

J. Philip Welch

FOUR MEDICAL CASES

submitted for

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Ward D1. Western General Hospital

Patient's Name John Wilson
 " Age 31
 " Address 10 Camp View, Bonnyrigg.
 " Occupation Labourer
 " Doctor Dr. Somerville
 Doctor's Address High Street, Bonnyrigg
 Date of Admission 30d. May 1957
 " " Examination 28th. May 1957

COMPLAINTS

1. Passage of frequent motions - up to 13/day - containing blood, mucus and pus.
2. Abdominal colic immediately prior to defaecation.

(Both the above symptoms during last 12 years until his operation six weeks ago.)

PRESENT HISTORY

In May 1945, Mr. Wilson had an acute onset of diarrhoea. Although he does not remember the exact date, he recollects that it was a Saturday on which he had previously consumed a considerable quantity (6/- worth) of ice cream. The diarrhoea persisted at a rate of about 13 stools/day, and the patient, seeing blood in his motions, and wondering if he had a cancer, did not go to see his own doctor until August 1945. His doctor then treated him with Sulphonamides but his condition did not improve, and in December, he asked to be sent to the R.I.E., to which he was admitted (Ward 29), and treated with penicillin. Under this therapy he was slightly improved - 8 motions a day - and was discharged in March 1946 on 'a diet'. After discharge, his motions rapidly returned to their original rate.

His symptoms continued, unaffected by any form of treatment, but with the addition, over the last few years, of colicky pain immediately prior to defaecation; and in 1957 he was again admitted to the R.I.E. under Prof. Bruce. He was then consequently treated with with Cortisone, but without any improvement in his condition, and subsequently he was transferred to the Western General Hospital for further investigation with a view/

a view to operative treatment. Following investigation an ileostomy and total procto-colectomy was performed by Prof. Bruce and Mr. Falconer on 17.4.57.

Following the operation he had some 'ups and downs' and has lost nearly a stone in weight; he now feels much better however, and considers the operation to have been well worth while.

No history of chest pain, breathlessness, or ankle swelling.

Mict. - n.a.d.

PREVIOUS HISTORY

Whooping cough as a child

Mumps at age 16

No other illnesses or opn.s of note other than above.

Never been abroad.

FAMILY HISTORY

Father is aged 63 - suffers from 'rheumatism' (retired on this account 10 wks. ago)

Mother is aged 58 - alive and well

Sibs - two married sisters. One, married 10 yrs ago, lives at Hawick; the other, married 5 yrs ago, lives at Arniston.

Both sisters A & W.

No member of the family has ever had any serious bowel upset.

SOCIAL HISTORY

Mr. Wilson is unmarried, at present living with his parents, and for some years has worked as an itinerant labourer, always aiming for the job which paid most money.

His last years at a country school do not appear to have held many opportunities for useful effort, and he was glad when his father obtained 'special permission' for him to leave at the age of 13½ in order to help the family finances.

Although he would have preferred to 'do something mechanical' there was no local opportunity for this - the family were living in the Haddington district at this time and the nearest garage was several miles away.

After leaving school he 'managed a horse' on a farm for a year, then joined his father and worked as a shepherd for two years. Following this he worked on various farms, and 1944 found him at the age of 18 working on a farm, about 300 yds from his home, near Carlisle. At this time he was 'living in' at the request of the farm manager and with parental approval.

At/

At this time he received his 'calling up' papers, but, being a farm labourer, he was entitled to deferment should he so desire it. His mother was anxious that he should not join H.M. Forces, though his father was quite willing for him to go. It seems that the patient devoted considerable thought to this decision, eventually deciding not to enlist. On looking back he now wishes that he had decided to join, but this regret did not appear to be causing him any mental or emotional upset at the present time. (It is perhaps noteworthy that another interviewer - a lady almoner - provoked a considerable emotional (and tearful) reaction, on touching upon this subject in a prior interview.) He did, however, confess to feeling very uncomfortable when in the company of men of his own age speaking of their wartime experiences.

About this time his father was becoming incapacitated through 'rheumatism' and as a result the family moved back to Scotland (Hawick) in November ('44); leaving the patient on the farm. Shortly after this he 'lost his temper' with a cow (over some triviality) and struck it with an iron bar, as a result of which the cow died. This affair ~~xxxx~~ terminated with the farm manager demanding payment for the cow (and for 'putting its death through the books as natural') which caused the patient great distress. (He claims to have been a 'very hard saver' at this time.) He 'decided to leave' this farm and did so in March '45, moving to join his father. (This movement gave rise to some dispute with the Min. of Labour due to the wartime control of employment.) He then took a job driving a tractor on a farm, leaving abruptly (after 2/52) when he was taken off the driving job - thus causing another dispute with the Min. of Labour. He then remained unemployed for some 3-4 weeks (occasionally helping his father who was again working as a shepherd) and while unemployed suffered a further upset, as his previous farm manager sold his motor cycle which he had been forced to leave behind on the farm. He considers that he thereby lost about £15.

Finally he took employment on the farm at which his father was working to lend a hand with the harvest. While thus employed his illness started with the onset of diarrhoea as described above.

Since this date he has been employed doing 'labouring jobs', changing them fairly frequently. In 1954 he started a taxi business as a sideline and seems to have developed it fairly successfully. He now owns a 'Volkswagen' 'Minibus' (9 seats) which he is busy 'paying off'. His father is at present paying the instalments.)

Personality of the Patient

Mr. Wilson does not smoke and drinks little (only at Weddings etc). He gives a strong impression/

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impression of being a very quiet, retiring man of unemotional nature - difficult to reconcile with a story of loss of temper. One incident may be cited to illustrate his character - he is accustomed to arranging an evening cup of tea for himself and the other fitter inmates of the ward; during the first evening interview however he did not do this and missed his own cup of tea - because he 'did not like to mention it'. In all other interviews he was, in the writers opinion, abnormally accommodating.

He is an ambitious man, indeed he has so many ambitions that it is very difficult to decipher exactly what he really wishes to do with his life. He claims to be interested in things mechanical - does not wish to remain an employee - would like a small business of his own - has considered or is considering emigration - the stream runs on interminably. He has taken out an Endowment Policy to help finance his future schemes.

In the writer's opinion, this man has an I.Q. unsuited to the job of a labourer, and his assessment is an essential part of his future treatment and rehabilitation. (As an aside it is interesting to note that in the middle of one interview the patient remarked - quite out of the blue and irrelevant to the current conversation - 'of course I think that this is really a nervous complaint'. On questioning he claimed that he had evolved this theory for himself; he thought that, in his case, the causative factor was his upset over the death of the cow. (see above)

He has no particular hobbies or interests; in the last few years he has taken to doing a little 'Olde Tyme' dancing, but cannot do modern ballroom dancing (though he has been taking private lessons). He has had no dealings, so far as can be ascertained, with the opposite sex. It is perfectly feasible, of course, that his lack of social intercourse results more from the nature of his illness than from a facet of his personality. (Card, 1952. q.v.)

GENERAL EXAMINATION

The patient was walking in the ward when first seen; a tall, 'Big boned' man with a rather lean pale face, prematurely grey hair, and walking with a slight stoop. Temp. 97.8 F

ALIMENTARY SYSTEM

Edentulous. Tongue, gums, and fauces - all healthy. No halitosis.

Abdomen/

Abdomen -

Inspection. Good musculature, moves on respiration. There was a long (pubis-costal margin), well-healed, L paramedian scar, and an ileostomy opening slightly to the R and below the umbilicus to which a rubber (Down's) bag was attached. About 2" of ileum was exteriorised (not including the amount which had been 'turned back'). The ileostomy appeared healthy and there was no excoriation of the skin.

Palpation. No tenderness. Hernial orifices closed. No visceromegaly, no masses.

Percussion. No visceromegaly confirmed (liver extended up to 5th space).

Auscultation. Borborygmi normal.

CARDIOVASCULAR SYSTEM

Radial Pulse - rate 82/min. Regular in time and force and of good volume. Vessel wall not palpable.

Hands - minimal nail bed fluctuation. Nil else of note.

Neck - J.V.P. = 0 H.J.R. neg.

B.P. - 110/70 mmHg.

Oedema - Nil.

Retinae - Both normal

Heart - Apex beat difficult to locate, sited in 5th space on M.C.L.

Auscultation. H.S. quiet, closed; no murmurs or added sounds detected.

RESPIRATORY SYSTEM

Rate - 17/min, regular.

Chest symmetrical with good expansion

Trachea central.

No lymphadenopathy.

P.N.=P.N.= Normal (Slight kyphosis noted)

B.S. vesicular all areas, without accompaniments.

CENTRAL NERVOUS SYSTEM

Higher Functions - Attention and powers of concentration good. A quiet, superficially unemotional man of above average intelligence.

Cranial Nerves

I -No history of upset in taste or smell

II -Glasses not worn. Full visual fields.

III } (Pupils equal, round, reacting to light &

IV } accommodation. Movements full without

VI } strabismus, diplopia, or nystagmus. No ptosis.

V/

- V -Facial sensation unimpaired.
 VII -Facial muscles of good power, equal both sides.
 VIII-Hearing unimpaired.
 IX - No difficulty in swallowing
 X -Palate moves equally both sides.
 XI -Good and equal power of sternomastoids and Trapezii.
 XII -Tongue - protruded without tremor, deviation.
 No atrophy.

Motor Functions - No involuntary movements
 - Muscle tone normal
 - No paralysis or wasting
 - Co-ordination good.

Reflexes -

Superficial

Plantars	R. ↓	L. ↓
Abdominal	✘	

Deep

	R.	L.
Ankle	+	+
Knee	++	++
Biceps	+	+
Triceps	+	+
Supinator	+	+

Sensation -

Touch, pain, vibration,
 proprioceptive sense - all intact.

LOCOMOTORY SYSTEM

No signs of inflammation or deformity in any joint. Full range of movement in all joints.

SKIN

There was an area of reddened, scaly skin about 2½ x ¼ cm, half way up the medial surface of the R. tibia. This lesion had been present some 9/12 and although better than it has been, is still sometimes itchy. ('I still have to scratch it occasionally'.) This lesion could well be self-induced - the so-called 'neuro-dermatitis'.

INVESTIGATIONS

3.4.57 E.S.R. 10 mm R.B.C. 5M W.B.C. 7,600
 Hb. 95% Stool Benzidine POS.

6.4.57 Sigmoidoscopy. Some ulceration and bleeding was seen, with the impression of a stricture at 10 cm.

Chest X-ray - Lung fields clear. N.A.D.

