A NOTE ON GASTRIC ECCHYMOSIS, GASTROSTAXIS, AND SIMPLE GASTRIC ULCER: THEIR POSSIBLE RELATIONS TO HÆMORRHAGIAS AND MUCOSTYSES.

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The term "simple gastric ulcer" requires no definition nor does "gastric ecchymosis," beyond that of extravasation of blood into gastric mucosa; but the term "gastrostaxis" demands some notice because there seems to be some misconception as to its proper use. Sir R. C. Perry and Dr. W. Hale White have suggested that the term should be applied to a disease hitherto unnamed. Herringham, Løjars, Bertrand Dawson, Hale White, Mayo Robson, and others are its discoverers, in that they have called attention to an oozing of blood from gastric mucosa which is not obviously related to, or accompanied by, ulceration. But the word is not capable, because, though it accurately describes the phenomenon, it only labels a symptom that is common to many diseases.

I. Gastric ecchymosis.

The many conflicting views at present held as to the pathogenesis of gastrostaxis will be the subject of a subsequent paper. Here it may be said that gastrostaxis, and gastric ulcer may be thus epitomised.

1. Gastric ecchymosis and gastrostaxis are in no way related to each other or to gastric ulcer. 2. The cause of gastrostaxis without ulceration is quite unguessed at, the cause
of ecchymosis being equally obscure. 3. The occurrence of gastric ulcer depends on a breakdown of normal immunity of mucosa against autodigestion through destruction of protective to the rabbit, is cytolytic to the mucosa of the guinea-pig. Again, injection of pure cultivations of streptococcus pyogenes aureus and of bacillus coli communis will produce in dogs characteristic necrosis, ulceration of the stomach.

Weinland once more has postulated the existence of fixed antipeptic and antitryptic bodies antagonistic to peptic and tryptic bodies supposed to reside normally in gastric and pancreatic secretions. In the light of this and similar work I hope to be able to show, by presenting a broader conception of the pathology of the phenomena than is generally accepted, that it is not impossible (1) that some forms of gastric ecchymosis, gastrosis, and gastric ulcer are but local expressions of general blood dyscrasia hitherto unrecognized; (2) that ecchymosis may be intimately associated with gastrosis and that both may be associated with ulcer; (3) that as a symptom of an unnamed general blood dyscrasia ulcer may also occur without preceding ecchymosis or gastrosis; and (4) that (a) ecchymosis and gastrosis are due to the presence in the blood of endothelial and mucolytic bodies, and (b) gastric ulcer itself is due to the presence in the blood of floating hemorrbagins, mucolysins, and other cytolysins, affecting gastric mucosa through one of two channels—either from lymph stream constantly flooding epithelial cells with the specific toxins (mucolysins) or from escaped blood charged with the same bodies (hemorrbagins, mucolysins). The suggested course of events, or of alternative events, may be for the sake of clearness thus tabulated:

**Unnamed Disease.**

1. **Pure Hemorrhhagin Type.**

Gastric ecchymosis.


(b) Local. Cure by absorption. Secondary hemorrhhagin. No gastrosis. No ulcer.

2. **Hemorrhhagin plus Mucolysin Type.**

Gastric ecchymosis.

General gastrosis. Local gastrosis.


(a) Primary hemorrhhagin. Death with or without secondary gastrosis. Post mortem. Nothing found.

Symptoms of hemorrhhagis only.


3. **Pure Mucolysin Type.**

Ucer without preceding hemorrhhagis.


N.B.—Secondary hemorrhhagin is meant the result of ulceration of large vessels as opposed to primary hemorrhhagin, which may be of the nature of gastrosis.

The foregoing table requires explanation. There are clinically four sets of cases requiring elucidation: (1) those of gastrosis without ulceration; (2) those of ulceration without gastrosis; (3) those of ulceration with hemorrhhagin, (a) of the gastrosis order and (b) of the secondary hemorrhhagin order, and (4) those of pain and vomiting without apparently either gastrosis or ulceration.

