Clinical medicine Cases.

Cases of

1) Coronary Occlusion
2) Valvular Disease of Heart: Hypertension - Failure.
3) Diffuse Glomerulo-nephritis.
4) Peptic Ulcer: Haematemesis.
5) Lobar Pneumonia: Empyema.
Case of
George Roberts.          Aged 56.      Boilerman
4 Grindlay Place.

Admitted: 21.2.40 to Ward 26 (Prof. Davidson).
Discharged: 9.4.40.

Complaint: Pain in precordial area, which "gritted the breathing," of twelve hours duration.

History (32.2.40) At 12.30 a.m. on 21.2.40 patient was awakened from sleep by an intense pain in the precordial area, which was worst in the lower left substernal area, and radiated "like a knife" to the left nipple region, and to the left shoulder and down the inside of the left arm to the tips of the 4th - 5th fingers, which were "numb and prickling." There was slight radiation of the pain to the other side of the chest.
The pain was "gritting" in nature and gave him a tight feeling in his chest. The patient (who was very co-operative throughout) added the information that the skin of the left side of the chest was tender and irritated by his pajamas. He tossed and turned with the pain associated with the pain there was severe dyspnoea, breathlessness, and a feeling of faintness.

Consciousness was never lost.

Ascribing his pain to indigestion, the patient tickled the back of the throat to produce vomiting. He retched and brought up a little wind and phlegm, but got no relief.
There was never any nausea.
HISTORY
For the rest of the night the pain " went back and forward" and during one of these remissions the patient got a little sleep. As the night went on, though the pain maintained its severity, the breathlessness and faintness lessened.

In the morning he rose to dress; almost at once he had a severe attack of pain with great breathlessness; he " broke out in a cold sweat " and his face in the mirror was " as white as his shirt. " The doctor was now sent for, and the doctor sent patient straight into the Infirmary. There he got an injection which sent him to sleep.

Now (22.2.40) 36 hours after the onset, the pain is almost gone, he says, there is no breathlessness and he feels much better.

PREVIOUS HISTORY
The patient has " never had a pain like this before " - so severe, with such a distribution, or occurring, like this, during sleep. However, for the last 2 or 3 years he has noticed increasing breathlessness on exertion, and slight praecordial pains on climbing hills. For example when he walks up Leith Street nowadays there is a slight pain localised to the praecordium, not severe enough to notice unless he is thinking about it, and disappearing when he reaches the level and walks along Princes St.

Swelling of his feet at night has been noticed lately - his feet are marked by the laces on his
Previous History

shoes.
Occasionally he suffers from headache, of no definite
distribution. He has never had spasmodic
headaches, loss of memory, amnesoes or anything
in the nature of a hypertensive cerebral episode.

Previous illnesses

Whooping cough. Measles. Boils. — all in the
distant past.

Family History

His father died aged 81. of "just old age"
His mother 98. " " "
He has 8 children — 6 of these are
alive and well today. One died neonatally; one
died a few years ago of a Perforated Gastric Ulcer.

Habits: Use of Alcohol and Tobacco — moderate.
He is a "hearty eater." His present
occupation — Boilerman in a picture house, which
he has been for a few years only, is much less
strenuous than his old one of mining and has
made him put on a lot of weight in the last
2 or 3 years.

11.2.49 Examination: Patient is grossly obese, dull
with morphia. He lies in bed, restless, and
groaning intermittently. Respiration are
stertorous and otherwise not obviously
laboured. The patient is a short man, with
a ruddy face, a good musculature, a wide
costal angle.

CARDIO-VASCULAR System.

Complains now only of a dull precordial pain which has ceased to radiate. There is now no faintness, dyspnoea or palpitation.

Pulse: 64. Regular in time and force, of good volume, the vessel wall is just palpable. None of the palpable arteries show signs of sclerosis.

Heart:

No apex beat is visible or palpable. The chest wall is very fat. (No beat ever became palpable the whole time patient was in the ward.) Percussion gave no evidence regarding the size of the heart, owing to the thick chest wall.

On Auscultation - In all areas the heart sounds were faint, but pure and closed. In the Aortic and Pulmonary areas, the heart sounds were almost inaudible. There was marked Tic-Tac rhythm. A Pericardial friction rub could be heard in the fifth space and over the fifth rib near an area from the 2" to 4" from the midline. It was faint & silky. Nothing was heard in the intercostal region.

The Neck Veins were not engorged. There was no oedema of the neck and sacral region.

The FUNDUS Oculi was of normal appearance. The vessels were not sclerotic, the veins were not compressed; the reflex from the artery walls seemed normal.

RESPIRATORY System.

No breathlessness or any respiratory symptom is now complained of.

Respiration: Rate 21/min. Respirations are thoracico-abdominal in character, and the
RESPIRATORY SYSTEM

Chest and upper abdomen move freely and equally. This is confirmed by palpation. The vocal phoniics is faint but equal on both sides. The percussion note is normal, and equal on both sides.

Breath sounds vesicular, with no abnormal accompaniments. Vocal resonance normal.

ALIMENTARY SYSTEM

Slight occasional flatulence is complained of, after over-eating.

Teeth - all false. Tongue - clean and moist.

Abdomen: Very obese, but otherwise examination reveals no abnormality. Particularly, there is no liver tenderness or demonstrable enlargement.

Renal System: No complaints. No physical signs of disease of urinary system. Urine is acid, contains no albumen or sugar. Specific Gravity 1.018. Microscopic examination reveals urate crystals, debris, no other deposit.

Other Systems: No abnormality was revealed.

Laboratory Investigations

22.2.40. Blood Wassermann was Negative.
Blood Urea Nitrogen - 31 mg.

21.2.40. Haemoglobin 94% (Sahli).
White Blood Corpuscles - 14,100.

22.2.40. ELECTROCARDIOGRAM.

Rate - 95/minute.
Normal sinus mechanism.
ECG.

P-waves are of low voltage in lead II, inverted in lead II, diphase in III.
PR interval - .14"
QRS - upright in lead I, with slight depression of the ST segment. - diphase in lead II, with elevation of ST segment. - displaced downwards in III, with an enlarged Q wave and elevation of the ST segment.
T waves - upright in lead I, inverted in leads II, III.
"The changes" to quote the report "are therefore those of a recent coronary thrombosis situated posteriorly.

A diagnosis of coronary occlusion was arrived at, in consideration of the history of onset during sleep, with pain persisting for hours; leucocytosis; low blood pressure; pericardial friction; and the Electrocardiogram.

Treatment was on the usual lines.

The patient was kept at absolute rest in bed. Repeated morphine injections were given whenever pain and restlessness required them - which was frequently in the first few days. Hot bottles were provided. Otherwise, no "circulatory stimulants" were given, and digitalis was also avoided. Laxatives were not given. The first day no desire for food was expressed by the patient, but fluids
TREATMENT

were permitted as required. Next day a 1000 Calory diet was begun with, providing small meals, 50 gm protein, designed both to reduce the patient's obesity, and to diminish metabolism and lighten the task of the heart. Laxatives were withheld.

TREATMENT and PROGRESS NOTES

On 22.2.40, the second day of the illness, the patient's general condition was much improved. Pain was gone, or almost so, being reduced to a vague precordial unease. The Pulse was 80, Respirations rate 20, blood-pressure 120/60. Even the very considerable exertion required when the patient took off his tight-sleeved bed-jacket for blood-pressure estimation produced no dyspnea. [with some models of bed-jacket, absolute rest and the regular estimation of blood-pressure are mutually exclusive] The heart-sounds were faint, as they have remained throughout, but the friction-sound had disappeared, and the tic-tac rhythm was no longer in evidence.

Next day I saw the patient at 11:30 a.m. He was in no obvious distress, and on being questioned, said the old pain was quite gone, adding however, as an afterthought, the information that since he woke up that morning he had been a new pain in his chest, "not the same sort of pain as the old one"
and much less severe. It did not radiate; and unlike the first pain, it was increased by deep inspiration. Pulse was 93, regular in time and of good volume. Blood-pressure was 105/70.