Ba. enema - showed the typical features of chronic ulcerative colitis. (Shortened colon, lack of haustrations, ragged mucosal outline, and presence of pseudo-polypi.)

Fat balance test - showed some disturbance of absorption.

Ascorbic/

Ascorbic acid absorption test - also showed some impairment.

Repeated cultures failed to isolate organisms of the dysentery group, and searches for *Entamoeba histolytica* proved fruitless.

Cultures of the pus from the stool revealed the presence of *E. coli* and *Staph. aureus*. (Coag. +ve)

Liverfunction tests - Serum Alk. Phosphatase 22 K.A. units otherwise normal.

Plasma Electrolytes - normal.

DISCUSSION

A. DIAGNOSIS Ulcerative Colitis

B. REASONS FOR DIAGNOSIS

The presence of longstanding diarrhoea accompanied by the passage of blood and pus without the isolation of a possible causative organism from the stools. The diagnosis is clinched by the sigmoidoscopic and radiological findings.

C. DIFFERENTIAL DIAGNOSIS

This condition must be distinguished from

1. Bacillary dysentery
2. Amoebic dysentery
3. Carcinoma

Bacillary dysentery produces a similar picture but the causative organisms can be isolated from the stools in the vast majority of cases. (Particularly in the early stages of the disease.) This condition practically always responds to drug treatment, but in this case drugs (Sulphonamides prescribed by own G.P.) failed to affect the disease even in its early stages.

Amoebic dysentery does not usually produce pus in the early stages, and a history of foreign travel is usually - though not necessarily - obtained. The characteristic 'amoebic ulcers' were not seen in this case, and attempts to isolate amoebae from the stools were unsuccessful.

A carcinoma does not usually present with this type of history, nor is it commonest in this age group. The findings on sigmoidoscopy and by Ba. enema lend little support to this diagnosis.

It may be noted that with any of these alternative diagnoses the condition would be unlikely to smoulder on for 12 years, the patient remaining comparatively fit and free from complications.

It/

It may also be noted that according to Goligher (1957), 5-6 % of cases are in fact carcinoma, and this may be shown only by histological examination after operation. Whether this is the primary condition or a secondary complication, (perhaps more likely), is debatable.

D. AETIOLOGY, ANATOMY, PATHOLOGY, AND FUNCTION

The aetiology of this condition is still somewhat obscure but it may be helpful to review the theories in the light of current knowledge.

For many years gastro-enterologists, determined to prove that this condition is due to a specific infection, sought to find a causative organism. A small number of cases are due to chronic bacillary dysentery (probably only about 5% - even the most enthusiastic supporters of this theory claim only about 15% 'proved' cases); but Gaertner's bacillus, Morgan's bacillus, and other more obscure organisms have been isolated from the stools of patients suffering from this condition and branded as the causative agent.

Many authorities claim this as a psychosomatic disease, and even ardent opponents of this theory admit that psychological upsets in patients suffering from this condition often causes a relapse. It should be said however that some authorities categorically deny that this condition has any psychosomatic origin (Kennedy, 1957). This may be associated with their own failure to treat the condition by certain psychotherapeutic techniques. (Kennedy, 1957 Brit. Med. J. I, 1319)

It might be helpful at this point to give a brief review of the relevant literature on this last theory.

- 1930 - Cecil D. Murray - classic work which initiated the psychosomatic hypothesis. Stated that the onset of the disease often coincided with a difficult emotional situation - particularly marriage.
- 1942 - Daniels - found that UC patients are apt to be too dependent on some member of the family ('important person'); usually the mother; and developed conflicts in situations threatening this dependence.
- 1948 - Lindemann - showed a close time relationship between the loss of this 'important person' from the patient's social sphere and the onset of illness. (Demonstrated this in 26 cases from a series of 45)
- 1948 - Daniels - stated that 75% of patients had an emotional conflict as a primary causative factor/

factor, but that this emotional factor was not 'the whole reason'.

- 1948 - Kirsner et al - showed in a number of cases that recurrences were most frequently associated with emotional stress, upper respiratory tract infections, and fatigue. (In that order.)
- 1948 - Meyer et al - showed that the enzyme 'lysozyme' is present in higher concentrations in the stools of UC patients than in others. Concomitant with clinical improvement, lysozyme titre fell. Also demonstrated that administration of oral lysozyme in the dog produced ulcers of the large and small intestine. They assumed two stages - (a) removal of surface mucus (b) necrosis of denuded tissue by proteolytic enzymes presumably furnished by micro-organisms. They felt that the reason for the overproduction of lysozyme by the mucosal cells was obscure.
- 1949 - Lindemann - Pointed out that the disappearance of UC is often associated with psychotic illness, and when this passes, colitis reappears.
- 1949 - Stewart - showed that the basic personalities of UC patients are often similar - extreme dependence, emotional immaturity, subservient yet subversive attitude to domination, overt passivity, and high intelligence. He advocated a course of reassuring psychotherapy.
- 1950 - Glass, Grace, Pugh and Wolf - showed lysozyme to have no lytic action on human gastric and colonic mucus in vitro. They admitted that this did not preclude the possibility of its destroying or initiating the destruction of mucosal cells in vivo.
- 1950 - Grace, Wolf & Wolff - by observation of fistulous subjects showed that feelings of anger and resentment were associated with hyperfunction of the colon. (Hypermotility, hypersecretion of mucus and lysozyme, hyperaemia and engorgement.) This resulted in increased fragility of the mucosa, submucosal bleeding, and ulceration.
- 1950 - Lindemann - demonstrated in a UC patient the blockage of the transition from impulse to execution, resulting in a poverty of expression.
- 1950 - Paulley - was impressed by the similarity regarding parent dependency and history in 173 UC patients.
- 1950 - Gray et al - inhibited lysozyme by means of a detergent ('aerosol O.T.') in a series of UC patients without any change in their condition. He emphasized that this may not have been effective in the activation of lysozyme within the mucosal cells.

1951/

- 1951 - Almy - demonstrated that UC patients and others showed engorgement and increased motility of the sigmoid colon under stress by experimental stimuli.
- 1951 - Investigating Committee under Stewart Wolff stated that 'the G.I. tract shows significant changes in form and function under situations regarded as threatening by the individual, but evidence of relevance to structural disease is incomplete.' They went on to say that these changes 'may be of first grade importance in the pathogenesis of such disorders as peptic ulcer and ulcerative colitis'.
- 1951 - Grace, Wolf & Wolff - conducted detailed direct and indirect observations of the colon in a wide variety of persons - some healthy and some suffering from large bowel diseases including UC. They concluded from these observations that in all persons life situations of abject fear and depression produces hypofunction of the colon, and situations provocative of conflict (with anger, resentment, hostility and apprehension) are associated with hyperfunction of the colon. These changes were found to be much more pronounced and sustained in UC patients. They viewed UC as a stress disorder, and were of the opinion that a constructive physician-patient relationship was of the greatest benefit in treatment - good results (they claimed) are not obtained without this. They found UC patients to be characteristically outwardly calm, superficially peaceful individuals of more than usual dependence. They went on to say that on probing beneath this exterior it became apparent that this outwardly placid person was 'sitting on a powder keg' of intense hostility, resentment and guilt. (Similar work on the colon has also been carried out by White et al. 1938; Almy & Tulin, 1947; Groen & Van der Valk, 1956.)

From the above literature the following characteristic features of the UC patient emerge -

1. Mental immaturity and childishness
2. Extreme dependence and passivity
3. Strong parent (or substitute) relationship with suppressed resentment
4. Poverty of expression
5. Above average intelligence.

Assuming the disease to have a psychosomatic aetiology, we might conclude that psychotherapy would have a considerable place in the treatment of this condition; this is borne out by such figures as are available. Groen & Bastiaans (1951) treated 33 cases by psychotherapy supplemented by only general medical measures (e.g. bed-rest, rehydration and blood transfusion when their condition was serious.) No drugs were given. Of this series, 18 patients remained clinically cured or with signs and symptoms

remained clinically cured or with signs and symptoms well controlled, from 1939-1951.

A smaller series started in 1953 (all severe cases) produced results which compare favourably with a similar series treated by surgery.

Similar work has also been done by Paulley (1956) - 40 cases - and by Grace, Wolf & Wolff (1951) - 19 cases.

To turn from the general to the particular; the aetiology of Mr. Wilson's condition is now almost certainly lost in the past, and only speculation is open to us. Furthermore, there is a strong temptation to allow it to run away with us. It would be easy, for example, to pin the blame on the emotional crisis caused by his call-up papers in 1944; or to suppose that his regret at having made a 'wrong' decision became resentment against his mother for having influenced him - resentment which the very nature of the man precluded him from showing openly. To add weight and colour to this theory, (which may be the correct one), the patient's mother stated in an interview that for years she has worried about him, afraid that he might die from his complaint. Might not this be his way of taking a childish 'revenge' on his mother?

Alternatively the upset over the cow and/or the further upset over the sale of his motor cycle - either of these might be regarded as the psychosomatic cause of his condition. Then again, the loss of temper (which does not 'fit' his personality) leading to the death of the cow with its subsequent complications; might not this be due to his call-up worries?

One salient fact emerges from this mass of data, viz., that the character of the patient has most of the features described as typically occurring in the patient suffering from ulcerative colitis of psychosomatic origin.

Anatomy, Pathology and Function.

The disease usually appears first in the rectum, spreading to involve the colon, and in severe cases, the small intestine. In chronic cases the mucous membrane may become hyperplastic, resulting in a pseudo-polypoid appearance. This hyperplastic mucous membrane has an undoubtedly increased tendency to malignant change. Multiple small ulcers are formed, and in the chronic case the colon is transformed into a rigid, inflamed tube; of no value to the patient; which may absorb toxins through its raw surface, while depleting the body of blood and protein.

E. TREATMENT/

E. TREATMENT

Radical treatment had already been carried out on this patient when he was first seen, so that this aspect will be only briefly considered.

General supportive medical treatment should always be given; fluid lost must be replaced and anaemia corrected (if necessary by transfusion); and a high calorie, high protein diet is necessary to combat the loss of protein in the stools.

Local treatment with demulcents, bismuth sub-gallate etc, is now falling into disrepute; as are other quaint methods of treatment ranging from Bergen's serum, (prepared from one of the micro-organisms in vogue some years ago), to the wearing of a body belt of silk and wool round the abdomen.

