

amount of fluid injected—one and three-quarter gallons in all—which in the first place relieved shock and afterwards enabled the emunctories, especially the kidneys, to dispose of the poison, and also to the very able assistance which enabled me to complete the operation in just over the hour.

Plymouth.

THE CONTENTS OF THE STOMACH IN THE GASTRIC CRISES OF LOCOMOTOR ATAXIA:

A CLINICAL AND CHEMICAL STUDY.

By CARSTAIRS C. DOUGLAS, M.D., B.Sc. EDIN.

GASTRIC crises hold a prominent position among the visceral symptoms of locomotor ataxia and no one observing a patient suffering from them when accompanied by much vomiting can fail to be struck with the enormous quantity of fluid which is often rejected, even when the patient is taking little by the mouth. As a rule, first of all the stomach is emptied of what food it contains; then there is vomiting of a clear liquid which later may be stained with bile or occasionally with blood. Marie¹ says: "There is vomiting, at first of food, but afterwards exclusively of slimy liquid or perhaps of a clear liquid mucous in character and more or less abundant. At times the vomited matter is coloured by bile and more rarely by blood; it may quite exceptionally present the appearance of coffee-grounds." With this general description that by Gowers agrees closely. He states that the vomiting is at first of food and then of clear liquid which may be very abundant; ultimately bile is vomited and sometimes blood. As regards detailed chemical examination of the vomited fluid there are on the whole but few reports, especially by English workers. Sahli² some years ago stated that he had found excessive acidity of the gastric juice in this condition, but his analysis is unreliable as he made no exact quantitative estimation but merely relied on the intensity of the colour reaction as judged by the eye. Marie quotes Rosenthal (1886) who found 0.3 per cent. of HCl during the first days of the crisis and 0.10 per cent. during the latter days, so that, taking Richet's figure of 0.174 per cent. as the normal, Rosenthal's figures show an increase in at least part of the attack. The same was found by Simonin of Lyons (1886). Hoffmann in one of his patients found the gastric juice to be secreted in excess and to contain temporarily an excess of acid. The quantity of HCl varied, often amounting to twice the normal quantity and rarely to less than the ordinary amount, and this only when owing to the abundance of the matter vomited the acid was somewhat largely diluted. The only other point in this connexion to which reference is made by Marie is that in some cases small and varying amounts of lactic acid have been found. Grasset³ gives no details regarding the composition of the fluid, while Möbius⁴ considers hyperacidity to be the exception in these cases and that the amount of acid is usually small.

In a recent paper Michell Clarke⁵ gives a good account of the stomach contents in a patient suffering from early tabes both between and during the crises. The chief points as regards the chemistry are: 1. In the intervals gastric digestion was rapidly performed and there was excess of HCl during the process (sometimes 0.4 per cent.), whereas in the intervals between meals there was no evidence of secretion of HCl. 2. During the whole duration of the gastric crises free HCl was absent from the stomach contents altogether while lactic acid was abundantly present. In his analyses, however, no exact quantitative estimation of the different acids was made, the result being simply deduced from the amount of dilution which the fluids would stand while still exhibiting the colour reaction for the given

acid. In Dr. Michell Clarke's case, as in the one which I am about to describe, there was marked hæmatemesis. In drawing any conclusions as to the presence of hyperacidity it must be remembered that the amount of acid secreted may vary, according to Verhaagen,⁶ considerably even in health. He found that there might be variations of one per mille, 1.5 per mille, and even two per mille, the acidity from two to two and a half hours after a test meal ranging from 3 to 4.8 per mille. As regards lactic acid our latest knowledge of the gastric chemistry shows that it may occur in the fluid rejected in locomotor ataxia as in any other condition provided the stomach is emptied within an hour of eating and provided the last meal contained at least some carbohydrate. Here it is formed as a result of bacterial action upon maltose taking place within the stomach. This formation of lactic acid, which is quite physiological, ceases with the secretion of HCl and the lactic acid appears to be absorbed—at all events, it disappears from the stomach. Various observers, however, have found, as I myself did, that lactic acid may be present during gastric crises hours after food has been ingested and subsequently vomited. In the absence of dilatation of the stomach it is difficult to explain this, as recent observations point to the fact that when HCl is not secreted lactic acid does not take its place.⁷

Having recently had the opportunity, through the kindness of Dr. James Finlayson, to whom I desire to express my obligation, of studying the gastric crises in a case of early tabes where much hæmatemesis occurred I made a number of analyses of the stomach contents which I shall give in detail after a short account of the general features of the case.

