THESIS

ON

1. OBSERVATIONS ON PEPTIC ULCER IN INDIA

2. WATER EXCRETION IN THE TOXEMIAS OF PREGNANCY

FOR THE DEGREE OF

M.D. EDINBURGH,

by

E.G.L. MARK, B.Sc., M.B., Ch.B.
SOME OBSERVATIONS ON PEPTIC ULCER IN THE ARMY IN INDIA.

While serving in India I was attached to a large base hospital through which passed all British cases recommended for evacuation ex India, or for discharge from the Army. Amongst these was a high percentage of cases of peptic ulcer - gastric, duodenal or simply labelled peptic ulcer - mostly depending for their diagnosis on a radiological report. Because of shortage of films the barium meals were not repeated in our hospital.

After spending a month writing up these Peptic Ulcer cases for the Review Board, I was struck by the number which presented many features unlike those I had been accustomed to meet at home. This was most forcibly brought home to me, when I was taking the history of one case. He was a young man, aged 24, who had had no previous digestive trouble until he was seized in China with severe pains in the abdomen and passed frequent stools containing blood and mucus. He belonged to a party, 250 strong, which had no medical officer with them, and at the time he was troubled with his pains and loose watery motions, a large percentage of the others were also stricken, a fair number dying from the illness. This man remained on duty, but had loose motions off and on for 6 months before leaving China, when his stools returned to normal. After that he began to get symptoms of indigestion after meals with some epigastric pain and discomfort. He put up with his troubles until he arrived back in India when he reported sick. He was eventually sent to our hospital with the diagnosis of duodenal ulcer on the strength of an X-Ray report of deformity of the duodenal cap. His case notes contained no reference to the attack of dysentery in China.
After seeing this case, careful records of all succeeding cases were made, and my opinion of the true incidence of peptic ulcer amongst the cases coming under my observation was formed from the examination of 93 cases.

On admission to hospital all these cases were put on a gastric No. 1. diet, which consisted of 2 hourly feeds of milk with arachis oil and Tinct.Belladonna being given alternately before 6 feeds. Mist. Alkali containing Mag. Carb. Pond. and Sod. Bic. was given after feeds. Ascorbic Acid Tabs, mgm 100 were given daily. The patients were kept on this diet for one week, at the end of which, bread, butter, eggs, custard and jellies were added. They remained on this diet for 2 weeks - Gastric No. 2. - or longer if their symptoms had not cleared up, after which they were given Gastric No. 3. diet, which contained boiled fish, sieved vegetables and chicken in addition. The patients remained on this diet until all symptoms and signs - this latter meaning the disappearance of deep tenderness - had disappeared. The oil and belladonna before, and the alkali after feeds were reduced in the usual way.

ABSTRACTS FROM CASE RECORDS.

Case 1. Sgt. S. Aged 27. Service 7 years. India 1 year.
Diagnosed G.U.

History. Symptoms for 3 months, no spontaneous remissions.
Started with pain in the epigastrium associated with tenderness over the R. upper rectus. After the first day the pain had always been in the R.I.F. Pain comes on at any time; no relation to meals; Alkalies gave partial relief; always flatulent; frequently nauseated especially in the mornings. He dieted himself mostly
on steamed fish, avoiding meats and fried foods, and
felt much better as a result. General health good; no
diarrhoea or dysentery; no blood or mucus ever noticed
in the stools; no haematemesis or melaena.
Admits to smoking 30 cigarettes a day.

O/E.
Slight diffuse tenderness in epigastrium.
Definite tenderness over McBurney's point in R.I.F.
No rigidity or muscle guarding.
Liver, spleen and sigmoid colon not palpable.
Progressed favourably on gastric regime.
Tenderness in R.I.F. remained, but disappeared in
epigastrium.
Ba. Meal was reported as showing an ulcer on the lesser
curve of the stomach.
Patient refused to have his appendix removed.

Comment. The entire clinical picture seems to me to be that of
Braithwaites Ileo-gastric syndrome - probably an
appendicular dyspepsia with a degree of alcoholic
gastritis superadded.

Case 2. Pte T. Aged 32. Service 3 years. India 1 year.
Diagnosed D.U.

History. 8 months ago started with pain in the epigastrium and
flatulence p.c. Nauseated but no vomiting; no remissions;
no relief from alkalis, but pain passes off spontaneously
after about ½ hr. Appetite poor. Loss of weight 10 lbs.
No previous history of dyspepsia.
No family history of dyspepsia.
Ba: Meal: - "No evidence of any gastric lesion but the filling of the duodenum was difficult to obtain, and in my opinion there is a small ulcer at its base. Small residue of Barium remained in the stomach 5 hrs. after the meal."

Stools: - Occult blood negative on 3 occasions.

When I saw him he complained of pain in the epigastrium 20 min. p. c. lasting 1/2 hr. The pain was sharp even after drinking milk. He got no relief from alkalis. He also had slight flatulence and waterbrash.

On questioning him he admitted having been in hospital for 2 weeks with diarrhoea immediately before he developed his symptoms, and said that ever since he had had a dull ache below and to the left of the umbilicus.

O/E. Tongue fissured and furred; definite tenderness high up in the epigastrium and in the L.I.F. Sigmoid colon palpably thickened.

Comment. The history is not that of a duodenal ulcer. The symptoms started with diarrhoea, after which he had constant epigastric pain which was not relieved by alkalis. His appetite was poor, he had lost a lot of weight and complained of pain in the L.I.F.

In my opinion the X-Ray report is valueless, and clinically he appears to be a case of reflex dyspepsia from colonic infection.