There is now considerable evidence to show that Cortisone (and ACTH) is able to produce a remission in many cases, though this may be only temporary. In a disease subject to natural remissions as this is, it is difficult to establish the value of any method of treatment; indeed some authorities affirm that Cortisone is of no value and may be actually dangerous by increasing the likelihood of perforation. (Goligher, 1957)

With the increased safety of major surgery, operative procedures have come to play an increasing part in the treatment of this disease. The usual procedure is to carry out an ileostomy and procto-colectomy (as in this case) as a one stage operation. The indications for surgery are usually given as -

- 1. Chronic invalidism - with a possibility of malignant change and with physical retardation.
- 2. Local complications - perianal and ischiorectal abscesses, fistulae, strictures.
- 3. General complications - non-suppurative multiple arthritis, iritis.
- 4. To save life in the florid form of the disease. (Goligher, 1957) Many authorities consider this to be rather an indication for Cortisone. (Toxaemia and high fever may be a feature in these cases.)

Other complications which are sometimes seen are -

- 1. Pyoderma gangrenosum (and sometimes other skin lesions).
- 2. Finger clubbing.

Perforation of an ulcer may also require surgical intervention, though it may be noted that according to Handfield-Jones (1956) perforation does not occur in this disease as the muscle coat of

of the bowel is not invaded.

Anastomosis of the terminal ileum and the rectum has been carried out in the U.S.A. with some apparent success, but the operation has not met with great favour in this country as yet. One of the essential pre-requisites for such an operation is that the rectum should be uninvolved in the disease process - a somewhat uncommon finding.

It might be advocated that psychotherapy combined, in suitable cases (the majority being suitable), with hypnosis (Ambrose & Newbold, 1956) should be employed far more often than is at present the case. In any event there is a good case to be made for all UC patients to have a full psychiatric investigation.

This, of course, does not decry the advisability of an operation at this stage in the disease process in this particular case. It may be, however, that had treatment on the above lines been carried out at an earlier stage; this man might have avoided a mutilating operation and subsequent ileostomy life, though this is not the major handicap of former years.

PROGRESS NOTES

Operation - Carried out on 17.4.57 by Prof. Bruce and Mr. Falconer.

There was some degree of peri-proctitis and a palpable polyp in the transverse colon. Also some reddening and thickening of the terminal ileum.

A total procto-colectomy and ileostomy was carried out and 12" of ileum was also removed. A separate incision was made for the ileostomy and the mucosal edges were sutured to the skin. Two pints of blood given during the opn.

Postoperatively - some jaundice appeared after operation, probably due to the blood transfused during the operation. This faded after two days.

19.4.57 - Required fairly rapid blood transfusion due to continued loss from perineal wound. 3+ bottles given.

23.4.57 - Further transfusion, 2+ bottles given.

Although losing about 3½ litres/day he remained fairly well until about

7.5.57 - Gradually became depressed with feeling of general malaise - continued high fluid loss per ileostomy.

18.5.57 - Found to have Serum Na. 295 mgm. %
" Chlorides 422 mgm. %
B. U. N. 39 mgm. %

E. I.V. N saline with remarkable change for the better in the patients well-being.

Continued/

Continued well with gradual healing of his perineal wound and diminishing loss of fluid per ileostomy until on

18.6.57 - fluid loss per ileostomy down to 1200 ml/day.

PROGNOSIS

The prognosis for this man is good. On discharge there is no reason why he should not lead a normal life, his ileostomy merely a minor inconvenience. The two main problems are -

(a) to guide him into a more worth-while job than he has hitherto been accustomed to occupy. This must essentially be guidance as distinct from compulsion, or persuading him into a job against his own wishes. In order to accomplish this he should have a full psychological assessment ~~of~~ including his aptitudes and capabilities.

(b) to provide him with superficial psychotherapy and reassurance, both as regarding his ileostomy life and his old problems which existed concurrently with his disease process.

These two aspects of future treatment cannot be over-emphasised.

(It was noted that this man is very quick to snap up tit-bits of medical information, especially bits of chance conversation or injudicious comments made on his own case during ward rounds. Having absorbed them it is quite clear that he tends to brood on them and their significance.

It is also worth noting that this man has never met a fit and healthy ileostomy patient, either before or since his operation; this defect should be remedied with a carefully chosen ex-patient at the earliest opportunity.)



FURTHER PROGRESS

Following his discharge this man reported to the O.P. clinic at monthly intervals, where it was noted that his perineal discharge was becoming chronic. In view of this he was re-admitted on 11.12.57.

At this time his weight was 14 st 2½lb and he was 'in excellent spirits'. His stools were now more formed and he was 'grateful for the peace of mind' which the operation had given him.

Laboratory findings - E.S.R. 15 mm
Electrolytes satisfactory (normal limits)
Alk. phosphatase 27 K.A. units.

His perineal sinuses were curretted and packed (17.12.57) and he was discharged on 31.12.57 - to have further dressings done by the District Nurse.

Mr. Wilson/

Mr. Wilson had a full psychological assessment and was found to be of approximately normal intelligence. (A little higher in Arithmetic only.) He spent some time at the Industrial Rehabilitation Unit but does not appear to have found his stay at the I.R.U. altogether satisfactory, perhaps because of the financial aspect although his reason was that 'it was too much like school'.

Eventually he obtained a job as a general labourer in a carpet factory. One might have wished for better things for him; however he seems well pleased with his state which is, after all, the most important consideration.

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and also

Groen & Van der Valk, 1956 Practitioner 177,572

CASE II

Ward C5 Western General Hospital

Patient's Name	Alexander Mc.L. Whittet
" Age	59
" Address	244 Crewe Rd. North. Ed. 5.
" Occupation	Sheet metal worker.
" Dr.	Dr. Donald
" " Address	Ferry Rd, Leith.
Date of Admission	11.5.57
" " Examination	13.5.57

COMPLAINTS

1. Intermittent epigastric pain - 17/12
2. Gripping pain in chest - 2 years
3. Slight morning naeasa with a little vomiting - 5/12

PRESENT HISTORY

One day about two years ago Mr. Whittet experienced a sudden, 'tight' pain in his chest which arose without warning and, so far as he remembers, while he was at rest. He ascribed the pain to 'heartburn' and took some Aspirins which gave him 'a little relief'. Since that date this same pain has recurred at intervals of about 2/52. It seems to occur at any time, bears no relation to exercise, and usually lasts about ¼-½ hour; it did not radiate into either arm or neck. After he had these symptoms for some months he took the advice of a friend ('my father's heart trouble started like that') and visited his Dr. who referred him to Dr. R.W.D. Turner (Cardiologist); who saw him on 2.2.56. Thereafter he was treated by his own Dr.

Two months prior to his visit to Dr. Turner however, after New Year celebrations, he experienced an attack of nausea with fairly severe pain related to the umbilical region. This was quite a new experience for him and he describes it as 'a dull pain' quite different in character to the chest pain which he had previously experienced. This pain too has recurred at intervals, and although he initially had periods of up to 2/52 completely free of it, it has gradually become more persistent. Eventually he mentioned this pain to his own Dr. who referred him to Dr. Strong (W.G.H.). He was seen by Dr. Strong on 4.4.56; by this time the pain was a daily occurrence and had its maximum intensity in the epigastrium (having gradually shifted to this position during the first 2/12 of its history).

The pain occurred most frequently about 3 hours after meals and was relieved by taking food. ~~He~~
He/

He has never been wakened by the pain, nor has it ever kept him from his work prior to 17.4.57. He has never tried to relieve the pain by taking baking soda or proprietary stomach powders, but he found that Aspirin gave him some relief. The pain is now a continuous dull ache, and in the last 6/12 has radiated through to the back (to just below inf. angle of L. scapula). On Dr. Strong's recommendation the patient was in bed at home (with frequent administration of alkalis) from 17.4.57 until his admittance to W.G.H. (He got up for toilet purposes only - including a weekly bath.) He was admitted to W.G.H. as his own Dr. did not think he was making satisfactory progress at home.

He states quite definitely that all his attacks of chest pain have been similar, but totally different from his abdominal pain.

During the last 5/12 he has had a little vomiting in the morning (first thing) but never at any other time. The vomitus has apparently always consisted of a small amount of mucoid material and he has never seen recognisable food in it; occasionally it has been 'brackish' in character. He has never seen blood or coffee-ground material in his vomitus with the exception of one morning 6 days prior to admission when it was 'streaked with blood' which he ascribes to coincident nose-bleeding. For the last few months he has felt 'blown up' after fairly small meals and he thinks he has lost a little weight - now 11st. 4lb. (pyjamas only) previously 12st. 4lb. (usual clothes).

He has never seen any frank blood in his motions, nor has he noticed any change in their colour.

He has never suffered from headaches but in the course of the last 6/12 he has had about half a dozen 'black-outs'. These have all occurred while he was at home and he has always had a few seconds warning - 'felt things going' - in time to sit down. He estimates that he loses consciousness for a few seconds only, and he has never injured himself or passed urine while in one of these 'blackouts'. There are no epileptiform prodromata.

PREVIOUS HISTORY

At age 13 - 'Rheumatic fever', treated in Leith Hospital for about 2/12.

In 1921 he had jaundice while working abroad. This was painless but with considerable malaise - cleared up under medical treatment in hospital - ? infective hepatitis.

About 1951 he began to suffer from pain in the back of his neck, treated as 'rheumatism' by his own Dr. On visiting Dr. Turner on 2.2.56 this was/

was diagnosed as cervical spondylosis and was treated with physiotherapy. He has not had any further discomfort in his neck following treatment.

FAMILY HISTORY

Mr. Whittet's father died aged 80+
- 'old age'
Mother died aged 70 (approx.)
- 'stoppage of the bowel'
Sibs - four sisters; one elder,
three younger, all A & W.
one younger brother - A & W
In addition,
1 brother killed 1914-18
2 sisters died v. young
(one from Diphtheria)

SOCIAL HISTORY

Mr. Whittet is married with 6 children, 5 of whom are married and live away from home. (3 ♀, aet. 36, 34, 28; 2 ♂, aet. 38, 22; all these married plus 1 ♀ aet 24 at home.)

Wife and all children are A & W.

Smokes 10-12 'Capstan' per day; prior to his visit to Dr. Turner the figure was 30-40. At this time he was also in the habit of consuming about 2 pints of beer per night and about 10-12 at the weekend with some 6-7 glasses of whisky or brandy. He now has about 2 pints of beer per week and has cut out the spirits.

It is interesting to note that his abdominal pains also improved considerably after this change of regimen. His main hobby is gardening.

He does not confess to any financial or other worries.

GENERAL EXAMINATION

The appearance was that of a fit-looking tanned man, sitting up in bed and quite at ease. Temp. 97.5

ALIMENTARY SYSTEM

Edentulous.