On looking at his chart, there was a newly done blood pressure recording of 38/25. Inquiring established that there had been a new coronary infarction that morning; the 38/25 B.P. reading had only been done one hour previously. And yet now, except for the pain, the patient’s general condition and that of his blood pressure were satisfactory, a rapidity of recovery which seemed to promise well for the future, if it could be taken as an index of his cardiac reserve.

Examination of the chest revealed no change from the previous day. Sounds faint; no pericardial or pleural friction; no tic-tac rhythm.

An ELECTROCARDIOGRAM taken later that morning revealed some changes compared with the one done 6-8 hours before. There was more elevation of S-T in Leads II, III. T was now inverted in lead III. These changes were taken to denote an extension of the original infarction.

From today, the 23rd, Phenobarbital 400 i.e. t.i.d. was given regularly as a sedative, and morphine injections were only required once or twice in the succeeding weeks, for restlessness. Charcoal 3 i. t.i.d. was also begun; to diminish his slight tendency to flatulence.
Treatment. Progress Notes

24.2.40. Today the condition was good. Pulse regular, rate 73, blood pressure 125/70. On the 22nd & 23rd the urinary output was respectively 12 oz. & 22 oz.; today the circulatory improvement is documented by a urinary output of 60 oz.

26.2.40. W.B.C. 8,000. Hb. 94%. (Sahli). Improvement maintained.

From now on, progress was good and uneventful. There has never been any return of pain, though occasionally at night there would be an awareness of slight uneasiness in the left side of the chest. Even when, eventually, he was discharged, to the convalescent home, there was still this "tightness" in the chest now and then, after meals.

Breathlessness has never returned. The bowels move, without difficulty, once a day.

For the first 10 days or so, the urinary output showed a rising trend - of 12, 22, 60, 56, 50, 56, 52, 56, 48, 60, 62, 76, 90. Concentrating power of the kidneys was good (in the 24 hrs. static gravity was 1.034) so this rise in urinary output, in the absence of diuretics, was a sign in all probability of increasing efficiency of the heart, and evacuation of occult oedema.

Unfortunately, no records of weight could
be taken, as there would have been too much disturbance of the patient.

From the 3rd week onwards the urinary output was normal, and fairly constant.

By now the risk of cardiac rupture was no longer present.

On 2.3.40, the electro-cardiogram was taken once more. The report was that there were changes characteristic of a healing infarction.

Blood Pressure readings on March 4th, 7th, 11th, 13th, 21st. were respectively 120/78, 115/65, 112/80, 120/75, 122/80, 118/75.

Another E.C.G. on 12.3.40 showed "changes compatible with an old healing infarction."

Further E.C.Gs taken on March 20th and April 1st revealed only slight changes.

Blood Haemoglobin readings (sahli) on 21.2.40, 26.2.40, 4.3.40, 12.3.40, and 3.4.40 were respectively 94%, 95%, 83%, 89%, 92%.

The pulse rate which for the first week or so varied between 90-100, subsequently settled down to a more or less constant 85.

On March 29th patient was thought to be a little oedematous round the ankles. 2cc. of 1% strychnine were given intramuscularly.

The urinary outputs from 25.3.40 to 30.3.40 were (oz) 48, 46, (10st) 88, 60, 22, 34.

Towards the end of his stay in the ward, the heart sounds were a little louder, but the apex beat never became palpable.
On April 3rd, 6 weeks after the first attack, he was allowed up for quarter of an hour. He felt a little weak and shabby, but none the worse. The periods out of bed were slightly increased in the next days; and on April 9th he was discharged to the convalescent home.

**Discussion**

**Diagnosis.** It is a convention to produce a long list of diseases which may simulate coronary occlusion — Angina Pectoris, Perforated Peptic Ulcer, Dissecting Aneurysm etc., but it would be carrying convention too far to pretend that any difficulty ever existed in the diagnosis of this case. The character of the pain — "gritting," its distribution, and the associated breathlessness and fall in bloodpressure, localised it to the heart. The fact of its onset during sleep, the observed blood-pressure figures following
**Diagnosis:**
the second attack, the restlessness of the patient, the leucocytosis, the tic-tac character of the heart sounds, and, above all, the persistence of the pain — these signs and symptoms together provided conclusive evidence that actual infarction of the myocardium had occurred.

Certainly, the patient's history does indicate that for some time Angina Pectoris has existed, and that the contrast between the previous mild anginal attacks, and this, is very striking.

In addition to the clinical evidence there is the testimony of the Electrocardiogram.

**ETIOLOGY:** Cardiac ischemia may be due to an absolute diminution of coronary blood-flow; or it may result from failure of the coronary circulation to keep pace with the expanding requirements of an overworked heart. Specifically, cardiac infarction — acute cardiac ischemia, may arise from one or more of the following causes.

1) **Coronary Arteriosclerosis**, with usually Atheromatous; with or without thrombosis
2) **Coronary occlusion by Syphilitic Aortitis**
3) **Coronary Embolism** (rarely)
4) Where there are causes of work hypertrophy of the heart, necessitating a greater coronary blood-flow, any of the above causes (1, 2, 3) will be facilitated.

There is no clear explanation why, in the absence of sudden thrombosis, coronary arteriosclerosis should sometimes produce a slow
myocardial fibrosis, with occasional anginal attacks, perhaps, but terminating in congestive failure, and why at other times the premonitory anginal attacks are succeeded, more dramatically, by acute cardiac infarction, without obvious precipitating cause. Syphilitic arteritis is suggested by nothing in the history or examination of this case. There is no reason to think of embolism. But everything in the patient's history, age, and appearance is suggestive of coronary artery disease. A man past middle age, grossly obese, short, full-bodied, ruddy, with a wide costal angle — that is the "musculo-digestive habits" straight from the pages of a clinical atlas, and one would be mildly surprised if such a patient did not develop arterial disease, whether essential hypertension, or coronary arteriosclerosis or both. That may be exaggerating a little, but degenerative vascular disease in general, and hypertensia and coronary disease in particular are, of all systemic diseases, the most selective in their common constitutional background.

Apart from the diastasis, and since clinical examination failed to establish the size of the heart, there is only the presence of left-axis deviation to support a diagnosis of antecedent hypertension. The history was not suggestive: histories are often unhelpful in essential hypertension. Retinal arteriosclerosis was not present; but, again, though the presence of sclerosis of the retinal vessels is pathognomonic of
previous chronic elevation of blood pressure, its absence by no means rules it out.

On the whole, therefore, it seems almost certain that there is cardiac infarction, the result of coronary occlusion by arteriosclerosis, with perhaps antecedent essential hypertension.

CLINICAL ASPECTS.

The disease, like the diathesis, here provided a text-book picture. The sudden severe constricting pain, its characteristic radiation, its very slow disappearance from the periphery inwards, so that the praecordial pain was the last to go, these points are all characteristic of cardiac ischaemic pain. The restlessness and the slowness of disappearance of pain, the shock, the leucocytosis are the accepted characteristics of coronary "thrombosis" as compared with Angina Pectoris. The praecordial hyperaesthesia is interesting; I have not heard of its occurrence in non-infarctive cardiac disease.

The course of convalescence was chiefly notable for the considerable recuperative powers displayed by the heart which, after a severe two-stage infarction, could yet keep the circulation at a level of efficiency compatible with absence of dyspnoea, congestion, and, most strikingly, could restore the clinical state and condition of pulse and blood pressure within an hour after the prostration of the second infarction, to a satisfactory level.

It was therefore early apparent that congestive failure was not greatly to be feared in the immediate future, in the absence of fresh infarction. And the first 3 weeks, safely past, removed the fear of delayed rupture of the organising
Discussion: Clinical Aspects

Infarction — but not the danger of aneurysm formation at a later date.