History. Landed in Egypt in July 1942. 1 month later he had
clinical dysentery. Stools D. 6-7. N. 6-7. Blood and mucus were present. He was treated in hospital for 3½ weeks with fluids and Sod. Sulph. On returning to the desert the diarrhoea started again 10 days after his discharge from hospital, and with it he had pains across the upper abdomen 1 hr. p.c. and vomiting which partly relieved his pain. From that time he was in and out of 5 different hospitals for varying periods up to 10 weeks, each time being treated with rest, alkalis and restricted diet. An X-Ray at one hospital showed a gastric ulcer. He was finally shipped to India. In India he complained of pain in the epigastrium which came on at any time without relation to meals or the type of food he ate. The pains would last for 2-3 days, and be clear for a day or two. Flatulence was very troublesome. He had slight diffuse tenderness all over the upper epigastrium, but much more marked tenderness in both the R. and L.I.F. The caecum and sigmoid colon were palpably thickened and tender. He had lost 13 lbs. in weight. His bowels were only open once in 24 hrs, but were loose. Nil was found on laboratory examination of the stools.

Comment. The history is not that of gastric ulcer. There is little doubt in my mind that this is a case of reflex dyspepsia from a residual low grade infection of his colon following his attack of dysentery. The X-Ray report of a gastric ulcer was either wrong or was merely an incidental finding. The tenderness and
thickening of the colon is characteristic of chronic amoebiasis of the colon which so frequently gives negative results on stool examination.

Case 4.

History. Duration 4 years. Father had "stomach trouble" and Brother G.U. and Anxiety Neurosis. Complained of continuous pain in the epigastrium for 4 years with one remission of 5 months. The pain comes on ½-l hr. p.c. and lasts 1-2 hrs; slightly relieved by food and alkalis; self induced vomiting; several small haematemesis. Symptoms much worse since an attack of dysentery 1 yr. ago, which was untreated and lasted 5 months. Lost 20 lbs in weight.

When seen by me he complained of pain l hr. a.c., relieved by food and vomiting. Flatulence ++. Belches acid and is nauseated ½ hr. p.c. (on milk diet). Frank blood in the stools on several occasions and some mucus.

F.T.M.:- Hyperchlorhydria. Peak type of curve, not sustained.


He was tender in the mid line of the epigastrium just above the mid point between the Xiphisternum and the Umbilicus. The Sigmoid colon was also palpable and tender.

Comment. This history which includes several small haematemesis
The shaded area represents the limits for free HCl (dimethyl indicator), of 80% of normal (Englishmen) individuals.

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represent free HCl.

represents total acidity.

<table>
<thead>
<tr>
<th>Fasting Juice.</th>
<th>Volume</th>
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<th>Organic Acid</th>
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<td>Lactic Acid</td>
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<td>Acetic Acid</td>
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Date

Pathological Laboratory

Diabetes Laboratory

Officer Incharge District Laboratory.
HOSPITAL ROUTINE.

1. At 7 A. M. of the morning following a light supper, without taking further food or fluid, the stomach is emptied by a Ryles tube with the help of record syringe and the volume of the "resting" gastric contents is noted.

2. A test meal is prepared by boiling 2 tablespoonfuls of fine oatmeal in two pints of water until the volume is reduced to one pint which is filtered through coarse muslin, salted if desired, and swallowed by the patient.

3. Ten to fifteen c. c. are withdrawn every fifteen minutes thereafter for 2½ hours or until nothing further is aspirated.

4. The specimens are sent in the numbered test tubes to the District laboratory as soon as possible.

Nami Press, Lucknow.
is characteristic of ulcer. Whilst its earlier features are those of G.U., the history obtained in our hospital is that of D.U. This is supported by the F.T.M. and X-Ray report. A year ago he had severe untreated dysentery, which aggravated his ulcer symptoms, which had dated for 2 years prior to it.

**Case 5.** Sigm W. Aged 27. Service 4 yrs. India 3 yrs. Diagnosed G.U.

**History.** Well until August 1940 when he had diarrhoea for 4 days and was admitted to hospital with discomfort in the centre of the abdomen and vomiting after eating the battalion food. He was treated with castor oil and salts and discharged 3 weeks later. He remained well until July 1941 when he had Amoebic Dysentery and was treated in hospital with Emetine Hydrochloride by injection. (Despite the fact that this patient passed through several hospitals after this, none of his documents contained any reference to his attack of Amoebic Dysentery. On being asked why, the patient said that no one had ever asked him about it.) He remained symptom free until Nov. or Dec. 1942 when he started with a burning pain below the Xiphisternum, acid eructations, a sickly feeling and occasional vomiting about 1/2 hr. p.c. These symptoms came on if he went any length of time without food. He carried on like this until Jan. 1943, when he reported sick, and was admitted to hospital where he was put on a gastric diet, which eased his symptoms in a week. He took his own discharge
then, but as soon as he went back on ordinary food his symptoms returned. He took McLeans powder which gave partial relief. He carried on until May when he was posted to another station, where he had to go longer without meals. He was admitted to hospital there on May 12th, where he was put on a gastric diet, but he never became free of symptoms and has never been at any time since. Ba. Meal revealed a Gastric Ulcer. F.T.M. revealed a complete achlorhydria. He had 8 weeks treatment in that hospital and then was transferred to another one where he was kept a further month on restricted diet. He continued to have discomfort in the abdomen and belched up acid and the milk he was taking. He was then transferred to another hospital where he remained 3 weeks before coming to us. In our hospital he had loose motions 3 in 24 hrs. for 3 days. He had no tenderness in the epigastrium, but was very tender in the R.I.F., and to a much lesser extent below the R.C.M. and in the L.I.F. The caecum was palpably thickened.