The theory here advanced presupposes the existence in the blood in such cases of (a) two or more toxins, which may occur either alone—(a) of the purely endothelial or hemorrhhagin type, causing ecchymosis only; and (b) of the purely epithelial or mucolytic type, causing ulceration only; or (b) of the hemorrhhagin and mucolysin type, causing first ecchymosis and then gastrosis; and (b) of the same type but with mucolytic toxins in the majority, with first ecchymosis, then gastrosis, and finally ulceration. Here, apparently, by the injection of macrocated mucosa there is produced in the serum of rabbits a substance of the nature of an immune body which, while protective to the rabbit, is cytolytic to the mucosa of the guinea-pig.
the existence of both of which is essential. (a) Given order or the existence of a large store of anticytolysins. (b) The function of normal gastric juice. (a) Not normally occur. The explanation rests on two factors, one of which toxaemia takes a share. So that in these nine cases association of cytolytic toxins in cases that do. The explanation is simple. The cytolytic bodies are, in this case, of the nature of immune bodies and either because of an absence of antibody or an excess of immune body they may be accepted by the body as a foreign substance. A solution is thus offered of some of the clinical problems presented by gastric ulcer. If the dose of immune body in this disease be in excess of the ability of the body to remove it, by toxoid formation, anticytolysin, and anti-haemorrhagin, or other anticytolysins, the progressive nature of an ulcer will depend on the absence of immunity production. Hence unsuitable food and gastric movements will be recognised as only subsidiary factors. If the dose is reduced, the response to the demand for the production of immunity readier we can at once see why some cases get perfectly well by rest, &c. For the delicate processes of repair will go on in spite of gastric secretion, which, in any case, according to this view, has no digestive effect on gastric mucosa, though it may interfere with healing. Wounds of the stomach, other than those made for excision of ulcers, will heal readily enough in the absence of blood-carried cytolysins. The explanation is that if mucosa is damaged by trauma, or embolism, or what not, such immunity is broken down and the organism is exposed to the toxins for which it may interfere with healing. Wounds of the stomach, other than those made for excision of ulcers, will heal readily enough in the absence of blood-carried cytolysins. The explanation is that if mucosa is damaged by trauma, or embolism, or what not, such immunity is broken down and the organism is exposed to the toxins for which it is not normally immune. If the dose is reduced, the response to the demand for the production of immunity readier we can at once see why some cases get perfectly well by rest, &c. For the delicate processes of repair will go on in spite of gastric secretion, which, in any case, according to this view, has no digestive effect on gastric mucosa, though it may interfere with healing. Wounds of the stomach, other than those made for excision of ulcers, will heal readily enough in the absence of blood-carried cytolysins. The explanation is that if mucosa is damaged by trauma, or embolism, or what not, such immunity is broken down and the organism is exposed to the toxins for which it is not normally immune.

5. What is the relation, so often seen, of chlorosis to gastrectasis and to ulceration? If it be true that gastrectasis and ulceration are due to the presence in the blood of toxins which are cytolytic to vascular endothelium and to mucous membrane, then it follows that chlorosis will in some way or other be closely connected with the existence of either toxins. But an ecchymosis into gastric mucosa, as elsewhere, is a primary lesion, and we may assume that it is due in the majority of cases to vitamin C deficiency in the blood. Many, however, believe that it has nothing whatever to do with either. Among these is Sir C. Ball who says: "If auto-digestion is the cause of ulcer how can the delicate processes of healing by granulation and epithelialisation go on while gastric juice is being removed?" Such a statement as this, coming from the lips of surgeons who at times patients get well by rest, rectal feeding, little fluid, and no solid food by the mouth, when we know that gastric juices on the whole are not the only food for the stomach and well in spite of the fact that the line of suture may be bathed in secretion? Is not the continuance of an ulcer, once established, due to gastric movements and solid food? These arguments are based upon post-mortem evidence, clinical observation, and surgical experience. Before a satisfactory answer can be given to these questions, the history of the ulcer in question must be closely studied. If the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. If the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that