TREATMENT:

Little comment is necessary on the treatment. Morphine is the first indication, and as soon as possible thereafter, absolute rest in bed for 6 weeks at least, rest being aided by sedatives, avoidance of stimulants, a low-calorie diet. All these were provided. In this case especially, good results may be hoped for from a continuance of the low-calorie diet; if the obesity can be much reduced, that will be so much gained for the cardiac reserve.

In hospital, a beginning was made later by increasing the patient's activity.

In the convalescent home, this will be the most important part of the treatment, a gradual exploration of the possibilities of the cardiac reserve, with moderate exercise within this. I do not know how long a supervised convalescence is possible, the ideal would be for the patient to remain in the home while very gradually, the amount of exercise he takes is increased. Pain or dyspnoea will be indications (which should not be sought) that the patient is doing too much. Eventually the capacity of the recovered heart will be learnt by the patient and this future life regulated accordingly. Diet too will be important. Eventually of course, a somewhat larger allowance than 1000 calories will be needed; but this may well be continued till there has been a substantial weight reduction.
Even now, however, while it is permissible and advisable to starve the patient as far as calories are concerned, PROTEIN must be given, to repair wear and tear. 50 gms. of high-class protein is a good allowance — enough for the above purpose, not enough to have significant metabolic stimulus. Vitamins too must be given plentifully. Otherwise the diet may be mainly carbohydrate, and, incidentally, if the patient is to bum his own fat he must have carbohydrate enough to do it with and to prevent the appearance of acetone bodies in the breath and urine.

Sleep, both during convalescence and after, will have to be secured, whether by abstinence of injudiciously late or heavy meals, worry, excitement, or by the administration of necessary of mild hypnotics (soporific, paraldehyde, phenobarbital, bromide etc.)

Work should be given up. However, if economic necessity prohibits this, probably the patient’s present occupation which has regular hours and is not arduous, would have to do.

For the rest of his life the patient will have to concentrate on living easily. Medical advice may help him in this respect, but mostly he will have to rely on his own intelligence.

Prognosis: If the patient conscientiously follows the principles of life which will be recommended to him, then he may expect to live as long as his coronary arteries let him — that is a period difficult to estimate and impossible to affect by direct treatment; but it may be far longer enough yet. If the coronary disease does
Discussion - Prognosis

Progress, then there may be another and probably fatal interaction; or there may be slow fibrosis of the myocardium with the gradual onset of congestive failure.

The return of any hypertension there may have been before is unlikely; apart from the fact that the heart could probably no longer sustain a high blood pressure, it is a well recognized fact that frequently, after a markedly hypertensive patient has had a coronary thrombosis, hypertension never returns in spite of the fact that the patient may make a good recovery and go back to work. This is far from being evidence that the heart is the "cause" of the hypertension, but perhaps it breaks a vicious circle of increasing arteriolar spasm - increasing cardiac work - increasing arteriolar spasm.
Case of
Alexander Lawrence. Age 60. Retired Steelman.
3 Guthrie St.
Admitted to Ud. 26 9. 4. 40.
Discharged 3. 5. 40 to convalescent home.
Complaint: Breathlessness, Cough, and a
"Growing in the chest" of 8 weeks duration.

History: The first intimation the patient
had that there was anything wrong with
him was in 1914. He was at that
time in the Navy, and, before joining a
new ship, was up before a medical
board. They told him that he had
Valvular Disease of the Heart and he had to
leave the service. He insists that he had
been perfectly fit till then. Before he
actually was discharged he was
sent to a naval hospital where he
was kept for 14 weeks. He thinks that
while in hospital he had pains in
his chest. He was eventually
discharged, and since then has
worked from time to time in shipyards.
and at sea. Between then and now his memory is vague. However, he thinks that ever since 1914 he has suffered with freedom intervals of months, sometimes intermittently from GIDDINESS and CONSTRICTING PAINS in the chest. "For a long time" also he has had SLEEPLESSNESS caused by a gnawing feeling in his chest. He has never had palpitations, or, till recently, dyspnoea. The Giddiness tends to come on while he is active, for example climbing stairs, rising suddenly to his feet, tying his bootlaces etc. He feels that "if he stopped he would faint" but he never does faint, and the giddiness passes off. The Pain, (which seems to be of different varieties, as he has repeatedly described separate "gnawing" and "tight" chest pains) almost invariably comes in bed; though once or twice he has had pains related to severe exercise.

8 weeks before admission DYSPONEA appeared for the first time, quite suddenly. This affects him in five ways. First of all, it...
HISTORY

seems to have been entirely as a result of exercise. If he climbed stairs quickly or even walked quickly along the level, he has a sudden "tightening" in his chest and has to gasp for breath; breathing is difficult. There is no pain with these attacks. They never last more than a minute if he stops the exertion. Since these attacks started there has been a very distressing unproductive cough: he feels as if there was "string in his windpipe" but can never bring anything up. The cough comes in paroxysms and is worse after exertion.

Secondly there has developed an apparently distinct postural dyspnoea. Since over the cough and the exertion dyspnoea began, he has been aware of a discomfort in bed 4 or 5 nights before admission, he went to bed, and, no sooner had he lain down than there was an intense "thumping of his heart" (never felt before) and a
feeling of choking and breathlessness. He got out of bed and walked up and down the corridors, gasping and coughing and breathless, but without the choking sensation. At last, cautiously, he tried to lie down again, but the choking feeling threatened once more, and he spent a sleepless night sitting up in bed, leaning forward with his head on his knees. Every one of the subsequent three nights before he was admitted, he has been sleepless all night long, and unable to lie down. The cough which follows this paroxysmal postural dyspnea is paroxysmal too, but never productive. Certainly he has never brought up pink watery sputum.

He likes to wash with cold water, but these last days he has been unable to, as he finds that cold water produces the same choking feeling and cough. He has never been woken from sleep by these attacks; but then he doesn't believe he
has ever gone to sleep since the nocturnal attacks began. Pain has been absent.

Lately his **Memory** has been getting bad. He says his memory of the past in general is all right (personally I don't think it can be, for he is very vague about the details of his history); what has deteriorated is his memory for day to day events. He forgets what day it is; he forgets to collect his money at the labour exchange, which till this year he never forgot to do; he suddenly forgets the names of his friends; he forgets what he was going to say; and he takes these "stupid turns" with increasing frequency. This mental change is of recent origin, definitely within the last year.

Before now, at various times since his discharge from the naval hospital, he has thrice been a patient in the old Glasgow hospital. He is completely vague
about dates though he thinks the last

time was "just before Craighead Hospital

was given up." These hospital stays

have been on account of the old giddiness

and growing pain in the chest. The

giddiness and pain have sometimes been

entirely absent for months, and

sometimes have been present for months

at a time, the giddiness in several attacks,
in the day, the pain more or less continuously

but worse at night. In general however

these, the original symptoms, have shown

no progression in severity or frequency of

attacks since 1917.

Since this recent phase of his illness

he thinks he has passed less water

than usual.

He is always more or less constipated.

In the last 2 years he has been on the

Labour exchange.
Previous History:

He was in the Boer War and
the Great War, and, till his discharge
in 1919, never had an illness or suffered
any ill-effects.

Previous Illnesses. Measles is the
only childish ailment he remembers.
Apart from that, he was kicked by a horse
in the face while a child, and subsequently
the sight of his left eye became bad.
There is no suggestion of any Rheumatic
illness. Once (it may have been the
occasion for one of the Craigleith visits)
he had a severe bleeding from the
nose which was treated in hospital
for a day or two. He was told it
was due to "Blood Pressure." He thinks
that was in 1924.

Social History

He has lived in a lodging-house for
some years, since his wife died.

Tobacco: smokes 4-5 oz. a week.
SOCIAL HISTORY

Alcohol "Whenever he can get it" has been his motto since youth. If he can't afford to eat and drink, he doesn't eat. Whiskey he never touches; he has for years confined himself to Burgundy and Rum, equal parts, which he warmly recommends for its tonic effects.

