxis, or oozing of blood from gastric mucosa, is merely a symptom that may occur in many diseases, just as epistaxis, or uterostaxis, or enterostaxis may occur in many diseases. To ask, therefore, if "gastrostaxis exists as an independent disease" is almost equivalent to asking if epistaxis exists as an independent disease. Diseases and their symptoms are surely not interchangeable terms. That the confusion created by not distinguishing in this case cause from effect is a real one is shown by the opening paragraphs of Dr. Bolton's paper. He there differentiates, in nomenclature, the oozing of blood from gastric mucosa that may occur in absence of demonstrable ulcer and of any other disease in which bleeding is likely to occur from the oozing that may occur in many conditions of toxaemia. To oozing in the first group he gives the name of "gastrostaxis"; to oozing in the second group he gives the name merely of "haemorrhage." Now, the object of his paper appears to be to show reason for believing that where oozing occurs in the absence of demonstrable ulcer, and in the absence of recognized toxaemic disease, the absence of ulcer is only apparent. Hence, if the term "gastrostaxis" be applied to this group only, the inference is that ulceration is not responsible for the oozing that occurs in the toxaemic group. This suggestion can scarcely be intentional, and, even if it were, could not be maintained until it had been shown that minute ulceration may not occur in cases of the toxaemic group in which the mucous membrane is ostensibly intact.

It is now many years since it was pointed out by Dr. Donald Hood that oozing of blood may take place from gastric mucosa without there being any demonstrable ulcer present to account for its production. The point was taken up also by Dieulafoy in 1898 and later by Dr. Hale White. The word "gastrostaxis" was then coined by Sir Edwin Cooper Perry (on the analogy of "epistaxis"), and if properly used it well describes the phenomenon. Its use was, however, immediately perverted, as I pointed out in December, 1907. The reason for perversion was the undoubted fact that oozing of blood from gastric mucosa may occur, mainly in young women, and may be accompanied or preceded by pain, in the absence of ulcer demonstrable by ordinary methods. This, of course, apart from its occurrence in the demonstrably toxic conditions in which gastrostaxis, or other forms of viscerostaxis, may occur in its purest forms. In order, therefore, to distinguish the apparently peculiar syndrome of oozing and pain without discoverable ulcer from the familiar syndrome so often accompanied by demonstrable ulcer the word "gastrostaxis" was swept from its proper place in medical nomenclature and was quite inadmissibly applied in the sense in which Dr. Bolton uses it.

It is to be hoped that this criticism, which is offered in no spirit of pedantry, will help to check a growing abuse that tends to add needless difficulty to a subject already sufficiently intricate.

To the many excellent points made by Dr. Bolton against acceptance of the view that gastrostaxis, whether accompanied by pain or not, may be symptomatic of a disease not associated with ulcerative lesion, I should like to add one or two remarks.

It is generally assumed that if, on macroscopic inspection, above all in the deadhouse, no ulcer or scar of ulcer be found, there is not, and has not been, an ulcer. Dr. Bolton has sufficiently dealt with the view that macroscopic evidence of the absence of ulcer is, unless the examination be conducted with detailed care, not necessarily of any value in proving absence. Certainly the position of those who maintain that failure to find ulcer is no proof of its absence, since a minute ulcer is easily overlooked, is at present unassailable by those who suggest that gastrostaxis without ulcer may be symptomatic of a distinct clinical entity; but of equal importance with ability or failure to demonstrate the existence of open ulcer is the question of scars. Some gastric and duodenal scars there are which from their nature cannot, on inspection, escape notice. Others there are that require most careful search; but—and here is the crux of the matter as regards acute gastrostaxis—are we sure that ulcers need leave any scar at all—any scar, that is, that the eye unaided by the microscope can detect? There is much suggestive, if indirect, evidence to the contrary. The one characteristic of experimentally produced gastric ulcers in animals is their fugitive nature, also always acute. The same is true of the natural disease in man. A patient with an acute ulcer—often, it may be, missed—either dies, or merges into a chronic stage, or gets rapidly well. Acute ulcer in man, apart from the obvious toxaemias, is a commoner event than is often supposed. Why should acute ulcers of the stomach, single or multiple, which rapidly get well, necessarily leave any scar at all, recognizable by macroscopic methods? If this reasoning be sound, and if it should eventually prove to be the case that wherever there is profuse gastrostaxis there is also a condition of ulceration, minute or gross, we should not always expect to find scars. The position, therefore, of those who think that gastrostaxis may occur without ulceration cannot be strengthened by an appeal to the apparent absence of scar any more than to the apparent absence of ulcer.

Once more, it is assumed by many writers that a chronic gastric or duodenal ulcer is essentially different, as regards its genesis, to an acute ulcer. This view I believe to be wholly erroneous. The fact that chronic ulcers are more often found on inspection to be single than multiple proves nothing. The distinguishing anatomical differences in morphology and site between an acute ulcer and a chronic also prove nothing as regards different genesis. Both in animals and in man multiplicity of acute ulcers, and often of chronic, is a well marked feature, particularly as regards acute ulcers in demonstrable toxaemias. If it be true that a chronic ulcer is merely an unhealed relic of a crop of acute ulcers that have healed, the conception of a clinical entity characterized by gastrostaxis and pain without ulcer becomes still more difficult to sustain. For if all single chronic ulcers be only the remains of an acute multiple outbreak the connecting link between acute gastrostaxis and multiple points of ulceration is greatly strengthened.

At the best, then, it is difficult to see how the view that gastrostaxis is independent of precedent ulceration can at present be more than a matter of speculation. From the argument that the conception is based on incomplete observation there seems to be no escape. Of far greater interest and practical importance, however, than the relation of ulceration to these rare cases of oozing of blood from no readily demonstrable source is the essential cause of ulceration itself. Reflection shows that both haemorrhage and ulceration must almost certainly be only symptoms of a disease of which we know nothing. Till this problem of the initial cause of ulcer be solved energy is wasted on academic issues, particularly as the need for differential treatment of the two conditions in practice very seldom arises.—I am, etc.,

E. C. HORT, F.R.C.P.Edin.,
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May 20th.