SIMPLE MORBID ANATOMICAL STUDIES OF THE HUMAN STOMACH

- AN ANALYSIS OF 78 CASES.

by

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Being a Thesis submitted for the Degree of
Doctor of Medicine in the University of Edinburgh.

C O N T E N T S

INTRODUCTION - Aims and methods.

A vast literature exists on the subject of gastric pathology but we are still ignorant of the true incidence and severity of "gastritis" among the general population. For obvious practical reasons it is impossible to derive direct histological proof of the existence or absence of gastritis among the general population during life. However, valuable information concerning the state of the gastric mucosa can easily be obtained if stomachs are fixed immediately after death.

Proper fixation is essential for accurate results. Magnus (1937) re-stressed the necessity of this. He demonstrated that fixation by intraperitoneal injection, as first practised by Faber 50 years ago, is inadequate, but that direct intragastric injection of fixative within ½ to ¾ hour after death gives excellent results.

Magnus showed that antolysis usually commences within ½ to ¾ hour after death in adult stomachs though much later in foetal stomachs.

The simple technique of direct intragastric fixation requires the minimum of skill and material. It is very surprising that it has been so much neglected. So far, no-one has examined a large series of stomachs

adequately fixed immediately after death.

My series of 62 adult and 10 foetal stomachs, all formalin-fixed, according to Magnus' rulings, represents the crude attempt of one individual to assess certain gastric lesions. As far as I am aware, it represents the largest series of stomachs fixed by the intra-gastric method immediately after death.

Ideally, in the investigation of abnormalities of gastric structure among the general population, only cases of sudden death should be chosen, for, in theory, many unselected post-mortem stomachs would be expected to show appreciable neo-mortal change. The selection of cases only of sudden death, however, would be a very considerable task and, as the largest number of post-mortem stomachs fixed immediately after death is 32 (Magnus' own series) I thought it would be interesting to see the state of health or disease of a larger series of stomachs selected more or less at random.

My primary aim was to assess the pathological changes in a more or less heterogeneous series of stomachs, i.e. "gastritis", erosions, ulcer, etc., and to attempt some correlation between the age and general pathological condition of the subject and the state of his stomach.

As a secondary aim, some special histological details have been investigated, these being more of academic than practical interest.

In addition to the 72 post-mortem stomachs, 4 gastrectomy specimens were fixed in formal saline immediately after their removal from the body and later compared with the autopsy specimens.

My heartfelt thanks is due to the large number of the medical staff of the Central Middlesex Hospital, London, who helped me, especially to the sisters and nurses, Dr. F. Avery Jones for his constant encouragement, Drs. J. D. Allan Gray and Pagel for unfailing material assistance and to Dr. George Discombe for statistical help. Also to Prof. H. A. Magnus of King's College Hospital for his opinions.

TECHNICAL PROCEDURES.

Fixation.

All the stomachs were fixed with 10% formol saline. The time of fixation and the quantity of fixative used differed according to the age of the stomach as follows:-

Adult Stomachs were fixed within ½ to ¾ hour after death. An ordinary oesophageal tube, permanently strengthened with a length of strong, pliable wire, was passed over the tongue and along the posterior pharangeal wall into the stomach. One and a half to two pints of fixative were quickly poured down the tube through a large glass funnel.

There is little difficulty in passing the tube into the stomach. The error of passing it into the trachea is one that sometimes happens but can be avoided by taking care and not hurrying. The sensation of the tube within the oesophagus is quickly appreciated. Confirmation of correct filling can be got by prodding the anterior abdominal wall with the fingers and eliciting a "stomach splash".

If much difficulty arises in attempting to pass the tube, the stomach may be filled by the

alternative method of passing a large bore needle into the stomach through the 7th or 8th left intercostal space or through the epigastrium. This method, however, is uncertain in its results.

Child and Infant Stomachs. For these an appropriately smaller quantity of fixative was passed into the stomach through either a No. 4 Jacques stomach tube or a wide-bore needle.

These small stomachs are not always easy to fill with fixative.

Foetal Stomachs. Antolysis occurs in these much later than in adult stomachs. Nevertheless, it is advisable to fix with formalin within 2 to 3 hours of death for the sake of uniformity. The stomachs of large foetuses were filled by direct intubation or else by needling. Those of small foetuses were removed from the abdomen and immediately immersed in formol saline.

Macroscopic Examination.

At autopsy the oesophagus, stomach and duodenum were removed as one piece. The stomachs were opened longitudinally by an incision in the anterior wall midway between the curvatures. The mucosal surface was gently washed with a stream of water and then carefully examined, first with the

naked-eye, then with the aid of a hand lens. The specimens were then carefully kept wholly immersed in a bath of formol saline for seven to ten days before pieces were taken for section.

Representative pieces, 2 to 3 cm. long, were cut from the wall in 5 places: (a) the oesophago-gastric junction, (b) the fundus, (c) the body at the lesser curvature, (d) the antrum and (e) the pyloro-duodenal junction. They were fixed in formol-saline for a further 48 hours. They were afterwards washed, dehydrated, cleared, embedded in paraffin and stained with haematoxylin and eosin.

If a stomach presented unusual features, more pieces than the standard five pieces were removed for section and staining.

Ideally, special staining methods should have been used for the fullest examination of the cut sections, e.g. special stains for mucin, collagen, argentaffine cells, Paneth cells, etc., but as I was obliged to do all my own cutting and staining only the simplest procedures were used; but for most purposes these were entirely adequate.

GENERAL PATHOLOGICAL FINDINGS.

Individual cases are described in the appendix.

The stomachs were carefully examined macroscopically and microscopically. The condition of each stomach, considered as a whole, was assessed, and then each stomach was placed in one of 5 grades:-

Grade 1 : normal or almost normal.
" 2 : mild deviation from the normal.
" 3 : moderately severe deviation from the normal.
" 4 : severe deviation from the normal.
" 5 : very severe deviation from the normal.

The assessment depended principally upon the general condition of the mucosa and principally upon the severity of so-called "gastritis".

Normal Mucosa.

Histologists are more or less agreed upon the glandular structure of the mucosa but there is still some confusion concerning the cellular components of the inter-glandular connective tissue. Maximow and Bloom (1948) define the cells of the

lamina propria as being: (a) fibroblasts or reticular cells, (b) numerous small lymphocytes and some plasma cells, eosinophil lencocytes and mast cells, (c) sometimes Russell body cells. They admit that the cells have not been satisfactorily investigated. In Schaffer's Essentials of Histology (14th Edition) the normal mucosa is described as consisting of glands and reticular tissue, the latter containing many leucocytes and basiphil connective tissue cells in its meshes. The description of the mucosa in Gray's Anatomy (28th Edition) is simpler: the glands are embedded in mixed fibrillar and reticular connective tissue containing lymphoid cells.

Bloch, Faber, Lange and Wimtrup investigated 30 formalin-fixed stomachs of newborn infants and described the normal appearance of the stomach. They found no interstitial cell infiltration and also that the same picture was present in many older children and adults. They described the normal stomach as showing only a fine network of fibrillary threads and a few scattered plasma cells or lymphocytes. Other investigators, such as Saltzmann and Orator, later arrived at the same conclusions.

Magnus (1937) as a result of studying formalin-fixed foetal and normal adult stomachs

defined the connective tissue of the stomach mucosa as a delicate network of collagenous and reticulum fibres in which there were sparse cells: occasional fibroblasts and reticulum cells and scattered lymphocytes and eosinophil leucocytes. Plasma cells occurred very infrequently and polymorphs were not found in the foetal stomachs, although they were seen very occasionally in 2 normal adult stomachs. Russell body cells were never found in the normal material.

My findings on normal stomachs, foetal and post-natal, confirm Magnus' descriptions except: (1) that no plasma cells were ever found in foetal or early post-natal life stomachs and (2) I would consider more plasma cells to be present in the normal mucosa of adults.

Pathological Elements.

The principal ones seen in the various stomachs under review were the following:-

Congestion; oedema; fibrinous exudate; haemorrhage; inflammatory cell infiltrations .... neutrophils, plasma-cells, lymphocytes, eosinophils, monocytes, Russell cells; degeneration, including cloudy swelling, fatty degeneration,

hyaline degeneration and hydropic degeneration; atrophy; cyst-formation; hyperplasia, innocent or malignant; necrosis; erosion; ulceration; fibrosis; amyloidosis; coccal or tuberculous infection; secondary carcinomatosis.

Normally the covering epithelium of the stomach mucosa and the glandular epithelium are clearly defined. Swelling, cloudiness and vacuolation are early signs of disease. With further disease these cells became increasingly opaque and shrunken and finally disappear altogether.

Acute inflammation in the mucosa is characterized by vascular congestion and oedema and neutrophil migration. The neutrophils, often in great number, stream into the mucosa and some into the lumen of the stomach. Often they are seen within small or large vacuoles in or between gland cells.

The commonest mucosal lesion found was that of "chronic gastritis" ..... an infiltration of the mucosa with inflammatory cells, accompanied by variable vascular congestion and oedema and variable epithelial degeneration and atrophy. The

most numerous inflammatory cell was the plasma cell, although lymphocytes were sometimes very numerous also. It is a popular idea that lymphocytes are the predominant cells in chronic gastritis but plasma cells are usually more plentiful in this condition.

Sometimes plasma cells degenerate to give rise to the conspicuous Russell body cells.

Variable numbers of polymorphs are always found in chronic gastritis.

"Intestinalization" of the gastric mucosa, viz. the appearance of goblet cells and Paneth cells, is characteristic of the severe grades of chronic gastritis.

A mucosa very severely affected by chronic gastritis shows great devastation. There is considerate glandular atrophy and there is frequently enormous stuffing of the connective tissue with plasma cells and lymphocytes. Goblet cells are often very numerous.

No case of diffuse non-inflammatory atrophy of the gastric mucosa, corresponding to Lubarsch's diffuse atrophic pan-gastritis, was found. Magnus described six such cases. Of these two were cases of pernicious anaemia and one a case of carcinoma ventriculi.

A characteristic of chronic gastritis is its patchy quality. Although the antro-pyloric part of the stomach was commonly diseased there was no obvious predilection for this region.

The severity of inflammatory lesions in the immediate vicinity of ulcers as contrasted with the mildness of such lesions in the mucosa a little more distant from the ulcers was not infrequently observed, suggesting that the inflammation was secondary to the ulcer and not vice-versa.

Grades of Stomachs, arranged in age-groups. The stomach grade in brackets is multiplied by the number of stomachs in that grade:-

(a) 72 autopsy stomachs:-

10 and under (19 stomachs):-

10 foetal ..... x (1)

Of the remaining 9:-

4 x (1)
4 x (2)
1 x (3)

11 - 20 : 1 x (1)

21 - 30 : 2 x (3)

31 - 40 : 1 x (1)
1 x (2)

31 - 40 (cont'd)

1 x (3)
1 x (4)

41 - 50 :

1 x (1)
1 x (2)
3 x (3)

51 - 60 :

5 x (3)
6 x (4)

61 - 70 :

3 x (2)
5 x (3)
1 x (4)

71 - 80:

2 x (1)
3 x (2)
2 x (3)
8 x (4)
1 x (5)

81 - 90 :

2 x (2)
1 x (4)

91 - 100 :

2 x (3)

FIG I. GRADES OF GASTRIC LESION ··· DISTRIBUTION PER AGE GROUP & NO. STOMACHS PER GROUP.

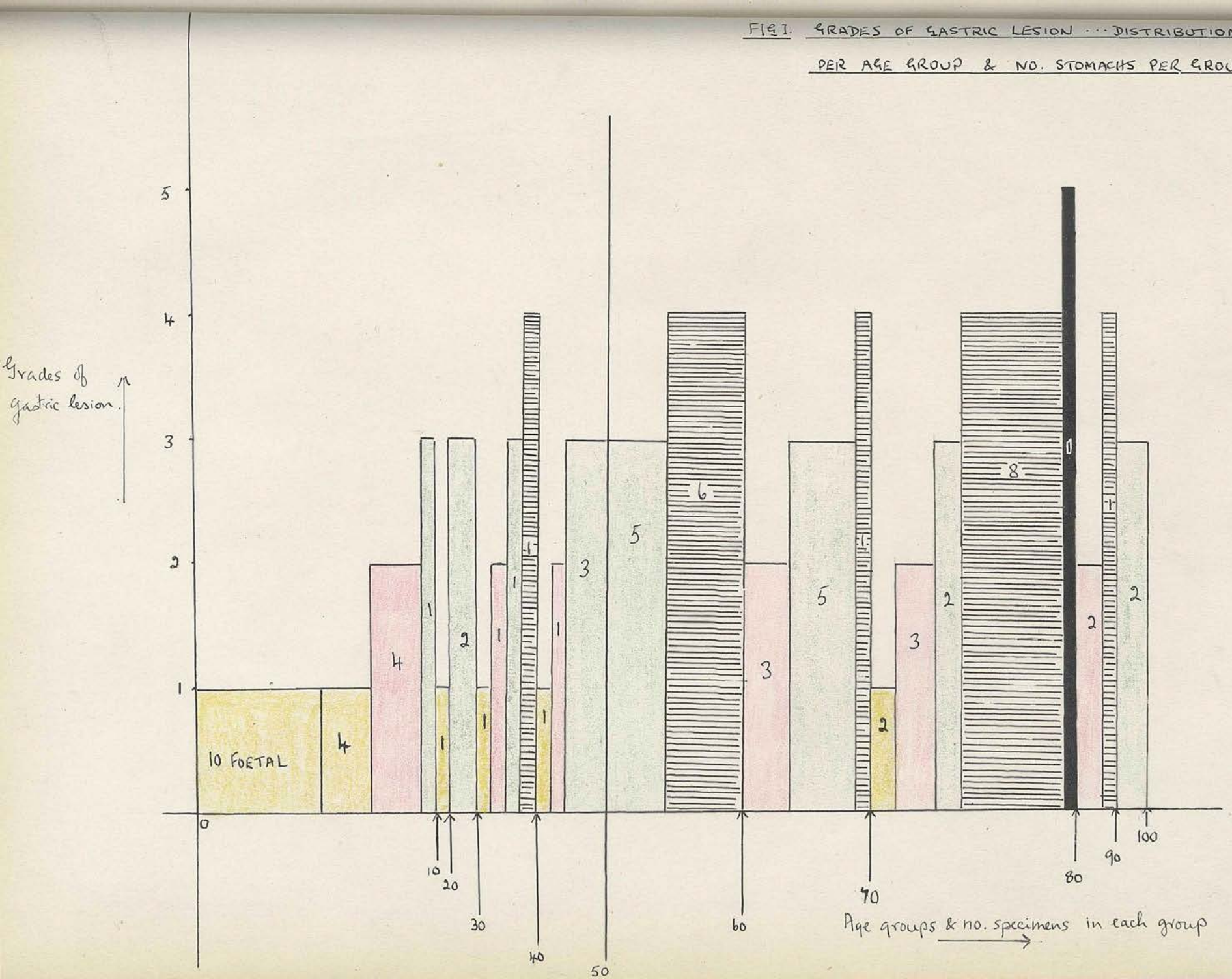
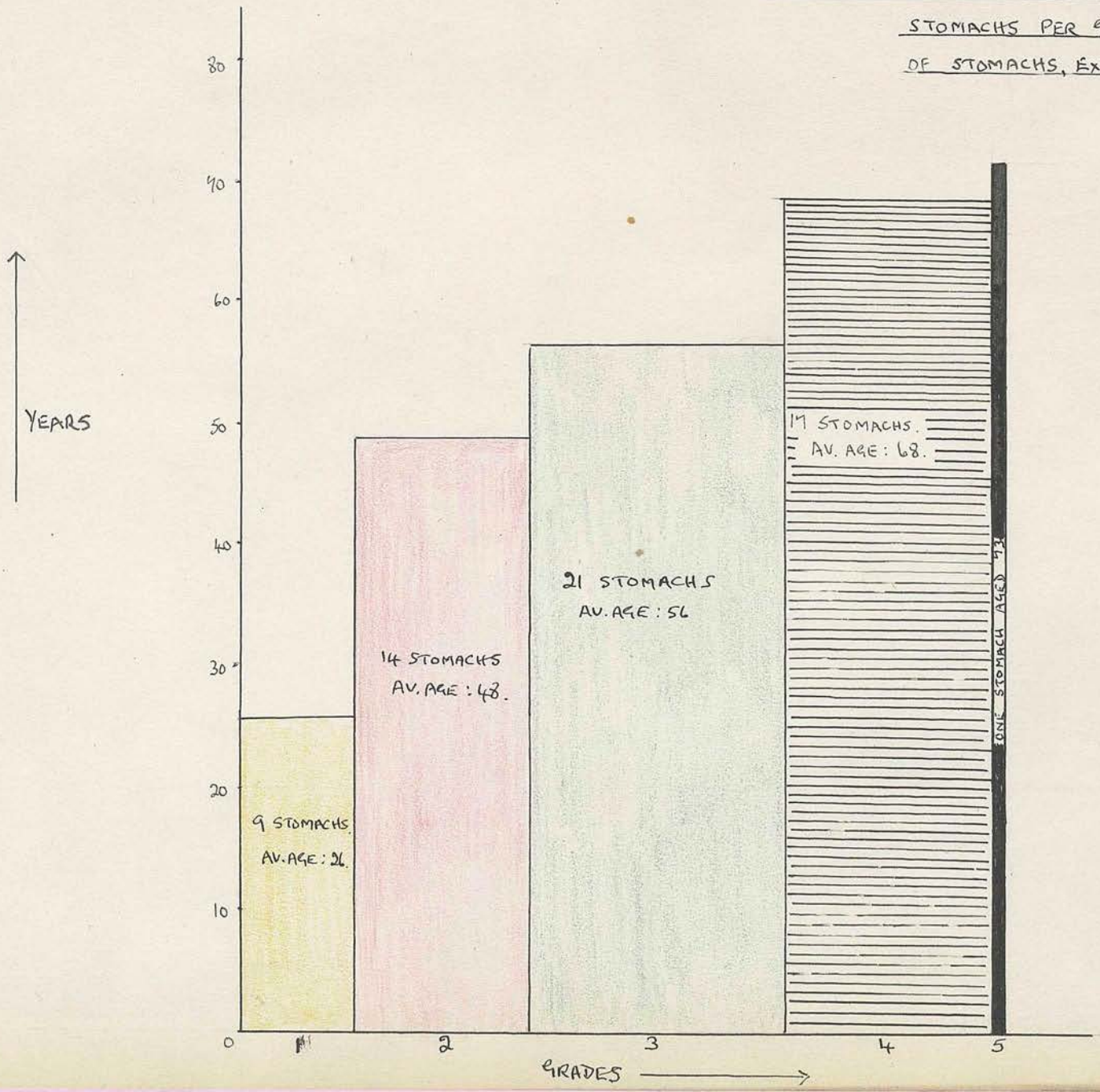


FIG. 2. GRADES OF GASTRIC LESION – NUMBER OF STOMACHS PER GRADE AND AVERAGE AGE OF STOMACHS, EXCLUSIVE OF FOETAL STOMACHS



(b) 4 gastrectomy specimens:-

31 - 40 : 1 x (2)

41 - 50 : 1 x (4)

51 - 60 :

1 x (3) }
1 x (4) }.

Fig. 1. illustrates the distribution of grades of stomach per age group and the number of stomachs per age group.