A man, aged 27 years, was admitted to the Western Infirmary, Glasgow, on Oct. 14th, 1898, under the care of Dr. Finlayson, complaining of sickness and vomiting and weakness of the legs. The history of the patient's illness is taken in part from the ward journal. Eighteen months previously he had noticed some weakness of the legs and unsteadiness on going downstairs or when he was in the dark. Six months later girdle pain was experienced, but only for a fortnight. At the same time attacks of vomiting began. When the attacks were about to come on he had a feeling of nausea and uneasiness in the epigastric region; also great restlessness, so that he always tossed about in bed. When vomiting began he first rejected any food that was in the stomach and as emesis proceeded he brought up mucus mixed with blood and bile. The vomiting had no special relation to food either as regards the nature of the latter or the time of ingestion. The attacks occurred at first every fortnight but latterly every two or three days and might last from one hour to a couple of days. Eructations occasionally occurred before the attacks but as a rule the latter began suddenly. For nine months he had had some bladder trouble and a tendency to constipation. As regards his previous health, he had had gonorrhœa but not syphilis, nor was there any history of any stomach disturbance or of any gastric symptoms in the past. He was temperate in the use of alcohol. As regards his state on admission, the patient was pale, rickety, and undersized, but fairly well nourished. His weight was 7 st. 7 lb. As to his nervous system it may be briefly stated that there was some impairment of the tactile and temperature sense in the toes and in an area extending from the umbilicus to the fifth rib. Romberg's sign was well marked; the superficial reflexes were present and were normal; the patellar reflexes were absent. The pupils were equal and medium in size; the Argyll-Robertson phenomenon was present; ptosis existed and was most marked on the left side; the fundus was normal. Examination of the blood between the attacks gave 5,500,000 red cells and 9000 leucocytes per cubic millimetre, 80 per cent. of hæmoglobin, and a specific gravity of 1060. The urine was clear, amber-coloured, and acid, and its specific gravity was 1026; no albumin, sugar, blood, bile, or albumoses were present; occasionally it was neutral or alkaline even apart from the attacks, and it then showed a deposit of pus and ammonio-magnesium phosphate. As regards the alimentary system the patient after admission exhibited marked ptialism, sometimes rejecting from a pint to a pint and a half of colourless, slightly

¹ Lectures on Diseases of the Spinal Cord, 1892, p. 266.

² Ueber das Vorkommen abnormer Menge freier Salzsäure bei den Gastrischen Krisen eines Tabetikers, Correspondenzblatt für Schweizerische Aerzte, 1885, No. xv., S. 105.

³ Traité pratique des Maladies du Système Nerveux, Paris, 1894, quatrième édition, p. 513.

⁴ Twentieth Century Practice of Medicine, 1897, vol. xi., p. 837.

⁵ A Contribution to the Clinical Study of the Gastric Juice, Brit. Med. Jour., vol. ii., 1898, p. 1863.

⁶ La Physiologie et Pathologie de la Sécrétion Gastrique, Paris, 1898, p. 9.

⁷ Schäfer: Text-book of Physiology, vol. i., 1898, p. 358.

opalescent, thin mucoid fluid which readily rendered starch-paste soluble even at room temperature and converted it into maltose and dextrin. It also faintly gave the reaction for sulphocyanide of potassium. The salivation went on during sleep; it was always less at the end of the gastric crises. The patient did not swallow much saliva when sick. Crises took place after admission every three days from Oct. 14th till Nov. 8th. There was then an interval till Nov. 18th to 20th and after this the patient had a period of immunity lasting from Nov. 20th to Dec. 23rd (four and a half weeks). There was then another short attack which was repeated on Jan. 10th and Feb. 3rd, 1899, and so on. The general features of the crises were the same as before admission. Blood occurred frequently in the vomited matter, sometimes at the beginning and sometimes at the end of the attack. On several occasions it was very plentiful, amounting to about a pint. In colour it did not