6. Gastric ecchymoses are so commonly seen in the post-mortem room that if they have any relationship to gastrectasis and to ulceration we ought to find them in association. Many, however, believe that it has nothing whatever to do with either. Among these is Sir C. Ball who says: "If auto-digestion is the cause of ulcer how can the delicate processes of healing by granulation and epithelialisation go on while gastric juice is being removed?" Such a statement as this, coming from the lips of surgeons who at times patients get well by rest, rectal feeding, little fluid, and no solid food by the mouth, when we know that gastric juices on the whole are not the only food for the stomach and well in spite of the fact that the line of suture may be bathed in secretion? Is not the continuance of an ulcer, once established, due to gastric movements and solid food? These arguments are based upon post-mortem evidence, clinical observation, and surgical experience. Before a satisfactory answer can be given to these questions, the history of the ulcer in question must be closely studied. If the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that the ulcer is one in a patient who is suffering from a chronic disease, the reason for the presence of the ulcer must be sought in the disease itself. The conclusion is that
So the true value of the argument, that because ulcers are rare in malignant disease, abcess, peritonitis, tubercle, and pneumonia therefore they can have no connexion with the simple ulcer under consideration, and found no support. The doctrine that they can have no causative connexion is, of course, clear, but if we postulate the existence of cytolytic toxin or toxins behind both sets of conditions a definite connexion is at once established.

S. Some cases of gastric ulcer never have hematemesis at all. It cannot be asserted that there has in any given case been no hemorrhage, unless both gastric and intestinal contents have been repeatedly examined and at the same time. That there may be that large hemorrhage is not at all necessary for the production of an ulcer. A small hemor-

rhage may be, and sometimes is, entirely missed. An analogy may be found in those not infrequent cases of pulmonary tuberculosis, who from first to last show no sign of rhagopytis. It cannot be entirely because vessels are not involved in direct tuberculous ulceration. There must be some other factor to account for such cases. In these cases, where perhaps there is not even streaking of sputum, it may be that the natural store of antihemorrhagins is large and that of hemorrhagins small. In cases of gastric ulcer where precedent hemorrhage can be absolutely excluded hemorrhagins might be demonstrated. In such cases ulceration may be initiated by a deposit in mucous epithelium of mucolytins from the lymph stream, as suggested in the scheme under the heading of the purely mucolytic type.

S. Many cases of gastric pain and vomiting (non-hemorrhagic) continue for years, and yet post mortem no ulcer or traces of ulcer can be found. If the hemorrhagin theory of emesis or emesis be adopted the difficulty at once disappears. That an emesis should be accompanied by pain is only to be expected, as the hemorrhagic form would determine the amount of pain and both would depend on the degree of extravasation. Recurring attacks of pain would be explained by recurrent hemorrhagins. The absence of hemorrhage and of cytolytic toxin would depend on the amount of immunity to its action. It is notorious that vomiting may be produced by different kinds of acute pain. Pain pro-

duced by the tension of an emesis or emesis might be called vomiting. Again, it has often been noticed how frequently an attack of emesis may relieve pain.

4. Perforating cases of ulcer are seldom preceded by recent severe hematemesis. If the cytolytic theory be adopted a severe hemorrhage is not at all likely to be followed by ulcer and perforation because of the draining off of hemorrhagins and mucolytins caused by the hemorrhage. Gastric mucosa would, in fact, be more or less free of them.

Again, the acute perforating ulcer would be more likely to consist of gastric regions than in the case of hemorrhagic ulcer, caused in a different way than by the route of emesis and ooze. Here the plural conception of cytolytic toxins goes far towards explaining these acutely progressive cases, in addition to epithelial cells, sub-mucous, muscular, and peritoneal tissue would each fall a victim to their own specific cytolytin.

From these considerations it is clear, if it can be established, (1) that no other theory as yet put forward brings into line so apparently incongruous manifestations as ulcer without hemorrhage, hemorrhage without ulcer, perforation without hemorrhage, &c.; (2) that it is useless to expect to find in the deadhouse conclusive evidence of cause and effect of hemorrhage and ulcer beyond that of extravasation; (3) that the present-day medical treatment of the conditions must be modified; and (4) that the use of surgery in combating a profound toxemia must be carefully restricted.