Fig. 2. illustrates the relationship between the age of the stomach and its grade.

Inspection shows that the severity of the histological lesions tends to increase with age. By applying the Chi square statistical test (the "exact X^2 method"), it can be demonstrated that the odds are 100 to 1 that histological grading does depend on age. Eliminating foetal stomachs, the odds are about 50 to 1.

But the statement "the older a stomach the more unhealthy it is" is a statement of a tendency not an absolute certainty. Five out of sixteen stomachs in the 71-80 years group and two of the three stomachs in the 81 - 90 years group were relatively remarkably healthy.

There is definite proof that with increasing age there is a diminished power of

gastric secretion (Bloomfield and Polland; Davies and James). After the age of 40 the production of HCl and pepsin by the stomach of both sexes decreases. Above the age of 60 about 30% have histamine-fast achlorhydria. But no unchallengeable evidence concerning the effect of senescence on the morphology of the stomach exists (Ivy 1942).

The examination of the present series of 72 autopsy stomachs revealed no obvious cause-and-effect relationship between age per se and disease per se. Atrophy without inflammation, for example, was not seen, nor was there any outstanding increase in atrophy of the stomach with age. It seems that time in relation to mucosal atrophy is important mainly in so far as exposure of the body as a whole to disease-producing agents is concerned, though there may be exceptions to this statement.

NON-GASTRIC DISEASES AND THEIR RELATION TO CHRONIC GASTRITIS.

Many agents are said to be capable of producing gastritis. They may be summarized briefly as follows:-

1. Exogenous agents: mechanical and chemical irritants.
2. Endogenous agents:-
   - (a) Infections.
   - (b) Hyperchlorhydria.
   - (c) Metabolic substances e.g. in uraemia and in burns.
   - (d) Avitaminoses, e.g. sprue.
   - (e) Venous stasis. e.g. in heart failure.
   - (f) ? nervous stimulation of the stomach.

Attention will be focussed on the influence of infections and some reference will be made to other possible agents.

Hayem was the first to emphasise bacterial toxic gastritis. He observed gastritis in various acute infections and

confirmed his belief by injecting tuberculin and diphtheria toxin into animals.

Many other workers have correlated infection with gastritis ..... Beaumont observed the effect of infectious illness on the stomach of Alexis St. Martin, Chauffard noted gastritis in typhoid fever, Dieulafoy in pneumonia and acute appendicitis, Wimtrup in influenza and Nyfeldt and Wimtrup in diphtheria.

Workers other than Hayem have produced experimental gastritis by the injection of bacterial substances ..... Enriques and Hallion, also Einar Thomsen, by injecting diphtheria toxin, Charrin by introducing P. pyocanea, etc. Glassner observed achylia in man following the injection of tuberculin, typhus and gonococcal vaccine.

"Metastatic" gastritis, due to bacteria themselves, has been observed in septicaemia, pneumonia, influenza, enteritis etc. It has been proved experimentally by Rosenow.

Kauffmann noted severe erosive gastritis in association with fixation abscesses.

Théohari and Babes showed that the parenteral injection of protein could produce severe parenchymal damage.

Konjetzny found severe acute gastritis in patients who had died of severe burns. He believed in the production of gastritis by the excretion through the gastric mucosa of protein metabolic products, especially in infections .... the more the inflammation, the more the gastritis.

Fenwick thought that uraemia produced gastric catarrh.

Faber and Holsti were of the same opinion. Many have described the acute ulcerative gastritis of uraemia.

An analysis of the 72 autopsy specimens revealed the following data:-

Group 1. Specimens.

19 stomachs, including 10 foetal and 5 adult stomachs. Of the 5 adults, 4 had mild to moderate terminal broncho-pneumonia. One showed, in addition, severe urinary infection, one fairly severe intestinal infection and one marked bronchiectasis and uraemia.

Arterio-sclerosis of any magnitude was found in only one of the five adult stomachs .... one of the cases with moderate infection.

The four infants, whose stomachs were in this group, had no infection at autopsy.

Group 2 - Specimens.

14 cases, 11 of which were more than 2 years of age.

There was infection in 10 cases, principally pulmonary in 7, principally urinary in 2, wholly peritoneal in one. Uraemia was present in only one case. Arteriosclerosis of any magnitude was found only in 2 stomachs.

Group 3 - Specimens.

21 cases, twenty of which were above the age of 2.

There was infection in 12 cases .... principally pulmonary in 7 (including 2 cases of tuberculosis), principally urinary in one; one case was an ulcerative carcinoma of the hepatic-flexure with a small paracolic abscess, another a tuberculous spine, and two cases were cases of acute gastro-enteritis.

There were 2 cases of uraemia, one of which showed infection also.

Arteriosclerosis was prominent in only 2 stomachs.

Of the cases without infection or uraemia there was a case of multilobular cirrhosis of the liver with death from oesophageal haemorrhage

a case of Hodgkin's disease with sudden death from cerebral haemorrhage and a case of status asthmaticus.

Group 4 - Specimens.

17 adults.

Infection was present in 8 cases, principally pulmonary in 6, principally urinary in 2. Uraemia was present in 5 cases, accompanied in 4 of these by infection. Arteriosclerosis of moderate severity was present in 3 stomachs.

Two of the cases in this group were cases of suspected nutritional deficiency.

Group 5 - Specimen.

The patient was a hypertensive with senile cerebral changes and a fractured femur. There was no mention of infection in the autopsy report but this cannot be excluded and was, in fact, probable.

Summary of the extra-gastric infections:-

Group.	Principally Chronic.	Principally Acute or Subacute.
1	3 cases	2 cases.
2	6 "	4 "
3	8 "	4 "
4	7 "	1 case.
5	? 1 case.	-
	Total: 24 or 25 cases.	Total: 11 cases.

These figures represent infections proved before or after death.

The overall incidence of infection is probably somewhat higher than this, as occasionally the autopsy examinations were necessarily limited.

Thus infection was established in roughly one half of the 72 cases, or excluding the 10 foetal stomachs in about 60% cases.

The infections were mostly mixed chronic and acute infections. Chronic infection was twice as common as acute infection and of the chronic infections pride of place went to bronchiectasis and pyelonephritis.

Whatever the importance of extra-gastric infection in the pathogenesis of gastritis, it cannot be the only important factor as it is not present to a significant degree in all cases of severe gastritis yet may be present to a marked degree in some cases of mild gastritis or in association with anatomically - normal stomachs.

Uraemia was present in 9 cases, but of these extra-gastric infection was present in 6. It was almost invariably accompanied by gastritis,

sometimes severe chronic gastritis.

Arterio-sclerosis and avitaminosis did not appear to have any powerful influence in the production of gastritis.

ULCERS.

10 of the cases showed one or more ulcers in the stomach, i.e. 15% of the adults.

Ulcer.	No. of stomachs.	No of ulcers in each stomach.
Acute	4	3 1 3 1
Subacute	2	1 1
Chronic	5	1 1 1 3 1

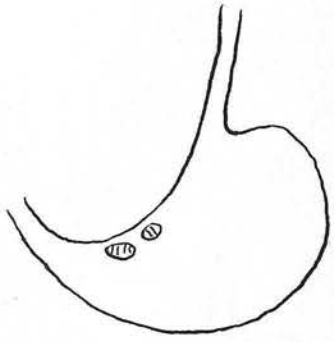
The average age of the patients was 67! In no instance had the ulcer been diagnosed during life. Six of the stomachs showed severe gastritis. Three of them showed moderately severe gastritis. Carcinoma ventriculi was present in one case.

Seven cases had extra-gastric infection. The single remaining case showed no extra-gastric infection.

Gastritis was frequently of a patchy character and, with the exception of the mucosa immediately around the ulcer crater, the mucosa around the ulcer was not infrequently milder in severity than that distant from the ulcer, suggesting that the inflammation was secondary to the ulcer and not vice-versa.

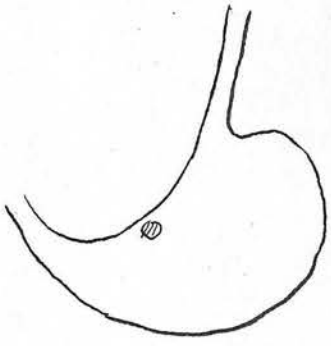
A brief description of the individual ulcer cases follows:-

Female, 36.



Linitis plastica. Two deep acute carcinomatous ulcers on the lesser curvature in the lower body and antrum respectively. The diameters were 1.5 c.m. in one ulcer, 3 c.m. in the other. Severe chronic gastritis.

Male, 76.



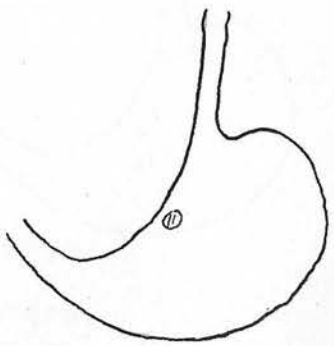
Carcinoma of bladder. Death from uraemia. Very severe cystitis and pyelonephritis. Purulent bronchitis and early brocho-pneumonia. One subacute or early chronic ulcer, 2.5 c.m. in diameter, on the lesser curvature, 6 c.m. from the pylorus. Severe chronic gastritis with a few small acute necroses and erosions in some areas of maximal inflammation.

Female, 91.

Emaciated, incontinent old lady. Bilateral bronchiectasis. Marasmic pulmonary thrombosis. Acute purulent pericarditis. Multiple 'senile' changes.
Moderately severe chronic gastritis. Very patchy. One subacute or early chronic ulcer, 1 c.m. in diameter, just proximal to the pylorus on the anterior wall. A similar ulcer just beyond the pylorus. Two ? scars of old ulcers on the posterior wall of the body and antrum respectively, 2 c.m. and 1 c.m. in diameter. One small acute haemorrhagic erosion in the fundus.

Male, 77.

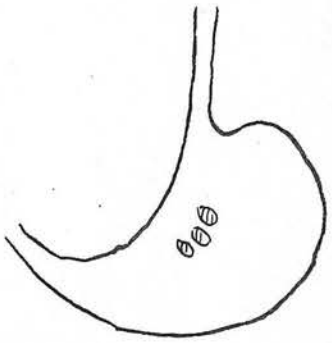
Benign prostatic hyperplasia. Cystitis. Chronic pyelonephritis. Death from ureaemia.
Severe chronic gastritis. A chronic ulcer 1.5 c.m. in diameter, 6 c.m. from the pyloro-duodenal junction, on the lesser curvature. One small erosion at the pylorus, with polymorphs.



Male, 78.

Bronchiectasis and broncho-pneumonia. Hypertension. Cardiac failure.

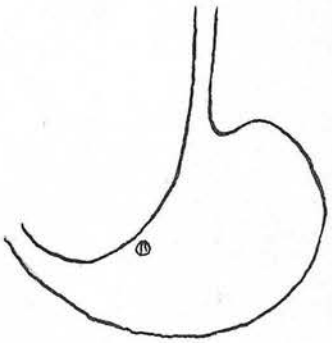
Severe chronic gastritis. Three acute ulcers on the anterior wall of the body, varying in diameter from 0.5 to 1.5 cm.



Male, 61.

Carcinoma of bronchus.

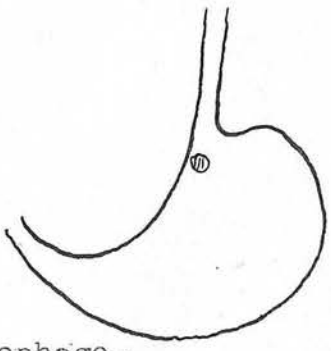
Diffuse chronic gastritis of moderate severity. Chronic ulcer, 0.5 cm. in diameter, on the posterior wall near the lesser curvature, 6 cm. from the pylorus.



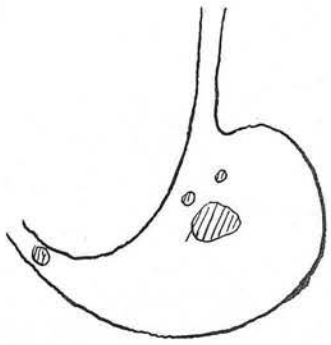
Male, 57.

Hypertension. Coronary occlusion. Cardiac failure. Bronchiectasis and broncho-pneumonia. Uraemia.

Severe chronic gastritis. A partially healed chronic ulcer, 0.5 cm. in diameter, 3.5 cm. from the oesophago-gastric junction adjoining the lesser curvature. Very severe arterio-sclerosis in the stomach.



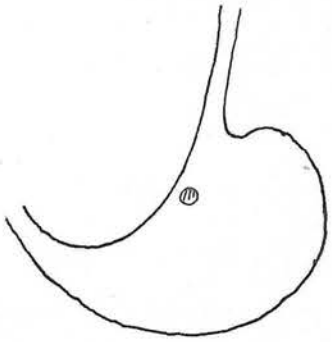
Female, 77.



Very obese. Patient complained of upper abdominal pain for the 4 years prior to her death. She was admitted to hospital as an "acute abdominal emergency". At laparotomy and at autopsy acute pancreatitis, recent cholecystitis and multiple abdominal adhesions were found. The patient died from a sudden pulmonary embolism.

There was a severe, very patchy chronic gastritis, three chronic ulcers in the body, two on the posterior wall and one on the anterior wall, the smallest 0.2 cm. in diameter, the largest 3 cm. in diameter and covered with laminated blood clot. One small acute erosion, with polymorphs, in the fundus. One very large post-pyloric chronic ulcer.

Male, 53.



Syphilitic sortitis with ruptured aneurysm of the ascending aorta. Bilateral haemorrhagic congestion of the lungs with pneumonic consolidation of part of the right upper lobe. Purulent pericarditis. Diffuse moderately severe sub-acute gastritis. Shallow acute ulcer, 0.7 cm. in diameter, on posterior wall of the body adjoining the lesser curvature.

Male, 74.

Chronic bronchitis and emphysema. Cor pulmonale. Severe malaena and anaemia. Thought ante-morten to have carcinoma coli.

Severe chronic gastritis. Large chronic ulcer, 4 x 5 cm., on the posterior wall of the cardia, just below the o-g junction.

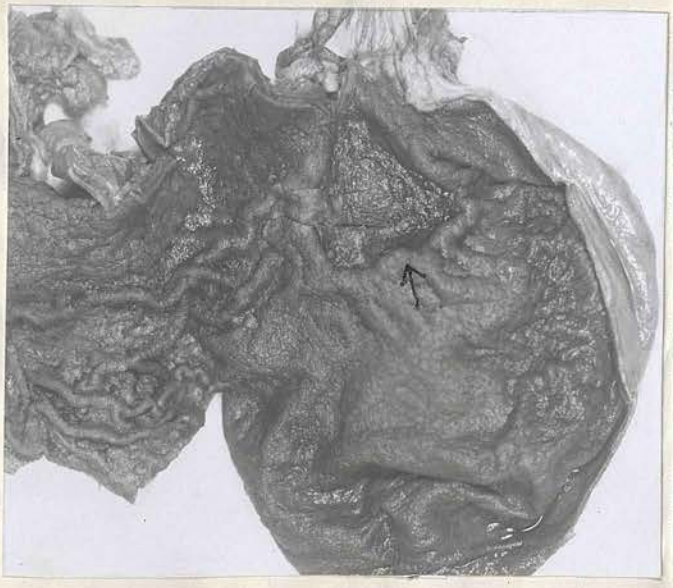
Hart (1919 in Germany) and Stewart (1929, in England) found chronic peptic ulcers, or scars thereof, in 10% of all necropsies.

Doll (quoted by Avery Jones 1947) made a statistical survey of working men in industry and transport and found a 5 - 7 % incidence of peptic ulcer.

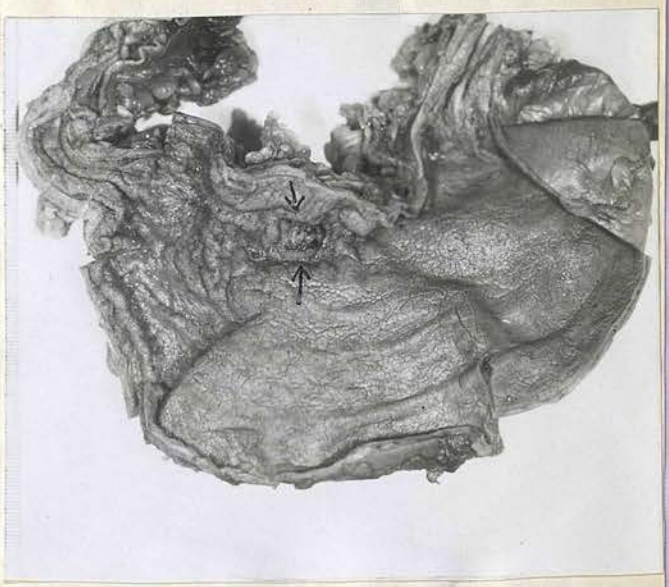
Avery Jones (1947) studied 615 peptic ulcer patients. Of these two-thirds were males and 33% were over 60. He drew attention to the apparently increasing age of the ulcer population and to the considerable rise in mortality in those patients over 60 years of age who have not had surgical assistance.

A study of the material under review suggests that the increasing incidence of ulcers in the older age groups is related more to failure of the general health of the patient than to local factors. Excluding the case of carcinoma ventriculi, 7 out of 9 cases of gastric ulcer were associated with extensive extra-gastric infection... one patient had pneumonia and purulent pericarditis, another bronchial carcinoma, 3 had bronchiectasis and 2 had severe urinary infection.

ULCERS



S4. MALE 74. CHRONIC "INNOCENT" ULCER IN CARDIA & UPPER BODY. IT MEASURED 4 × 5 cm.



S32. MALE 44. CHRONIC ULCER ON LESSER CURVATURE.

S21. FEMALE 91. CHRONIC ULCER IN PYLORUS & ANOTHER IN THE FIRST PART OF THE DUODENUM.

S60. MALE 61. DEEP "PUNCHED OUT" CHRONIC ULCER ON POSTERIOR WALL OF BODY NEAR THE LESSER CURVATURE.

EROSIONS.

Erosions were found in 18 stomachs i.e. in 30% of all stomachs exclusive of foetal stomachs. Four of these eighteen stomachs also showed ulceration.

These erosions had the following characters:-

1. Distribution.

They showed no characteristic distribution. They appeared in any part of the mucosa.

2. Size and shape.

They varied in size and shape from minute, round specks, just visible to the naked-eye, to round, oval or irregular-shaped excavations. They varied in depth from simple desquamations of surface epithelium to neo-ulcers.

3. Number.

They were single or multiple, frequently multiple.

4. Relation to gastritis.

They were or were not associated with gastritis.

5. Relation to haemorrhage.

They frequently showed evidence of recent or old haemorrhage.

6. Histology.

Some erosions ... the simplest ... were minute deficiencies in the superficial part of a congested but otherwise normal mucosa. The edges of these erosions had a pale, blurred appearance due, apparently, to the digestive action of the gastric juice.