Family: Mother died of old age. Father, previously a temperate man, whilst engaged on the construction of the Forth Bridge, fell into drinking habits and subsequently the Forth, and thus died in his prime. Patient is the only survivor of 8 brothers and 1 sister, though he was the third eldest. One died in infancy. One died of war wounds; one of a "child." The most recent death was of a brother who was told in Dundee that he had a very rare form of heart disease. The other 4 died of causes unknown to patient.

Patient is a lean, sallow old man, who, propped up in bed, breathes loudly, and with obvious effort. The neck veins are engorged and pulsating visibly. No obvious cyanosis. His head nods and trembles with every pulse-beat.

CARDO-VASCULAR. For symptoms, see history.

HEART:—

There is diffuse precordial and epigastric pulsation. The apex-beat is strong and 2" square, which the apex is the lower outer angle. The presystolic murmur is best heard just inwards from and above the apex. The first sound is loud, and associated with it is a soft blowing systolic murmur which is conducted out to the anterior axillary line. Aortic Area:— There is a rough systolic aortic murmur, sharply localised to the second right intercostal space just lateral to the sternum. It accompanies but does not replace the first aortic sound which is fairly loud. I could not hear it in the vessels of the neck. The second sound is of slightly reduced strength, and is accompanied by a soft blowing murmur which continues for a short way into diastole. This murmur is fairly evident in the aortic interspace but is best heard over the right half of the sternum at that level. It is transmitted for about 2½" down the sternum and in the direction of the apex.
HEART— Auscultatory Findings

In the Pulmonary area, the first sound is associated with a soft but distinct murmur. The second sound was unduplicated. The second sound, as well as being unduplicated, was abnormally loud.

No abnormality of the sounds in the Tricuspid area could be made out.


Optic Fundi: Owing to an opacity of the lens of the Right eye, only a red reflex can be obtained, nothing can be seen of the vessels. Left Fundus—no retinitis. There seems, to my rather unpractised eye, to be a thickening of the arterial walls, seen as a distinct double line in each side of the blood columns. No arterio-venous thickenings. The reflex of light from the arterioles is unusually bright. In short, there are signs of retinal arteriosclerosis as yet uncomplicated by changes in the veins, or Retina.

Jugulars: congested and pulsating. Ankle and Sacral Region show considerable pitting oedema.

RESPIRATORY System.

Symptoms—cough, dyspnoea (see History). Examination—The chest is narrow and the musculature poor, but it moves well on respiration, which is thoraco-abdominal in type, and laboured. The voice is not harsh.
Respiratory System

Palpation revealed no abnormality of the chest, and the vocal prominences was unimpaired. The percussion note was unimpaired. Auscultation showed the breath sounds to be normal in character in all areas; there were numerous fine inspirations at both bases.

No "tracheal tug" could be detected.

Alimentary System

Patient has always been constipated, and there has been no change in this lately. The appetite is good. For years he has suffered from intermittently descending piles, which have bled occasionally, never seriously; but have never been painful. They have not come down recently. There is some pain and "heaviness" in the upper right abdomen; this has been present for 2 or 3 weeks and has not been noticed before. No indigestion except when drinking heavily.

Tongue is slightly covered with a whitish fur. Teeth are not good, carious, but without obvious sepsis.

Abdomen: fairly well covered, but not in any way distended. No local swellings. There is diffuse epigastric pulsation, rather more obvious on the right side.

Palpation reveals marked deep tenderness in the liver area. The liver is enlarged two fingerbreadths below the rib margin at the 9th cartilage, and is felt to be pulsating. The enlargement is diffuse, and the liver smooth.

The spleen is not enlarged.

Otherwise, the abdomen reveals no abnormality.
ALIMENTARY SYSTEM

No abnormality can be made out on Rectal examination.

CENTRAL NERVOUS SYSTEM

MENTAL FUNCTION: the patient is co-operative and intelligent, and the history he gives of the course of his trouble, though vague in the remote details, is coherent. I have not observed any of the "stupid fits" or sudden attacks of what seems to be sensory aphasia, from which he mentioned and which have been referred to under the history. However, he is perfectly definite that there have occurred, and that, in addition to such momentary aberrations, his memory for recent events has deteriorated lately.

He has had headaches for the past 2 or 3 years. They are frequent, 2 or 3 a week perhaps, "grumbling" in character, usually frontal, and they "come on in a minute" and disappear as quickly. Often they follow his giddy fits; sometimes they precede them; sometimes they are quite unrelated. They occur mainly in the early part of the day, but no relation to alcoholic indulgence is noticed.

Apart from the loss of memory, the headaches and the long-standing poor vision in the right eye, no other mental or nervous symptoms have been noticed.
Central Nervous System

 Cranial nerves: no impairment of cranial nerve functions could be found on examination. Pupils are equal, and of normal size and reaction. The deep and superficial reflexes were all normal. Sensation is intact and unimpaired, as is motor power.

Urinary System: Recently the patient believes he has been passing less water than usual, and it has been of dark colour. He says he has sometimes a little difficulty in initiating urination. There was no tenderness in the renal angles. The kidneys were not palpable. The prostate felt on rectum was smooth, perhaps a little enlarged, but not more so than many asymptomatic old prostates.

Urine: (Morning Specimen 10.4.60)


Haemopoietic System

(9.4.60) W.B.C. - 9000 /c.mm.
- Haemoglobin (Sahli) 14%.

(11.4.60) Film showed no obvious abnormality. Cells well filled with haemoglobin; no poliloiocytes or anisocytosis.

Other Systems: Nothing abnormal demonstrable.

WASSERMANN REACTION NEGATIVE.
DIAGNOSIS:
Aortic Insufficiency and Mitral Stenosis and insufficiency
with Heart failure, predominantly left Ventricular

TREATMENT and Progress Notes.

The patient has been in bed, at complete rest, throughout his stay in hospital. He has been propped up in the position most comfortable, half inclined to his right side, and in a sitting position.

Diet was of the usual type; 1000 Calories, low fluid (about 1 pint daily) moderately restricted in salt, rich in vitamins and of small bulk. It has never satisfied the patient's hunger or thirst, and he complained bitterly that "milk sticks half-way down his throat"—this bears all the signs of a functional disability, because he has no trouble with solid food.

For the first few nights hypnotics were required to secure sleep. During the first night Morphine grm. hypodermically had to be given at 10.30 p.m. + 2.30 a.m. The next 2 nights it was required only once, at 4 p.m. each night. For a night or two after, a mixture of Ataral, Bromide and Tinct. Opis was given, and thereafter hypnotics of the milder sort were only occasionally required.

"Attention to the bowels" is represented on the chart by an initial dose of Licorice Powder, and Cascara subsequently daily. Tinct. Cardamom Co. has been given in 10 minc. doses after meals for the
relief of the patient's tendency to flatulence, and distension.

Digitalis has not been thought necessary, and the patient has made very good progress without it.

**PROGRESS NOTES.**

There is no point, in this case, of making any attempt at a day by day record of the course of improvement, for it was smoothly progressive (though naturally more dramatic in the first 10 days or so of hospitalization) and uninterrupted by any complication.

**SYMPTOMATIC** — The **DYSPEA**, which on admission was constant and distressing, even in the most comfortable position, rapidly improved, and within 3 or 4 days was "almost gone." The **PRÆCORDIAL DISCOMFORT** also diminished quickly and on discharge he had been free from any trace of it for at least a week. The **COUGH** and the "tightness in the chest" had disappeared after two weeks. **FAINTNESS** had never been complained of since he was admitted.

The **PAROXYSMAL POSTURAL DYSPEA** was the most obstinate of the symptoms. Even just before discharge, although he could lie down without an attack coming on, there was a feeling of respiratory oppression in the recumbent position. The last actual attacks occurred at the end of the first fortnight, when twice as he was falling off to sleep in the same
PROGRESS notes

night the old chesty feeling, with paroxysms of coughing and laboured breathing, occurred. That was on the 20th; on the 22nd there was slight return of oedema of ankles, and a few crepitations were heard at the bases of the lungs — the first for a week. The ELEFACTA Dysuria had disappeared.