Comment. This case to my mind is clearly one of chronic Amoebiasis with an associated gastritis. The history is quite unlike that of a gastric ulcer, and his symptoms are those of a reflex dyspepsia. It is difficult to refute an X-Ray report, but clearly the patient was not suffering from a Gastric Ulcer. The fact that his symptoms did not clear up after prolonged treatment on an ulcer regime supports this view.
GASTRO-INTESTINAL ANALYSIS


**Date**: 11/18

**Fractional Test-Meal**

- **One-Hour Juice**
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Date

- **Two-Hour Juice**
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Hour
  - Date

**SUMMARY**

The stippled area indicates the limits for free HCl of 80% of normal people.

- **Vomiting**
  - Volume
  - Cells
  - Blood
  - Mucus
  - Total Chloride
  - Active HCl

**Twelve-Hour Test-Meal**

- **One-Hour Juice**
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Date

- **Two-Hour Juice**
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Hour
  - Date

**Summary**

- **Twelve-Hour Test-Meal**
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Date

**One-Hour Juice**

- Free HCl
- Blood
- Mucus
- Food
- Volume
- Date

**Two-Hour Juice**

- Free HCl
- Blood
- Mucus
- Food
- Volume
- Hour
- Date

**Vomiting**

- Volume
- Cells
- Blood
- Mucus
- Total Chloride
- Active HCl

**Twelve-Hour Test-Meal**

- One-Hour Juice
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Date

- Two-Hour Juice
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Hour
  - Date

**Summary**

- Twelve-Hour Test-Meal
  - Free HCl
  - Blood
  - Mucus
  - Food
  - Volume
  - Date

**Patients Name**: May 3/74

**Gastro-Entestinal Analysis**
of tenderness in both iliac fossae, and the history, suggest that the symptoms were reflex in origin from an old lesion in the colon, probably amoebic in origin. The radiologist's report in my opinion is quite insufficient on which to base a diagnosis of D.U. The marked loss of weight is also unlike a D.U.


F.H.: No relative ever troubled with Peptic Ulcer.

History. 3 months ago started with severe stabbing pain over the R. lower chest in front, at the side and behind, 1 hr. post-const. and lasting 2 hrs. The pain was relieved for about an hour by taking Bismuth, and also by vomiting. Associated with the pain he had acid eructations, flatulence, and a tender area immediately below the R.C.M. He had had no history of dyspepsia previous to this. About 1 week after he started with the pain he noticed bright red blood and "white stuff" in his stools. He had no pain on passing his motion. He continued passing a small quantity of blood in his motions off and on for 2 weeks. His stools were never examined. He was given Bismuth, which relieved his symptoms for a week. Then he reported sick again and was told he had muscular rheumatism. On the third occasion he reported sick, he was referred to a Medical Specialist, who admitted him to hospital, where a gastric ulcer, high on the lesser curvature, was demonstrated.

On a milk diet all his symptoms except the flatulence
11.

cleared up. He had no loss of weight.

He had an area of tenderness below the R.C.M. only.
The caecum and sigmoid were not palpably thickened.

O/E.

The occurrence of pain 1 hr. p.c. is unusual in an ulcer
high on the lesser curve of the stomach. The fact that
he had lost no weight was also against the diagnosis.
It is much more difficult in this case to be definite
about the diagnosis, though it would be consistent
with a localised infection of the colon in the area of
the hepatic flexure. There is definite evidence in his
history that he had had a colonic infection.

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the hepatic flexure. There is definite evidence in his
history that he had had a colonic infection.

CASE 8.

Gunner W. Aged 29. Service 2½ yrs. India 1 yr. Diag¬
nosed G.U.

HISTORY. Before joining up this patient had been canvassing and
in doing that work had had irregular meals. In Nov.
1938 he first noticed discomfort in the epigastrium.
The discomfort had no relation to meals and lasted 1-3 hrs.
Bismuth gave him partial relief, and usually resulted in
his breaking wind. He was very constipated. He continued
to have this epigastric discomfort off and on until he
joined the Army in Jan. 1941. He then remained symptom
free until he was admitted to hospital with diarrhoea
in April, 1942.

Stools:— D.5. N.3. He was treated with castor oil and
salts and discharged in 7 days. 10 days later he started
having severe pains all over the epigastrium with
flatulence, acid eructations and vomiting 1½ hrs p.c.,
which were relieved by belladonna but not by alkalis.
He continued to have these symptoms on and off until July 1943, when he had bacillary dysentery. After that he was never free from symptoms and a Ba Meal then showed an ulcer on the lesser curviture of the stomach.

**COMMENT.** This case seems to present the features of a chronic gastritis before joining the army, the result of his irregular meals in civilian life. His symptoms in the Army dated from an attack of diarrhoea. The character of the pain suggested a colonic spasm and not ulcer pain. The fact that it was relieved by belladonna and not by alkalies also supports this view.

**CASE 9.** Dvr. G. Aged 32. Service 2 5/12 yrs. India 1 yr. Diagnosed D.U.

**HISTORY.** This patient started having upper abdominal pains mainly during the night with heartburn and vomiting 1-1½ hrs p.c., almost after every meal. These symptoms had been on for 3 months before admission to our hospital. He had no previous history of dyspepsia. On being asked specifically he admitted having had loose motions for about 2 weeks, some three months before the onset of his symptoms. He never noticed any blood or mucus in his stools at any time.

F.T.M.:— showed marked hyperchlorhydria and excess total acid.

Ba. Meal:— "Irregularity of duodenum and rapid emptying of stomach - duodenal ulcer."

He was treated with Hurst's gastric régime for a month,
Fractional Test Read in Tannery
No. 255-71-63 Perrier Guenter, Royal Signals.

Case 9.

Slight Fever - Short except for Pusings.

Left Arm

Activity 100% best read in Second of C to Hke.