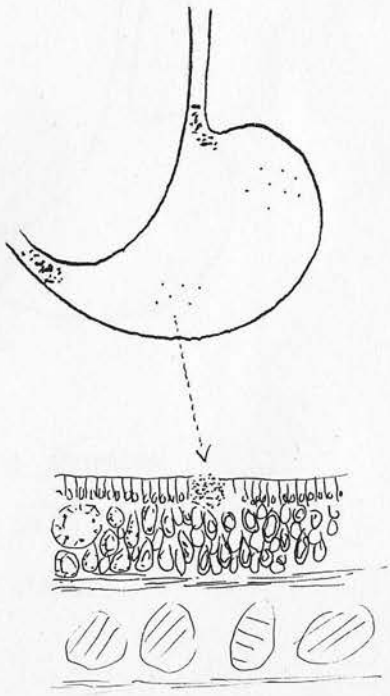
Polymorphs were or were not present. Sometimes they formed a cuff around the edges of an erosion.

Other erosions were simply miniature ulcers, stuffed with polymorphs and set in a very inflamed mucosa.

A brief description of the cases with erosions follows:-

Male, 69.

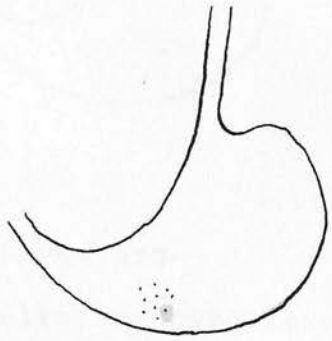
Silicosis. Bronchitis. Broncho-pneumonia. Cor Pulmonale. Moderately severe chronic gastritis with patches of acute inflammation at the cardia and at the pylorus. Small erosions on the summits of the gastric folds in the fundus and antrum .... breaches in a relatively healthy mucosa, unaccompanied



by any signs of active acute inflammation.

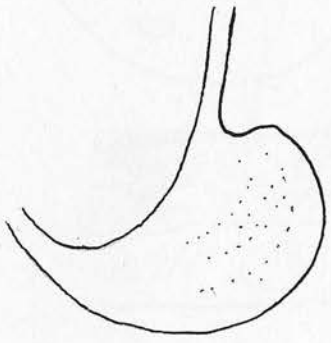
Brisk acute oesophagitis and duodenitis with erosions and ulcers.

Male, 4 weeks.



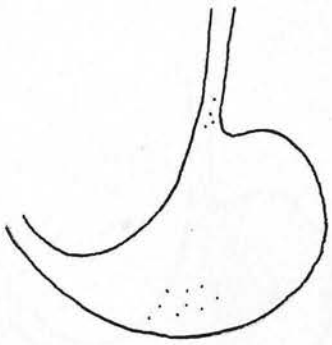
Premature. 4 lb. 7 oz. and 17 inches long at birth. Asphyxiated. Lobeline 1 cc. injected into cord. 23 days later developed frequent stools. Died 9 days later. At autopsy, some emphysema, very mild chronic gastritis. Very well defined erosions in the lower body and antrum with no cellular reactions.

Female, 43.



Chronic asthmatic. Status asthmaticus not responding to adrenaline. Given paraldehyde 6 cc. intramuscularly 2 hours before she died. Mild chronic gastritis. Multiple acute erosions on the summits of the gastric folds in the fundus and body.... breaches in the surface with no inflammatory cell reaction. Many eosinophils throughout the mucosa.

Male, 37.

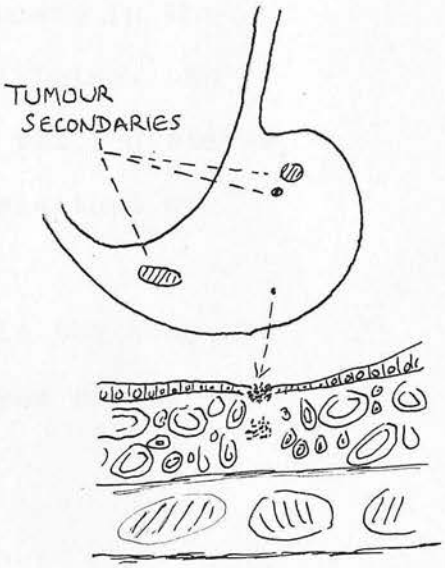


Carcinoma of pancreas. Obstructive Jaundice. Chronic alcoholic. Died from haemorrhage, either from the oesphagus or the duodenum.

Mild chronic gastritis. A few erosions in the body and antrum. No cellular response.

Female, 60.

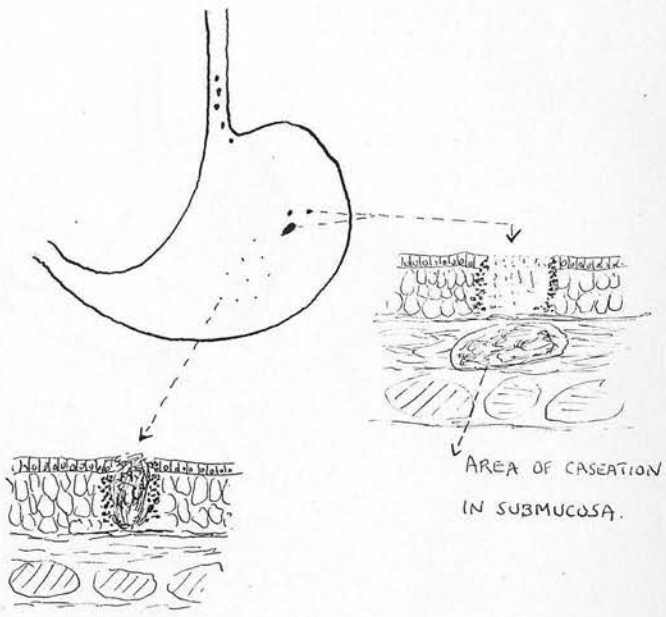
Carcinoma of cervix. Pulmonary embolus. Severe chronic gastritis. Tumour nodules in the mucosa and submucosa of the antrum and fundus. One erosion in the body... a surface deficiency in a chronically inflamed mucosa containing polymorphs and fibrinoid material and overlying a small lymph follicle.



Female, 44.

Bilateral active pulmonary tuberculosis. Uncontrollable diabetes.

On the day before death developed acute pain in the mid-chest and epigastrium and vomited. At autopsy: black faeces; acute oesophagitis with multiple acute erosions .... appearance suggestive of tubercle infection.



Moderately severe gastritis. Areas of mucosal necrosis in the body, overlying ? tuberculous follicles. No tubercle bacilli were found with the Z-N stains but the histological appearance was that of caseation.

Multiple acute erosions in the body and antrum, with a mantle of polymorphs around the necrotic core.

Male, 78.

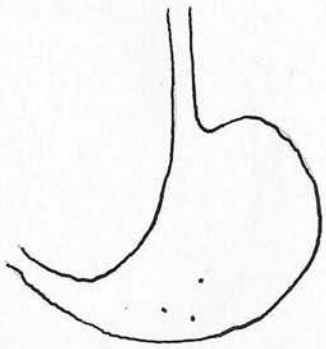
Ulcerative carcinoma of the hepatic flexure with a local abscess. Duodeno-colic fistula. Some myocardial scarring.

Male, 78 (Cont'd.)

Moderately severe chronic gastritis. Minute surface erosions in the pylorus, with many polymorphs, almost certainly post-haemorrhagic.

Male, 87.

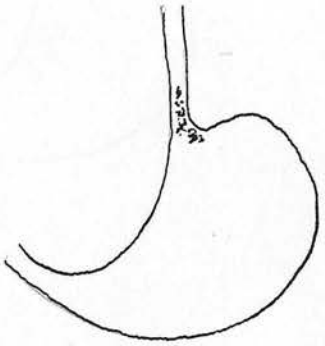
Carcinoma of prostate.
Cystitis and pyelonephritis.
Infarct right upper lobe. Mild chronic gastritis. Mild chronic gastritis.



One small acute haemorrhagic erosion in the body, another 2 in the antrum. No polymorphs present.

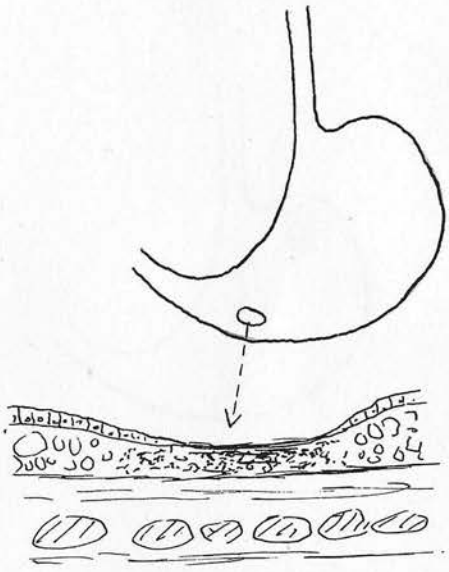
Male, 57.

Congenital cystic kidneys. Severe uraemia. Severe chronic gastritis. Severe acute erosive oesophagitis. Acute erosive inflammation in the cardia, continuous with the oesophageal lesions.

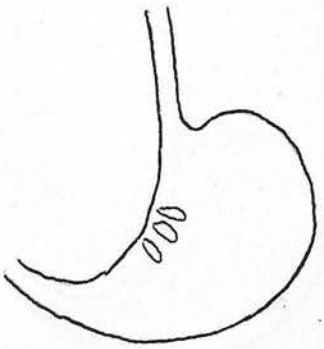


Female, 57.

Squamous-celled carcinoma of lung. Old tuberculous focus of lung.
Moderately severe chronic gastritis with acute elements.
A large erosion in the antrum on the posterior wall. Many polymorphs present.

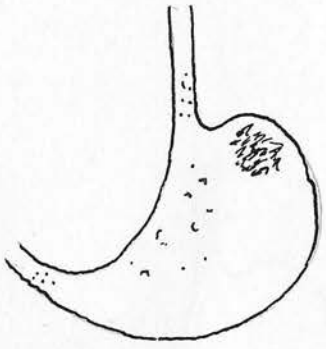


A small polymorph erosion present in the first part of the duodenum, in an area of severe inflammation.



Male, 55.

Carcinoma left lower lobe of lung.
Brisk acute oesophagitis.
Moderately severe chronic gastritis with acute elements. Three almost healed large polymorph erosions in the body on the lesser curvature.

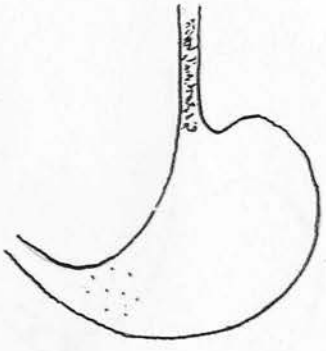


Male, 3 months.

Transposition of the viscosa with partial obstruction of the small intestine.
Patches of acute erosive oesophagitis and duodenitis. Much of stomach healthy. Patches of mucosal lysis. A large patch of acute erosive gastritis in the fundus. One small acute erosion, with polymorphs, in the body.

Female, 5 years.

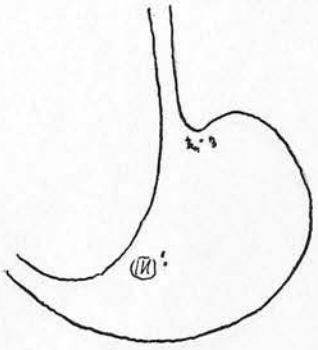
Primary tuberculous focus right upper lobe of lung. Generalised miliary tuberculosis. Tuberculous meningitis. Acute erosive oesophagitis. Much of mucosa of healthy appearance. Patches of congestion and oedema, and patches of lysis. Some of the latter showed some definite small haemorrhagic erosions. Many mitotic figures in the gland cells.



Male, 62.

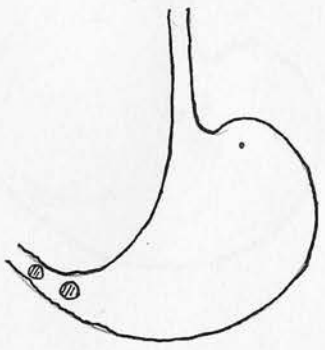
Bronchiectasis. Empyema. Widespread loculated peritonitis. Resolving cholangitis. Amyloidosis of spleen and kidneys. Diffuse monitial infection of oesophagus. Moderately severe chronic gastritis. Acute haemorrhagic erosions in the lower body and antrum.

Male, 76.



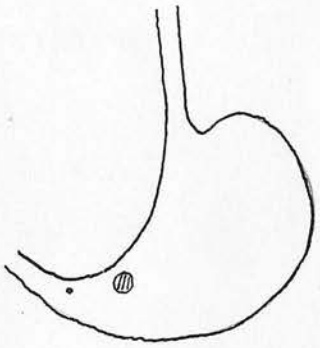
Carcinoma of bladder. Death from uraemia. Very severe cystitis and pyelonephritis. Purulent bronchitis and broncho-pneumonia. Severe chronic gastritis with acute elements. One subacute or early chronic ulcer in the body on the lesser curvature. Erosions, with polymorphs, bordering the ulcer. Small acute superficial necroses in the cardia, in areas of considerable inflammation.

Female, 91.



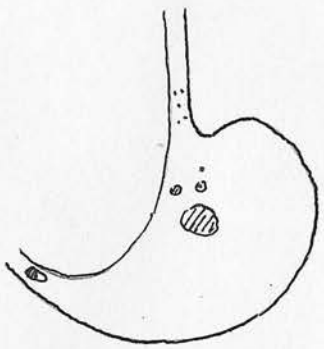
Bilateral bronchiectasis. Acute purulent pericarditis. Marasmic pulmonary thrombosis. Moderately severe, very patchy, chronic gastritis. One subacute or early chronic pyloric ulcer. One acute haemorrhagic erosion in the fundus.

Male, 77.



Benign prostatic hyperplasia. Cystitis. Chronic pyelonephritis. Death from uraemia. Severe chronic gastritis. Chronic ulcer in the body on the lesser curvature. One small acute polymorph erosion at the pylorus, apparently post-inflammatory.

Female, 77.



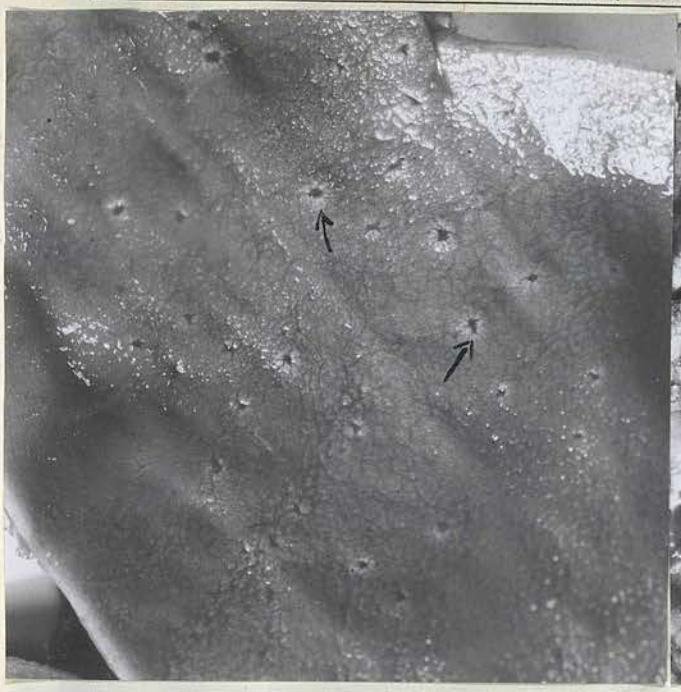
Upper abdominal pain for 4 years. Admitted to hospital as a case of ? perforated gall-bladder. At laparotomy: acute pancreatitis. Pulmonary embolus during convalescence.

Severe chronic gastritis. Three chronic ulcers in the body, one covered with laminated

Female, 77. (Cont'd.)

blood clot. One chronic duodenal ulcer. One acute erosion in the cardia, with polymorphs.

ACUTE EROSIONS OF STOMACH & DUODENUM. LOW MAGNIFICATION.



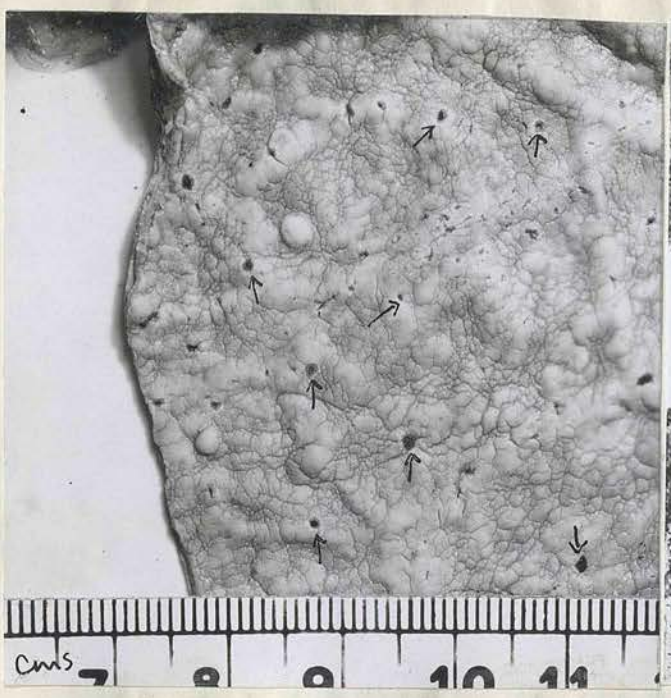
S5. MALE . 4 weeks.
"SOLUTION" EROSIONS OF PINHEAD SIZE IN THE ANTRUM. NO POLYMORPHS PRESENT.

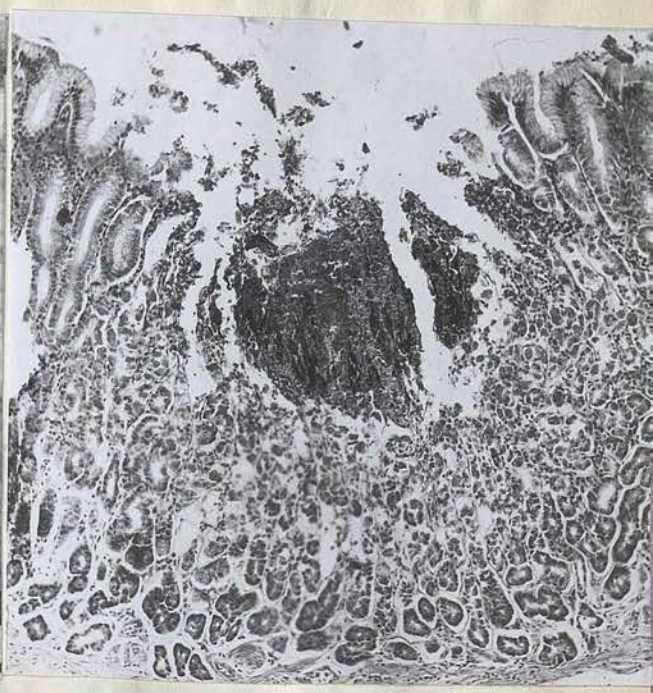
S9. FEMALE . 43.
HAEMORRHAGIC EROSIONS IN THE FUNDUS & BODY.

S64. MALE 62.
HAEMORRHAGIC EROSIONS IN THE LOWER BODY AND ANTRUM.
VERY FEW POLYMORPHS PRESENT.

S3. MALE .69.
EROSIONS IN THE FIRST PART OF THE DUODENUM.
NUMEROUS POLYMORPHS PRESENT.

ACUTE EROSIONS OF STOMACH & DUODENUM.