Tongue, gums, fauces - all healthy.
No halitosis

Abdomen - Inspection. Good musculature, moves on respiration. No veins, scars, or prominences. No visible peristalsis. 'Pointing' sign noted to be positive. Palpation - A fair amount of resistance was present, particularly well marked in the epigastrium where there was marked central tenderness. No rebound tenderness. A minimal degree of splashing could be elicited. No masses, no visceromegaly. Hernial orifices x closed
Percussion - Confirmed the non-enlargement of spleen and liver (which extended up to/

to the 5th space)

Auscultation - borborygmi heard.

Rectal Examination - n.a.d.

Faeces - POS. for occult blood (Gregerson)

CARDIOVASCULAR SYSTEM

Radial Pulse - 78/min. irregular, with multiple extrasystoles (every third or fourth beat). Regular in force and of good volume; character of the wave was within normal limits - no 'plateau' characteristics were detected. Vessel wall just palpable.

Hands - No cyanosis, no clubbing.

Neck - J.V.P. = 2 cm H.J.R. pos.

B.P. - 112/82 mm Hg.

Oedema - nil.

Retinae - both normal.

Heart - no visible pulsations. A/B difficult to locate but in 5th spce, 1/2" outside MCL. No heaves, no thrills.

Auscultation - the first sound was poorly marked and there was a well marked coarse systolic murmur heard along the left sternal edge, propagated up for a short distance towards the neck. This murmur could be heard through most of systole, beginning gently, reaching a peak of intensity, and slowly fading. No diastolic murmur detected.

RESPIRATORY SYSTEM

Rate - 15/min. regular

Chest symmetrical, well covered.

Numerous Campbell-de-Morgans spots. Good expansion.

Trachea central

No lymphadenopathy

P.N.=P.N.= resenant

Auscultation - B.S. vesicular without accompaniments. V.R. normal and equal both sides.

CENTRAL NERVOUS SYSTEM

Higher Functions - No obvious defects. Emotionally stable with good concentration and powers of attention

Cranial Nerves

- I -No history of upset in taste or smell
- II -Glasses worn for reading. Full visual fields.
- III /

- III Pupils equal, round, reacting both to light
 IV & accommodation. Movements full without
 VI strabismus, diplopia, or nystagmus. No ptosis.
 V -Facial sensation unimpaired.
 VII -Facial muscles of good power, equal both sides.
 VIII -Hearing - could not hear watch ticking, but no
 difficulty with normal conversation. Weber's
 test - no lateralisation. Rinne's test POS. for
 L. ear, NEG. for R. ear.
 Indicative of some slight conduction-type
 deafness, more marked in the R. ear.
 IX No difficulty in swallowing
 X Palate moves equally both sides
 XI Good and equal power of sternomastoids and trapezii
 XII Tongue - no deviation, tremor or atrophy

Motor Functions - No involuntary movements
 Muscle Tone normal
 No paralysis or wasting
 Co-ordination good

Reflexes -

		R	L
Superficial	Plantars	↓	↓
	Abdominal	—	—
Deep	Triceps	-	-
	Biceps	+	+
	Supinator	-	-
	Knee	+	+
	Ankle	?+	?+

Sensation - Touch, pain, vibration,
 proprioceptive sense, all intact.

LOCOMOTORY SYSTEM

No signs of inflammation or ~~xx~~
 deformity in any joint or limb. Full range of
 movement in all joints.

Three subcutaneous nodules were
 found on the L. forearm, one near the proximal end
 of the ulna and two more, fairly close together,
 about half way down the medial border. Their size
 varied from $\frac{3}{4}$ - $1\frac{1}{4}$ cm. diameter; they were slightly
 fluctuant, not fixed to skin but fixed to deeper
 tissues. They had 'always been present' and had
 never grown any larger or given rise to any trouble.

(Δ = ? Ganglia)

GENITO-URINARY SYSTEM

No history of frequency,
 dysuria, or nocturia. He had never noticed any
 abnormal colouration of his urine.

INVESTIGATIONS

11.5.57 Hb. 104% E.S.R. 16 mm/Lst hr. W.B.C. 6,100

Barium Meal - very ragged duodenal cap and active ulcer crater could be seen. No gross delay in emptying. (A moderate amount of fasting juice was present.)

Histamine Secretion Test (0.04 mgm Histamine per Kg. body wt., covered with mepyramine maleate.)

Fasting volume 20 ml.

Free acid output (in post-histamine hr.)
- 18.2 mEq.

Total acid output (in same period) - 20.8 mEq.

Chest X-Ray Lung fields clear, some cardiomegaly consisting of L. ventricular hypertrophy, bulging of R. atrium, and ? some enlargement of R. ventricle. Also some unfolding of the aorta.

Neck X-Ray - Cervical spondylosis with disc degeneration between C6 & C7.

E.C.G. Evidence of minimal L.V.H. Some conduction damage - shown as a slightly lengthened QRS interval. (Indicating prolonged intra-ventricular conduction.)

Blood Grouping Not done.

DISCUSSION

A. DIAGNOSIS Active duodenal ulcer with some coincident aortic stenosis following Rheumatic Fever.

B. REASONS FOR DIAGNOSIS

The history of ulcer-type pain, particularly its time and conditions of onset. The presence of maximal tenderness in the epigastrium and of occult blood in the stools. The history is quite typical - initially acute exacerbations of pain with periods of complete remission, the latter becoming gradually shorter until the pain is almost always present as a dull ache. The characteristic relief of pain by taking food or alkalis should also be noted. The diagnosis is confirmed by the X-ray appearances.

C. DIFFERENTIAL DIAGNOSIS

Duodenal ulcer must be distinguished from -

- (a) Gastric ulcer
- (b) Chronic Gastritis
- (d) Gastric Carcinoma
- (d) Nervous dyspepsia
- (e) Cholecystitis

(a)/

- (a) This case is rather difficult to differentiate from one of gastric ulcer, and recourse must be made to X-rays to clinch the diagnosis. The relation of the pain to meals and its relief by food point to a duodenal rather than a gastric ulcer; these symptoms are not altogether reliable however. The maximal histamine response, though distinctly higher than normal, is a little lower than is usually found with a D.U.
- (b) Heavy drinking, morning nausea, and the vomiting of swallowed mucus are characteristic of this condition - the factors which militate against it are -
 - 1) The presence of localised pain and tenderness - as distinct from mere discomfort - which is relieved by alkalis.
 - 2) the occurrence of the pain at a definite time after meals.
 - 3) the presence of a considerable quantity of acid in the gastric juice
 - 4) the X-ray findings.
- (c) These same factors are against the diagnosis of gastric carcinoma.
- (d) The factors mentioned above, plus the finding of occult blood in the stools, are very much against this possibility.
- (e) The site and character of the pain plus factor 4) render this diagnosis unlikely.

Three other conditions which might be mentioned in passing are chronic pancreatitis, biliary dyskinesia, and hiatus hernia. These are unlikely for the same reasons as in (e) above. It is possible that this patient may have a hiatus hernia in addition to ~~his~~ his duodenal ulcer; the two quite often co-exist. This could be giving rise to peptic oesophagitis and thereby causing the chest pain of which Mr. Whittet complains. Such a hernia would probably, though not necessarily, have been demonstrated at the Ba. meal examination.

Mr. Whittet's heart condition might conveniently be considered at this point. It seems fairly certain that he has aortic stenosis - the characteristics of the murmur, the radiological findings and the blood pressure, all point towards the presence of this condition. Aortic stenosis rarely exists alone and when the result of old rheumatic fever is usually accompanied by some degree of mitral disease, though no evidence of this was found in this case.

The aortic lesion almost certainly accounts/

accounts for his 'black-outs' and may also be contributing to ~~his~~ myocardial ischaemia which is almost certainly the cause of his chest pain. - This despite the fact that his chest pain is not absolutely characteristic of myocardial ischaemia, e.g. no relation to effort and no radiation. (See above for another possible explanation.) It might be worthwhile to supply him with a few tablets of 'Trinitrin' to take next time he has the pain as a diagnostic measure.

The impairment noted in the conduction of the impulse is probably due to a lesion in the bundle of His or one of its branches - this may be rheumatic (an Aschoff node) or ischaemic in origin.

D. AETIOLOGY, ANATOMY, PATHOLOGY AND FUNCTION

The chronic duodenal ulcer is said to be more common in males than in females. (Ratio 8:1 - Handfield Jones) 95% occur in the first part of the duodenum, and all occur proximal to the ampulla of Vater. They are rarely multiple except in the case of contact ulcers (these constitute about 10%); in the majority of cases the ulcer is found in the anterior wall.

Histologically five characteristic features are commonly found -

- (a) complete destruction of the muscle coat in the centre of the ulcer.
- (b) dense fibrosis in the base.
- (c) fusion of the muscularis mucosae with the muscle coat at the margin of the ulcer
- (d) presence of endarteritis obliterans in the vessels round about
- (e) presence of an overhanging proximal edge and a sloping distal edge

(Handfield Jones 1957)

Aetiology

Historical - at one time the cause of duodenal ulceration was thought to lie in the blood supply to the duodenum; one particular artery, the 'supra-duodenal artery of Wilkie', was thought to be an end artery supplying the 'anaemic spot' of Mayo. This theory gave rise to the 'physiological gastrectomy' of Somerwell. (The 'anaemic spot' being resected.) This theory is no longer recognised in this form.

Many factors are now regarded as relevant and are here briefly considered -

1. Environment

(a) Geographical - the incidence of peptic ulceration and the G.U./D.U. ratio varies widely, e.g. in different parts of India (Dogra, 1940) and between Scotland and London. (Lower G.U./D.U. ratio in Scotland - Tidy, 1944) The reason for this is not definitely known.

(b) Social Class - contrary to some beliefs, duodenal ulcers are equally distributed throughout the population. (In Great Britain.) (Doll et al 1951)

(c) Occupation - the only definite findings are an increased incidence of D.U. in men holding responsible positions in business, also in doctors and some groups of unskilled workers (the latter two may be artefacts); together with a decreased incidence amongst agricultural workers. (Doll et al 1951)

(d) Season - the mortality rate and the rate of recurrence of symptoms tends to reach its lowest level during August and September, rising in October and often with a further exacerbation in the Spring. The cause is unknown.

2. Constitution

(a) Sex - the M:F ratio is about 8:1 ; though the incidence of acute ulcers is approximately equal. (Jones & Pollak, 1945) In addition, pregnancy seems to confer some partial immunity, indicating a humoral effect.