But from then on, symptomatic improvement was quite unmistakable.

The Urinary Output, in the patient's first 3 days in hospital was low; then as the heart began to regain its strength, it rose from the original 15-20 oz figures, into the forties and fifties, in spite of the continued restriction of fluid intake; this excess of output over intake was maintained for about 3 weeks, while the patient's weight fell; then, as the weight became stationary, the urinary output declined again. Concentrating power had not at any time been impaired significantly; on the first morning after admission the morning specimen had a specific gravity of 1028. A day or two before his discharge however, I found that the morning specimen had a specific gravity of 1035, in spite of the fact that that day's output was high.9 than the first days, and that the protein intake was certainly not more; so perhaps, though the blood urea was never estimated, there may have at first been a certain degree of pre-renal oedema, unimportant and temporary. The Albuminuria did not seem to diminish, in spite of the clinical improvement.
Progress notes

Body weight provided perhaps the most obvious index of the efficiency of treatment; it declined at first rapidly, and later as the last of the oedema was evacuated, more slowly. The decline had ceased in the last week, except for the fractional decline which is to be expected as long as a calcinically inadequate diet is continued. Figures are worth recording.

9.4.40  Body weight 11st. 4 lbs. (Standard 11st. 6.5 lbs.)
12.  10  12½
14.  10  10½
16.  10  10
18.  10  2 2½
22.  10  1½
24.  10  1
26.  10  ½
3 5.45  9  12½

Thus in the first seven days, he lost 11 lbs; in the next eight he lost 6 lbs; and in the last eight he only lost 2½ lbs.

Heart, Pulse and Blood-pressure.

Electrocardiogram was taken on 18.4.40. 9 days after admission, and after symptomatic improvement was well established. The summary of the report was to the effect that "there is a regular sinus mechanism, with marked left axis deviation. The ST segment is elevated in the standard lead, and depressed in the apical lead, and T is inverted in the apical lead, which is suggestive of hypertension."
On 16.4.40 a chest X-RAY was taken. It showed “Gross cardiac enlargement; Left Ventricles, Right Ventricles and Right Auricles are all prominent.”

Apart from the improvement in urinary output, the first signs of the circulatory improvement were the return of the liver to a more normal size, and the disappearance of extrasystoles which had been so prominent on admission.

On the 14th - 5th day after admission the liver was no longer palpable, and percussion showed that the dullness was less than one fingerbreadth below the 9th costal cartilage. In another two days its size was normal and the epigastric pain and tenderness were quite gone.

About the same time, the extrasystoles, which for the first 2 or 3 days had been very frequent indeed, disappeared fairly rapidly, both from the radial pulse; and the heart on auscultation. On the 15th I noted only one extrasystole in 3 minutes.

Pulse rate. Pulse rate came down steadily. On admission it had been 116 / minute, and a steady fall in rate brought it down to 95 on 14.4.40 and 85 on 18.4.40. It has remained around 85 since.

Sp. Res. I could detect no change in the force of the apical beat. Its position however changed noticeably. On admission it was in the VI space 15 cm. from mid-clav. It remained throughout in the VI space; but it came in towards the mid-clav. On the 22nd it was in 1 cm. - 14 cm. from mid-clav. On the 26th it was 13.5 cm.
Progress Notes

The murmur, i.e. only ½ cm. outside the mediastinum, and there, until discharge it stayed, which probably meant that the downward, and a little part of the outward displacement represented hypertrophy and compensatory dilatation, and the rest of the outward displacement was caused by the dilatation of cardiac failure.

The auscultatory findings changed distinctly and rather puzzlingly as the heart recovered its functional capacity. After about a week, one began to notice, as would be expected, an increase in the loudness of the heart sounds. A few days later a change in the characters of the individual adventitious murmurs became obvious. On the 24th (after five weeks in hospital) I recorded in my notes: a) that the harsh presystolic sound in the mitral area was distinctly louder, both absolutely, and in relation to the stronger first sound. The systolic mitral murmur was less obvious, and was not transmitted quite so far into the axilla.

b) The pulmonary reduplication was no longer audible, and the pulmonary systolic murmur, previously distinct, was now doubtful. c) In the aortic area, the first sound was still accompanied by a rough murmur, but this was not so loud. The diastolic murmur was very much less distinct, not transmitted for more than half the distance down the sternum and towards the apex that it had been originally. The second sound, previously faint, was now more obvious.
Progress Notes

The **Blood Pressure** and the **Pulse Pressure** both fell steadily. Originally it had been 180/90 (on 9.4.40). On 12.4.40 it was 160/90; on 16.4.40 160/85; on 22.4.40 132/90; on 24.4.40 130/90. From then till discharge it stayed around the latter figures 140/45. In this connection, it may be mentioned that whereas at first one could count the patient's pulse from 10 yards away by watching the trembling of his head with every systole, after a week this trembling had quite disappeared.

On May 3rd he was **DISCHARGED** to the convalescent home, where his cardiac reserve will be gradually gauged, so that eventually he may be advised as to what sort of life and activity will be possible for him in the future.
DIAGNOSIS: The two-line diagnosis on p. 14 made after my first examination of the case, looks rather frayed at the edges now, in the light of the changes in the stigmata findings which have been recorded under "progress," but I leave it as originally made because it was more or less justified at the time. The mitral presystolic murmur was characteristic, and, I believe, pathognomonic of Mitral Stenosis; it is still there, louder than before, so that part of the diagnosis needs no defense. The mitral systolic murmur too, is present, though diminished and there is probably some degree of Mitral Insufficiency still. Lessened by the amount which the dilatation of the left ventricle and mitral ring had contributed to it.

AORTIC INCOMPETENCE, which I had placed first in importance in the original diagnosis, was then undoubtedly present; the characteristic murmur and the collapsing pulse were indubitable evidence. But on discharge, the patient's aortic diastolic murmur was greatly diminished, though still present, and the pulse pressure was a high normal, or at the most, only slightly above the limits of normality. I had regarded the aortic insufficiency as the primary cause of the cardiac failure; the cardiac failure, although generally distributed and obviously affecting the right side, seemed most severe on the left side, in view of the extreme dyspnea, the cardiac asthma, and the signs of dilatation of the left ventricle, and therefore it appeared to be the primary failure.
DISCUSSION

And when, in association with failure of the left ventricle there are obvious signs of mitral incompetence, one need usually look no farther for a cause. Therapeutic incompetence had produced the heart failure, I thought, and this incompetence since it was associated with mitral stenosis—a lesion of rheumatic origin nearly always was probably rheumatic too. The original infection the patient did not recall; but it must have been years ago if he had "V.D.L." in 1914. But, if it was an old infection and the damage to the valves had ceased to progress and had passed beyond hope of repair, what had precipitated the failure now, and why did the incompetence seem to have been improved by treatment?

It was not difficult to find plausible reasons for the onset of failure. The patient's age and mode of life (too much drink and too little nourishment) would have sufficed as explanation if necessary. But a far more likely precipitating cause might well have been the ESSENTIAL HYPERTENSION which had already been suggested (1) by the cerebral symptoms detailed in the history which sounded rather like mild hypertensive encephalopathy (sudden amnesias, sudden headaches), (2) by the electrocardiogram, and (3) — rather speciously — by the failure of the albuminuria to clear up, as the other signs of congestive failure cleared up. The fact that renal function was good is by no
DISCUSSION.

means exclusive of Essential Hypertension, especially of relatively early hypertension, as this probably is judging by the excellence of the response to rest. (4) Finally the history of Epistaxis due to "Blood Pressure" is suggestive.