TABLE I.—*Examination of the Contents of the Stomach removed by Stomach-tube during Intervals of Freedom from the Attacks. In all cases the lavage was performed after a test dinner.*

	No. 1. One and a half hours after test meal.	No. 2. Two and a half hours after test meal.	No. 3. Two hours after test meal.*	No. 4. Two hours after test meal.
Reaction ...	Acid to litmus and to Congo red.	Some amount of fluid, neutral to litmus and negative to Congo red.	—	Acid to litmus and to Congo red.
Total acidity ...	0·292 per cent.	—	—	0·39 per cent.
Free HCl ...	Present (scanty).	Contained no HCl or peptones or sugar.	—	Present (scanty).
Lactic acid ...	Faint trace.	—	—	Absent.
Volatile organic acids ...	Absent.	—	—	Absent.
Peptones ...	Present.	Pepsin present and active in acid solution.	—	Present.
Sugar ...	"	—	—	"
Starch ...	"	—	—	"
Blood ...	Absent.	—	—	Absent.
Bile ...	"	—	—	"
Pepsin ...	Present and active.	—	—	Present and active.

* Stomach found to be perfectly empty.

TABLE II.—*Detailed Analysis of Total Acidity on Two Occasions during Intervals between the Attacks.*

1. Total acidity = 0·292 per cent.	2. Total acidity = 0·39 per cent.
0·216 = combined HCl + trace of lactic acid.	0·253 = combined HCl.
0·022 = free HCl.	0·045 = free HCl.
0·054 = acid salts.	0·092 = acid salts.
0·292	0·390
	No organic acids were present.

present the ordinary coffee-ground appearance, but was generally of a deep-red or plum colour. Apart from the crises the patient had no indigestion and physical examination revealed no abnormality in the stomach. When the crises came on the abdomen seemed to bulge and after they ceased it subsided again. The bowels at these times were usually costive and irregular. During the attacks the urine became neutral or alkaline and tended to deposit phosphates.

While the patient was in hospital I made a number of careful examinations of the contents of the stomach, the results of some of which will be found in the accompanying tables.

Nothing in Table I. suggests the least defect in digestive power. As a matter of fact digestion always appeared to proceed evenly and rapidly, and, as noted above, on two occasions the stomach was found to be quite empty at from two to two and a half hours after a plentiful test dinner. On one occasion the total acidity was somewhat high (0·39 per cent.) without any obvious cause. (Table II.)

In Tables III. and IV. are shown the results of some examinations made when the attacks were present. It will be seen from Table III. that on no occasion did the gastric contents exhibit any hyperacidity, the highest figure obtained being 0·3 per cent. which included free and combined HCl, a trace of lactic acid, and the acid salts. Free HCl itself was generally absent. There is no indication that the total acidity diminished as the crises continued, as will be gathered from analyses 2, 3, 5, and 6, where on consecutive days the acidity was found to have slightly increased though it never rose above normal. Traces of lactic acid were present on three occasions and as a rule peptones were found in the fluid. On all occasions save one blood formed a large part of the vomited matter; on several occasions it was very copious and was generally deep-red or plum-coloured in hue.

TABLE IV.—*Detailed Analyses of Total Acidity during the Crises.*

1. Total acidity = 0·109 per cent.	3. Total acidity = 0·219 per cent.
0·036 = combined HCl.	0·149 = combined HCl + trace of organic acid.
0·073 = acid salts.	0·070 = acid salts.
0·109	0·219
There was no free HCl, volatile, or lactic acid.	There was no free HCl.
2. Total acidity = 0·233 per cent.	4. Total acidity = 0·215 per cent.
0·124 = combined HCl + trace of lactic acid.	0·146 = combined HCl.
0·109 = acid salts.	0·069 = acid salts.
0·233	0·215
There was no free HCl.	There was no free HCl or organic salts.

These detailed analyses indicate that on no occasion was there any hyperacidity and particularly no hyperchlorhydria, the highest figure for combined HCl being 0·149 per cent., which included also a trace of organic acid. The greater part of the total acidity was composed of this combined acid, the remainder being due mainly to acid salts.