(b) Age - the expectation of an adult developing a D.U. in any age group is approximately equal. (Avery Jones, 1957)

(c) Blood Groups - It has recently been shown, (Aird et al, 1953; Clarke et al, 1955), that persons in blood group 'O' show a higher incidence of gastric and duodenal ulcers than those in groups 'A' and 'B'. (100:72 as O:A in cases of peptic ulcer.) The blood group substances are mucopolysaccharides, distinguished by their antigenic properties. There does not appear to be a specific factor for group 'O', but those in this group appear to have increased amounts of a mucopolysaccharide called 'H substance'. These substances are present in small quantities in R.B.C.s and in much larger quantities in the body tissues and fluids. Their mode of action in influencing the stomach has not yet been determined.

(d) Heredity - there now seems little doubt that there is some independent inheritance of gastric and duodenal ulcers. (Doll & Buch, 1950; Doll & Kellock, 1951) Whether this is due to environmental factors - "hereditary habits" - or to genetical tendencies is still in doubt.

(e) Acid Secretion - it has been shown that hypersecretion occurs in the majority of patients with a duodenal ulcer (Kay, 1953). This hypersecretion appears to be due to an increase in the parietal cell mass rather than to an increase in the activity of individual cells (Hunt & Kay, 1954). This view is supported by the finding that ulcer patients tend to have a stomach rather above average in size (Cox, 1952). A derangement of the duodenal mechanism/

mechanism which normally inhibits secretion in response to the acidity of the effluent gastric contents may also be a factor.

(f) Blood Supply - this is the modern version of the old theory of the causation of ulcers (see above) - thrombosis, mucosal shunts (suggested by Bentley & Barclay), and the occurrence of vascular spasm have all been suggested. The basic theory is that local ischaemia cuts off adequate supplies of CO₂, leading to an intracellular necrotising concentration of alkalis. Ulcers have actually been produced in this way (Davies, 1952), but although this may be a factor in the causation of the acute ulcer, it does not explain why some ulcers become chronic.

(g) Rate of Emptying - it has been suggested that this is increased in patients with a duodenal ulcer, but investigations have not so far substantiated this suggestion. It may be that the important factor is nervous tension causing temporary fluctuations in gastric motility.

(h) Anxiety and Personality - though difficult to prove it seems probable that such factors as anxiety, frustration, resentment, fatigue, smoking, or irregular meals - any of these may determine whether or not an acute ulcer becomes chronic. The condition is said to be commoner in the leptosomatic or asthenic type.

(i) Past History - it has been shown that those with chronic bronchitis have an increased incidence of peptic ulceration (Weber & Gregg, 1956). Persons with hypertrophic pulmonary emphysema have an incidence of peptic ulceration three times that of the general population (Latts et al, 1956).

Integration The real problem now seems to be not to explain the occurrence of ulceration so much as to explain the development of the chronic ulcer. As has been demonstrated, many factors are involved; these may operate either on cellular repair mechanisms or by modifying the physical character of the mucus (Hirschowitz et al 1955) thus allowing cytotoxic substances in the stomach more ready access to the damage mucosa.

Regarding the cause of the acute ulcer, it might be added that gastric irritants, swallowed in sufficient quantity, will sometimes produce an acute ulcer which may give rise to considerable bleeding. The commonest culprits in these cases are alcohol and common aspirin. (Wuir & Cossar, 1955)

Duodenal ulcers have certainly become more prevalent in this country during the last thirty years (Avery Jones, 1957), and at least one contributory factor - though difficult to prove - must surely be the increased tempo and stresses of these/

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these times in which we live. Another factor may be the increase in tobacco consumption, though no figures are available on this as yet.

In the case of this particular patient, his acid secretion is a little low for the usual D.U.; he appears not to have any outstanding worries - indeed he seemed far removed from the 'anxious type' - and he does not hold a responsible business position. Positive findings are his sex, his tobacco consumption, and, in the past, alcohol. His blood group and secretor status were not determined.

E. TREATMENT

The most effectual ^{/single} medical treatment for a duodenal ulcer is bed rest - preferably in hospital so that the patient is completely removed from his usual environment with all its possible undesirable effects. The cessation of smoking has also been shown to encourage healing. The evidence of the good effects of dietetic treatment is regrettably slender. Recent work (Doll et al, 1956) has not shown any significant advantage gained by prescribing special diets. Similarly the time honoured milk drip and alkali therapy have not so far been shown to have any marked effect on the rate of healing of an ulcer; though they do, of course, considerably relieve the pain.

In the early stages of treatment the patient is certainly relieved and made more comfortable by small, frequent meals consisting of bland foods; when he becomes symptom free a practically normal diet (~~excluding alcohol and other irritants~~) may very soon be given. Alkalis should be given for the pain; these should be gradually withdrawn on the subsidence of symptoms. Initial slight sedation is often very useful (e.g. phenobarbitone) and iron may be given if anaemia is present.

Anti-cholinergic drugs, such as 'Probanthine', may be used and are particularly helpful if pylorospasm is present. Oedema may play a large part in this condition, which should be carefully distinguished from pyloric stenosis. If Belladonna is prescribed it should be given in full doses - sufficient to cause drying of the mouth and blurring of vision - and care should be taken if it is prescribed for ambulant patients.

Although hospital bed rest (see above) is probably the best single line of treatment (it has been said that if symptoms do not subside after a few days in bed the diagnosis should be reviewed); this is not always practicable, and it should be borne in mind that the possible financial worries, fear of loss of employment, etc, entailed by this procedure may considerably affect the healing/

healing of an ulcer. If pain persists however, resort should be made to a period of bed rest, as in this case.

From the psychological point of view, the patient should feel that he has a sympathetic, independent person anxious to help him. Under these circumstances he is more likely to unburden himself of his worries and difficulties - a cathartic process of not inconsiderable therapeutic value. The state of the patient's dentition should also be checked.

Considering the long term view, it must be remembered that an ulcer may take six weeks or longer to heal, despite the early subsidence of symptoms. It is important that it should be given every opportunity to heal completely, thereby considerably reducing the chances of recurrence. Patients should be advised not to take spirits or to smoke on an empty stomach - complete abstention is perhaps the ideal. The beneficial effects of reasonable hours of work, thorough mastication, regular meal hours, and an adequate amount of physical and mental rest, should all be made clear to him. The question of change of occupation arises here, and the medical attendant must take care not to dogmatically prescribe a change of occupation without carefully considering its practicability. The practitioner should also keep in mind the possibility of the 'milk-alkali syndrome' and possible calcinosis with impairment of renal function in ~~xxx~~ patients on long term milk and soluble alkali. (Avery Jones, 1957). All the above points are highly relevant to this particular patient.

Surgical treatment of this case is not indicated since -

- (a) No complication (see below) has arisen as yet.
- (b) Symptoms have not been severe enough to compel him to lose time from his work until the present treatment started.
- (c) He is progressing satisfactorily (see below).
- (d) The history is relatively short.
- (e) the maximal histamine response is not unduly high.

PROGRESS NOTES

Mr. Whittet was seen again on 21.5.57. He looked very fit and thoroughly pleased with himself. The pain had completely disappeared and there was now very little tenderness in the epigastrium and no tenderness elsewhere; the whole abdomen now being considerably more relaxed. He had had no morning vomiting since admission.

His treatment had been on the lines outlined/

outlined above, with bed rest, a bland diet, alkalis (Mg. trisilicate & Al. hydroxide), and full doses of Belladonna (as the tincture).

PROGNOSIS

This man had some signs of developing 'pyloric' constriction, viz.

- some slight weight loss
- morning vomiting (prior to admission)
- feeling of 'fullness' after meals
- ⊖ some ? splashing
- moderate amount of fasting juice present on Ba. meal examination
- small difference between 'free' and 'total' acid in maximal histamine test. 'Total' acid is said to be greater than 'free' acid on account of the regurgitation of alkaline duodenal contents.

As these signs have settled down, as far as can be judged, with treatment, it is likely that they were due to so-called pylorospasm (some authorities claim that this is really mostly oedema).

It is possible that this patient will develop some complication in the future such as -

- 'pyloric' stenosis - considering the above this may be the most likely complication
- perforation
- penetration to pancreas
- haematemesis and/or melaena
- alkalosis and/or calcinosis (if he changes to soluble alkali)

Malignant change has not been shown to occur in D.U.; may occur in 0.5 - 5 % of cases of G.U.

If the long-term treatment, as outlined above, is conscientiously carried out by both patient and doctor, there is at least a fair chance that his ulcer may not bother him again. If symptoms do recur, he may well be able to control them with the help of his G.P.

It has been said (Avery Jones 1957) that approximately one third of all patients with duodenal ulceration eventually merit surgical intervention. In this patient the recurrence and persistence of symptoms and more particularly the development of one of the above complications, would suggest the consideration of surgical intervention.

In view of Mr. Whittet's age, his cardiac condition may well limit the number of years in which he is liable to present with duodenal ulcer complications.

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For references see over -

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NEUROLOGICAL CASE

CASE III

Ward 2. Northern General Hospital

Patient's Name	Mr. Robert Hoggan
" Age	66
" Address	33 Drumbrae Terrace, Clermiston
" Occupation	Retired for last 15 years - previously Brewery Cellarman
" Doctor	Dr. Howell
" " Address	19 Parkgate Terrace, Clermiston
Date of Admission	3rd April 1957
" " Examination	29th April 1957

COMPLAINTS

1. 'Lightning', 'shooting' pains in arms and legs - 20 years duration
2. Some 'difficulty in walking' - 15 yrs.

PRESENT HISTORY

Mr. Hoggan has experienced shooting pains in his arms and legs for about 20 years - first noticed in his legs though he states that his arms were affected 'shortly afterwards' (as far as can be judged about a year later). He describes the pain as 'severe' - sometimes it brings tears to his eyes - points out his tibiae (bilateral subcutaneous parts) as its most frequent site; and says that 'it is as though the skin were being stripped off' his legs.

Sometimes he has periods of up to three weeks during which he is completely free of the pains. He has not noticed anything which tends to bring on the pains and nothing really relieves him. (The pains are not brought on by exercise, nor are they relieved by rest.) They seem to occur at any time of the day or night and sometimes keep him awake. He thinks that the pains are not now so bad as they were three years ago (see notes below). Sometimes there is a prodromal sensation of coldness in the legs but frequently the pains come on without any warning.

In the past fifteen years he has noticed himself falling forwards if he closes his eyes while washing his face and considerable 'unsteadiness' on his feet when walking in darkness. This latter he first noticed during the wartime 'blackout' '39-'45.