The addition thus made to the original diagnosis makes the course of the improvement much more understandable. Suppose that the aortic value was damaged by an old rheumatic process, but was still competent if only just; if then a true hypertension of any origin appears, the raised diastolic intra-aortic pressure might render the valves incompetent by means of the additional strain on them, or, (much more probably) the sclerotic aorta which often goes with Essential Hypertension might dilate as a result of the pressure, with consequent dilatation of the aortic ring and incompetence of the value. If, then, as a result of treatment (rest) the blood pressure falls, and the aortic dilatation diminishes at all, a lessening of the clinical signs of aortic regurgitation would appear. [In the case of heart failure due to pure aortic incompetence, improvement would bring a rise in systolic and diastolic pressures, and in pulse pressure, and an increase perhaps in the diastolic murmurs - because since the valvular lesion has not improved, the higher cardiac output would produce greater regurgitation with exaggeration of the signs of regurgitation.]
DISCUSSION

To conclude on the evidence of a cleared diastolic murmur alone that the amount of regurgitation was less would be unsafe; but when in addition the pressures fall and the pulse pressure is halved, then I think that the assumption of some such mechanism as that described is unavoidable. Therefore the diagnosis, modified by events, is now

DIAGNOSIS

ESSENTIAL HYPERTENSION, arising on top of old-standing MITRAL STENOSIS, INCOMPETENCE, and a "potential" or actual AORTIC INCOMPETENCE, previously compensated; DECOMPENSATION having been effected by the hypertensin, and also the amount of increase in Aortic incompetenec which it caused.

ETIOLOGY: To attempt any discussion of the original causes of Rheumatic infection, a hypertensiun would be out of place. That both contributed to the heart failure in their separate ways seems certain. The association of cardiac valvular lesions with Rheumatic (rarely arteriosclerotic) origin with Essential Hypertension is quite common; and mitral stenosis at least is compatible with considerable true hypertensiun. It is possible for Aortic incompetence to be the result of degenerative changes, but rare; mitral disease is said by some to be occasionally thus caused, but this is very rare indeed, and even in the absence of a rheumatic history, a rheumatic origin for the valvular lesions is much more likely.

TREATMENT: The effect of simple rest, sleep, and dietary measures has been most dramatic. Although some authorities recommend digitalis for every case of congestive failure, its avoidance seems to have been fully justified.
in this case, so quickly did the failure improve without it.

It is difficult to evaluate the extent to which the bed rest, the securing of untroubled sleep by adequate hypnotics, and the depleting diet respectively were responsible for the result. But they are all complementary measures, and directed to a single end, the relief of the heart from more than the minimum necessary amount of work. The diet, being poor in fluids, promoted a daily reduction of oedema by the amount by which the urinary (and respiratory and sweat) outputs exceeded the intake. The low calorie content of the diet, and particularly its low protein content, reduced metabolism to the basal level.

The only drugs of any importance in the treatment were the hypnotics; of these morphine was essential in the early stages when distress was severe; later, the milder chloral, bromide, Tinct. opii mixture was adequate for securing sleep.

Future treatment: The immediate treatment in the convalescent home will no doubt be on the same lines as those mentioned in the discussion of the first case of this collection. A graduated increase in activity within the limits of the cardiac capacity, (which itself should gradually improve to some extent with increasing activity) will be the main form of treatment. Regular hours, plenty sleep, a nourishing
and especially a vitamin-rich diet (but not so nourishing as to make the patient put on more than a few pounds weight) — these are all cardinal points in convalescent treatment. If, by such a comfortable régime, the patient can be seduced from his present attachment to alcohol and tobacco at the expense of his diet, so much the better; but it is difficult to believe that such an estrangement can be more than temporary. In general it is obvious that if the improvement obtained is to be lasting, the patient must mend his ways considerably; not so much because of any intrinsically deleterious effect of either alcohol or tobacco on hypertension or heart disease, as because their indulgence leads to inadequate nutrition in this case. And, in addition, though there is no convincing proof of their bad effects, still there must be some substance in the widespread disapproval of their use in such cases. In addition, after convalescence, the patient will have to be advised of the necessity of regular hours, adequate sleep, suitable meals etc. Whether he adopts such a régime, which in any case will not be easy in a working-men's lodging house, will rest with himself. But it is unlikely that he will easily find anything to substitute for the loss of his banned pleasures.

**CLINICAL ASPECTS:** The most striking clinical feature in this case was the **CARDIAC ASTHMA**, otherwise called Paranymal Nocturnal Dyspnoea — here the best name would be Paranymal Postural Dyspnoea. The extreme dyspnoea, with wheeziness, cough, and
Discussion - Clinical Aspects

A little viscid sputum, resulted almost immediately when the patient assumed a posture approaching the horizontal, and nothing else but posture seemed to have anything to do with it. Numerous explanations of this phenomena are given; the most plausible seems to be the following. When the patient lies down, there is an immediate redistribution of blood to what are now the most dependent parts, notably the lungs. This is brought about by an increased return of blood to the right heart, which is still capable of responding by an increased output. The consequent increased inflow to the left heart whose failure is relatively more severe, cannot be properly dealt with by increased output, perhaps because of the production of an excessive dilatation of the left ventricle beyond the limits of efficiency. And therefore an excessive blood volume piles up in the pulmonary capillaries, stretching the alveolar walls; perhaps interfering with the Hering-Breuer reflex, and inducing abnormal breathing-paroxysmal dyspnea. The element of broncho-spasm and hypersecretion of mucus which seems to be present in such cases is probably reflex. Fortunately in this case the pressure in the pulmonary capillaries never became so severe as to cause the sudden massive transudation of plasma, and the small hemorrhages, which produce the clinical picture of Acute Pulmonary Edema.
As the heart began to recover, its least affected component recovered first - i.e. the right ventricle. This was shown by the rapid rise in urinary volume, the shrinkage of the congested liver, and the disappearance of oedema. The left heart, more seriously decompensated, recovered more slowly, but eventually the dyspnoea, and the postural dyspnoea paroxysms abated, which showed that at least the ventricle had acquired enough reserve to cope with postural adjustments of circulation.

It was interesting to note in the history that washing in cold water also brought on similar dyspnoea paroxysms - due doubtless to the resultant peripheral vasconstriction producing sudden insufficiency of the left ventricle. It is well known that the pressor response to cold stimuli is greater in Essential Hypertension than in normal subjects, which may have contributed to this mechanism.

The central episodes in the history were not unfortunately observed in the ward. But the descriptions given of the sudden headaches and sudden confusions is very suggestive of the central phenomena observed in hypotension; and due either to the ischaemia produced by cerebral angiospasms (themselves often the result of sudden blood pressure rises), or to the local congestion following the relaxation of such angiospasms, produced by the local accumulation of tissue metabolites.
Discussion, Prognosis.

The only other aspect of the clinical picture which was of more than routine interest was the way the extrasystoles disappeared with improvement in the degree of cardiac failure. Why heart failure should predispose to the occurrence of extrasystoles I do not know; perhaps an overstretched myocardium in a diluted failing heart becomes somehow more irritable — not that that is an explanation.

Prognosis: If one believes that the failure here was due to the effects of increasing hypertension on a damaged but unprogressive and compensated heart, the prognosis is rather more difficult to assess than had the failure been due to the vascular lesions alone. Essential hypertension is so variable in its course. But the good response to treatment inclines me to believe that it may be controllable for some time yet by a proper régime, of quiet living and all that that implies. If the patient can be got to adopt such a mode of life, then he may well be good
Prognosis.
for some years yet. But for an old and lonely man, whose interests and companions must be those of the public house and casual ward, and who has no shell within which to retire, proper adjustment of his life and habits must be very difficult, if indeed it is desirable at all. And the prognosis is to that extent impaired.

If, every time the hypertension reaches a certain degree, aortic incompetence is going to develop and precipitate cardiac failure which can be recovered from by bringing down the blood pressure, then to some extent, the renal lesion might almost be useful, as a kind of warning signal. Much of the prognosis depends on the speed with which the blood pressure rises again after being lowered by hospital treatment.
Case of 
Mr. Cullen. 
25, Middle Bank St. 