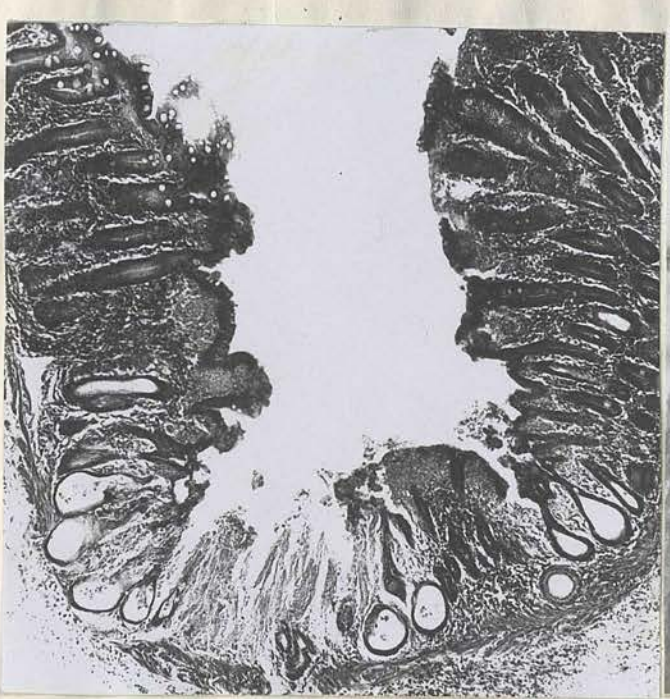


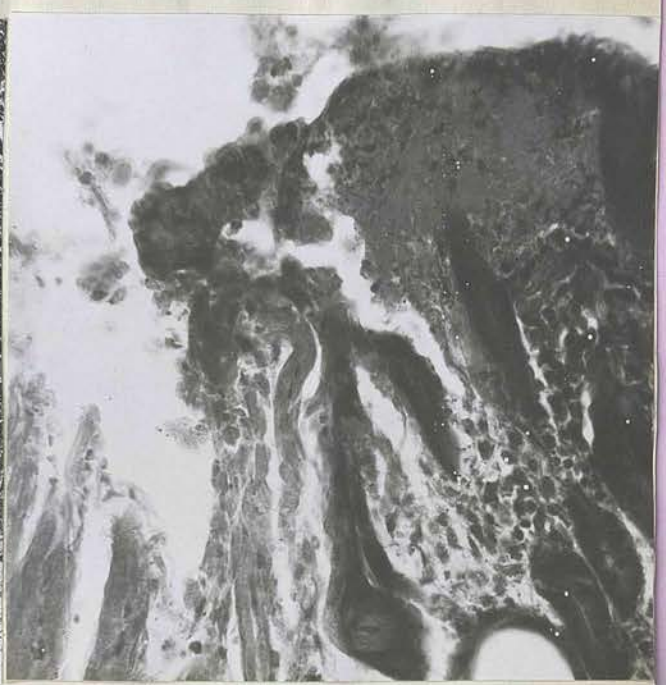


a. LOW MAGNIFICATION

b. HIGHER MAGNIFICATION.

S. 33. MALE 84. HAEMORRHAGIC EROSIONS IN LOWER BODY & ANTRUM THE PHOTOGRAPH ON THE RIGHT IS OF A SINGLE EROSION.





a. LOW MAGNIFICATION.

b. HIGH MAGNIFICATION.

S 54. MALE 59. ACUTE EROSION IN THE FIRST PART OF THE DUODENUM. NOTE THE EXUDATE IN THE SUPERFICIAL PART OF THE MUCOSA.

PATHOGENESIS OF ULCERS AND EROSIONS.

Although they can arise de novo, it is probable that in many instances ulcers are preceded by erosions.

Brummelkamp (1938) reviewed the theories concerning the pathogenesis of peptic ulcer. There are 3 principal theories:-

(1) Peptic Theory: that erosions and ulcers are produced by the action of gastric juice.

(2) Inflammation Theory: that erosions and ulcers are preceded by mucosal inflammation.

(3) Vascular Theory: that erosions and ulcers are preceded by vascular lesions.

Each theory has many adherents. Most people have favoured either the peptic theory or the inflammation theory, or both together. The vascular theory has never been very popular and consequently little attention has been paid to it, yet the influence of the blood vascular system of the stomach should not be underestimated

in a consideration of peptic erosion and ulceration.

Von Bergmann propounded the theory of ulcer patients having a disharmonious nervous system with vagal preponderance and an increased tendency to muscle spasm leading to local ischaemia, and superabundant secretion of gastric juice. Westphal furnished proof of this theory by subcutaneous injections of pilocarpine and physostigmine in cats, guinea-pigs and dogs, also by faradic irritation of the peripheral stump of the cut vagus. Haemorrhagic erosions and ulcers were produced. Westphal believed that the contraction of small arteries as well as the strong secretion of gastric juice were ultimately responsible for the lesions.

Nakashima produced haemorrhagic erosions in rabbits by pilocarpine injections. He believed that spasm of the muscle wall of the stomach was responsible for the haemorrhage.

Hauser believed that erosions were originally small haemorrhagic infarcts which became secondarily invaded by polymorphs.

Rokitanski, Kundrath, Orth, Kaufmann, Ribbert and Schmans - Herxheimer all supported the theory of haemorrhagic infarction. Hans Hanke (1933) produced acute erosions in cats by the administration of drugs (morphine, pilocarpine and caffeine) and hormones (insulin and suprarenin). Acute erosions occurred also in the oesophagus. Although Hanke believed the erosions were peptic in origin, they bore a close resemblance to those of other haemorrhagic erosions which were probably of primary vascular origin.

Beaumont described the production of congested patches and erosions in the stomach of St. Martin when the latter was the subject of emotional turmoil. Davies and Wilson (1939) and Gainsborough and Slater (1946) have described the effect of acute emotional crises in producing multiple haemorrhages in the gastric mucosa. Avery Jones (1947) describes the mucosa of a woman of 49 as seen through the gastroscope, where acute haemorrhages were succeeded by acute ulcers. Finally, Wolff and Wolff (1947) describe minute haemorrhages frequently following vigorous contractions of

the stomach when the mucosa was engorged. They suggested that vascular engorgement might predispose to erosions and secondary inflammation.

It is probably true to say that peptic juice, inflammation of the mucosa and vascular lesions can all play an important part in the genesis of erosion and ulcer.

Hydrochloric acid in physiological concentration can produce damage in the gastro-intestinal mucosa (Mathes; Cummins, Grossman, Ivy, Bachrach) although healthy living gastric tissue is much more resistant to acid-pepsin digestion than unhealthy tissue (Grossman). Price and Lee, by producing hyperacid juice by parenteral injections of histamine in beeswax, have corroded the gastric mucosa of dogs.

Wolff and Wolff also have drawn attention to the resistance of the lining cells of the stomach. They showed that strong irritants, e.g. mustard, while producing inflammation, including tissue

destruction, and tissue destruction when applied to the skin of their patient "Tom", failed to cause more than a slight to moderate erythema in the gastric mucosa. They suggested that thick mucus might protect against irritants. They showed also that prolonged contact of acid gastric juice with a minor erosion produced a peptic ulcer.

Many authors are agreed that inflammation of the mucosa can lead to erosion and ulceration. The enormous stuffing of the mucosa with blood, oedema fluid and inflammatory cells, and the degeneration and death of the surface cells of the mucosa all predispose to erosion and ulceration. But so many authors believe that haemorrhage in erosions is a secondary and not a primary phenomenon.

Hamperl (1932) examined 120 autopsy stomachs fixed not later than 5 hours after death and found erosions in some stomachs which were distinguishable from post-inflammatory erosions. Some of the erosions were haemorrhagic, others apparently were not although he stated

that all showed fibrinoid material on their surface. He believed that these erosions arose from peptic digestion.

Most, if not all, of the erosions which showed no polymorphs in my series of stomachs were agonal, but this is not to say that the underlying mechanism of production may not play an important part in the pathogenesis of some erosions and ulcers during life. One may suggest that contraction of the muscle wall of the stomach and vascular engorgement may lead to haemorrhages which burst through the covering epithelium.

Support is lent to the importance of a vascular basis for these agonal erosions by 3 facts: (1) their localisation to minute areas of mucosa, (2) their frequent presence in an otherwise normal mucosa, (3) the almost invariable finding of whole or altered blood in them.

Henning and Norpeth conducted experiments on rats, producing a corrosive gastritis by treating the mucosa of fasting animals with 0.7% Hcl. A severe acute inflammation was produced immediately and necrotic areas

appeared after 15 minutes. At first small white dots appeared on the summit of the gastric folds. Later, haemorrhages appeared in the erosions. They stressed the importance of HCl in the production of erosions but they emphasised also the peculiar localisation of the erosions to the summits of the ridges. They could offer no explanation except a suggestion of a possible "peculiarity of the circulation".

The gastro-intestinal mucosa is immensely rich in blood-vessels and it is not improbable that rupture of these vessels from many possible causes is a frequent pre-erosion phenomenon. It is likely, therefore, that many so-called primarily peptic, secondarily haemorrhagic erosions are really primarily haemorrhagic, secondarily peptic. In the present series of stomachs all degrees of erosion - formation were to be seen, from gross congestion of sub-surface vessels, their rupture and the earliest phases of peptic digestion to polymorph infiltrations and the formation of ulcers-in-miniature.

Nevertheless much greater elucidation of the pathogenesis of erosions and ulcers is required before we can speak with any convincing authority. Carefully designed experiments concerned with each of the suggested aetiological factors is necessary before there is any striking advance in our knowledge.

To summarize the possible aetiological factors concerned in the pathogenesis of peptic erosion and ulceration:-

1. Vascular Factors: congestion and ? vaso-spasm (in connection with contraction of the muscle wall of the stomach, nervous stimulation, acute inflammation or drugs). Rarely embolism (tumour or bacterial) and thrombosis.

   The end result is haemorrhage.

2. Inflammation: acute inflammation, arising either de novo or superadded to chronic inflammation.

   Leads to degeneration and necrosis.

3. Peptic Juice: irritating, even necrotizing, and digestive.

4. Nervous Influences: psycho-somatic stimulation; cerebral haemorrhage.

5. Metabolic Factors, e.g., altered pH of the blood.

It is very likely that several of these factors combine in the production of an erosion and ulcer.

SOME CELLS OF THE INFLAMED GASTRIC MUCOSA.

PLASMA CELLS AND LYMPHOCYTES.

The healthy foetal mucosa contains no plasma cells and very few lymphocytes. The inter-glandular connective tissue is composed of reticular cells and their fibrils.

After birth, the healthy mucosa contains more lymphocytes, perhaps a few polymorphs and a number of plasma cells. The lymphocytes are sometimes aggregated into follicles.

Even in the healthy mucosa it is not uncommon to find that the plasma cells outnumber the lymphocytes, although the total number of both is not very considerable.

It is a mistake to think that because plasma cells and lymphocytes are fairly numerous that they are an indication of an inflammatory process. Magnus (personal communication has suggested that fair numbers of plasma cells, greater than was previously allowed, may be associated with normal stomachs and may be a physiological phenomenon and as a result of my studies I am in agreement with this view.

Astrid Fagraeus (1948), amongst other workers, has demonstrated a relationship between plasma cells and antibody formation, showing that immature cells produce most antibody and confirming this theory by the in vitro production of antibodies using splenic explants.

Many descriptions of chronic gastritis have given priority of numbers to the lymphocyte but in the series of stomachs referred to here plasma cells appeared generally to outnumber the lymphocytes.

The origin of plasma cells has been debated. It was suggested that they were derivatives of lymphocytes (Michels; Nicholas). Eliz. Lowenhaupt and Bessis, however, consider them derivatives of the histiocytic series of cells and Fagraeus believes that they develop from reticulo-endothelial cells during the formation of antibody.

The richness of the mucosa in reticulum cells, the numbers of plasma cells compared with the number of lymphocytes in many stomachs, and the absence of transition forms between lymphocytes and plasma cells, suggest that plasma cells are derived not from lymphocytes

but from the reticulum cells of the mucosal connective tissue in response to an unknown stimulus.

EOSINOPHIL LEUCOCYTES.

The eosinophil still is a mystery cell. The ease with which it can be identified contrasts with the lack of knowledge of its function.

Of the 72 autopsy specimens, the overall incidence of eosinophils in the mucosa was as follows:-

Nil found	2 stomachs.
Slight numbers	36 "
+	22 "
++	10 "
+++	1 "
++++	1 "

The greatest numbers were found in a female patient of 43 who died in status asthmaticus.

Of the 4 gastrectomy specimens the incidence was:-

+	1 stomach
++	1 stomach
+++	2 stomachs.

Eosinophilia did not appear to be related to the severity of uncomplicated chronic gastritis. It could not be correlated with increased tissue destruction unless possibly such destruction was considerable and fairly acute.

Code, quoted by Rebuck, has stated that eosinophils are an important source of blood histamine; but the possible connection between this fact and the observed mucosal eosinophilias is unknown.

Hamperl (1932) reported finding eosinophils in the majority of autopsy stomachs. He also found them to be more frequent in cases where death was very sudden and also in operation specimens.

RUSSELL BODY CELLS.

The incidence in the autopsy specimens was as follows:-

0	36
Slight numbers	27
+	4
++	4
+++	1
++++	0

In the gastrectomy specimens the incidence was:-

0 3

Slight numbers 1

At first one was tempted to assume that the incidence of these cells is directly proportional to the severity of the gastritis but many cases of severe gastritis showed no Russell body cells, or hardly any.

The highest incidence occurred in a stomach the site of an anaplastic carcinoma and with severe chronic inflammation.

That they are degenerated plasma cells seems likely but the mechanism of their production is unknown.

GOBLET CELLS.

In the autopsy specimens the incidence was:-

0 42

Slight numbers 13

+ 10

++ 6

+++ 2

++++ .....nil.

In the operation specimens the incidence was:-

0 1

++ 3

The number of goblet cells ran parallel with the severity of the chronic gastritis.

PARTICULAR STOMACHS.

A few stomachs presented such unusual features that they deserve special mention:-

1. A case of ? gastrostaxis.

The patient was a man of 74 who had had a gastro-enterostomy 9 years before his death for chronic duodenal ulcer. He was in relatively good health until three weeks before his death when he developed melaena after taking several aspirins for a cold. Twelve to fifteen pints of matched blood were transfused into him but his melaena continued to be very severe.

At autopsy the stomach was filled with fresh blood clot. The mucosa showed moderately severe chronic inflammation and multiple brown granules in the mucosa, particularly in the surface layers, some intra- and some extra-cellular. There was a healed chronic duodenal ulcer in the duodenum, first part.

No bleeding point was found anywhere, despite very careful search of the mucosa. The possibility of gastrostaxis was raised.

Several cases of this condition have been described in the literature. The granules in this case were never proved to be altered blood and they might well be an artefact.

2. Probable tuberculous lesions in the Stomach.

The patient was a female of 44 who was admitted to hospital 8 days before her death with uncontrolled diabetes and bilateral phthisis. On the day before her death she developed acute pain in the midchest and epigastrium and vomited. She later became comatose.

At autopsy the large intestine contained tarry faeces. The oesophagus showed multiple acute erosions as well as lesions very like caseous tubercles.

The stomach contained a moderate amount of coffe-ground material. On the posterior wall of the body there were 3 brown "scorched" areas, the largest about 2 cm. in diameter. Microscopically, these areas consisted of well-defined submucosal

necrotic lesions, strikingly like caseous tubercles, over which the mucosa was necrotic. The necrotic mucosa was very sharply defined and had undergone partial digestion by the peptic juice and showed early polymorph infiltration at the margins.

Although tubercle bacilli were searched for they were never found in the stomach and oesophageal lesions, but the resemblance of the tubercle-like lesions in a patient with marked pulmonary tuberculosis seems more than coincidental.

3. The stomach of a patient with Hodgkin's disease.

The patient was a woman of 38 who had been treated with "nitrogen mustard". She had developed a severe aplastic anaemia and died following a sudden massive cerebral haemorrhage.

The stomach showed considerabl congestion and oedema and numerous small fresh haemorrhages. There was a brisk parenchymal atrophy in some parts of the mucosa. Muscle degeneration and atrophy was marked.

Typical Hodgkin cells were present scattered throughout the connective tissue of the mucosa.

4. The stomach of a patient with carcinoma of the prostate.

The patient was a man of 87 who died of a pulmonary embolus a few days after a pre-urethral partial resection of the prostate.

At autopsy, although the bones were not examined for secondary deposits, no secondaries were found in any organs except those mentioned below.

A secondary nodule of well-differentiated adeno-carcinoma, 1 cm. in diameter, was found bulging into the lumen of the lower third of the oesophagus. The oesophagus was very severely and very acutely inflamed, eroded and ulcerated. A nodule of similar quality but if slightly smaller size was found in the mucosa of the fundus of the stomach; and there was a similar but still smaller nodule in the mucosa of the first part of the duodenum.

5. Stomach showing amyloid deposit in the mucosa.

The patient, a female of 64, died of uraemia following chronic cystitis and bilateral pyelonephritis. At autopsy amyloidosis of the liver, spleen, kidneys and large bowel was found.

The stomach showed an extensive amyloidosis. The gross thickness of the mucosa was only slightly diminished but individual glands were often markedly degenerate and atrophied.

MISCELLANEOUS.

S 22. FEMALE 44. PART OF OESOPHAGUS – LOW MAGNIFICATION – TO SHOW AN EROSIVE OESOPHAGITIS PROBABLY TUBERCULOUS.

S 22. FEMALE 44. PART OF BODY OF STOMACH – LOW MAGNIFICATION – TO SHOW A NECROTIC PATCH OF MUCOSA, PROBABLY TUBERCULOUS; ALSO SOME HAEMORRHAGIC EROSIONS.

S 58. MALE. 3 MONTHS. CURIOUS LESIONS IN THE BODY OF THE STOMACH. LOW MAGNIFICATION. SEE TEXT.

S. 18. FEMALE. 38. MULTIPLE PETECHIAL HAEMORRHAGES IN THE MUCOSA OF THE STOMACH. A CASE OF APLASTIC ANAEMIA.

MISCELLANEOUS

a. OESOPHAGUS.

b. STOMACH MUCOSA.

S.33. MALE 84. CARCINOMA OF PROSTATE WITH A METASTASIS IN THE OESOPHAGUS AND TWO METASTASES IN THE FUNDUS OF THE STOMACH. THERE IS ALSO A SEVERE EROSIVE OESOPHAGITIS

S 64. MALE 62. MONILIAL INFECTION OF THE OESOPHAGUS.

SUMMARY.

1. This work deals with the morbid-anatomical examination of 72 autopsy and 4 gastrectomy stomachs, all formalin-fixed.

2. The specimens were fixed according to Magnus' rulings within a very short time of death.

3. The stomachs are divided into 5 histological grades according to their health or disease.

4. It is shown that the severity of "chronic gastritis" tends to increase with age, although it is pointed out that there are exceptions to this statement.

5. Proof of the existence of physiological ageing of the stomach was not found.

6. A correlation was attempted between the incidence and severity of chronic gastritis on the one hand and infection, uraemia and other factors on the other. Uraemia was constantly associated with chronic gastritis, often of

severe degree. Severe chronic infections were often associated with severe chronic gastritis but sometimes such infections were associated with an almost normal gastric mucosa.