No. 1 0.5 1 2 3 4 5 6 7 8 9

Content:

- Free Water
- Free Alkali
- Total Alkali
- Total Organic
- Free Alkali
- Total Alkali

Activity:

1. 16 + - - - + 23 34 -
2. 14 + - - - + 44 87 -
3. 20 + - - - + 70 54 -
4. 15 + 2 - - + 90 110 -
5. 6 + Jr. - - + 100 116 -
6. 6 + Jr. - - + 80 76 -
7. 6 + - - - + 56 64 -
8. 7 + - Jr. - + 38 14 -
9. 7 + - Jr. - + 26 44 -

Elizabeth M. Granger
Copr. R.A.M.C. 17/118.

of Brigade Laboratory 1942.
GASTRO-INTESTINAL ANALYSIS

Patient's Name

Fasting Juice

One-Hour Juice

Twelve-Hour Test Meal

Date

Volume

Food

Mucus

Blood

Free HCl

EXCESS

The stippled area indicates the limits for free HCl of 80% of normal people.

Summary

The stippled area indicates the limits for free HCl of 80% of normal people.

Volume

Food

Mucus

Blood

Free HCl

Doctor

Patients Name

2/2/43

CISTRO-INTESTINAL ANALYSIS
at the end of which he still complained of upper abdominal discomfort after meals, and pain in the back. 2 weeks later he was reported as still having pain in the upper abdomen and right flank, with tenderness in the epigastric region and some rigidity of the R. Rectus. The stools were reported as being normal.

On being transferred to our hospital the F.T.M. and Ba. Meal were repeated and both were normal. Vegetative E.H. were found in the stools.

This history suggests a peptic ulcer, and the first F.T.M. supports the diagnosis. The X-Ray report is quite inconclusive. The unusual frequency of vomiting may have been related to a coexistent gastritis.

In our hospital he was proved to have Amoebiasis. The F.T.M. and Ba. Meal were normal, and since his symptoms did follow an attack of diarrhoea, and the presence of an amoebic infection was proved, it is not unreasonable to suggest that the whole picture was due to a chronic amoebic infection and that the dyspeptic symptoms were reflex in nature.

CASE 10.

F.H.: - No member of family troubled with peptic ulcer.

History. He complained of pain in the epigastrium immediately after food, with nausea, but no vomiting, continuously for 3 yrs. One year ago in Burma he passed blood for a week per rectum. His motions were loose at that time. 3 months before admission to hospital he reported sick
with diarrhoea 5-6 times in 24 hrs., and pains in the epigastrium. He passed mucus but no blood. 2 weeks before admission his symptoms became worse, and for the last 2 or 3 days he had nausea and vomiting after each meal, with epigastric pain 5 mins. p.c., lasting 10 mins., which nothing relieved. He also had discomfort, belching and flatulence with some sharp pains in the epigastrium, which bore no relation to food. He had lost 20 lbs. in weight.

F.T.M.: - Low total and free H.Cl. Achlorhydria from 1½ hrs on. Traces of blood first 3 specimens (no significance.) Some mucus in most specimens.


In our hospital he passed many stools with blood and mucus in them. Laboratory examination revealed an indefinite exudate but no E.H. or cysts. Culture was negative.

He was tender in the mid upper epigastric region and also on inspiration below the L.C.M. The spleen was not palpable. The sigmoid colon was tender and thickened.

COMMENT. The patient has a 3 year history which does not suggest ulcer. There have been no remissions and nothing relieves the pain which comes on 5 mins. p.c. and lasts 10 mins. The bouts of sharp pain unrelated to food suggest colonic spasm. The physical signs and clinical
picture suggest chronic gastritis and a reflex dyspepsia following an attack of what was probably amoebic dysentery.

In face of the clinical picture I have no faith in the X-Ray report.

**Case 11.**
Gnr P. Aged 30. Service 2 8/12 yrs. India 1 yr.
Diagnosed D.U.

**History.**
This patient's history began with an attack of dysentery in Oct. 1942, when he was in hospital for 2 weeks, and apparently cleared up.

In Dec. 1942 he had spasms of pain in the upper epigastrium lasting 1 hr. These returned at weekly intervals until he found he couldn't eat anything heavy without having pain. In his unit he had to live on bread and jam. The pain gradually increased and lasted for longer periods. It came on immediately p.c. He was troubled with flatulence and acid eructations. When he took only a milk diet his symptoms improved.

Stools negative occult blood.

**Ba. Meal:** "1- Gastroptoses. 2- Gastritis. 3- D.U."

**Weight:** Lost 1st. 6 lbs in 7 months. 2 st. 2 lbs since he came to India.

On arrival at our hospital he complained of pains across the upper abdomen continuously, but aggravated by taking food. The pain was in the mid-epigastric region and below the R.C.M. particularly when he took a deep breath. He was vomiting back his feeds of milk. He had pains during the night, and a great deal of
GASTRO-INTESTINAL ANALYSIS

Patient's Name: Um.

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Date: 3/1/34

Doctor: Lieut. Colonel

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FRACTIONAL TEST MEAL

Date

Volume

Food

Mucus

Blood

Fasting Juice

---

SUMMARY

The stippled area indicates the limits for free HCl of 80% of normal people.

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TWO-HOUR

TWELVE-HOUR

Free HCl

Active HCl

Total Chloride

---

VOMIT

Volume

Hour

Food

Mucus

Blood

---

ONE-HOUR JUICE

---

ONE-HOUR JUICE

---

Fasting Juice

---

FREE HCl

---

FREE HCl

---

FREE HCl
flatulence. He also had pain over the 11th and 12th ribs behind, associated with hyperaesthesia of the overlying skin.