It may well be asked what proof is there (1) that the forms of gastric ecchymosis, gastrostaxis, and gastric or duodenal ulceration here discussed are, individually or collectively, responsible for those manifestations of ulceration and (2) that the cause of such disease is the presence in the blood of cytolytic toxins? The evidence to submit is of two kinds. 1. Direct evidence of cytolytism in man in the form of hemorrhage or emesis and of ulceration afforded by (a) blood examinations and (b) the remarkable results of treatment by serum and by vaccines that I have undertaken with the object of proving the exact cachexia caused by the specific toxins. 2. Collateral evidence of a more indirect nature. In other diseases characterised by gastrostaxis or other hemorrhagic fluxes or other signs of cytolytism, as hematemesis, afforded by (a) blood examinations and (b) the encouraging results of serum and vaccine therapy that others as well as myself have undertaken with a view to the production of immunity in such cases; and (2) cytolytism in the animal kingdom afforded by (a) the artificial production of cytolytic phenomena in certain animals and (b) the establishment of immunity in such animals by (a) the preparation of sera and vaccines. Such evidence will form the subject of a subsequent communication and will embrace the results obtained, hematological and clinical, by serum therapy applied to the following conditions: (1) gastrostaxis and duodenostaxis without apparent ulceration; (2) with a microscopic ulcer or lesion; (3) acute gastric pain and vomiting without hemorrhage, suggesting ecchymosis; (4) chlorosis; (5) pernicious anemia; (6) purpura, and especially purpura hemorrhagica; (7) hemophilia; (8) leucocytthemia; and (9) the incidence of capillary hemorrhage (a) tubers, as acute lobar pneu-

monia and (b) in certain other conditions.

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THREE CASES OF INTESTINAL OBSTRUCTION.

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CASE 1. Vomitus of the small intestine.—The patient, a man, aged 32 years, was admitted to the Bolton Infirmary on April 13th, 1906, with a history of about a fortnight's illness. On April 10th he was seized with abdominal pain and vomiting, becoming gradually worse till the 12th, when the vomits became fecal. There had been complete constipation since the 9th, not even flatus having been passed. On admission he was evidently in a very serious condition and an immediate operation was decided upon. On opening the abdomen in the middle line below the umbilicus a very distended coils of small intestine was found apparently twisted on itself, but it was so tense that it could not be untwisted without emptying it of its contents. It was therefore incised and a quantity of liquid and feces allowed to escape. It was then clear that a loop of small intestine had passed over a band of omentum which was attached to the abdominal wall in the region of the appendix and the loop had become twisted on itself through a complete circle. It could now be readily un-

twisted and the omereal band was divided and removed. It was considered advisable to drain the bowel for a time, so a Paul's tube was tied into the incision already made. The bowel having been carefully cleansed without the use of an assistant the pledge was removed and the peritoneum was sutured round the neck of the tube just above its exit from the bowel. The rectus muscles and skin were also brought together for the greater part of the wound. The tube was allowed to remain in for six days, during which time there was a passage of a few flat and a considerable amount of flatus. It was then removed and for some days there was a copious discharge from the opening, as well as some from the anus with the help of enemata. At first the skin round the wound became very red and raw but by keeping it coated with zinc ointment it soon resumed the normal condition. The opening closed very rapidly and on May 10th only allowed the escape of gas. On the 13th it had quite closed and on the 20th the patient was discharged from the infirmary. Since then he has had good health, except for an attack of indigestion caused by eating new bread.

CASE 2. Subacute obstruction of bowels probably due to adhesions and kinking.—The patient, a man, aged 36 years, was admitted to the Bolton Infirmary on July 8th, 1906. His illness began with flatulence and on the following day there was abdominal distension with vomit-

ing of everything and a passage of feces and flatulences. There had been complete constipation since April 13th, 1906, with a history of about a fortnight's illness. On admission, after about a week's illness, the abdomen was much distended and he complained of lancinating pains, mostly round the umbilicus. Shortly before admission he had a passage of feces and a flatus. The temperature was 97°F. and his pulse was 140. Immediate laparotomy was decided upon and an opening was made below the umbilicus. There had been complete constipation since April 6th, 1906, and on admission the abdomen was much distended. From the not very acute onset below the umbilicus. The intestines were found to be very flabby and macerated, and as the patient's condition was very bad it was decided to defer an immediate operation. On the 15th the abdomen was much distended and he complained of lancinating pains, mostly round the umbilicus. Shortly before admission he had a passage of feces and a flatus. The temperature was 97°F. and his pulse was 140. Immediate laparotomy was decided upon and an opening was made below the umbilicus. There had been complete constipation since April 6th, 1906, and on admission the abdomen was much distended. From the not very acute onset round the wound became very red and raw but by keeping it coated with zinc ointment it soon resumed the normal condition. The opening closed very rapidly and on May 10th only allowed the escape of gas. On the 13th it had quite closed and on the 20th the patient was discharged from the infirmary. Since then he has had good health, except for an attack of indigestion caused by eating new bread.

Three cases of intestinal obstruction.