7. Ulceration of the stomach was found in 15% of the adult cases. The average age of the patients was 67 and the ulcers were undiagnosed during life.

8. Erosions were discovered in 30% of all stomachs exclusive of foetal stomachs.

9. The pathogenesis of erosions and ulcers is briefly discussed. It is suggested that a vascular origin for erosions and ulcers is commoner than has frequently been supposed. This is supported by the common finding of haemorrhage in the mucosa and of all grades of erosion between 'pure' haemorrhagic erosions and typical miniature ulcers.

10. Reference is made to particular cells of the inflamed mucosa and to 5 stomachs of particular interest.

A P P E N D I X.

CASE NOTES.

1. Autopsy Specimens.

2. Operation Specimens.

1. AUTOPSY SPECIMENS.

S.1. R.R. Male, 74.

Clinical Data.

Gastro-enterostomy 9 years before death for D.U. Felt well until 2 weeks before admission when he developed melaena after taking several aspirins for a cold. Was transfused with 12-15 pints of blood.

General Autopsy Findings.

Large, obese, pale subject with generalised oedema. Some exophthalmos. Thyroid x $1\frac{1}{2}$ in size, with some cystic nodules. Generally dilated heart. Fatty liver. Terminal basal pneumonia.

Stomach and Duodenum.

Large stomach containing much fresh blood clot. Quite thick wall. Mucosa stained dark red or brown. Operation stoma healthy. Healed D.U. in 1st part of duodenum .... mucosa thin and blood-stained. No bleeding points seen in stomach, duodenum or jejunum.

Microscopically there was a diffuse chronic gastro-duodenitis of moderately severe intensity. There were multiple brown granules in the mucosa...

altered blood from haemorrhage ? or else artefact ? The possibility of that vague entity "gastrostaxis" was raised.

Very few eosinophils were present in the mucosa. There was moderate arteriosclerosis.

S.2. Infant, W. Male, 8 days (? 38 weeks).

Clinical Data.

Asphyxia neonatorum. Multiple congenital deficiencies and deformities.

General Autopsy Findings.

No obvious prematurity. Mongoloid facies. Deformity left thumb. Some atelectasis. "Double" kidneys with 4 ureters. Interventricular and interatrial septal defects.

Stomach and Duodenum.

Gastric and duodenal mucosa normal except for a few minute scattered haemorrhages. Fairly abundant eosinophils, especially near the base of the mucosa.

S. 3. W.E.L. Male, 69.

Clinical Data.

An ex-miner from S. Wales. Bronchitis and cor pulmonale.

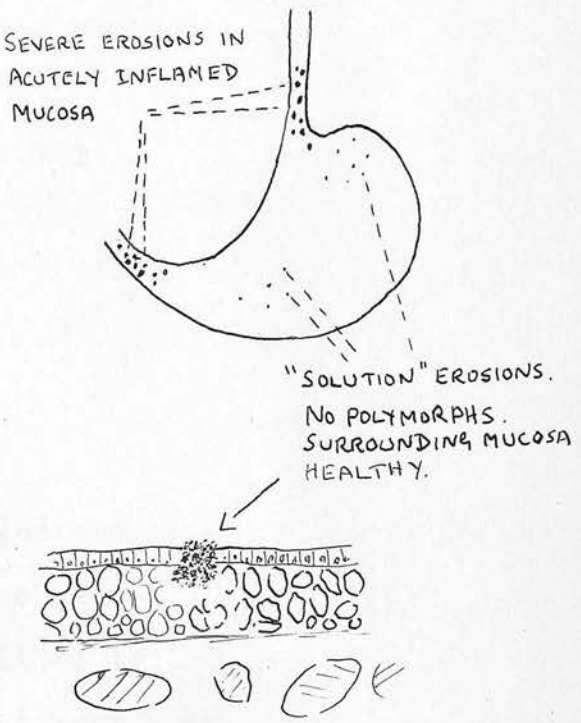
General Autopsy Findings:

silicosis, broncho pneumonia, cor pulmonale.

Oesophagus, Stomach and Duodenum.

Severe acute oesophagitis with surface erosion and ulceration. The inflammation extended from the

mucosal surface into the depths of the wall ....? peptic in origin.



Stomach of normal size and shape. Wall of average thickness. Small scattered erosions in the body and antrum, mostly on the summits of the rugae. Microscopically the stomach showed the following features: mild to fairly severe diffuse chronic inflammation, worse in antrum and pylorus. Acute inflammation in the terminal pylorus. Degeration and autopsy most in the depths of the mucosa. Acute erosions at the oesphago-gastric junction, with many polymorphs, presumably peptic like the oesophageal erosions "Solution" erosions in the summits of ridges in the fundus and antrum viz. breach in mucosal continuity filled with degenerate nuclei, mostly ex gland cell nuclei but showing also some plasma cells in the antrum. Multiple minute mucosal haemorrhages.

1st part of duodenum showed a very severe acute inflammation of the mucosa with multiple acute erosions, containing numerous polymorphs.

Eosinophils Sl. → + in the gastro-duodenal mucosa.

S.4. C.C. Male, 74.

Clinical Data. Prostatectomy 11 years before death. Operation for acute pancreatitis 3 years later. Admitted to hospital 10 days before death in a very weak condition. Very anaemic. Occult blood in the stool. ? Carcinoma coli. For 2 weeks prior to admission was in ill-health with dyspnoea, oedema of legs and ascites; the patient had also pain in the left side of the abdomen not related to food, increased by effort, relieved by defaecation. Appetite at that time was good.

The patient was found to be emphysematous.

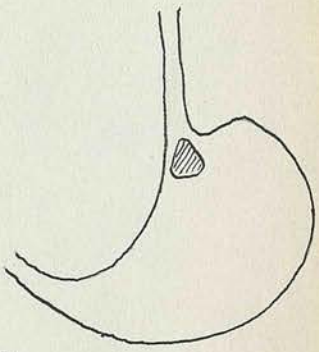
General Autopsy Findings.

Large malignant-looking gastric ulcer. Lungs congested and very emphysematous.

Small fibrinous pleurisy R. base.
Moderately thickened L. ventricle.
Moderately dilated heart. Granular kidneys.

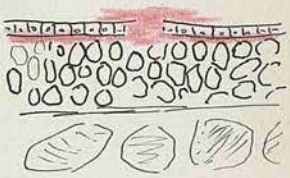
Stomach and Duodenum.

Stomach rather small; adherent posteriorly to the pancreas. It contained a large amount of fresh blood clot. There was a large ulcer, 4 x 5 cm., just below the oesophagus on the posterior wall, with a firm, granular, greyish-yellow floor and rolled edges.



LARGE GASTRIC ULCER

Microscopically, the stomach showed a mild to severe diffuse chronic inflammation, mild in the fundus, severe elsewhere. Mucosal "intestinalization" present in the body, antrum and pylorus. Russell cells sl. → +++ A few cysts. Eosinophils slight. Surface epithelium intact in all sections except that of the pylorus where a minute mucosal haemorrhage was seen which had just burst through the covering epithelium.



FRESH MUCOSAL HAEMORRHAGE.

Brown pigment granules were present throughout the mucosa.

There was moderate arterio-sclerosis.

The large chronic ulcer was not neoplastic. It showed considerable fibrosis.

The duodenum showed no special features.

S.5. Michael A. Male 4 weeks. (36 weeks' gestation).

Clinical Data.

Premature. Normal delivery. 4 lbs. 7 ozs. at birth and 17" in length. Asphyxia at birth. Lobeline, 1 cc. injected into the cord.

12 days after birth developed conjunctivitis due to Gram-negative 'rods'. Four days later developed a sterile abscess of the L. arm.

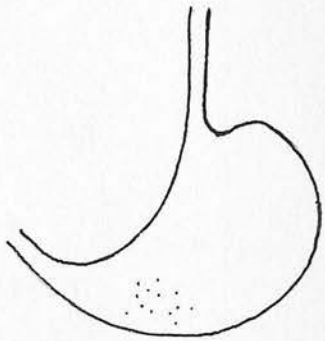
23 days after birth developed frequent stools. Died 9 days later.

General Autopsy Findings:

premature, dehydrated infant. Some emphysema.

Stomach:

normal size and shape. The mucosa had a fine velvety appearance and was dusky in the body, pale elsewhere.



Scattered over an area of about 2 sq. cms. in the lower body and antrum, in the region of the greater curvature, were several erosions of pin-head size, giving this portion of mucosa a striking sieve-like appearance.

Microscopically, there was a very mild chronic gastritis of equal intensity everywhere .... minimal changes. The erosions were a very striking feature: very well defined deficiencies in the mucosa extending to the muscularis mucosae.

No polymorphs or other inflammatory cells were seen and haemorrhage was also absent. Eosinophils slight throughout the mucosa.

S.8. Foetus. ? 18 weeks. Female.

Clinical Data.

Mother aet 28 had had 10 former miscarriages. She had 2 children, aged 10 and 9 years respectively, alive and well. The mother herself was healthy. Seven days before the foetus was passed, the liquor began to drain.

General Autopsy Findings:

Female foetus of healthy appearance.

Stomach:

was of normal size and shape. The

mucosa was of a uniformly fine velvet appearance. Microscopically there were no abnormal features. The covering epithelium was columnar and mostly one cell thick. There were acidophil cells present in the body glands. The connective tissue of the mucosa and submucosa was rich in reticulum cells. Lymphocytes were very scanty. Neutrophils were not observed. No eosinophils were found in the mucosa although a few were found in the pyloric submucosa.

S.9. Daisy T. Female, 43.

Clinical Data.

The patient was a chronic asthmatic. The day before her death she had an acute attack of asthma which did not respond to repeated injections of adrenaline. About 2 hours before her death she seemed wildly hysterical and was given 6 cc. paraldehyde by i.m.i.

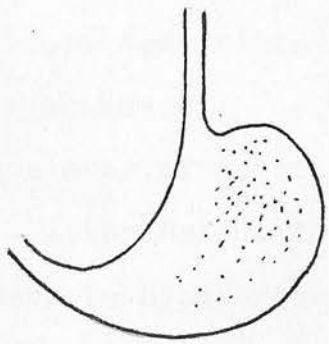
General Autopsy Findings.

Pale, emaciated female. Very emphysematous lungs. Small heart and liver.

Stomach:

was of normal size and shape. Rugae were prominent in the body.

Numerous brownish erosions were present along the rugae .... shallow and irregular in shape and 1 - 4 mm. in diameter. Microscopically they consisted of well-defined shallow deficiencies in the mucosa on or near the summit of the gastric folds. The surrounding mucosa was healthy apart from some congestion. There were no polymorphs or other inflammatory cells in the erosions. Using the Lepehne-Pickworth technique, some small amounts of blood were detected in some of the erosions. The impression given by the erosions was that they were agonal, peptic erosions, probably post-haemorrhagic.



There was a mild chronic gastritis in the cardia and the pylorus. Eosinophils were very numerous throughout the mucosa and were even more numerous in the duodenal mucosa.

S.10. K.E.L. Female, 48.

Clinical Data:

Left ventricular failure secondary to coronary thrombosis.

General Autopsy Findings.

Old scarring and recent infarction of the left ventricle. Nutmeg liver. Moderately contracted kidneys. Some broncho-pneumonia.

Stomach:

showed a very mild chronic gastritis in the cardia, with cysts +. Mild chronic gastritis in the pylorus. Remainder of mucosa healthy. Eosinophils slight → + throughout the mucosa. Arteries show considerable hypertrophy of their walls. Muscle healthy.

S.11. Albert C. Male, 48.

Clinical Data:

Admitted in coma. Died on the same day. B.P. 290/130. Diagnosed as a case of cerebral haemorrhage. Peptic ulcer 5 years before death. Dyspeptic.

General Autopsy Findings:

Small granular kidneys; hypertensive heart with some scarring of the left ventricle. Marked cerebral atheroma. A large cerebral haemorrhage.

Stomach:

Normal size and shape. ? old ulcer on the lesser curvature, 8 cm. from the pylorus. Microscopically: moderate to fairly severe chronic inflammation in the fundus, antrum and pylorus, more or less normal elsewhere. Goblet cells ++ in the areas of maximal inflammation. No Russell cells. Eosinophils slight $\geq$ + ... more numerous than in normal stomachs; + $\geq$ ++ in the section of antrum. Well-marked arterial hypertrophy. No ulceration past or present.

S.12. Mrs. E.K. Female, 36.

Clinical Data:

Poor appetite, painless vomiting, loss of weight, dyspnoea, cough, sputum ++ for 6 months before death. Diagnosed as having carcinoma ventriculi with pulmonary metastases.

General Autopsy Findings.

Considerable emaciation and pallor. Oedema of the legs. Blood and lymph pulmonary metastases. Transverse colon, gall-bladder and bile-duct ensheathed by tumour. Ante-mortem thrombus in portal vein.

Stomach:

Normal shape and size. Wall firm, whitish because of scirrhous carcinoma .... 6 to 15 mm. in thickness, and thickest in the antrum and pyloric canal. Two deep carcinomatous ulcers on the lesser curvature of the body and antrum, one 1.5 cm. the other 3 cm. in diameter. There was a diffuse chronic gastritis, moderate to severe, rather worse in the distal part of the stomach. Considerable glandular atrophy in places. Russell cells slight → +. No goblet cells. Neutrophils quite numerous. Eosinophils slight → +, most numerous in the ulcer bed.

One small post-haemorrhagic erosion in the body, with a few neutrophils.

S.13. W.C.L. Male, 76.

Clinical Data.

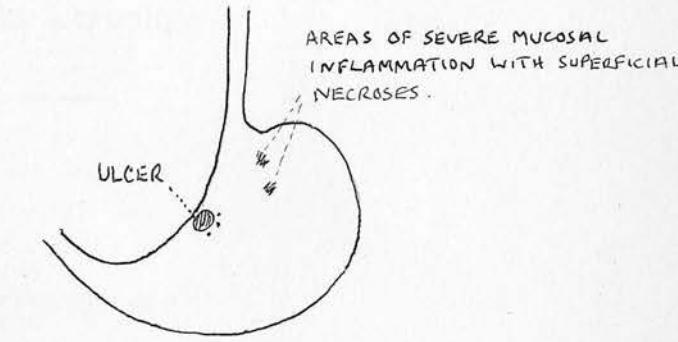
Recurrent haematuria for 5 months before death. Anaemic. Partial cystectomy 19 days before death for carcinoma of the bladder. Death from uraemia.

General Autopsy Findings.

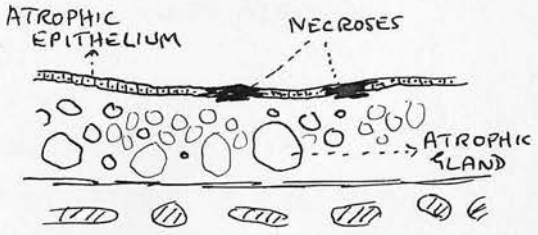
Emaciation ++. Extensive carcinomatous infiltration of the bladder. Very severe sloughing cystitis and severe pyelonephritis. Malignant infiltration of right suprarenal. Fatty liver. Purulent bronchitis and early broncho-pneumonia.

Stomach:

Normal size and shape; contained a small quantity of altered blood. Diffuse moderate to severe subacute to early chronic gastritis, maximal in the cardia and upper body and around a subacute or early chronic ulcer, 2.4 cm. in diameter, on the lesser curvature of the body. Fairly considerable glandular atrophy in some places. Intestinalisation +. Very few



ULCER & NEIGHBOURING MUCOSA.



SEVERE MUCOSAL INFLAMMATION IN CARDIA AND BODY

Russell cells and eosinophils. Considerable neutrophil migrations.

Areas of severe subacute inflammation in the cardia and upper body showed considerable epithelial atrophy and small acute necroses in the most superficial part of the mucosa. Around the ulcer crater were some small erosions with many polymorphs. There was considerable congestion and oedema of many gastric tips.

There was moderate arterio-sclerosis. Many portions of the muscle of the stomach showed fatty degeneration and atrophy.

S.14. A.E.L. Male, 37.

Clinical Data.

Abdominal pain, vomiting and haematemeses for 3 months before death. Developed obstructive jaundice and became more and more anaemic. The patient had been a heavy consumer of alcohol.

General Autopsy Findings.

Only the abdomen was examined.

Carcinoma of pancreas or common bile duct.

Oesophagus, Stomach & Duodenum.

The veins of the middle third of the oesophagus were more prominent than normal. Some

of them had ruptured. There were several small acute erosions, with polymorphs in the lower third of the oesophagus.

The stomach was large and thin-walled. It contained a large quantity of "coffee-ground" material. It showed the mildest chronic inflammation, maximal in the cardia where there were a few cysts and a small number of goblet cells. There was more glandular atrophy than inflammation Eosinophils were few. In view of the past alcoholic history much more damage to the stomach was expected. In the antrum and body were several ? "solution" erosions. There was a small nodule of glandular hyperplasia in the fundal mucosa, differing from the surrounding mucosa only in its greater thickness. The muscle of the stomach showed a fair amount of fatty degeneration.

The tumour had eroded into the second part of the duodenum.

The most likely source of the haemorrhage was, however, the oesophagus as no bleeding point was found in the duodenum. The first part of the duodenum showed only minimal inflammation.

S. 15. George R. Male, 80.

Clinical Data.

Urinary frequency for 18 months before death. Admitted with acute retention 4 days before death in a uraemic state.

General Autopsy Findings.

Emaciated male. Prostate 2 x normal size because of senile hyperplasia. Marked congestion and trabeculation of bladder. Bilateral hydronephrosis. Kidneys small, pale and irregularly scarred. Brown atrophy of liver.

Stomach:

Mild to moderate chronic inflammation of cardia and fundus, mild inflammation elsewhere. Very few goblet cells and Russell Cells. A small fibro-myoma in the deeper part of the wall of the fundus. Moderate arterio-sclerosis.

S.16. Albert B. Male, 76.

Clinical Data.

Frequency of micturition for 2 years. Haematuria and overflow incontinence for a few months. Admitted 25 days before death with

acute retention for 16 hours. Treated by suprapubic cystostomy and decompression. Conditions deteriorated, became drowsy and died a month after operation.

General Autopsy Findings.

Obese old man with carcinoma of bladder, purulent cystitis and bilateral pyelonephritis.

Stomach:

Very mild diffuse chronic inflammation. More or less normal stomach apart from ? atrophy of deeper mucosal parenchyma in proximal part of stomach. No goblet cells or Russell cells. Neutrophils slight in numbers very few eosinophils. Definite moderate arteriosclerosis.

S. 17. Caroline, P. Female, 60.

Clinical Data.

Carcinoma of cervix treated with radium and deep X-rays. Four months later developed anorexia and lassitude. Two months later developed thrombosis of deep veins of calf-thigh and external iliac vein.

General Autopsy Findings:

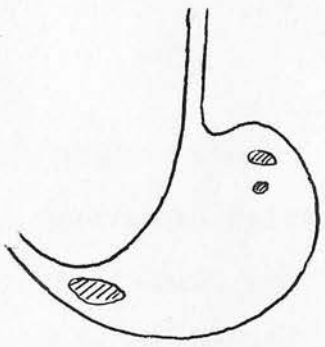
Emaciated female subject. Secondary

carcinomatous nodule in the umbilicus. Thromboses of external iliac, femoral, popliteal and saphenous veins. Thrombosis of portal vein in the porta hepatis with partial infarction of the liver. Pulmonary arteries filled with antemortem clots. Carcinoma of cervix ulteri with extension into vagina, bladder and rectum. Right hydro-ureter and hydro-nephrosis. Finely granular kidneys.