The conclusions which we are entitled to draw from the study of this case are: (1) that during the crises the stomach secreted a large quantity of digestive fluid, inferior

TABLE III.—*General Analyses of Fluid Rejected during the Gastric Crises.*

	No. 1.—Oct. 17th.	No. 2.—Nov. 14th.	No. 3.—Nov. 15th.	No. 4.—Nov. 22nd.	No. 5.—Dec. 23rd.	No. 6.—Dec. 24th.
Reaction ...	Acid to litmus, faintly to Congo red.	Acid to litmus, negative to Congo red.	Acid to litmus and to Congo red.	Acid to litmus, faintly to Congo red.	Faintly acid to litmus, negative to Congo red.	Acid to litmus, faintly to Congo red.
Total acidity ...	0·30 per cent.	0·211 per cent.	0·233 per cent.	0·262 per cent.	0·109 per cent.	0·233 per cent.
Free HCl ...	Present (scanty).	Absent.	Present (scanty).	Absent.	Absent.	Absent.
Lactic acid ...	Trace.	"	Absent.	Traces.	"	Trace.
Volatile organic acid ...	Absent.	"	"	" (?)	"	Absent.
Peptones ...	Present.	Present.	Present.	Present.	"	Present.
Sugar ...	"	"	"	"	Traces.	"
Starch ...	"	Absent.	"	Traces.	Absent.	Absent.
Blood ...	"	Abundant.	"	Absent.	Abundant.	Abundant.
Bile ...	"	Present.	"	"	Present.	Present.
Pepsin ...	Present and active.	Present and active.	Present and active.	Inactive.	—	—

in but slight degree to that poured out during health; (2) that there was no indication that this secretion was associated either with hyperacidity or hyperchlorhydria; and (3) that apart from the presence of blood there was nothing in the fluid indicative of a true lesion of the stomach.

It has sometimes been suggested in cases like this, where there was abundant vomiting of blood, that we might be dealing with an ulcer of the stomach—an ulcer of the same type and significance as the perforating ulcer of the foot. But it seems to me that, in this case at least, such a hypothesis is untenable and in other reported cases it appears equally wanting in proof. It could hardly be expected that an ulcer which bled freely every two or three weeks would not leave some trace of its presence in the shape of disordered digestion, pain, or other symptom between the attacks. But it has been pointed out that this patient enjoyed excellent gastric health between the crises, exhibiting no trace of stomach lesion on careful physical examination, and digesting so rapidly and perfectly that it was not possible to find any trace of a test dinner two and a half hours after its ingestion. It is quite possible that in such cases the source of the bleeding lies in engorged or varicose veins around the cardiac end of the œsophagus.

Glasgow.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

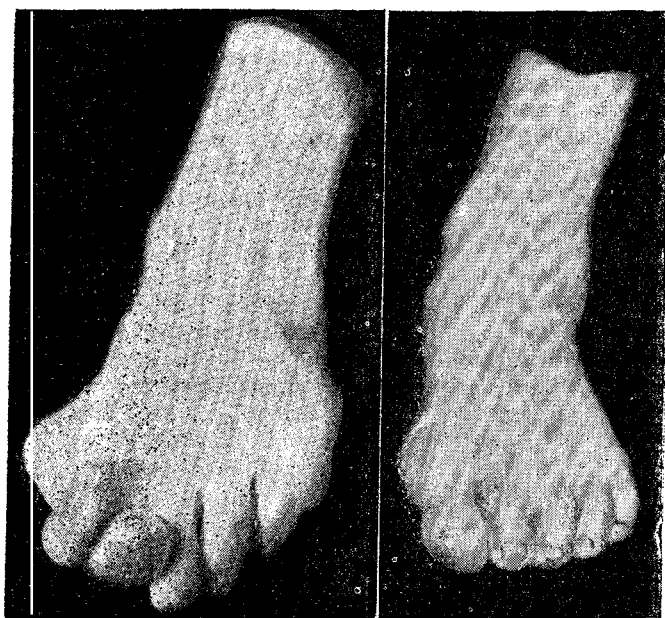
HALLUX VALGUS AND HAMMER TOE.

By W. J. COLLINS, B.Sc., M.S., M.D. LOND., F.R.C.S. ENG.,
SURGEON TO THE LONDON TEMPERANCE HOSPITAL AND TO THE
ROYAL EYE HOSPITAL; FELLOW OF THE UNIVERSITY OF LONDON.