F.T.M. showed free H.Cl and total acid within normal limits. Blood present 1\(\frac{1}{2}\)-2\(\frac{3}{4}\) hrs. Mucus in nearly all the specimens.

Ba. Meal: "D.Cap appears normal. ? nearly healed ulcer on the lesser curvature of the stomach."

Repeated 10 days later "Stomach and duodenum N.A.D."

The day before he was boarded he developed loose stools with abdominal pains. His temp. rose to 102 deg. Blood appeared in the motions 4 days later.

Repeated examinations were made of these stools in the laboratory, but not until 6 days later were vegetative E.H. demonstrated. Stools, after that one positive, showed no Amoebae but Charcot Leyden crystals were reported to be present.

He was tender in the mid epigastric region below the R.C.M. and in the L.I.F. The Sigmoid Colon was palpably thickened.

Comment. This history is suggestive of a gastric ulcer high up on the lesser curvature, but not a D.U. He rapidly lost weight, the pain in the epigastrium appeared immediately p.c. and he had marked vomiting. The F.T.M. meals supports a G.U., blood being present in many specimens. Physical signs suggest some hepatic disturbance in the wide area of tenderness, its site, and the hyperaesthesia over the 11th and 12th ribs behind.
The second X-Ray, though doubtful, accords with the clinical history, but the physical signs suggest a more widespread disturbance no doubt associated with his Amoebic infection.

Pte A. Aged 40. Service 2 yrs. India 1 4/12 yrs.
Diagnosed D.U.

Family history - nil relevant.

For the past 12 years he had had pain in the centre of the epigastrium very soon after meals which was relieved by alkalis. There have been no remissions. On questioned he stated that he had diarrhoea for a few days at that time, but passed no blood. His stomach trouble became worse after arrival in India.

By April 1943, one year after his arrival in India, the pain was almost continuous, but was relieved temporarily by eating food. He had had an attack of acute bacillary dysentery with abdominal colic and blood and mucus in the stools just before this.

On admission to our hospital he complained of pain in the mid-epigastric region 2 hrs. p.c. This pain was much more severe than the old one he used to get very soon after meals. The pain was relieved by food and alkali.

The character of the pain varied with the movement of the bowels, being worse when he was constipated.

F.T.M.: - Achlorhydric type with acid appearing between $1\frac{1}{4}$-2 hrs.

Ba. Meal: - "Orthotonic stomach, mobile, empties well. No G.U. or filling defect. D.Cap deformed. Incisura on
lateral wall, tenderness on opposite border. No niche demonstrated. Appearances strongly suggest D.U."

He was tender in the centre of the epigastrium and along the whole descending colon. He became very much better on the gastric regime, although not completely free of symptoms.

The history is confusing. The epigastric pain shortly after p.c., relieved by alkalis, but lasting 12 years practically without remissions, does not suggest ulcer dyspepsia. That it may have been due to gastritis is more likely since his recent history is given as epigastric pain 2 hrs. after p.c.(the pain being more severe than the former one,) relieved by food and alkali. In this the ulcer picture is clearer, but he did have acute bacillary dysentery just before his dyspeptic symptoms became much worse, and though he improved on an ulcer regime, he did not become symptom free.

I am inclined to view this man as a long standing gastro-duodenitis, in which the added strain of colonic infection may have precipitated the ulceration to which he was prone, and is now responsible for the continuance of symptoms beyond the usual period on a strict ulcer regime.


History. This patient had had loose motions for 9 months off and on with blood and mucus in them frequently. He also complained of epigastric pain and pain in the R.I.F.
GASTRO-INTESTINAL ANALYSIS

Patient's Name 3597843 Sgt. PARTINGTON. I.A.O.C.

Case 13.

FRACTIONAL TEST-MEAL

Date

Fasting Juice

Volume
Cells

One-Hour Juice

Free HCl.
Active HCl.
Total Chloride

VOMIT

Date
Hour
Volume
Food
Mucus
Blood
Free HCl.

TWELVE-HOUR TEST MEAL

Date
Volume
Food
Mucus
Blood

The stippled area indicates the limits for free HCl. of 80% of normal people.

SUMMARY

indicates free HCl.
indicates total acidity.

associated with vomiting 2 hrs. p.c. and not relieved by food or alkalis.

He reported sick and was treated for gastritis. He took his own discharge from hospital before his stools had settled down. Later he again reported sick because he had lost such a lot of weight. This time he had his chest X-Rayed and was returned to duty when a negative result was obtained.

About 4 weeks before he was sent to us he was again admitted to hospital and there he developed a sore tongue and was unable to eat anything hot or spicy. Ba. Meal revealed an "ulcer high on the lesser curve of the stomach."

He was grossly under-weight. (lost 3 stones.)

Within a week of his arrival in our hospital, on an ulcer regime, his tongue had become red and raw and he was again unable to eat or drink anything hot. His stools were bulky, pale and greasy. He had diffuse tenderness in the epigastrium, but much more in the L.I.F., and to a lesser extent throughout the whole colon. The lower abdomen was distended.

F.T.M. almost complete achlorhydria.

Blood picture Normocytic Anaemia.

The ulcer regime was stopped and he was put on a high protein with restricted fat carbohydrate diet, and in addition given nicotinic acid and liver parenterally, and iron orally. His response was dramatic, and within
6 weeks he had regained 1½ stones, and was completely symptom free. Against our advice he took his own discharge because he felt so well.

The entire clinical picture and progress of the case is against the diagnosis of G.U., and this was confirmed by the appearance of the characteristic tongue and stools of sprue.

This case is typical of so many in this country, who go from hospital to hospital with diarrhoea. The stools are examined on many occasions without any E.H. or dysenteric organisms being found. On a restricted diet their diarrhoea eases up and they are discharged as fit. Eventually when they have lost a tremendous amount of weight (in one case as much as half his weight) they appear in their true colour, as cases of sprue.
ANALYSIS OF CASES.