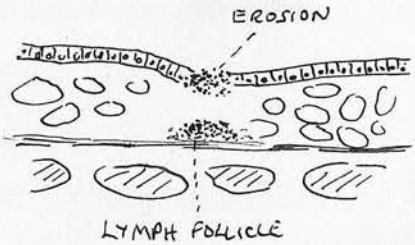
Stomach.

Normal size and shape. Three carcinomatous nodules in the mucosa and submucosa as illustrated below.

Diffuse chronic gastritis, very severe in the fundus, moderately severe to severe elsewhere. Parenchymal degeneration and atrophy were often considerable. Fibrosis of the mucosa sometimes well marked. The thinning and destruction of the



CARCINOMA NODULES.



ACUTE EROSION

fundus was striking.

Lymphocytes were as numerous as, sometimes more numerous than, plasma-cells. Neutrophils and Russell cells were fairly numerous. Goblet cells were present in most sections. Eosinophils were few.

Altered blood pigment was commonly found in the mucosa.

There was a microscopic erosion in the body, showing a few neutrophils and some fibrin, and overlying a lymph follicle.

The mucosa overlying the nodules of carcinoma was comparatively healthy.

S.18. Ellen, O. Female, 38.

Clinical Data.

Admitted 3 months before death with a history of rapid loss of weight, weakness, pallor, fever, anorexia, generalised abdominal pain and vomiting. Found to be suffering from Hodgkin's disease. Was treated with "Nitrogen Mustard" 14 days before death. Developed agranulocytosis. Died suddenly of cerebral haemorrhage.

General Autopsy Findings.

Fairly well nourished female subject. Infected socket right first molar. Generalised

Hodgkin deposits. Multiple petechial haemorrhages. Large haemorrhage in right cerebral hemisphere.

Stomach.

Normal shape. Large in size. Chronic diffuse mild to moderate gastritis, mostly mild, worse in distal stomach. Fairly brisk parenchymal degeneration and atrophy in some areas. No Russell cells. No goblet cells. Eosinophils + → ++ throughout. considerable congestion and oedema of the gastric tips. Numerous fresh petechial haemorrhages. Tumour cells, isolated and in small collections, in the stroma and lymph follicles of the mucosa. Marked muscle degeneration and atrophy.

S.19. Baby K. Female. Still-birth - fulltime.

Clinical Data.

Mother's health good. Foetal movements not felt for 12 hours before delivery. Her first 2 children were born alive and well, Her third child was still-born (? a-p haemorrhage).

General Autopsy Findings.

Infant 20 inches in length and weighed 7 lb. 3 oz. Physically well-developed. Meconium-stained umbilical cord. Only gross finding was multiple petichial haemorrhages.

Stomach.

Normal apart from congestion +++ and small fresh mucosal haemorrhages. Eosinophils fairly numerous throughout the mucosa. Cells in the stroma were almost entirely connective tissue cells.

S.20. Annie D. Female, 84.

Clinical Data.

Patient had had several heart attacks for 4 or 5 years before her death. Her last attack occurred 5 days before her death. O/e: frail, delirious, dyspnoeic incontinent old lady. Cyanosis ++. Cheyne-Stokes' respiration. Oedema of legs. B.P. 180/80. Short aortic diastolic murmur. Auricular fibrillation. Basal vales. Diagnosis: chronic cardiac failure.

General Autopsy Findings. (Autopsy "limited to abdomen").

Emaciated female. Left ventricular

hypertrophy with myocardial scarring. Hepatic cirrhosis ("hob-nail" type). Granular kidneys. Suggestive basal broncho-pneumonia.

Stomach.

Normal shape. Rather small. Most of wall thin. Severe diffuse chronic inflammation, of about equal intensity everywhere, mucosal atrophy striking. Russell cells and goblet cells were found throughout the mucosa (+ to ++). Eosinophils fairly numerous. Brisk mucosal congestion with minute haemorrhages. Fairly severe arterio-sclerosis with well-marked muscle fibrosis.

S. 21. Lucy, R. Female, 91.

Clinical Data.

emaciated, incontinent old lady. Gradually weakened.

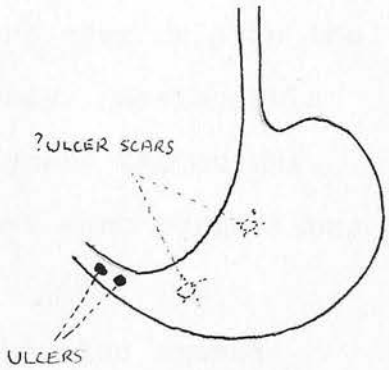
General Autopsy Findings.

An emaciated female subject. Bilateral bronchiectasis. Marasmic pulmonary thrombosis. Acute purulent pericarditis. Atrophic viscera.

Stomach and Duodenum.

Normal shape.

Contained a small quantity of altered blood. Macroscopically, there was a round,



fairly deep ulcer, 1 cm. in diameter and with a dark brown base, just proximal to the pylorus on the anterior wall. A similar ulcer was present on the posterior wall of the duodenum, just distal to the pylorus. In addition to the ulcer in the stomach there were 2 ? scars of old ulcers .... one 2 cm. in diameter, on the posterior wall 6.5 cm. from the pylorus, the other on the anterior wall, 2 cm. from the pylorus, and 1 cm. in diameter.

Microscopically, the ulcers were subacute or early chronic. Sections through the ? healed ulcers showed a thinned, atrophic epithelium and well marked fibrosis of the deeper layers.

There was a mild to moderate to severe chronic gastritis and a severe duodenitis. The gastritis was very patchy. In many sections the covering epithelium was remarkably healthy. Although glandular atrophy was quite marked in

places, it was never gross. Russell cells were few. Goblet cells were seen only in the pylorus and parts of the body. Eosinophils were fairly numerous throughout the mucosa.

All sections showed much congestion, some very much.

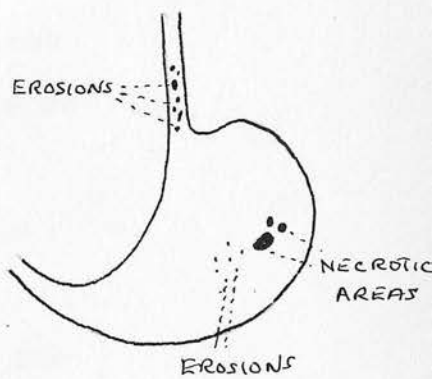
Nearly all sections also showed numerous fresh mucosal haemorrhages. Many showed altered blood pigment.

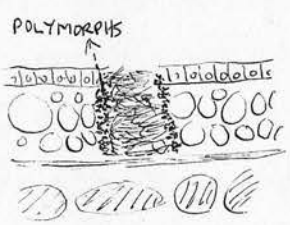
In the fundus there was a minute acute, haemorrhagic, "polymorph" erosion.

S.22. Caroline W. Female, 44.

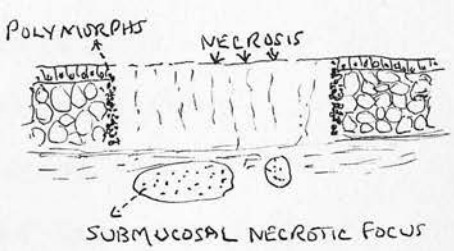
Clinical Data.

Admitted 8 days before death with uncontrolled diabetes and bilateral active pulmonary tuberculosis (multiple cavitation both upper lobes). On day before death developed acute pain in mid-chest and epigastrium





EROSION



NECROTIC AREA

with vomiting; respirations 40-50. Patient was given glucose-saline by rectal and intra-verrous drip but became drowsy and finally unconscious.

General Autopsy Findings.

Bilateral caseous tuberculous foci with early cavitation. Small pancreas. Large intestine contained black faeces. No ulceration seen in duodenum, jejunum, ileum or colon.

Oesophagus and Stomach.

Oesophagus showed severe inflammation, with erosion and ulceration. The appearance was strongly reminiscent of tuberculous infection.

The stomach was of normal shape and size. It contained a moderate amount of altered blood. On the posterior wall of the body, 7 cm. from the lesser curvature and 13.5 cm. from the pylorus, there was a brown "scorched" area, about 1.5 cm. in diameter. Two similar, though smaller, areas were seen just proximal to the large area. Microscopically, these areas were seen to be mucosal necroses overlying sub-mucosal lesions resembling early tuberculous follicles although, as in the case of the oesophagus, no tubercle bacilli were detected with

the Ziehl-Neelson staining method. The necroses were of recent origin and showed numerous polymorphs around the periphery.

There was a mild to moderate diffuse chronic gastritis, mostly mild.

The mucosa showed diffuse congestion and oedema, also some minute fresh haemorrhages. Eosinophils were fairly numerous throughout. There were no Russell cells and no goblet cells.

In the lower body there were several minute "solution" erosions, with polymorph margins.

There was some muscle degeneration and atrophy.

S.23. Alfred I. Male, 61.

Clinical Data.

A few months' history of fatigue and pallor. Was admitted to hospital 9 days before death. Two weeks and one week before admission passed dark red blood per rectum. On admission was comatose, very anaemic and ascitic and had melaena stools. Revived 4 days later after a blood transfusion. Died following a sudden massive haematemesis.

General Autopsy Findings.

Marked multilobular cirrhosis. Fatal haemorrhage from an oesophageal varix. Ascites. No gross infection.

Oesophagus: Varices +++.

Stomach.

Normal size and shape. Filled with a massive blood clot. It showed a diffuse chronic inflammation, severe in sections of fundus and body, mild to moderate elsewhere. Eosinophils fairly numerous throughout.

S.24. Robert, L. Male, 40 hours. 32 weeks.

Clinical Data.

B.B.A. Weight 3 lbs. 15 inches in length. Very cyanosed.

General Autopsy Findings.

Falx cerebri occupied by a large intra-dural haemorrhage. Lungs almost completely atelectatic.

Stomach.

Normal apart from small fresh mucosal haemorrhages. Mucosal stroma contains only a few lymphocytes, no plasma cells, very few neutrophils and small numbers of eosinophils.

S.25. George, C. Male, 78.

Clinical Data.

Several months' anorexia, loss of weight and alternating diarrhoea and constipation. Admitted 3 weeks before death with a large mass in the right hypochondrium and in a confused mental state. Hb 53%. Diagnosed as carcinoma coli.

General Autopsy Findings.

Ulcerating carcinoma of hepatic flexure. Small local abscess. Small duodenocolic fistula. Calcified coronary arteries with some myocardial scarring. No metastases.

Stomach.

Normal size and shape. Diffuse subacute or early chronic diffuse inflammation, worse in the distal ½. Very mild in the fundus, severe in the body (where there was much "intestinalisation"), moderately severe elsewhere. Very few Russell cells. Small numbers of eosinophils.

Parts of the mucosa were very congested and numerous fresh haemorrhages were seen. In the pylorus, some minute, microscopic erosions were observed, formed by the rupture of small haemorrhages through degenerate epithelium. These deficiencies contained many neutrophils.

S.26. Foetus. Female, 18-20 weeks.

Clinical Data.

Mother healthy. Had previously had one ectopic and two healthy pregnancies.

General Autopsy Findings.

Nil, abnormal.

Stomach.

Normal foetal mucosa. A few immature glands. Abundant connective tissue. Considerable congestion of the gastric tips with numerous fresh haemorrhages, particularly marked in the pylorus.

S.27. Thomas, B. Male, 53.

Clinical Data.

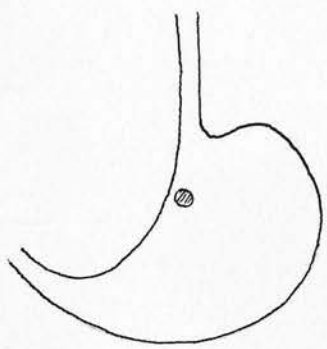
Sudden pain in the chest 3 days before his death and 2 days before admission to hospital. Patient very breathless. Vomited twice. Felt cold. O/e: cyanosed, hyperpnoeic, very restless; rales, impaired percussion and increased vocal resonance right upper lobe; pulse poor; skin cold and clammy. Sulphonamides given since onset of illness. Penicillin started immediately after admission. Heart sounds very faint. B.p. unrecognisable. W.b.c. 24,000. Died a few hours after admission.

General Autopsy Findings.

Syphilitic aortitis with aneurysmal dilatation of the ascending aorta and rupture of the aorta into the pulmonary artery. Endocarditis ? bacterial. Pneumonic consolidation of part of the right upper lobe and bilateral harmorrhagic congestion of the lungs,

Stomach.

There was a diffuse subacute gastritis, with numerous neutrophils and a round, acute ulcer, 0.7 cm. in diameter on the posterior wall of the body, just adjoining the lesser curvature, 4 cm. from the oesophagus. Eosinophils slight in the pylorus. There was considerable fatty muscle degeneration and definite moderate arterio-sclerosis.



S. 28. Foetus ?. 7½ weeks.

Clinical Data.

Incomplete abortion.

General Autopsy Findings.

Well-formed. A few subcutaenous haemorrhages.

Stomach.

Primitive glands in the body. Rich connective tissue.

ACTUAL SIZE & SHAPE

S. 29. Foetus, 24 weeks.

Clinical Data.

Mother had toxaemia of pregnancy.

General Autopsy Findings.

Nil gross.

Stomach.

Normal size and shape. Simple body glands present with faintly-acidophilic cells. A few definite lymphoid follicles. Very slight numbers of eosinophils present. Small fresh haemorrhages present in the mucosa, arranged mostly along the longitudinal rugae.

S.30. Cecil, S. Male, 51.

Clinical Data.

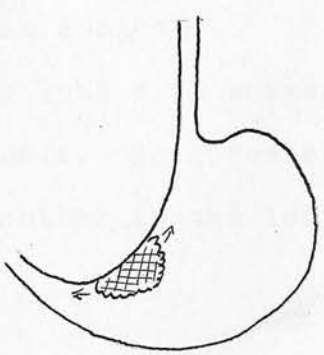
Deep thrombosis of left leg 6 months before death. Admitted to hospital 2 months later. O/e. hepatomegaly and gross ascites. Bipsy of supraclavicular lymph node showed a ? bronchial carcinoma. One week before death developed a staphylococcal cellulitis of the face which did not respond to penicillin.

General Autopsy Findings.

Carcinoma of lesser curvature of stomach. Englarged lymph nodes in the porta hepatis. Cirrhosis of liver. Splenomegaly. Thrombosis of inferior vena cava and superior mesenteric vein.

Stomach.

The stomach was of normal shape and size. Lymph nodes along the curvatures were infiltrated with tumour. Beginning 2.5 cm. from the pylorus, there was an extensive cauliflower mass of anaplastic carcinoma. There



were, microscopically, extensive local infiltrations of the stomach wall.

The mucosa showed moderate to severe inflammation, mostly severe, either chronic with acute features or else subacute, worse in the distal stomach. Russell cells, goblet cells and eosinophils were abundant. There were a few small mucosal haemorrhages.

S. 31. Arthur, A. Male, 57.

Clinical Data.

5 years before death had a left basal penumonia. Never fully recovered health. Four years later developed pleural pain on left side. Fifteen days before death was weak and irrational for one day. A few days later the patient had a fit and became unconscious.

General Autopsy Findings.

Thin, emaciated male subject. Bronchiectasis of left lower lobe with abscess-formation and broncho-pneumonia. An abscess in the left frontal lobe and another in the left occipital lobe of the brain.

Stomach.

Macroscopically there was a severe diffuse chronic gastritis, with a fair amount

of atrophy in places. The inflammation was worse in the distal stomach. Goblet cells were numerous. Russell cells were very few. Eosinophils slight to +. Only very slight fresh mucosal haemorrhages. No arterio-sclerosis.

S. 32. Augustus, H. Male, 77.

Clinical Data.

Bedridden for 1 year prior to death with arthritis of knees, shoulders and elbows; also had difficulty in passing water. Was admitted 8 days before death having had acute retention of urine for 3 days before admission. O/e: enlarged, prostate, enlarged bladder, uraemia, basal rales.

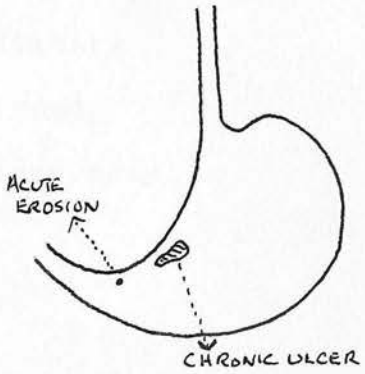
General Autopsy Findings.

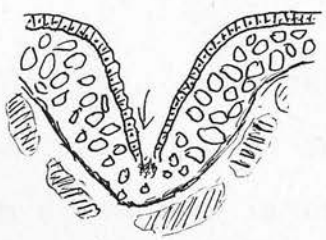
Obese male subject. Benign prostatic hyperplasia with local abscess formation. Cystitis. Chronic pyelonephritis. Death from uraemia.

Stomach.

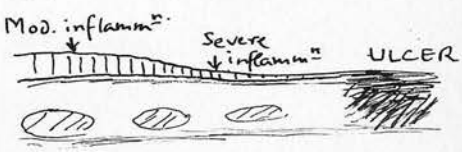
Normal size and shape. Chronic ulcer, 1.2 cm. in diameter. 6 cm. from pylorus on the lesser curvature.

There was a diffuse moderate to severe chronic inflammation, mostly severe, maximal (with the exception of the mucosa immediately skirting the ulcer) in the cardia and fundus. Goblet cells and eosinophils slight to ++. Very few Russell Cells.





ACUTE EROSION



CHRONIC ULCER & NEIGHBOURING MUCOSA.

A small acute erosion at the pyloroduodenal junction .... a little deficiency in a degenerate epithelium containing some neutrophils.

Whereas the covering epithelium in the areas of maximal inflammation was very degenerate, in other areas of lesser, but still severe inflammation the covering epithelium and the most superficial parts of the glands were remarkably healthy.

Note that the mucosa immediately skirting the ulcer was severly inflamed, but that a slightly increased distance away was only moderately inflamed.

S. 33. Frederick, J. Male, 87.

Clinical Data.

Carcinoma of prostate. Per-urethral resection of prostate performed. Died following a pulmonary embolism a few days later.

General Autopsy Findings.

Thin, aged male. Infarct right upper lobe. Cystitis and pyelonephritis.

Oesophagus.

Showed 2 features: (1) extensive acute erosion and ulceration; (2) a nodule of well-differentiated adeno-carcinoma, 1 cm. in diameter, bulging the epithelium.

Stomach and duodenum.

Normal size and shape. Very mild to mild chronic gastritis, maximal in the distal part. Russell cells slight to + in the pylorus. No goblet cells. Few eosinophils. Quite considerable congestion and oedema. A little fresh mucosal haemorrhage. Numerous haemorrhagic

erosions in the lower body and antrum. Two metastases in the mucosa of the fundus.

There was a mild duodenitis. The duodenum (first part) also showed in its mucosa a small metastasis and a small acute erosion with many polymorphs.

Mild arterio-sclerosis. No muscle changes.

S.34. Martin, A. Male, 24.