Complaint

"Swelling of the face, and blood in the urine." 6 weeks before admission patient had an attack of "flu," principally suffering from sore throat. He was in bed for over a week and then, apparently better, returned to school.

Another week passed, during which he felt quite well, and then one morning his mother told him that he was swollen around the eyes. At the same time he felt very tired, but didn't feel ill. There were no other symptoms. That morning he vomited once, without any preceding nausea, and brought up his breakfast. Later in the day, some time in the afternoon, passing water for the first time since the previous night, he noticed that the urine was red. It has remained so ever since.
The swelling of the face was present for about a week, more obvious in the mornings. After that week, to the best of his knowledge it went away and has not returned. His mother told him he was pale, and this has also continued.

The colour of the urine was the only urinary change he has noticed. He does not think he has been passing less urine. There has been no pain, no headache, drowsiness etc, and no vomiting after the first day.

The appetite, habitually good, has continued so. Since the illness began he has been distinctly constipated.

His doctor treated him with "a bottle of medicine" and what appears to have been a low-protein diet with milk, fish, eggs as the main constituents.

He was admitted to the R.I.E. on Dec. 16th. Since then, until this history was taken (9th January) the course of the illness has been devoid of subjective incident; the only symptom was and is
The unusual tiredness. His tonsils were removed on December 21st.

The only illnesses he remembers having are MEASLES, WHOOPING-COUGH, INFLUENZA. He had not previously ever had a sore throat.

He lives in a comfortable house and has a bedroom to himself. He gets on quite well at school. His description of his diet appears satisfactory.

His parents are alive and well. So is his only sister.

EXAMINATION

A pale languid boy of good physical development. Cheerful and co-operative.

URINARY System.

Complaints: as detailed in history. Polyuria, Haematuria, swelling of face, tachypnoea.

Examination: There is no obvious oedema. The kidneys are neither palpable nor is there any tenderness in the renal angles or along the course of the ureters.

The Blood Pressure is 125/75.

Results of Urinary examination etc. will be detailed under "Laboratory Findings". They were those typical of Acute Glomerular Nephritis.

CARDIOVASCULAR System.

Pulse: Rate 84. Regular in time and force and of good volume. Vessel wall not palpable.

Blood Pressure - 125/75.

Optic Fundus - no change in vessels.

Heart: Inspection reveals the apex beat in the 5th left space 4 cm. from midline. Mid-clavicular line is 4 1/2 cm. from midline.

This is confirmed by palpation. The beat is moderately forcible, no thrill. Percussion fails to reveal enlargement of the heart.
Examination

Heart. The heart sounds are pure and closed in all areas. There is no accentuation of pulmonary or aortic second sounds; the aortic is louder than the pulmonary.

Respiratory System.

Patient has a cold in his head, and is receiving 11-13 693 Gm. 1. 4 hourly. He is “stuffed up,” he says, but feels well.

Throat not inflamed.

Nose, inferior turbinates large, congested. Yellow mucous discharge.

Chest is well formed, skeletal and musculature. Movements are good and equal. Breathing Thoracico-Abdominal.

ALIMENTARY System - Abdomen.

Constipated. Receives laxative medicine, even so daily motion not always being secured.

Tongue moist, with a little whitish fur.

Teeth good. Breath without smell.

ABDOMEN Well covered. No rigidity or fullness. No tenderness or hyperaesthesia.

Liver dullness one finger above rib margin at 9th costal cartilage. No dullness in flanks.

CNS. No complaints referable to nervous system, except the fatigue.

Reflexes are all normal.

Opthalmoscopic Examination reveals normal fundus, retinal vessels.

LOCOMOTOR System.

General muscular development is good; and muscle-joint function is good.

HAEMOPOIETIC System. The skin - mucous surfaces are pale. Hb. 56%. Scler.

R. BC. 2.89 millions / c.m.

W.B.C. 10,800 / c.m.

Film showed normocytic normochromic red cells, with no obvious anisocytosis.
LABORATORY FINDINGS.

(Except for such investigations as throat swab cultures etc. which can suitably be mentioned incidentally under the Progress Notes, I think that the laboratory investigations, particularly the blood chemistry, Haemoglobin figures etc. are best listed together here, instead of punctuating the Progress notes chronologically)

BLOOD — Urea Nitrogen.

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<tr>
<td>14</td>
<td>39</td>
<td>19 mg %</td>
</tr>
<tr>
<td>18</td>
<td>39</td>
<td>29-3 mg %</td>
</tr>
<tr>
<td>15</td>
<td>40</td>
<td>23 mg %</td>
</tr>
<tr>
<td>19</td>
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<td>31 mg %</td>
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<td>31</td>
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<td>40</td>
<td>43 mg %</td>
</tr>
<tr>
<td>16</td>
<td>40</td>
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Plasma Protein.

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<td>3.0 mg</td>
<td>5.3 mg</td>
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### Laboratory Investigations

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<th>C.I.</th>
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<td></td>
<td></td>
<td>90%</td>
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<td>27/11/39</td>
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(The colour index is calculated with scale: 85% = 100%)

The definitely high colour index figures—even allowing for erratic technique—are sufficiently constant to allow us to say that the anaemia was **normochromic**.

Blood films done at different times by me confirmed the well-filled character of the red blood corpuscles.
LABORATORY INVESTIGATIONS e.t.

URINE.

**SPECIFIC GRAVITY:** (albuminuria has now risen above 6g/ml, so has not been corrected for)

The specific gravity of the morning urine has varied in these 3 months between a **MAXIMUM** of 1.019 and a **MINIMUM** of 1.008.

The average has been around 1.012.

Little trend has been noticed in one way or another throughout. If one were to express an opinion, there seems to have been the highest average S.G. in the first month, the lowest in the second, with a doubtful upward trend in the third, but with such a small overall variation as to make the significance of these trends very doubtful.

**DAILY OUTPUT OF URINE:**

[Since the edema disappeared in the first week, the patient has not been restricted in his fluid intakes]
LAbORATORY Investigations eti

DAILY URINE OUTPUT

In the first three weeks the weekly outputs were 210 g., 182 g., 206 g.
The next two weekly outputs were 288 g., 276 g.
A rising trend had thus appeared before the protein intake was increased from 40-60 grams daily, at the end of the 5th week.
The output for the next week was 311 g.
Thereafter the outputs remained at between 280 g. - 315 g. weekly till discharge.
In the first 3 weeks, day to day variations were sometimes considerable (from 20 ounces to 60 ounces was the biggest) but thereafter the daily variations were small, and the usual was about 40-45 g.

ALBUMEN, in Units

In the first 3 weeks, the albuminuria was considerable, and this was the usual indication on the charts. On admission, and after a week, and in the third week the Esbacher readings were 6, 6, - 4, (gms./l.)
Thereafter, over the next 2 weeks
LABORATORY INVESTIGATIONS

ALBUMEN
The albumen fell quickly and steadily to + on the charts, and on 17.1.40 the Eshad figure was 2 gm. 1 on the 25th it had fallen further to 2 gm 1/2.

Between now and 9.2.40, the Eshad figure was from 1/4 - 1 on different occasions. On 12.2.40 it was 1 1/2.

On 18.2.40, it was 5, and on 9.3.40 it was 5 again. From the 12th Feb. onwards it was always ++ on the charts.

"POS" cells have always been present, especially in the latter half of the patient's stay.

CASTS: In the first 3 weeks blood casts were numerous in the deposit; on Jan 11th Granular casts appeared, and from February on blood casts have been very few, and granular casts numerous.

Red BLOOD cells: Have diminished fairly steadily from a maximum in the
LAB. investigations

Blood & Urine

first 3 weeks until, latterly, they were only occasional in the deposit. But, even recently, a cold in the head with mild pyrexia has once or twice brought R.B.C.s back to the urine in their former profusion.

UREA RANGE, done on 15.1.43, confirmed the evidence of the blood urea nitrogen, and the specific gravity figures.