AMONG the minor deformities the pathology of which is obscure and the treatment of which has varied from elaborate trifling to unduly mutilative heroism is hallux valgus with the closely allied hammer toe. Congenital causation, ligamentous contracture, tendinous abbreviation, and external pressure have each and all been invoked to

FIG. 1.

FIG. 2.



Hallux valgus and hammer toe.
Before operation.

After resection of
joints.

explain this inartistic and most discomforting deformity. Treatment has, of course, varied according to the view entertained as to the general or particular cause, and even recent works recommend amputation in despair of securing success by any less radical procedure. It is curious that the "valgus" tendency in the toes

should be almost the exclusive prerogative of the hallux. The support afforded by the other toes might have been thought to operate in the direction of resisting such deviation. But, as in the case illustrated above, it will be seen that so strong was the valgus impulse that the coincident deformity of a hammer second toe afforded no obstacle. Similarly it is the second digit which most frequently, though not exclusively, exhibits the "hammer" deformity. Doubtless the free mobility of the metatarsophalangeal and inter-phalangeal joints, partaking of the nature of a universal and not only of a ginglymus movement, readily lends itself to extrinsic pressure. Any deviation from the normal line once started would tend to be stereotyped by intrinsic assistance. Thus the insertion of the innermost tendon of the extensor brevis digitorum into the base of the first phalanx of the big toe and the diagonal direction of its pull would coöperate in a valgus deviation. Similarly the second toe alone has inserted into the base of its first phalanx the tendons of two dorsal interosseous muscles. Here the malleus deformity once started would be assisted by muscular action and ligamentous shortenings would tend to perpetuate and to fix a dislocation in the first instance intermittent and reducible. The good result of aseptic excision of the heads of the proximal bones without division of any tendons is well shown by Figs. 1 and 2 reproduced from photographs taken before and after operation. The case was one of combined hallux valgus and hammer toe in a woman, aged 36 years. I operated on March 9th of this year, on the 18th the splints were left off, and on the 23rd the patient was discharged, walking well without pain and the toes in perfect position and naturally mobile.

Albert-terrace, N.W.

NOTES ON SOME CASES ILLUSTRATING THE ADVANTAGES OF PARTIAL CHLOROFORM ANÆSTHESIA.

By LIEUTENANT-COLONEL E. LAWRIE, M.B. EDIN., I.M.S.,
RESIDENCY SURGEON, HYDERABAD, DECCAN.

CASE 1.—The patient was a male native, aged 31 years. The operation took place on Jan. 14th, 1899. Food had last been taken at 7 P.M. on the previous day. The operation was the aspiration of an abscess of the liver. Mr. Salar Musood, a third-year student, was the chloroformist. The administration commenced in one drachm doses at—

H.	M.	S.	
9	24	0	Natural regular breathing; blowing into the cap.
9	25	0	Chloroform added to the cap; two breaths of air.
9	25	40	Unconscious; needle inserted; the patient shouted and struggled a little.
9	26	10	Pus was flowing; seven ounces were withdrawn.
9	26	57	The needle was removed.
9	28	7	The patient walked to bed.

The total duration of the operation was four minutes and seven seconds and the amount of chloroform employed was two drachms.

CASE 2.—The patient, a male native, aged 45 years, had last taken food at 6 A.M. on the day of the operation (Jan. 14th, 1899). This operation also was the aspiration of an abscess of the liver and the chloroformist was Mr. Salar Musood. The administration commenced at—

H.	M.	S.	
9	30	15	Blowing regularly into the cap.
9	31	30	Chloroform added; one breath of air taken.
9	31	50	Unconscious.
9	32	0	Needle inserted.
9	32	16	Pus was flowing; 10 ounces were evacuated.
9	33	13	The needle was withdrawn.
9	34	8	The patient walked to bed.

The operation lasted for three minutes and 53 seconds. The amount of chloroform employed was two drachms.

CASE 3.—A male native, aged 25 years, who had last taken food at 8 A.M. was operated upon on Jan. 14th, 1899, the operation being, as in the two former cases, one of aspiration of an abscess of the liver. Mr. Salar Musood