Clinical Data.

8 years before his death the patient was diagnosed as having Pott's disease of the spine .... affection of T 10, 11 and 12.

A fortnight before death developed a large pleural effusion and had abdominal pain. Suddenly collapsed and died.

General Autopsy Findings.

Pulmonary embolus originating from thrombosed veins in the thigh. Tuberculous osteomyelitis of spine. Cold abscess right thigh. Tuberculous pleural and peritoneal effusions.

Stomach.

Normal size and shape. Very mild to severe chronic inflammation, maximal in the

body and antrum, mild or very mild in the pylorus and fundus. Very few goblet cells. No Russell cells. Eosinophils slight to +. Congestion and oedema ++. Scanty small fresh mucosal haemorrhages.

Small areas of the body mucosa were packed with "foam-cells" .... ? significance.

Very severe fatty degeneration of muscle.

Some of the arteries showed thickening and hyalinosis of the intima..

S.35. Joseph, S. Male, 30.

Clinical Data.

Acute nephritis when 18 years old. Albuminuria for 12 weeks. No further symptoms until 8 months before death when there were the following: blurring of vision, diarrhoea, headache, polyuria, excessive thirst. Eight days before death there was bilateral papilloedema with exudate and small haemorrhages in the retinae; also purpuric haemorrhages on the chest and in the conjunctivae. B.p. 170/130. Blood urea 220 mgm. %. No oedema. Urine: protein +, hyaline and granular casts +. Death from uraemia.

General Autopsy Findings.

Chronic nephritis. L. ventricular hypertrophy. Uraemic pericarditis.

Stomach.

Mild to moderately severe chronic gastritis, mildest in parts of the body and pylorus. Very few goblet cells and Russell cells. Eosinophils scanty.

Marked arterial hypertrophy. No special muscle changes.

S.36. Jane, F. Female, 1 year.

Clinical Data.

A month before death began having difficulty in breathing. A week before death vomited, was drowsy and had one "stiff" turn. Remained drowsy. Diagnosis: either tuberculous meningitis or a cerebral abscess.

General Autopsy Findings.

Well nourished female child. Primary tuberculous focus in the lung. Calcified hilar and pre-aortic lymph nodes. Tuberculous meningitis.

Stomach.

Normal size and shape. No gastritis. Eosinophils very scanty. The section of

pylorus showed numerous mitotic figures in the gland cells.

S.37. Baby C. Stillbirth. Fulltime.

Clinical Data.

First child. Protracted labour. Final forceps.

General Autopsy Findings.

Nil significant found.

Stomach.

Normal apart from showing a few recent mucosal haemorrhages. Eosinophils slight to +.

S.38. Foetus ? $7\frac{1}{2}$ weeks.

Clinical Data.

Abortion. Mother's health good. Foetus 2.2 cm. long and somewhat macerated.

Stomach.

It consisted of a simple tube with no formed glands. There was a definite though immature muscle layer. Submucosa and mucosa consisted of rich

SIZE & SHAPE OF STOMACH

connective tissue. There was some slight orientation of cells at the lumenal surface of the "mucosa".

S.39. Baby W. Male, Three months.

Clinical Data.

Admitted 3 weeks before death with broncho-pneumonia. Later developed "gastro-enteritis".

General Autopsy Findings.

Pale, thin, cyanosed child. R. pleural effusion. Fatty liver.

Oesophagus.

There was an ulcer 1.5 x 0.9 cm. in the terminal 2 cm.

Stomach.

Showed no evidence of inflammation past or present. Eosinophils +. Multiple fresh haemorrhages in the stomach wall.

S.40. Baby H. Female, One day. 25 weeks.

Clinical Data.

Prematurity.

General Autopsy Findings.

Normal physical appearance for 25 weeks.

Stomach.

Slight haemorrhages. Eosinophils few.

S.41. Robert, S. Male, 62.

Clinical Data.

History of chronic bronchitis and auricular flutter controlled by digitalis. Admitted 6 days before death with broncho-pneumonia.

General Autopsy Findings.

Large, obese male. Bronchiectasis both lower lobes. Broncho-pneumonia. Fatty infiltration of heart.

Stomach.

Normal size and shape. Moderate chronic inflammation in the sections of cardia and pylorus. Elsewhere normal or only slightly chronically inflamed mucosa. Goblets cells + in the areas of maximum inflammation. Very few Russell cells or eosinophils. No arterio-sclerosis.

S.42. Alice B. Female, 57.

Clinical Data.

Congenital cystic kidneys. Severe uraemia.

Oesophagus.

Showed very severe acute inflammation with

erosions and ulceration.

Stomach.

Normal size and shape. Contained about a pint of fresh blood.... this presumably came from the oesophagus.

Severe diffuse chronic inflammation of about equal intensity everywhere.

Much glandular atrophy. Surface epithelium remarkably well preserved.

Goblet cells slight to +. Russell cells very few. Eosinophils slight to +.

Superadded acute inflammation in many areas, mostly mild to moderate but very severe in the cardia where there was an acute erosion as in the oesophagus. A few small fresh haemorrhages were present in the mucosa.

There was mild arterio-sclerosis and mild degeneration and atrophy of muscle.

S.43. Baby R. Male, 2 weeks.

Clinical Data.

Imperforate anus at birth. Vomiting persisted after surgical opening and stools became watery. Diagnosis: gastro-enteritis.

General Autopsy Findings.

Lungs showed grossly haemorrhagic upper lobes. Liver fatty.

Oesophagus.

Patchy moniliasis.

Stomach.

Normal size and shape. There was a diffuse, moderately severe "chronic" inflammation. The glands are swollen and basophilic. Some are vacuolated, others atrophied slightly. The predominant cells in the stroma are ? juvenile plasma cells and earlier cells.

There are a few small fresh harmorrhages. Eosinophils slight to +.

S.44. Infant T. Male, 7 days. 37 weeks.

Clinical Data.

4th child. The other 3 pregnancies ended as miscarriages. Normal L.O.A. Mild but persistent asphyxia livida. Birth weight 3 lbs. 8 ozs. Measured 16 inches. Developed generalised oedema on the second day which persisted. Unable to swallow properly, so fed by oesophageal tube.

General Autopsy Findings.

Immaturity., bilateral gross atelectasis. Cardiac dilatation.

Stomach.

Normal apart from considerable congestion and a few small fresh haemorrhages in different parts of the wall. Eosinophils few.

S.45. Foetus. 4 months.

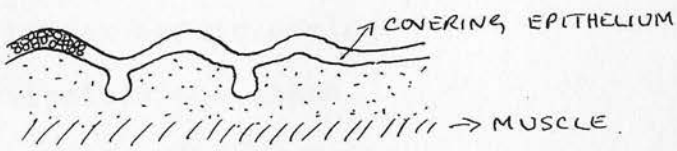
Clinical Data.

Ex-hysterotomy.

Stomach.

The surface epithelium was undulating and consisted of closely packed cells, basophilic and showing none of the clear, basophilic and showing none of the clear, columnar characteristics of mature covering epithelium. Arising from this

SIZE & SHAPE OF STOMACH.



epithelium were several little downward protrusions, from the sides and base of which little buds were growing.

The muscle layer proper was well developed but there was no muscularis mucosae. Between the covering epithelium and the muscle layer there was a thick layer of connective tissue containing many cells having poorly-staining cytoplasm and large nuclei of variable shape and also primitive blood vessels.

S.46. William, G. Male, 72.

Clinical Data.

6 months' history of flatulence and abdominal discomfort with watery stools. Developed an ischio-rectal abscess which was successfully drained. He was later discovered to have an adeno-carcinoma at the pelvi-rectal junction. An abdomino-perineal resection of the rectum was performed 1 week before death. The patient developed peritonitis and ileus and died despite penicillin and sulphamerazine.

General Autopsy Findings.

Well-developed muscular male. Leaking colostomy wound. Generalised peritonitis. No pneumonia.

Stomach.

Mild to moderately severe chronic inflammation of the cardia and pylorus. Elsewhere only mild inflammation. No goblet cells. Only one or two Russell cells. Very few neutrophils. Eosinophils few. No arteriosclerosis.

On the whole a comparatively healthy stomach.

S.47. Foetus. 3 months.

Clinical Data.

Ex-hysterotomy.

Stomach.

Similar to S.45 only less mature.

S.48. Foetus. 3 months.

Clinical Data.

Ex-hysterotomy.

Stomach.

Similar to S.47.

S.49. Howard, F. Male, 20.

Clinical Data.

10 years' peroneal muscular atrophy.

3 month's ulcerative colitis.

General Autopsy Findings.

Non-specific ulcerative colitis. Late subacute peritonitis. Broncho-pneumonia. Muscular atrophy in all 4 limbs. Very emaciated young adult male.

Oesophagus.

Severe moniliasis.

Stomach.

Normal size and shape. No gastritis, only mild atrophy of the glands. Few eosinophils.

S.50. Margaret, B. Female, 37.

Clinical Data.

Admitted 2 days before death in a state of shock. There had been pain in the left thigh for 7 days, a tender lump in this thigh for 4 days and swelling and blueness of the left lower limb for 24 hours. The diagnosis was made of deep thromboses of the left calf, femoral and external iliac veins. The patient remained very shocked and died suddenly.

General Autopsy Findings.

Very obese body. Marked oedema of left lower limb. There was marked pulmonary congestion with a large thrombus in the left pulmonary

artery. There was thrombosis of the saphenous and femoral veins in the left thigh and there were some small cortial infarcts in the kidneys.

Oesophagus.

There was an acute oesophagitis of moderate severity.

Stomach.

Apart from mild chronic inflammation in the cardia the mucosa was healthy. Eosinophils few. No arterio-sclerosis.

S.51. Lilian, R. Female, 57.

Clinical Data.

For nearly 3 months the patient was in hospital with a right-sided lymphocytic pleural effusion ? tuberculous. A fortnight before her death there was some cough and severe left-sided pleural pain.

General Autopsy Findings.

Pulmonary carcinoma, probably originating in a bronchus, with gross lymphangitis carcinomatosa. Also a healed tuberculous focus in the lung.

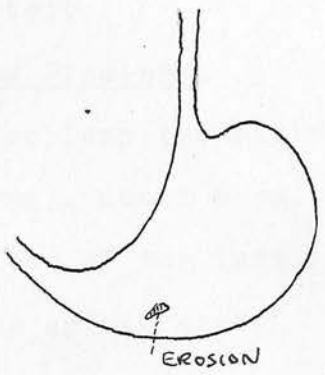
Stomach and Duodenum.

Stomach large and of normal shape. Lymph nodes along lesser curvature very hard. There was

a large erosion on the posterior wall of the antrum, 8.5 cm. from the pylorus, 1.5 cm. in diameter.

Moderately severe to severe chronic inflammation, with superadded acute elements, in the pylorus and around the central erosion. Moderately severe chronic inflammation in the cardia. Elsewhere mild chronic inflammation.

Few goblet cells and eosinophils. Russell cells vary few. The lymph foci in the pyloric mucosa were large and contained tumour cells. The section of the fundus showed a lymphatic in the extra-peritoneal fatty tissue packed with large tumour cells.



No arterial or muscle changes.

There was a moderately severe to severe chronic duodenitis with acute elements. In the tip of one villus, in the first part, where there was severe congestion, oedema and cellular infiltration, there was a small erosion containing fibrin and polymorphs.

S.52. Charles C. Male, 55.

Clinical Data.

Admitted 13 days before death with complete paralysis of the lower limbs, due apparently to a transverse myelitis. C.S.F. normal. No evidence of a spinal block. Four days before death a suprapubic cystostomy was performed. Two days later a Bact. Coli infection was found in the urine and a course of Sulphamezathine started.

General Autopsy Findings.

A peripheral neoplasm (possibly alveolar-celled carcinoma), about 6 cm. in diameter in the lower lobe of the left lung. Secondary deposit in the spinal cord.

Stomach.

Large stomach of normal shape. There was a mild to severe subacute or early chronic inflammation, maximal in the body. Few goblet cells and eosinophils. Neutrophils + to ++. Very few Russell cells. Some sections showed more parenchymal atrophy than cellular infiltration. On the lesser curvature there were three linear depressions which microscopically

showed considerable atrophy of the mucosa with much cellular infiltration .... ? healed erosions.

There was moderate arterio-sclerosis and slight to moderate muscle fibrosis.

S.53. Edward, P. Male, 80.

Clinical Data.

In hospital for 3 months prior to his death. Senile dementia. Hyperiesia: 210/120. ? nutritional deficiency .... sore, red tongue and red, oedematous hands.

General Autopsy Findings.

Very emaciated old man. Oedema of both upper limbs below the elbow. Skin red. Small brown heart: rather hypertrophied left ventricle with much fibrosis of the myocardium. No striking coronary atheroma. Lungs not examined. Small granular kidneys. Small brown liver. Spleen about twice expected size; firm; normal colour.

Stomach.

Normal size and shape. There was a diffuse chronic gastritis, very severe in the fundus and antrum, moderately severe to severe elsewhere. Atrophy everywhere, very marked in

the sections showing severe inflammation. Goblet cells ++ in cardia, +++ in pylorus. Very few eosinophils. Numerous neutrophils in some areas. Hardly any Russell cells. Surface epithelium showed much degeneration.

Moderate degree of arterio-sclerosis. Some muscle atrophy.

S.54. John, F. Male, 78.

Clinical Data.

Old bronchitic and hypertensive. o/e: afebrile, cyanosed, orthopnoeic, confused. Congestive cardiac failure.

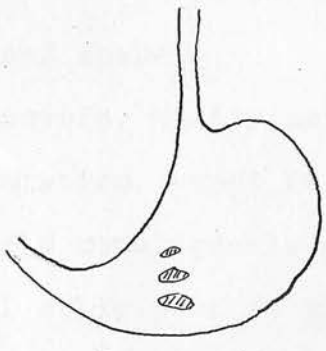
General Autopsy Findings.

Emaciated old man. Bronchectasis. Broncho-pneumonia.

Stomach.

Small. Normal Shape.

There were 3 acute ulcers in the anterior wall of the body, the smallest 0.5 cm. in diameter, the largest 1.5 cm.



3 ACUTE ULCERS

Diffuse moderately severe to very severe

chronic gastritis.

Marked parenchymal atrophy in many areas.

Surface epithelium largely degenerate. Many goblet and Russell cells. Neutrophils +. Eosinophils, hardly any. Fibrosis +.

Well-marked arterio-sclerosis and muscle fibrosis.

S.55. Jessie, W. Female, 69.

Clinical Data.

Cerebral thrombosis and broncho-pneumonia.

General Autopsy Findings.

Cerebral thrombosis with softening of the right internal capsule, and broncho-pneumonia superimposed on a bronchiectatic left lower pulmonary lobe.

Stomach.

Normal size and shape.

Moderate to severe, mostly severe, diffuse chronic inflammation, worst in the fundus, body and antrum. Fairly considerable glandular atrophy. No acidophil cells seen in glands. Fibrosis +. Goblet cells plentiful. Russell cells

hardly seen. Not many neutrophils. Eosinophils slight to +. Surface epithelium remarkably healthy.

Slight to moderate arterio-sclerosis. Well-marked muscle fibrosis.

S.56. Albert, M. Male, 45.

Clinical Data.

Pains in chest for 12 months, headaches and lassitude for 9 months, breathlessness for 3 months. O/e: apex beat one inch outside normal limit. Papilloedema.

General Autopsy Findings.

Bronchectasis and lobar pneumonia left lower lobe. Hypertensive heart with an old infarct in the left ventricle.

Stomach.

Normal size and shape.

Moderate chronic gastritis in the cardia, mild chronic gastritis in the fundus and pylorus. In the body and antrum there is a patchy atrophy of the parenchyma of the middle and deeper mucosa without inflammatory cell infiltration. The epithelial cells at the gastric tips in the cardia show an extraordinary vacuolation ... usually each cell containing one large vacuole.

No goblet cells, Russell cells or neutrophils. Hardly any eosinophils.

Well marked arterial hypertrophy. No muscle changes.

S. 57. Arthur, W. Male, 59.

Clinical Data.

Diagnosed as a case of bronchiectasis, with broncho-pneumonia. Clubbed fingers, feverish, rather dyspnoeic. Right empyema 8 years ago and pneumonia 2 years ago. Winter coughs.

General Autopsy Findings.

Emaciated male. Extensive bilateral bronchiectasis with broncho-pneumonia. Septic spleen. Fatty liver with vascular thromboses and extensive areas of necrosis.

Stomach and Duodenum (1st Part).

Stomach was of normal size and shape. It contained a small quantity of altered blood.

Fairly severe chronic inflammation in the cardia. Goblet cells +. Mild chronic inflammation in the pylorus. Remainder of mucosa quite healthy. No Russell cells. Very few eosinophils. No arterio-sclerosis or muscle change.

There was a fairly severe chronic duodenitis with numerous acute erosions, with polymorphs, and one acute ulcer formed from the extension of an erosion.

S. 58. Baby, H. Male, 3 months.

Clinical Data.

Diagnosed as a case of transposition of the viscera with pyloric or duodenal ileus.

General Autopsy Findings.

Transposition of the viscera, e.g. heart was on the right side of the thorax; partial obstruction of the small intestine... most of the small gut was twisted around the root of the mesentery.

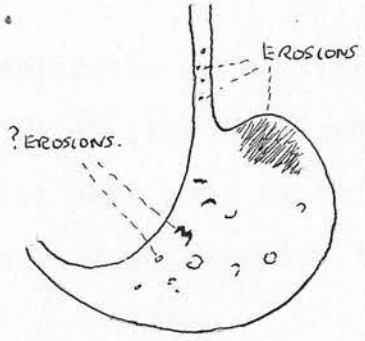
There was no severe obstruction. The whole of the small intestine contained green bile-stained mucus. Death from malnutrition.

Oesophagus.

Patches of severe acute inflammation, with erosion.

Stomach and Duodenum.

Macroscopically, the stomach showed a large patch of ? monilia infection in the fundus and a number of congested patches and ? erosions in the body and antrum. Microscopically the former was an area of brisk acute inflammation



EROSIONS & ?EROSIONS

of the surface layers of the mucosa, with much oedema and many polymorphs, and pseudo-membrane formation (fibrin ++, polymorphs +++ and bacteria ++). Monilia was absent. Section through the doubtful erosions revealed glandular atrophy with loss of covering epithelium at the surface of the mucosa, the surrounding mucosa being healthy. In one section there was a collection of polymorphs in the centre of the atrophied area.

The section of pylorus and many parts of the antrum and body were normal apart from slight degenerative changes in the parenchyma. Other parts of the antrum and body, together with much of the cardia and most of the fundus, showed considerable parenchymal degeneration and atrophy.

Eosinophils were very few and there were no chronic cell infiltrations.

Section of the first part of the duodenum showed interesting lesions in the tips

of some villi .... considerable congestion and oedema, and some superficial necroses leading to acute erosions. These were most striking. Some polymorph infiltrations accompanied these changes.

S.59. Frederick, S. Male, 69.

Clinical Data.

General debility. Had lost desire to live.

General Autopsy Findings.

Emaciated subject. Marked atheroma of the basilar and middle cerebral arteries. Atrophic brain with multiple cysts, especially in the putamen and thalamus. Broncho-pneumonia. Prostatic calculi. One small focus of pyelo-nephritis in the cortex of one kidney.