PAST/

PAST HISTORY

His childhood ailments consisted of Scarlet Fever and Measles (3 times). He gives no history of whooping cough or broncho-pneumonia as a child. He was however taken, because of 'a bad chest', to 'a Professor', who pronounced that he had 'congestion of the lung' and would only live to the age of 38 (!)

He then seems to have remained free of chest troubles for some time, and was 'passed A.I.' on joining the regular army in 1909. (He was in the army 1909-13 & 1914-18. In 1915 he was wounded in the region of the groin and subsequently had one bullet and one 'testicle' removed as ~~xxx~~ result. In 1917 he was 'blown up' and has been very deaf in both ears ever since; also in 1917 he was wounded in the L. forearm, a bullet passing through the forearm and fracturing the ulnar - the scar on the ulnar surface still 'breaks down' from time to time.

After leaving the army in 1918 he was operated on for 'rupture' in 1923; in 1934 he was admitted to R.I.E. with 'broncho-pneumonia' and it was at this time that the condition from which he now suffers was discovered. Since that date he has been troubled off and on with his chest, and has had 'a slight cough' with occasional yellow sputum.

A few years after his discharge from the R.I.E. the pains started and over the course of years his walking was affected - states that he 'couldn't control his legs properly' - so much so that by 1954 he could not walk at all and he was admitted to the Northern General Hospital. After a few weeks - 'getting injections of Penicillin' - he was discharged 'much better' but was re-admitted 12 months ago for similar treatment over a similar period. At present he can walk and get about quite well but his 'lightning pains' persist.

Appetite - is ~~good~~ poor, but no 'indigestion' of any kind.

Bowels - quite regular, no colour change noted

Mict. - no hesitancy, urgency, dysuria, ~~xxx~~ but x 2 nocturia. Good stream.

No history of chest pain, ankle swelling, or undue breathlessness.

FAMILY HISTORY

Father d. (45) - 'cancer of bowel'

Mother d. (80+) - 'heart attack'

Sibs. - ~~xxx~~ younger sister - A & W.

His wife died 12 years ago after being bed-ridden for 5 years following 'a stroke'. During her last years she x 'went a little queer in the head'.

Children - two married daughters aged 43 & 37
- both A & W.

SOCIAL/

SOCIAL HISTORY

Mr. Hoggan lives with his eldest daughter, and prior to his retirement worked for 25 years in a Brewery.

He is a fairly heavy smoker - 15-20/day - used to smoke more when he could afford it. He used to drink one (free) bottle of beer per day while working in the brewery, plus an indeterminate amount of whisky. Now drinks almost entirely whisky - 2-3 nips on Saturday nights.

GENERAL EXAMINATION

The appearance was that of an alert-looking, pale, thin, middle-aged man, sitting up in bed and quite at ease. He appeared to be of average intelligence and his temp. was 97.2° F.

CENTRAL NERVOUS SYSTEM

Higher Functions - attention and powers of concentration were good; there were no obvious memory or speech defects.

Cranial Nerves

- I -No history of smell or taste impairment
- II -Full visual fields. Fair visual acuity
(glasses worn for reading only)
- III -Both pupils small but regular, the L. being smaller than the R. Both irises atrophic.
- IV After several attempts there was a slight 'delayed' reaction to light, no reaction elicited to accommodation. No ptosis.
- VI Eye movements full and conjugate. No diplopia, nystagmus or strabismus.
- V -No impairment of facial sensation - power of muscles of mastication good.
- VII -Facial muscles good power, equal both sides.
- VIII -Hearing was grossly impaired on both sides, L. > R. (He wore a standard hearing aid in the R. ear, from which, he said, there was an occasional watery discharge.)

Rinne's Test - NEG. for both ears

Weber's Test - sound was heard best in the L. ear.

These tests indicate a 'middle ear' type of deafness.

A large perforation could be seen in each eardrum.

- IX -Swallowing reflex appeared unimpaired.
- X -Palate moves equally both sides.
- XI -Good and equal power of sternomastoids and trapezii.
- XII -Tongue protruded without tremor, or deviation. No atrophy noted.

Motor Functions/

Motor Functions -

1. No involuntary movements
2. Muscle tone in the lower limbs was slightly reduced; there were some signs of wasting in the calf and quadriceps of both limbs.
3. There was no gross ataxia on walking, but it was noticed that he walked with feet well apart. Romberg's test was strongly positive.
4. Past-pointing occurred (finger-nose test) with eyes closed but not with eyes open. The 'heel-knee' test ~~was~~ also showed some impairment with eyes closed but not with eyes open.

Reflexes -

1. Superficial

Plantars L. ?↑ R. ↓

The testing of the abdominal reflexes caused a generalised constriction of all the muscles of the anterior wall, but there was no localised 'flicker'. This means that the abdominal reflexes were probably present, but they could not be demonstrated.

2. Deep

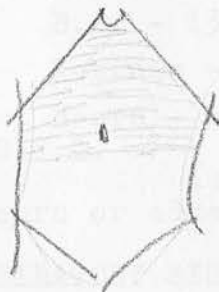
	R.	L.
Ankle	-	-
Knee	-	-
Biceps	+	+
Triceps	-	-
Supinator	-	-
Jaw	NEG.	

Sensation

1. Superficial

Light touch was unimpaired except for

- (a) ? impairment over ulnar border of L. hand.
- (b) anaesthetic oval area approx. 6x12 cm. over antero-lateral aspect, upper 1/3 of L. thigh.
- (c) hyperaesthetic area over ant. wall of abdomen as shown



The patient also stated that he had an 'itchy' skin, but that if he scratched it, it was painful, particularly over the chest.

~~xxxxxxx~~ Pain

= was considerably impaired over the entire trunk and limbs. ('Sharp' and 'blunt' pin test.) This finding was also noted over the face and neck, where it was more widespread than the 'tabetic mask' of Duchenne.

2. Deep/

2. Deep

Vibration sense was absent in all four limbs.

Position sense was considerably impaired in all the interphalangeal joints (hands and feet). There was also some impairment in the wrists and ankles but position sense appeared normal in the knee and elbow joints.

3. Cerebral

There was no defect of stereognosis. The patient's calves were fairly tender to pressure, as was also the tendo-achilles, particularly in the L. leg. Höman's sign was negative.

LOCOMOTORY SYSTEM

Joints - no deformity or inflammation, full range of active and passive movements in all joints.

Deformity - there was no gross deformity of any limb.

Muscles - there was a minor degree of wasting of the larger muscles - quadriceps, biceps, muscles of the calf.

Power - generally fairly good. Diminished grip and dorsiflexion of L. hand due to old injury.

CARDIOVASCULAR SYSTEM

Hands - no cyanosis or capillary pulsation. Although the nails showed no abnormal curvature and no nail-bed fluctuation, the nail - nail-bed angle showed considerable flattening - almost to 180°. This is said to be an important sign of early clubbing.

Radial Pulse - rate 76/min. regular in time and force. Good volume. Character of the wave was normal and the vessel wall was just palpable.

Neck - J.V.P. * normal H.J.R. NEG.

B.P. - 135/85 mm Hg.

Retinae - poor view but both appeared normal.

Heart - well-defined apex beat in 5th space on M.C.L.

Auscultation - Heart sounds closed, no murmurs or added sounds detected.

RESPIRATORY SYSTEM

Rate - 20/min, regular

Cough - moist

Sputum - increased quantity of tenacious, mucopurulent sputum.

Chest - Inspection - poorly covered, symmetrical movements, no gross increase in A-P diameter

Palpation/

Palpation - poor expansion, normal vocal fremitus, trachea central, no axillary or neck glands.

Percussion- increased P.N. over lower lobe of L. lung, particularly marked anteriorly.

Auscultation - bronchial breathing and increased V.R. over lower lobe of L. lung. Whispering pectoriliqy could not be elicited. Breathing otherwise vesicular, no other accompaniments.

ALIMENTARY SYSTEM

Edentulous - dentures worn
Tongue moist and heavily furred.
Mouth and fauces otherwise healthy
No halitosis.

Abdomen - Inspection - little subcutaneous fat, good musculature. Bilateral inguinal scars.

Palpation - was difficult as the patient did not relax well. No tenderness. No visceromegaly. No masses. The bladder was not distended.

Percussion - confirmed the above. There was no shifting dullness.

Auscultation - peristaltic sounds heard.

SPECIAL INVESTIGATIONS

Chest X-ray - showed the presence of brochiectasis in the L. lower lobe and some unfolding of the aorta.

N.B. The former was confirmed by bronchograms.

Blood W.R. Negative (on three occasions)

" Kahn test Negative " "

Blood picture - normal (no anaemia)

C.S.F. Protein - 30 mgm/100 ml
Cell Count - 1 cell/cu.mm.
W.R. - faintly positive.
Lange - 000,000,000,000

DISCUSSION

A. DIAGNOSIS
1. Tabes dorsalis
2. Bronchiectasis

B. REASONS FOR DIAGNOSIS

Tabes dorsalis is diagnosed in this case by the occurrence of the characteristic 'lightning' pains in the legs and arms, accompanied by other disturbances of sensation: i.e. areas of anaesthesia and also of hyper~~xxx~~aesthesia; loss of tendon reflexes (with some muscular hypotonia); sensory/

sensory ataxia (with Rombergism); absence of peripheral vibration and position sense; and abnormal pupillary responses. Although showing some of the features of the Argyll - Robertson pupil these pupils are not typical A-R pupils, showing as they do a Holmes-Adie type of tonic response to light.

Bronchiectasis is indicated by the long history of chest troubles coupled with the finding of mucopurulent sputum and bronchial breathing with increased V.R. over the L. lower lobe. These indications are confirmed by the bronchograms and the chest X-Ray.

C. DIFFERENTIAL DIAGNOSIS

Tabes dorsalis must be distinguished from -

- (a) Peripheral neuritis
- (b) Subacute combined Degeneration of the Spinal Cord.
- (c) Disseminated Sclerosis

Of these, (a) is supported as a possible diagnosis by the finding of muscle and tendon tenderness in the lower limbs. Sensation of deep pressure in these sites is commonly absent in tabes. Ataxia, absent reflexes, and disturbed sensation are all compatible with this diagnosis. Points against are that the pains are quite unlike the tingling usually found in peripheral neuritis, and are characteristic of tabes; there is no loss of ~~xx~~ muscle power such as would be expected ~~ix~~ with a peripheral neuritis of this severity; abnormal pupillary responses are present; and the C.S.F. W.R. was positive.