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<th>Numbers:</th>
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<tr>
<td>Age: (Average)</td>
<td>31.8 yrs.</td>
<td>29.9 yrs.</td>
<td>27 yrs.</td>
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<tr>
<td>(Range)</td>
<td>23-44 yrs.</td>
<td>24-36 yrs.</td>
<td>25-29 yrs.</td>
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<tr>
<td>Average in Civilians</td>
<td>38 yrs.</td>
<td>45 yrs.</td>
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The proportion of D.U. to G.U. corresponds with the accepted figures in ordinary hospital practice at home. The age incidence is naturally lower because of the selected age groups in the Army.

The number of cases with an unusual history aroused the interest in them.

Of the total of 93 cases
12% seemed to be true cases of ulcer.
33% seemed to me to be definitely not cases of ulcer.
49% were indefinite and might have been one or the other, or a double pathology may have been present.

DUBIOUS POINTS.

1. The time relationship of pain to meals was odd. Even in cases in which the X-Ray reports showed ulcers high on the lesser curve, the pain often came on 1½-2 hrs. p.c.
2. Flatulence was such a prominent feature of so many cases.
3. The pain was so frequently not relieved by a strict gastric diet, and in others it did so but tardily.
4. The tremendous loss of weight occurring in many cases of D.U.
5. The frequency of the association of diarrhoea with the onset or exacerbation of symptoms was too high to be merely a coincidence.
Frank blood was often found in the stools, and in others a history of dysentery was given or obtained after questioning. 

Vegetative Entamoeba Hystolytica were found in the stools of 2 cases.

The finger-point tenderness of typical ulcer was not the usual finding. Most frequently there was a diffuse tenderness, and in many cases the caecum and/or the sigmoid colon were tender as well.

The sigmoid colon and/or the caecum were obviously palpably thickened in an unusually high proportion of cases.

The fractional test meals showed an unusual incidence of achlorhydrias, and only a very small number, (under 10%) showed the typical ulcer curve.

The Barium Meals showed:

a. An unusual incidence of ulcers high on the lesser curve.

b. Duodenal Ulcers diagnosed on radiological findings such as deformity of the D. Cap or "D. Cap does not visualise normally suggestive of D.U. " etc.

c. Many of the reports showed a lack of confidence and a few films showed arrows pointing to minor irregularities, which I was not prepared to accept as ulcers. (Film attached.)

d. In cases in which the Ba. Meal was repeated in our hospital the reports did not agree with those obtained elsewhere.

**DISCUSSION.**

From an examination of these 93 cases which came under my control, I am quite satisfied that the diagnosis of peptic ulcers was inaccurate in about 40% of cases.
It is an unfortunate fact that when the troops arriving in India comes with the idea that they are in a happy land, or everyone to have diarrhoea as a matter of course, and the result that when he has an attack himself, which may be a perfectly natural thing, and does not report sick unless it is intolerably bad; or he begins to feel weak. A proof of this attitude is that the patients with peptic ulcer cases practically never report themselves sick when they have had it for a considerable period.

The overeating and excessive diet, as well as living in an unhealthy climate, may cause development of all sorts of digestive disturbances, at times has, an acute dysentery condition with a slight attack of dysentery, and particularly typhoid fever. It is sometimes difficult to cure these patients, and they will have some radical trouble in the future.
In the Army the two commonest causes of dyspepsia are chronic gastritis and gastric neurosis, while next in frequency come the reflex dyspepsias. True peptic ulceration is comparatively rare as one would expect, since the personnel belong to such a distinct age group.

The two commonest diseases in India are Malaria and Dysentery, and it is logical to assume that a fair proportion of those who contract dysentery, and particularly Amoebic Dysentery, which is so notoriously difficult to cure, as distinct from relieve, will have some residual trouble in the colon.

It is an unfortunate fact that the British troops arriving in India comes with the idea that it is a natural thing for everyone to have diarrhoea in this country, with the result that when he has an attack himself, he looks on it as a perfectly natural thing, and does not report sick unless it is intolerably bad, or he begins to pass blood. A proof of this attitude is that these "peptic ulcer" cases practically never volunteered the information that they had been troubled with diarrhoea, even though it may have persisted over a considerable period. Unfortunately it is just those cases who do not receive adequate treatment early, who so frequently develop chronic dyspeptic symptoms later on.

In the Army, the conditions of living in certain circumstances, undoubtedly do predispose to the development of an ulcer in the susceptible individual. The food at times has, of necessity, to be taken at irregular hours. Night duties will upset the normal daily routine. The food may be indigestible, because fresh meat out here has frequently to be cooked far too soon after the animal has been killed, with the result that it is usually very "tough".
And above all the excessive smoking, which is practically universal throughout the Army, would, in the individual with the hypersthenic gastric diathesis, precipitate ulcer formation by exaggerating through the autonomic nervous system the already excessive motor and secretory activity of the stomach.

Of all the unusual features which have made me doubt the diagnosis in many of these cases, the most striking has been the history. It must be more than a coincidence that so many of the cases in the age group 20-30, date the onset of their dypeptic symptoms from a varying period after an attack of diarrhoea or actual dysentery. It might be argued that the attack of diarrhoea or dysentery was the precipitating factor of the ulcer, but again the histories do not support this view. In many of the cases it was a discomfort and not a pain they complained of, and when there was pain, it was frequently diffuse, and often in more than one place. The usual exact localisation in cases of D.U. to a small area about 1" to the right of the mid-line, midway between the Xiphisternum and the Umbilicus, was only found in the small percentage of cases with the clinical picture of D.U. Those who gave a history of pain high in the epigastrium 1½-3 hrs. p.c., and in whom X-ray revealed a gastric ulcer, seem to me to give one of the typical histories of cases of gastric neurosis.