Stomach.

Mild chronic inflammation in the cardia and fundus and moderately severe chronic inflammation in the antrum and pylorus. Body healthy. Often rather more atrophy than inflammatory cell collections. Neutrophils +. Small numbers of goblet cells in the antrum and pylorus. Very few Russell cells. No eosinophils.

Moderate arterial hypertrophy. Slight fibrosis of muscle.

S.60. William, L. Male, 61.

Clinical Data.

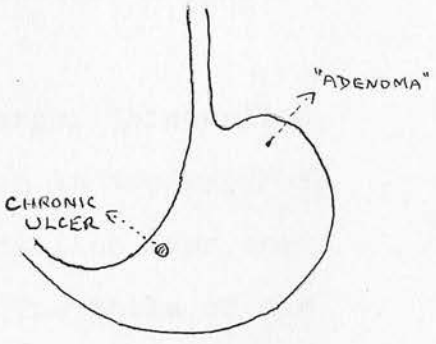
Crohn's disease diagnosed at laparotomy in 1944. i.e. 4 years before death. In 1946 developed right lower lobe pneumonia and had a slight cough for ever afterwards. In 1947, bronchoscopy showed a carcinoma of the right lower lobe bronchus. He was treated with deep X-rays.

General Autopsy Findings.

Carcinoma of the right lower lobe with abscess formation.

Stomach.

Normal size and shape. 6 cm. from the pylorus, on the posterior wall of the body near the lesser curvature, was a deep "punched-out" chronic ulcer, 0.5 cm. in diameter.



In the fundus there was an "adenoma", 2 mm. in diameter, of exactly the same structure as the surrounding mucosa and not encapsulated.

There was a diffuse subacute inflammation, mild in the cardia, fundus and body sections, moderately severe in the pylorus and very severe around the ulcer. Goblet cells slight to +. Eosinophils very scanty. Very few Russell cells. Striking brown-pigment - containing cells in the connective tissue of the mucosa of the pylorus.

S.61. Mrs. B. Female, 73.

Clinical Data.

Hypertension with senile cerebral changes. Fracture of right femur.

General Autopsy Findings.

Not available. (Coroner's Case).

Stomach.

Shape normal. Large, thin-walled. Severe chronic inflammation in the pylorus. Very severe chronic inflammation over the remainder of the mucosa. The whole of the mucosa showed terrible devastation. There

was very considerable atrophy of the mucosa, with complete disappearance of many glands. Also considerable degeneration of surviving epithelium and enormous stuffing of the mucosa with plasma cells and lymphocytes. Lymph foci were very prominent. Neutrophils were numerous, eosinophils fairly numerous. Only a few Russell cells present. Numberous goblet cells. Mucosal fibrosis +. Gross arteriosclerosis. Remarkably little muscle fibrosis.

S.62. Janet, M. Female, 5 years.

Clinical Data.

Diagnosed as a case of probable tuberculous meningitis. Unconscious for about a week before death.

General Autopsy Findings.

Primary tuberculous focus in right upper lobe. Miliary spread. Tuberculous meningitis.

Oesophagus.

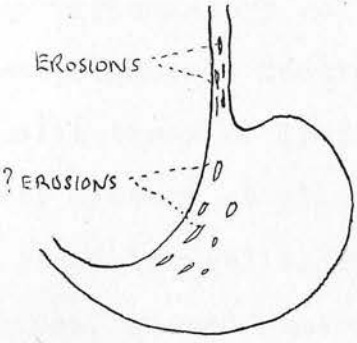
Showed acute inflammation with erosions and ulceration.

Stomach.

Normal size and shape.

Much of the mucosa was of healthy appearance.

Macroscopically there were 2 kinds of lesions in the mucosa of the body... areas of congestion and depressed areas that might be erosions. Both types measured from 2 x 2 mm. to 3 x 10 mm.



Microscopically the congested patches showed considerable congestion and oedema of the gastric tips. The depressed areas showed a transition from healthy mucosa to poorly-staining, digested mucosa. In the centre of the digested area of one of the body sections there was a small collection of fibrinoid material, a few polymorphs and a fresh haemorrhage.

The section of pylorus showed one small but very definite acute erosion, with fibrinoid material at the surface and a few polymorphs and a very definite fresh haemorrhage just beneath. The surrounding mucosa was beginning to show evidence of digestion. Neighbouring gastric tips were very congested and oedematous.

It would seem that the erosions and the digested areas had a common origin in small haemorrhages in the mucosa.

There were no inflammatory cells present except the few polymorphs mentioned and sometimes small collections of lymphocytes. Eosinophils were hardly present at all.

Many of the glandular cells, especially those in the upper mucosa, showed numerous mitotic figures.

There was some muscle degeneration and atrophy.

S.63. George, C. Male, 57.

Clinical Data.

Loss of weight for 18 months. Slight "stroke" 6 weeks before death. Two weeks before death. Two weeks before death sudden onset of dyspnoea and tightness in the chest. O/e: 240/160; cyanosis; dyspnoea; unfolded aorta; myocardial damage revealed by E.C.G; enlarged liver; consolidation left lung; enlarged heart. Diagnosis: coronary occlusion and broncho-pneumonia.

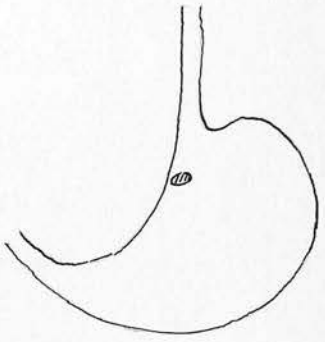
General Autopsy Findings.

Emaciated male. Enlarged R. & L.

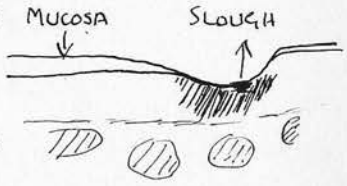
heart. Fibrosed patches in myocardium. Scarred kidneys. Marked pulmonary oedema. Emphysema present in upper lobes and bronchiectasis in the lower. Uraemic pericarditis and colitis. Death from ureaemia.

Stomach.

Normal size and shape. An ulcer, apparently 0.5 cm. in diameter 3.5 cm. from the oesophagus on the lesser curvature. Microscopically, it was seen to be a partially healed chronic ulcer. Only a small area showed true ulceration. On each side of this area the mucosa was very thin, extremely degenerate, very congested and haemorrhagic. Below this mucosa there was much fibrosis.



CHRONIC ULCER.



The remainder of the stomach mucosa showed a diffuse chronic inflammation with acute elements, fairly severe to severe, rather worse in the distal half of the organ and very severe around the ulcer.

There was much glandular degeneration. Goblet cells were numerous and so were neutrophils. Russell cells were scanty. Eosinophils were few.

The mucosa was much congested and there were multiple fresh haemorrhages in the mucosa,

especially in the gastric tips.

There was very severe arterio-sclerosis.

S. 64. Renfold, D. Male, 62.

Clinical Data.

First admitted 1 year before death as a P.U.O. Had an enlarged liver, some albuminuria and a small ascites. Readmitted with fever, jaundice and hepatomegaly 5 months before death. Three months later developed subacute intestinal obstruction with a tender mass in the umbilical region. At laparotomy straw-coloured fluid ++ was found in the peritoneal cavity and loculated pus between the liver and the hepatic flexure.

General Autopsy Findings.

Extensive bronchiectasis left lower lobe. Old empyema left chest. Widespread loculated purulent peritonitis. Macroscopic resolving cholangitis. Amyloidosis of the spleen and kidneys.

Oesophagus.

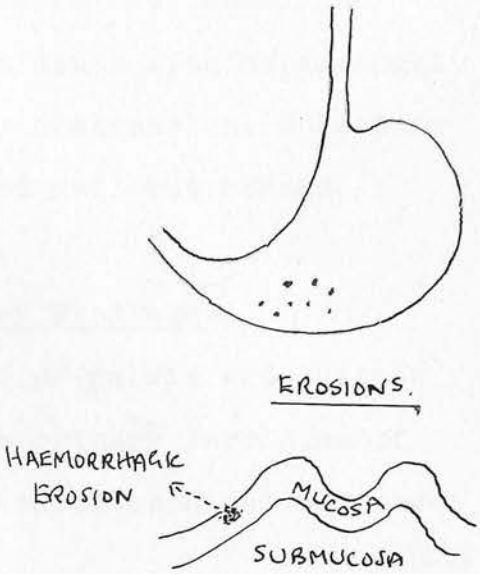
Diffuse monilia infection.

Stomach.

Normal size and shape. Some pus adherent

to the serosal surface.

The mucosa showed a very patchy chronic inflammation, very slight to severe in quality and worst in parts of the body. Many parts of the mucosa were remarkably healthy. Goblet cells ++. Neutrophils slight to +. Some gastric tips were very congested and haemorrhagic. There was much ? altered blood pigment in the mucosa.



Acute haemorrhagic erosions, varying in size from a pinhead to about 4 mm. in length and 2 mm. in breadth, were present in the lower body and antrum. They contained very few polymorphs and showed evidence of slight digestion around. The neighbouring mucosa was either quite healthy or showed moderate inflammation.

Some sections showed a resolving peritonitis.

S. 65. Alic, S. Female, 65.

Clinical Data.

Radium and Deep X-Rays for ? carcinoma vulvae 2 years before death. Admitted one month before death with hepatomegaly and subacute intestinal obstruction. Colostomy performed and functioned well but became increasingly jaundiced.

General Autopsy Findings.

Carcinomatosis of pelvis and abdomen arising from a probable primary carcinoma of uterus. Bilateral bronchiectasis and bronchopneumonia.

Stomach.

Normal size and shape. There was a mild to moderately severe diffuse chronic gastritis, more mild than otherwise and with rather more glandular atrophy than cellular infiltration in some parts. No goblet cells. Very few Russell cells. Eosinophils + throughout.

S.66. Arthur, S. Male, 79.

Clinical Data.

An old tabo-paretic. Six weeks before

death had diarrhoea and abdominal pain.
Cystostomy performed for urinary retention.
Began passing faeces through the catheter.
Steadily deteriorated.

General Autopsy Findings.

Ulcerative carcinoma of the pelvic colon. Faecal fistula connected with cystostomy. Pyonephrosis. One large secondary deposit in the liver.

Stomach.

Normal size and shape. Mild to moderately severe diffuse chronic gastritis of about the same intensity everywhere. Surface epithelium remarkably healthy. Goblet cells + in the cardia and pylorus. Very few Russell cells. Neutrophils +. Eosinophils slight to +. Quite a lot of atrophy ofthe glands. Mild arterio-sclerosis. No muscle change.

S.67. Ellen, W. Female, 77.

Clinical Data.

Long history of duodenal ulcer. Gastro-enterostomy a few months before death. Readmitted to hospital with melaena. Became uraemic.

General Autopsy Findings.

Obese old lady. Gross bronchiectasis of the right lower lobe and some broncho-pneumonia

of the left lower lobe. Small, scarred kidneys. Markedly calcified aortic valve.

Stomach.

The first loop of jejunum had been anastomosed to the posterior wall of the stomach. The pylorus was markedly stenosed. The pylorus showed a mild chronic inflammation of the mucosa. The remainder of the stomach showed no inflammation. There were no goblet or Russell cells, very few neutrophils and small numbers of eosinophils. There was moderate arteriosclerosis and slight muscle fibrosis.

Small Intestine:

There was a large chronic ulcer on the posterior wall of the first part of the duodenum. There was also a large chronic ulcer in the jejunum, just beyond the stoma. The whole of the small intestine was markedly congested and showed multiple small, intensely haemorrhagic ulcers along its length ....? uraemic.

S.68. Elsie, S. Female, 64.

Clinical Data.

Admitted 5 days before death with a 3 weeks' history of vague epigastric pain,

radiating to the right side of the chest. Vomited one day. Urine dark. o/e: T.100. P.104. Looked ill. Remained febrile. Deepening jaundice. Casts and red cells in the urine. Raised blood urea. Sank into coma and had convulsions. Diagnosis: acute cholecystitis and cholaemia.

General Autopsy Findings.

Obese cadaver. Marked jaundice. Biliary obstruction due to impacted metabolic calculi in the common bile duct. Cholangitis with "cholaemia" and jaundice. Biliary nephrosis.

Oesophagus.

There was a brisk acute oesophagitis with extensive erosion.

Stomach.

Showed a diffuse chronic gastritis, mild to moderately severe in the pylorus, severe to very severe elsewhere. There was well-marked glandular atrophy throughout, very striking in the areas of maximal inflammation. There were no goblet cells or Russell cells. Few neutrophils. Eosinophils slight to ++. The surface epithelium was remarkably healthy in comparison with the rest of the mucosa. There was no arterio-sclerosis.

S.69. Annie, R. Female, 73.

Clinical Data.

Sudden hemiplegia 2 weeks before death. B.p. 210/130. Fibrillating heart.

General Autopsy Findings.

Fairly marked cerebral atheroma. Large cerebral haemorrhage. Contracted kidneys. Hypertensive heart with a scarred left ventricle.

Stomach.

Normal size and shape. Mild chronic inflammation of the antrum. No goblet cells. Very few Russell cells and eosinophils. Surface epithelium very healthy. Moderate arterio-sclerosis. No striking muscle changes. A remarkably healthy stomach.

S.70. Margaret, B. Female, 77.

Clinical Data.

Upper abdominal pain for 4 years before death. Admitted to hospital 14 days before death as a case of suspected perforation of the gall-bladder. Laparotomy performed and a diagnosis of acute pancreatitis was made. The patient died of a pulmonary embolus.

General Autopsy Findings.

Marked obesity. Thrombosis of deep veins in the left calf. Large thrombus in the pulmonary

artery. Acute pancreatitis. Signs of a recent cholecystitis. Multiple abdominal adhesions.

Oesophagus.

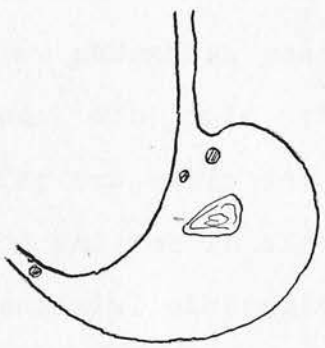
Showed acute inflammation with extensive areas of erosion.

Stomach.

There were 3 chronic ulcers in the body, 2 of them measuring about 0.5 in diameter, the third measuring about 4 x 2 cm. and covered with laminated blood clot.

There was also an acute erosion in the cardia.

Patchy chronic gastritis, as follows: mild to fairly severe in the cardia, moderately severe to severe in the pylorus, mild in the antrum, nil in the fundus and body except around the body ulcers where there was a margin of severe inflammation. There was striking health of the covering epithelium. The abrupt change from the healthy mucosa distant from the ulcers to the



CHRONIC ULCERS.

inflamed mucosa bordering the ulcers is to be noted.

There was no striking muscle change except in relation to the ulcer floors. Moderate arterio-sclerosis.

Duodenum.

There was a large chronic ulcer on the posterior wall 0.5 cm. from the pylorus.

S.71. Eliza J. Female, 64.

Clinical Data.

Admitted 6 weeks before death with the history of 8 months of weakness, loss of appetite, fainting attacks, abdominal pain and intermittent diarrhoea. O/e: mild pyrexia and low-grade leucocytosis; pus cells and Bact. coli in urine; blood and pus in stools. Sigmoidoscopy showed superficial ulceration of the pelvic colon. Later liver and spleen reported as palpable. Gradually sank into uraemia.

General Autopsy Findings.

Chronic cystitis. Bilateral pyelo-nephritis. Amyloidosis of liver, spleen and kidneys. Large bowel thickening, probably of amyloid origin. Advanced rheumatoid arthritis.

Stomach.

Normal size and shape. Diffuse

amyloidosis of mucosa. Generally speaking, the mucosa was not much thinned, but there was much glandular degeneration and atrophy. Many atrophied glands were widely dilated. Many chronic inflammatory cells present. Eosinophils very few. No goblet cells or Russell cells. Neutrophils numerous.

No arterio-sclerosis. No muscle changes.

S.72. Elizabeth, G. Female, 97.

Clinical Data.

Well until 3 weeks before death when had a stroke. B.P. 150/100. O/e: right Hemiplegia and deeply comatose. Started to recover consciousness after 3 days. Died suddenly.

General Autopsy Findings.

A remarkably well preserved old lady. Small, scarred kidneys, hypertensive heart, slight emphysema, small brown liver, very small fibrous spleen.

Stomach.

Normal shape. Small. Diffuse chronic inflammation, fairly severe in the cardia and pylorus, mild to moderately severe elsewhere.

Very large glandular "cysts" in the cardia.

Goblet cells +.

Very few Russell cells and eosinophils.

Few neutrophils.

Moderately severe arterio-sclerosis.

No striking muscle changes.

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2. OPERATION SPECIMENS.

S.73. J.F.L. Male, 57.

Clinical Data.

12 years history of gastric ulcer. In the year preceding operation, the patient lost weight. Gastroscopic examination suggested a neoplasm. Test meal: high free HCl and total acidity. Partial gastrectomy performed ... the distal two-thirds of the stomach were removed.

Stomach.

In the pylorus there was a partially healed chronic ulcer about 1.5 cm. in diameter. The mucosa in this part of the stomach showed a severe chronic gastritis with numerous goblet cells and much glandular degeneration. The remainder of the stomach mucosa showed a mild

chronic gastritis.

There were numerous eosinophils throughout the mucosa; also numerous fresh, traumatic haemorrhages. No Russell cells were seen.

There was no arterio-sclerosis.

S.74. E.H. Female, 52.

Clinical Data.

Carcinoma ventriculi. Gastrectomy.

Stomach.

A large portion of the pyloric canal was occupied by a soft polypoid mass of well-differentiated adeno-carcinoma. Just proximal to this, on the lesser curvature was an acute ulcer (2.5 x 1.5 cm.).

The mucosa around the tumour showed a severe chronic inflammation with many goblet cells. The mucosa in the remainder of the stomach showed a mild chronic inflammation. Russell cells absent.

The whole mucosa was very congested and oedematous. Eosinophils were abundant.

S.75. A.K. Male, 44.

Clinical Data.

Diagnosed as a case of simple gastric

ulcer. The distal two-thirds of the stomach and the first 1 cm. of the duodenum were removed.

Stomach.

Macroscopically there was a deep ulcer (1.5 x 1 cm.) 5 cm. from the pylorus on the lesser curvature. The whole wall of the stomach was thicker and whiter than usual.

Microscopically there was a widespread, partially differentiated adeno-carcinoma of the stomach. In many areas, it had a scirrhous quality. The ulcer was a carcinomatous ulcer.

There was a diffuse late subacute or early chronic inflammation of the mucosa, mild in the proximal part of the specimen, severe elsewhere. In the proximal part eosinophils were fairly abundant. In the distal part of the mucosa, where tumour cells were more numerous and inflammation was greatest, they were very numerous. There were numerous goblet cells in the areas of most inflammation. Russell cells were absent. There was no arterio-sclerosis.

S.76. W.W. Female, 32.

Clinical Data.

Partial gastrectomy for chronic duodenal ulcer.

Stomach.

The specimen consisted of the distal two-thirds of the stomach and the first 2 cm. of the duodenum. The proximal half of the specimen showed a mild chronic gastritis, the distal half a more severe chronic gastritis. Eosinophils fairly abundant.

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