The case for (b) is similar to that for (a) in that the same factors are in keeping with such a diagnosis, and the same factors are against it plus the very important finding of a normal blood picture.

In the case of (c) the reflexes are usually increased, the ataxia is motor in nature, and it is unusual to find such a gross sensory disturbance. The Lange test often shows a first zone curve in D.S. Additional factors against this diagnosis are the factors outlined as being against (a), plus the important absence in the history of any remission prior to treatment three years ago.

Two points deserve comment at this juncture -

1. Previous anti-syphilitic treatment probably accounts for the finding of a normal C.S.F. (apart from a faint positive W.R.) and the repeatedly negative blood W.R.

2. The plantar reflexes were highly equivocal - such movements as were recorded were very slight. The presence of more definite results would have aided the diagnosis, since they are/

are commonly absent in (a), extensor in (b) and (c) and flexor in the case of tabes.

D. AETIOLOGY, ANATOMY, PATHOLOGY, & FUNCTION

This is a late manifestation of Syphilis, usually occurring 10-20 years after the original or primary infection, affecting males more frequently than females, and occurring in the 35-50 age group. A rare congenital form occurs in childhood or adolescence.

The evolution of the essential lesion of tabes is still a matter of some controversy, but it seems likely that the initial lesion is a meningo-vascular one and is due to the spirochaetes affecting the dorsal roots between the ganglion and the spinal cord. The short and medium length fibres tend to degenerate earlier than the long fibres, the sensory fibres supplying the sacral region often being the first to be involved. The primary optic atrophy of tabes is probably the result of a similar process affecting the sheath of the optic nerve and not, as was formerly supposed, the result of a primary syphilitic degeneration of the optic nerve.

Neuroglial proliferation ^{occurs} at these sites of the disease and the end result is degeneration of the posterior columns of the spinal cord. This appears first in the lower lumbar and sacral regions and if untreated spreads upwards as far as the gracile and cuneate nuclei in the medulla. On microscopic section the affected posterior columns are shrunken and have a translucent, gelatinous appearance.

The pupillary changes are even more obscure in their origin. Some authorities confidently ascribe them to degeneration of the colliculo-ocular fibres in the mid-brain; while others with equal confidence claim that they are due to local atrophic changes in the pupil. Two other theories, not at present in vogue, concern either changes in the ciliary nerves or degeneration of the light reflex fibres.

In addition to producing the effects seen in this case, this affection of the sensory pathway also sometimes causes attacks of acute abdominal pain associated with nausea and vomiting - the so-called gastric crises - and sphincteric disturbances, often manifested as upsets in micturition. These latter are usually revealed as difficulty in starting and stopping, or as overflow. Such disturbances are due to the absence of reflex sensation - the patient does not know when his bladder is full.

Ptosis, diplopia, and squint may also occur, and the patient may suffer attacks of tenesmus.

ET TREATMENT/

E. TREATMENT

The sheet-anchor of all antisyphilitic treatment is Penicillin, usually given as successive courses of massive doses, each course consisting ~~xxx~~ of 10 - 12 mega units over 10 - 14 days. The penicillin may be preceded by a four week course of Potassium Iodide 10 - 20 gr. t.i.d., and Bismuth 3 - 5 gr. I.M. weekly. The rationale of this scheme is to avoid any possible Herxheimer reaction; it is said that this is more likely to occur with severe C.V.S. or C.N.S. involvement. In successfully treated cases the W.R. often becomes - and remains - negative, but a persistently positive W.R. is not necessarily a sign of active disease. Pyrexia, either electrical or malarial, is also still sometimes used for neurosyphilis.

In view of the C.S.F. and blood findings and the fact that this patient has had three courses of penicillin in addition to the older type of treatment (probably arsenicals) in 1934, it seems unlikely that he has active disease. Treatment should therefore be symptomatic, directed toward keeping the patient's present level of function. His ataxia has been very successfully treated by means of physiotherapy (re-educational exercises) and he now gets about extremely well. He should be encouraged to keep 'on the move' and out of bed, and every effort should be made to preserve his eyesight. He should have fairly frequent (about six-monthly) checks of his eyes with both his optician and his doctor. Watch should also be kept for painless ulcers, especially on the feet.

The 'lightning' pains are often greatly improved, as in this case, by the general treatment. Analgesics such as tabs. Codine Co. (1 - 2 every 4 hours) or tabs. A.P.C. should now be prescribed for the pain. In other cases in which the pain is more severe and/or with gastric crises it is sometimes necessary to carry out a cordotomy - section of the spinothalamic tracts - for relief.

If the micturition reflex becomes depressed he should be taught to empty his bladder at regular intervals - in any event such teaching would be no bad thing. Should this become ineffective Carbachol (0.5-1.0 ml S.C.) may be given. In some cases catheterisation may be necessary - a spell of tidal drainage is often very helpful if the patient develops incontinence with overflow. Special attention should be given to urinary sepsis should it occur (often the cause of such a patient going downhill) which should be treated along the usual lines.

It must be noted that the treatment of this patient's bronchiectasis is at least as important as the treatment of his tabes. This should be/

be on three main lines -

(a) he should be instructed in postural coughing so that he can carry on with it at home

(b) he should be given short sharp courses of a broad spectrum antibiotic e.g. chloramphenicol in the event of a disabling super-infection in his chest. Alternatively (but probably with less effect) he might be put on prophylactic penicillin. A suitable oral preparation of 'Estapen' (about 200,000 units/day) might be used. The makers of this product claim that it has a predilection for the lungs, in which it tends to occur in high concentration.

(c) he should be told to report to his doctor if his cough becomes worse or his sputum more copious, purulent, or foul smelling.

Removal of any septic foci (in sinuses etc), a good diet, and plenty of fresh air are useful adjuncts.

F. PROGNOSIS

While apparent arrest may occur, in all cases of tabes there is a tendency towards progressive deterioration; Neurons have been destroyed and their place taken by connective tissue; this unhealthy environment, accentuated by deficiency of the vascular supply due to endarteritis, must depress the vitality of the living neurons remaining in the area, and may eventually determine a spread of the degenerative process. From this it would appear that the most that can be expected from treatment is alleviation of the pains and such urinary disabilities as may occur, and prolonged arrest of deterioration.

In this patient arrest would appear to have been achieved; considering the age of patient and the presence of his lung condition it seems likely that he may live out his life without any new developments in his syphilitic disease process. Much depends on careful supervision - he should have a yearly check on his W.R. - then provided that his eyesight is kept in good repair and a watch is kept on his bladder, no spectacular new developments are likely, and this disease process is unlikely to be the primary cause of future death.

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CASE IV

Patient's Name	William Wilson
" Address	121 Waverly Cres. Bonnyrigg
" Age	14
" Occupation	Schoolboy
" Doctor	Dr. Penman
" " Address	Eckbank
Date of Admission	7th January 1956
" " Examination	10th January 1956

COMPLAINTS

Malaise, dizziness, anorexia, and vomiting - 2/52

PRESENT HISTORY

On Tuesday, 27th December 1955, this boy felt 'sick' and generally unwell, lost his appetite, and decided of his own accord to go to bed at 7.30 pm. The following day he got up but still felt rather unwell and slightly dizzy. On 29.12.56 he visited his grandmother; when returning by bus he felt very sick and dizzy, and on arriving home he went straight to bed - the same day his mother noticed that his skin had a slight yellow tinge.

Friday 30th he stayed in bed and the family doctor was called in; he left a bottle of medicine and promised to call again. Over the weekend the patient felt very ill and vomited (recent food only) on two or three occasions. On Monday 2nd Jan. he had very little to eat - in the latter half of the day he only had a little orange juice and some 'tonic' all of which he later vomited. The next day the doctor visited him again, gave instructions that he should remain in bed and promised to call again on 6th.

The week passed without any significant change in the boy's condition and as the doctor failed to appear on 6th the boy's father called on the doctor the following day, to discover that the doctor had been unwell. As there had been no real change in the boy's condition his doctor recommended his admission to the R.I.E. for investigation. He was admitted that day (7.1.56). The patient remembers nothing of the journey in the ambulance and claimed to have had a 'blackout' during the journey./

journey. During his stay in the R.I.E. he has felt gradually better apart from the presence of a slight but continuous itch in both feet and legs

The patient had never noticed any unusual colouration of his motions or his urine. He had not been taking any drugs other than the medicine prescribed by his doctor and states that he has never had any injections.

This boy's best friend at school has also had 'jaundice' and to the best of his knowledge still has it. Apparently he had not seen this friend for some three weeks prior to the onset of prodromal symptoms in his own case. This friend had been confined to bed with jaundice and although the patient had not been to see him, contact had been maintained through the brother of his friend. According to latest knowledge this brother has not been jaundiced. The patient was not aware of any other case of jaundice amongst his friends.

No history of any contact with damp or rat infested places. No history of any rash.

No history of any chest pain, ankle swelling, or undue breathlessness.

Appetite usually good, bowels regular.
Mict. n.a.d.

PREVIOUS HISTORY No previous specific illnesses, accidents, or operations.

FAMILY & SOCIAL HISTORY Parents both A & W
Sibs - 1 sister (age 11)
who attends a different school. No other member of the family has been jaundiced or has felt unwell.

The patient had typical schoolboy interests; reading, the cinema, ~~tz~~ and television featuring high on the list.

GENERAL EXAMINATION The patient was a fair-haired boy with a slight yellowish tinge to his skin and sclera. He was of average intelligence and his nutritional state was good. Temp, 96.5 F.

CARDIO-VASCULAR SYSTEM

Hands - no palmar erythema. n.a.d.

Radial Pulse - 60/min. regular in T & F
Good volume. Both synchronous.

B.P. - 110/70 mm Hg.

Neck - J.V.P. normal H.J.R. NEG.

Oedema - nil

Retinae - good view, both normal

Heart - A/B in 5th space on M.C.L.
H.S. closed. No murmurs detected.

RESPIRATORY/

RESPIRATORY SYSTEM

Rate 16/ min. regular
 Chest symmetrical, spine straight
 Trachea central
 No lymphadenopathy
 P.N.=P.N.=normal. Liver reached
 the level of the 5th space on R.
 Breath sounds vesicular without
 accompaniments.

ALIMENTARY SYSTEM

Tongue moist & healthy
 Teeth, tonsils, & fauces all healthy
 Abdomen - Inspection - smooth and symmetrical,
 no pulsations, no visible veins
 Palpation - Liver palpable two
 fingers breadth below the costal margin, slightly
 tender. No splenic enlargement, no other palpable
 viscera. No shifting dullness.
 Percussion - confirmed the above
 Auscultation - normal bowel sounds
 heard.