I have certainly never before seen a case of ulcer near the cardia, which did not have pain almost immediately after food.

Except in the cases which were accepted as true ulcers, there was no supporting evidence in the family history.

Another striking feature in the history of the cases of duodenal ulcer, which was unusual, was the gross loss of weight,
in some cases running into stones. This is such a common finding in the dysenteries, that it does support the view that these people were really suffering from a chronic colonic condition.

Finally, the failure to relieve the symptoms completely after several, sometimes prolonged, courses of strict ulcer treatment, points to the presence of another pathology.

In the physical signs the diffuse tenderness was unlike that usually found in ulcer cases. The unusual frequency in which the sigmoid colon and caecum were palpable was certainly not coincidence, as a check was made in one of the other medical wards of the hospital, and the cases there did not have palpable colons.

Fractional test meals are generally considered to be of little value in the diagnosis of peptic ulcer unless the typical climbing curve with hyperchlorhydria suggesting D.U. is obtained. It is however, more usual to accept achlorhydria as a supporting finding in the diagnosis of gastritis and not of ulcer, though of course both may co-exist. (Approximately 10% of the cases showed an achlorhydria, while the vast majority were within normal limits.)

The reports of the Barium Meals were quite unconvincing in many cases. Unless a definite ulcer niche which holds barium over a period, is demonstrated, the diagnosis of peptic ulcer by X-Ray seems to me to be uncertain. Certainly a mere deformity of the duodenal cap or a duodenal cap not visualising normally, does not of itself indicate a duodenal ulcer. A competent radiologist assured me that the appearances of the duodenum in the film submitted (diagnosed D.U.) were those usually found in stasis
of the duodenum which occurs in many cases of reflex dysfunction. Also any ulcer high on the lesser curvature of the stomach, as was demonstrated quite frequently, will not produce pain 2 hrs. after a meal is taken. In this connection it may be well to remember that most competent authorities report about 5% of peptic ulcers over a large series of consecutive post mortems.

In conclusion I would say that there are four interpretations of the 39% of cases which I do not believe to have been cases of ulcer, and also of the 49% doubtful cases:—

1. That there was an aggravation of a pre-existing gastritis by an attack of diarrhoea or dysentery.

2. That a toxic gastritis was produced by an attack of diarrhoea or dysentery, which was kept active by a chronic low grade colonic infection.

3. That there was a double pathology ulcer and colitis.

4. That the symptoms and signs were reflex in origin from a chronic low grade infection of the colon.

The last of these seems to me to be the most likely explanation.

It is unfortunate that further investigations were not completed, which, I feel, would have established my contention. Permission had just been granted to repeat the Ba. Meals and F.T.M’s, and, in addition, to have routine investigations of all stools for E.H. and occult blood, followed by sigmoidoscopy and a Ba. enema, when I was posted away from the hospital.

SUMMARY.

Cases diagnosed as gastric or duodenal ulcer have been described
and various points which suggest that the true pathology has not been recognised, have been mentioned.

The opinion is expressed that about 40% were reflex dyspepsias from chronic colonic infection.

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My thanks are due to Col. G. Moulson, Late R.A.M.C., the A.W. Commanding Officer, and Lt. Col. Leishman, R.A.M.C., the Officer Commanding the Medical Division of the hospital in which the cases were examined.
WATER EXCRETION IN THE TOXEMIAS OF PREGNANCY.

INTRODUCTION.

Many and varied have been the theories advanced to explain the toxemias of late pregnancy. Of these the hypothesis advanced by ZANGMEISTER in 1915 (Z. Geburtsh Gynak 78, 325), that retention of water in the body was responsible, led me to carry out water diuresis tests to see if the ability of the kidney to excrete water was in all cases impaired. It is well recognised that albuminuria and oliguria are often early signs of the toxemias of pregnancy.

Zangemeister had said that this water retention was due to a diminished urinary output caused by a toxin which produced an abnormal permeability of the capillaries, which he believed produced an oedema of the brain rendering it anaemic. The brain cells as a result became under-nourished, which in turn produced a greater irritability of the cells, leading finally to the convulsions of eclampsia. The rise in blood pressure he said was due to increased intracranial pressure, and the oedema of the kidney led to an ischaemic albuminuria and oliguria by causing swelling of the kidney within its rigid capsule.

TYPE OF CASE ON WHICH TEST WAS CARRIED OUT.

The test was carried out on fifty consecutive primiparae admitted to the antenatal wards of St. Mary Abbots Hospital, suffering from the toxemias of late pregnancy. All of these patients had been regular attenders at the ante-natal clinics from the twentieth week of pregnancy at the latest, and so the presence of essential hypertension or chronic nephritis would
have been recognised at an earlier date. It was a routine of the hospital that all patients with a blood pressure over 140 m.m. Hg (systolic) after 15 minutes rest, were admitted.

**CONTROLS.**

In addition, as a control, the same test was carried out on ten patients in the late stages of a normal pregnancy, and on ten non-gravid women admitted for conditions such as bronchitis, but in whom there was no impairment of renal function or rise of blood pressure.

**ROUTINE TEST OF WATER EXCRETION.**

In the routine test of water excretion the patient drinks 1200-1500 c.c.s of water within half an hour. A normal kidney will excrete the same amount within the next four hours, the larger part being excreted in the first two hours. If water elimination is impaired, only a small part, sometimes less than 200 c.c.s will be excreted in four hours.

**TEST EMPLOYED IN THE EXPERIMENT.**

The test consisted of giving a litre of water (four tumblerfuls) to the patient to drink as quickly as she could. This took from three to six minutes. A sterile rubber catheter was passed and the bladder emptied when all the water had been drunk. This urine was discarded. A Spencer Wells was applied to the end of the catheter, which was left in position. The bladder was then emptied every fifteen minutes for three hours, unless the 1000 c.c.s. was excreted before that time, in which case the test was stopped. The quantity excreted was measured each time. Blood pressure readings were taken every thirty minutes. No food was allowed to be eaten or any fluids - apart from the litre of water - to
be drunk from 10 p.m. on the night preceding the test, until it was completed. The patients were all kept in the recumbent position throughout the test. To assist in the emptying of the bladder, the hand was placed on the abdomen just above the symphysis pubis and pressure applied from there on to the bladder.

SPECIMEN ABSTRACTS FROM CASE RECORDS:

Mrs. H. Aged 23. Para 0., 32 weeks pregnant; R.O.A. General condition good; oedema of both feet and legs; cramps in the hands; Albuminuria and oedema increasing and blood pressure rising 12 days before admission. B.P. 144/98. Urine albumen 0.25 per cent. Blood Urea 36 mgms. per cent. Urea Concentration Test. 7 a.m. 3.20. 9 a.m. 1.1.

Water Excretion Test. 925 c.c.s in three hours.
One month later the oedema had increased and blood pressure risen to 174/126. Albumen in urine 1.5 per cent.
At this time the water excretion test was repeated and 398 c.c.s obtained in three hours.
On both occasions the patient took six minutes to drink the fluid and on each occasion there was a fall of blood pressure of 10 m.m. Hg at the end of the test.

Mrs. D. Aged 28; Para O; 30 weeks pregnant; L.O.A.; Varicose veins left leg; oedema of both legs; general condition very good; occasional vomiting; occasional pain in the epigastrium and heart-burn, occasional cramps. Urine Albumen nil; Blood Urea 28 mgm. per cent. Urea Concentration Test. 7 a.m. 1.54. 9 a.m. 2.46. Blood Pressure 144/98.
Water Excretion Test. 1280 c.c.s in three hours.
There was no change in the blood pressure during the test.
The patient took three minutes to drink the fluid.

Mrs. L.; aged 21; Para 0; 36 weeks pregnant; L.O.A. General condition good; frequent cramps in hands and legs; Blood Pressure 176/120; Urine Albumen negative; Blood urea 26 mgms per cent.

Urea Concentration Test 7 a.m. 1.8. 9 a.m. 1.6.

Water Excretion Test 648 c.c.s in three hours.
The blood pressure fell from 180/132 at the beginning of the test to 164/104 at the end.
The patient took six minutes to drink the fluid.
### Specimen Results (Tables and Graphs)

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<th>Mph 3</th>
<th>Mph 4</th>
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<th>Mph 5</th>
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**Total Exercised** 925 398 1023 1093 1280 814 648 198 278 1030 713 105
occurred in the cases examined.

13. One patient, on whom the test was repeated after her condition had deteriorated and blood pressure risen considerably, showed a marked decrease in the excretion rate, though on the first occasion it had been practically normal.

1st Test. Total excreted was 925 c.c.s in 3 hours.
2nd Test. Total excreted was 398 c.c.s in 3 hours.

CONCLUSIONS:

The experimental findings support the hypothesis that in the toxemias of pregnancy there is a retention of water in the body. They do not however, support the view that the increased blood pressure is due to oedema of the brain, as in the experiments there was usually a fall and never a rise in blood pressure following the sudden intake of 1000 c.c.s of water, nor did any signs or symptoms of increased intracranial pressure occur.

The findings would support the hypothesis advanced by Hoffman and Anselmino in 1931 (Archiv. Gynack 147, 652) that the posterior pituitary gland produced an excess of two of its secretions, the antidiuretic hormone causing oedema, and the pressor substance causing hypertension.

The results indicated that there was a definite relationship between the blood pressure and the urinary excretion rate in the toxemias of pregnancy, the higher the blood pressure the lower the excretion rate.

Purposely no attempt has been made to give a lengthy description and discussion of all the work done on the subject. The object of the investigation was simply to find out if the
rate of excretion of water by the kidney in the toxemias of pregnancy was reduced. This has been confirmed.

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My thanks are due to Mr. G. W. Theobald F.R.C.S., F.R.C.O.G., M.R.C.P. for the suggestion and the use of his patients to carry out this investigation.
RESULTS:

1. The excretion rate in the toxemias of pregnancy was in the vast majority of cases below that in a normal pregnancy.
2. The excretion rate during the later stages of a normal pregnancy was slower than in the non-gravid woman.
3. The average amount excreted in the toxemias of pregnancy in three hours was 713 c.c.s.
4. The average amount excreted in a normal pregnancy (late stages) in 2½ hours was 1030 c.c.s.
5. The average amount excreted in the non-gravid female in 2 hours was 1077 c.c.s.
6. The maximum amount excreted in 3 hours in the cases of toxemia of pregnancy was 1280 c.c.s. and the minimum 198 c.c.s.
7. The curve of excretion in the toxemias of pregnancy was much flatter than in the non-gravid woman.
8. In the cases of normal pregnancy and in the non-gravid female the maximum excretion was between 60 and 75 minutes. In the cases of toxemia of pregnancy it was between 45 and 60 minutes.
9. No rise of blood pressure accompanied the sudden intake of 1000 c.c.s of water. In most cases there was a fall often as much as 10 to 20 m.m. Hg.
10. Oedema of the legs alone was not associated with a fall in the excretion rate.
11. The higher the initial blood pressure the lower was the excretion rate.
12. No sign or symptom of increased intracranial pressure