CENTRAL NERVOUS SYSTEM

Higher Functions - no obvious impairment.

Cranial Nerves

- I -No history of upset in taste or smell
 II -Glasses not worn. Full visual fields
 III -Pupils equal, round, reacting both to light
 IV and accommodation. Movements full without
 VI strabismus, diplopia, or nystagmus. No ptosis.
 V -Facial sensation unimpaired
 VII -Facial muscles of good power, equal both sides
 VIII -Hearing unimpaired.
 IX -No difficulty in swallowing
 X -Palate moves equally both sides.
 XI -Good and equal power of sternomastoids and trapezii.
 XII -Tongue protruded without tremor or deviation.
 No atrophy.

Motor Functions - No involuntary movements
 -Muscle tone normal
 - No paralysis or wasting
 - Co-ordination good

Reflexes -

Superficial	Plantars R. ↓	L. ↓
Deep/	Abdominal	✓/✓ ✓/✓

Deep

	R.	L.
Ankle	+	+
Knee	+++	+++
Biceps	++	++
Triceps	++	++
Supinator	++	++

Sensation - Touch, pain, vibration, proprioceptive sense - all intact.

Urine Examination

10.1.56. S.G. 1.010 - Yellow in colour
 Reaction acid
 Alb., Sugar, Ketones - nil.
 Bile pigments, bile salts, - POS.
 Urobilinogen - POS.
 Pus cells, R.B.C.s, W.B.C.s, NIL
 No casts seen

Liver Function Tests

showed parenchymal damage with an obstructive element.

Agglutination Test for Weil's Disease - Negative
 (on 10th day of disease)

DISCUSSION

A. DIAGNOSIS Infective Hepatitis

B. REASONS FOR DIAGNOSIS

The occurrence of characteristic prodromal symptoms a few days before the appearance of a yellow tinge to the skin. Especially notable in this respect are anorexia, vomiting, and general malaise. The later appearance of pruritis is due to the presence of bile pigments in the skin.

The yellow colour of the skin and enlargement of the liver, coupled with the presence of bile and urobilinogen in the urine indicate some kind of liver dysfunction. The age of the patient together with the history of contact with another case of jaundice are strongly indicative of infective hepatitis.

C. DIFFERENTIAL DIAGNOSIS

Infective hepatitis must be distinguished from -

1. Homologous serum jaundice (syringe transmitted)
2. Spirochaetal jaundice (Weil's disease)
3. Acute massive necrosis
4. Extra-hepatic obstructive jaundice

In/

In the case of 1. there would be a history of previous injection(s), and the absence of this finding, coupled with the positive finding of a contact with another jaundiced person, makes this diagnosis very unlikely.

The history is also against a diagnosis of 2. as there is no history of exposure to infection. In Weil's disease haemorrhages are common (e.g. epistaxes and petichae); blood and casts are often found in the urine - these signs were absent in this case.

3. is unlikely as there is no history of exposure to hepatotoxic agents; had 3. been the cause of the illness one would have expected a much greater depth of jaundice and a far more severe illness.

It is worth noting that the most likely cause of jaundice in person of this age group is infective hepatitis; in a person over middle age 4. would be a strong possibility especially if there were no history of contact with jaundice. In this case the age and the history of contact virtually rules out 4. as a possibility.

ETIOLOGY, ANATOMY, PATHOLOGY, & FUNCTION.

The cause of infective hepatitis is believed to be a virus, spread by human faeces or pharyngeal secretions, and infection occurring by direct contact with either sufferers from the disease or apparently healthy carriers. The incubation period is 14-35 days and the disease is frequently epidemic.

The primary lesion is in the liver and consists of a zonal necrosis affecting the ~~xxx~~ centrilobular liver cells. As the reticulum framework of the liver is unaffected complete regeneration of the liver lobules without distortion or fibrosis usually follows, in the majority of cases, on recovery.

One of the first functions of the liver to be affected is the re-excretion of urobilinogen, so that one of the earliest signs is the excretion of abnormally large quantities of urobilinogen in the urine. This is followed later by an inability to excrete all the bilirubin, so that the next sign is increasing quantities of this substance in the urine. As the disease progresses the liver cells become increasingly swollen and may cause obstruction of the bile canaliculi; this means that more and more bilirubin spills over into the blood and thus into the urine while less and less finds its way into the intestine. This latter event gives rise to pale stools and the disappearance of urobilinogen from the urine.

The detoxification of pituitary anti-diuretic/

anti-diuretic hormone may also be affected, causing oedema in severe cases. The detoxification of other substances may also be disturbed - e.g. that of the naturally occurring oestrogens. This latter sometimes gives rise to slight gynaecomastia. (See additional note below re. water retention in these cases.)

E TREATMENT

There is no specific treatment; none of the present range of antibiotics have been shown to be effective.

There are good grounds for 'barrier nursing' the hospital case (Dunlop et al 1958); though the pre-icteric, and ^{therefore} usually pre-hospital, phase is the most infective.

The three most important considerations in treatment are -

1) Bed Rest - It is usually stated that this is best imposed until all evidence of disease activity has subsided; though this may be impossible in cases where the disease process goes on for months or years. Chalmers et al (1955) found that strenuous activity could be safely undertaken (checked by a one year follow-up) when the serum bilirubin has dropped below 1.5 mgm./100ml and the 45 min. Bromsulphalein test shows 5% retention or less.

Kramer & Fuchs (1953) reached similar conclusions, stating that bed rest should be continued as long as there are -

- (a) symptoms
- (b) enlarged liver
- (c) serum bilirubin above 2.0 mgm./100 ml
- (d) bromsulphalein 45 min. retention test above 10%

2) Diet - The usual regime is that a very light diet consisting largely of carbohydrate - e.g. fruit drinks and glucose - is given for the first few days and thereafter a diet consisting of normal amounts of carbohydrate with increased amounts of protein and decreased amounts of fat. (i.e. Carb. 350-400 Grms., Fat 40-50 Grms., and Protein 100-120 Grms.)

It has been found however that patients who are fed a high protein diet (19% protein) from the onset of the condition (if necessary by tube feeding) have a mean duration of illness 14% less than those given a 'normal' protein diet (14% protein) and allowed to eat what they wish in the initial stages. (Chalmers et al, 1955). The probability of this finding occurring by chance is 1 in 20. In the same series it was found that a high calorie diet, or dietary supplements (Choline and multi-vitamin preparations) did not make any difference to the disease process.

3) Drugs/

3) Drugs - It is advisable to avoid those drugs which are known to be detoxicated in the liver e.g. morphine and the barbiturates; also those which are potential hepatotoxic agents.

Cortisone and Corticotrophin appear to aid recovery but their use in the uncomplicated cases does not seem to be indicated.

If ascites arises it should be treated by -

- a. Bed rest
- b. Sodium restriction
- c. Mercurial diuretics
- d. High protein intake (unless there are signs of coma)
- e. Avoidance of abdominal paracentesis, except in the initial stages, as it drains off considerable quantities of protein. (Brit. Med. J. 1957 I, 1173)

In severe cases intravenous feeding may be necessary; a 10% soln. of glucose should be used, and up to 300 Grms./day may be given in this fashion. Vitamins of the B complex are also useful in the severe case. (e.g. Thiamine 5 mgm/day; Nicotinamide 50 mgm/day; Riboflavin 5 mgm/day.) Increased amounts of Thiamine (about 20 mgm/day) should be added to the infusion when large quantities of glucose are given.

In the acute phase the most important complication, which may occur in the severest cases, is hepatic coma. This is believed to be due to a failure of urea synthesis with subsequent intoxication by ammonia and other nitrogenous compounds.

It is only in recent time that medical treatment has had any success with this complication. It is most important that hepatic coma should be vigorously treated in cases where it is due to viral hepatitis, since the ultimate prognosis may be relatively good if the patient can be tided over his hepatic insufficiency. The basic principles of treatment are -

A. Protein intake should be reduced to a minimum. An intake of as little as 20 Grm/day can be tolerated for long periods. (Sherlock et al, 1956). Carbohydrate should be given in large quantities, if necessary by an intra-caval drip (50% glucose with a little heparin). Glutamic acid may be tried (Walshe, 1955), given as a slow I.V. infusion of sodium glutamate. (Up to 20 Grms in 500 ml. of 5% glucose soln. ~~ix~~ given over 3 hours.) The beneficial effect which is sometimes obtained has been ascribed to the binding of excess ammonia to form glutamine./

Recently l-arginine hydrochloride has been tried (Najarian & Harper, 1956). It is given as a dose of 25 Grms. dissolved in 500 ml. of 10% glucose, repeated if necessary.

B. Agents likely to produce nitrogenous intoxication should be avoided. i.e. ammonium compounds, amino acids such as methionine, ion exchange resins, and acetazolamide. Bleeding from oesophageal varices must be carefully controlled by such methods as the Sengstaken tube followed by gastric lavage, mild aperients, and high enemata.

C. Anaemia must be corrected - if necessary by blood transfusion.

D. Hepatotoxic drugs and those detoxified in ~~xx~~ the liver should be avoided if possible.

ADDITIONAL NOTE

It was later discovered that this boy's 'blackout' had all the characteristics of an epileptic fit. (From descriptions by observers ~~xxx~~ present at the time.) He is not a known epileptic, but presumably has an epileptic pattern of E.E.G., and was tipped into a convulsion by water retention caused by his damaged liver failing to detoxicate its usual quantity of anti-diuretic hormone.

PROGNOSIS

The vast majority of cases, of which this is typical, proceed to complete recovery in 3-6 weeks; mortality in children and young adults being 0.2%.

Very occasionally death ensues in the acute phase - these cases are usually those in which the disease is superimposed on a pre-existing dietary deficiency; e.g. alcoholics, pregnant women, and elderly patients. In similar cases the patient may survive the acute phase only to develop at a later date the signs and symptoms of chronic hepatic insufficiency and/or portal hypertension due to post-necrotic scarring.

A small number of cases produce a history of partial and alternating remissions and relapses. Biopsy studies show a continuation of hepatic cell degeneration and inflammatory cell infiltration with increasing fibrosis. As a result, chronic hepatic insufficiency, or portal cirrhosis, may occur. These cases may also be produced asymptotically, but more usually abdominal discomfort, bouts of diarrhoea, pruritis, and persistent elevation of the serum bilirubin are present.

There is no indication to treat this boy for his latent epilepsy (unless he has further fits), but he should, of course, be kept under observation.

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