Highest Concentration - 1.7% Urea (s. g. 1013)
Lowest " - 0.6% Urea (s. g. 1008)

WASSERMANN - negative

SUMMARY

The laboratory findings come under 3 headings - 1) Changes indicating of Renal Functional Impairment, and the degree of Compensation, 2) Abnormal Urinary Constituents. 3) Miscellaneous

RENALE FUNCTION: has been impaired considerably throughout, without much obvious change in the degree of impairment. In general, slight but definite decompensation has been present,
Summary of Laboratory Findings.

Renal Function

with intermittent and apparently "accidental" returns to a compensated state. The fast Blood Urea Nitrogen was normal.

2) Abnormal Urinary Constituents: 
in respect of these the course of the disease may be divided into 3 stages. Firstly, from the 5th to about the 9th week from the onset — Haematuria, at first massive, then, declining a little, with numerous blood-casts and no granular ones. Albuminuria copious, also declining later, with very slight decrease in total proteins; but diminution in albumen and increase in globulin, the A/S ratio being about 0.75.

Secondly, a kind of transition period lasting about a month. Haematuria and Albuminuria both less than before. GRANULAR casts appearing. Further slow decline in Plasma Protein.

Thirdly, a stage of rapidly rising (though not yet really massive) albuminuria,
Summary of Laboratory Findings

with many granular casts and few R.B.C.s. Plasma proteins still falling; the albumen is very low; the globulin is no longer absolutely increased. Total proteins are just above Van Slyke's "critical level," of 5.2%, on last estimation.

3. Miscellaneous

The state of the blood can be judged by the haemoglobin, as the cells appear of normal size and normal haemoglobin concentration in stained films done at intervals. On admission there was no anaemia; in 4 weeks the Haemoglobin was only 54%. Subsequently this level has been maintained.

Diagnosis: Diffuse Glomerular Nephritis which is passing into a "Subacute" phase.
TREATMENT; PROGRESS NOTES.

On admission the patient was put to bed, with the usual precautions against chilling etc. **Diet** was of high fat - carbohydrate, low protein character - protein intake daily being 40 grams. **Phenobarbitone** was administered from time to time throughout the illness when sleeplessness became troublesome.

A **throat swab** was at once taken from the tonsils, which were red and a little inflamed. This revealed the presence of **haemolytic streptococci** (Lancefield A) and non-haemolytic streptococci. **Tonsillectomy** was decided on, and performed on 21. XII. 39. This was followed by a transient mild pyrexia, and a leucocytosis of 19,600 c./mm. together with an increase in haematuria lasting for a day or two. But these changes were transient, and the disease resumed its undramatic course.

About this time (20. XII. 39) owing to the marked constipation, 17.1st. Cascara. Co. 3 b.i.d. was begun, and continued, as it produced a satisfactory result.
TREATMENT and PROGRESS NOTES.

I should perhaps mention that no fluid restriction was practiced; since then was no oedema, and the renal concentrating power was low. But the patient never drank much.

BLOOD-PRESSURE throughout was around 120/80.

So the disease continued, with gradual diminution in haematuria and albuminuria, and no subjective symptoms except weakness.

By Jan. 18th, owing to the patient's weakness, to the anaemia which had developed, and to the fact that he had already for 5 weeks been receiving only 40 gm. Protein daily, the protein-intake was raised to 60 gm daily. At the same time

Ferrous Sulfate (Glauber's) qv vi. t.i.d. was begun and has been given ever since.

Throughout, colds in the head, and sore throats have been not infrequent. They have all been treated with 17.13 643 Gm. four hourly, and this has usually arrested them. The first of these occurred
TREATMENT - PROGRESS NOTES.

on 14. I. 40. A "cold in the head" with nasal catarrh, headache, and malaise developed. A pyrexia of 101° - 103° was reached the next five evenings. **N.B. 693** was given on the 3rd day in the about dosage and the cold immediately subsided, as far as symptoms were concerned. After 4 days, the drug was stopped, and, next day the cold was back, with undiminished vigour. **N.B. 693** was again given for about 5 days, and this time the cure was real. The rest of his colds, etc. followed the same kind of course, and these were about five such episodes. One or two of them were associated with slight increases in haematuria; but in the case of the first cold this was particularly marked; previously the urine had been "smokey" for 2 days after.

The urine had been "smokey" with the onset of the cold; however, it was RED again, and this did not disappear till 3 days after the cold had been cured. In view of
TREATMENT, PROGRESS

the intractability of the nephritis, it was thought that the repeated colds were associated with a septic focus in one or more of the air-sinuses, and that this was perpetuating the glomerular inflammation.

On 29.7.40 therefore the Cranial Sinuses were X-rayed; the report stated that the frontal sinuses were absent, but the other cranial sinuses "showed opacities."

Percutaneous Puncture was the first, but no pus could be found in either antrum.

The disease had now entered its second phase, the transition period being at an end. [It should perhaps be emphasised that when I speak of "phases", I am referring to the results of laboratory examination. The clinical condition of the patient hardly changed from one month to another. Perhaps one should apologize for what may seem an excessive and unclinical pre-occupation with the protean microscopic character of the urine,
TREATMENT, PROGRESS.

and the silent fluctuations in blood chemistry, to the exclusion of the clinical condition of the patient himself. But I think that, of all diseases, nephritis is perhaps the one which most repays an interest in the (for the moment) clinically unimportant changes in urine microscopic findings, and blood chemistry. The second phase, as I say, had begun. Granular casts appearing with increasing frequency in the urine registered the beginning of degenerative changes in the tubules; the continued diminution in haematuria showed that the glomerulitis was subsiding; and the rising albuminuria, together with the changes in plasma proteins gave a clear hint as to what the clinical picture might soon be.

At this time (the beginning of the third month in hospital) it was thought that the amount of pus cells in the urine was more than could
TREATMENT, Progress

be explained by the diagnosis of glomerulo-nephritis. A superadded pyogenic or tuberculous infection was suspected, and an intensive search for evidence was begun. On 19.2.40 and on 24.2.40, mid-stream specimens of urine were sent for bacteriological examination. Both were sterile in culture, films, and the second gave no result by guinea-pig inoculation. On 26.2.40, a chest X-ray gave no evidence of a possible primary focus of Tuberculosis. On 5.3.40 and 12.3.40, the bladder and right and left urinary tracts were examined urologically. The examination was completely negative. And so the idea of possible infection of the urinary tract was abandoned.

There was no further change in the patient's clinical condition; and so, on 20.3.40 he was allowed to go home; the instructions for his subsequent
TREATMENT, PROGRESS, DISCHARGE

care were that he should lead a very sheltered life, with a moderately restricted protein diet. Ferrous Sulphate and yeast were prescribed for the anaemia.

COMMENTARY

DIFFERENTIAL DIAGNOSIS: There is no possible doubt whatever as to the diagnosis. The differential diagnosis is therefore non-existent, and it is hardly necessary to go into the reasons for saying so. It was always difficult to be enthusiastic about the necessity for excluding tuberculosis; the heavy deposit of leucocytes (NOT true pus cells) which started that here is a recognized manifestation of acute nephritis, particularly towards the end of the acute stage, when the extracapillary
COMMENTARY

Proliferation of glomerular epithelium, and the invasion of the capsular space with leucocytes, chiefly occurs.

AETIOLOGY:

Glomerular nephritis is recognized to follow infections, particularly haemolytic streptococcal ones. It occurs in the healing stage, and particularly in patients who, by skin-tests or blood antibody titres, can be shown to have an unusually high development of immunity. When the streptococcal infection is cured once for all, the nephritis is unlikely to become chronic; but when, as often in tonsillitis, and sinus infections etc., the organisms and the bodily defence forces adopt a kind of armed symbiosis, with intermittent revivals of the struggle, then the kidneys are subjected to repeated injury; recovery is never allowed to take place, and the condition becomes chronic. This case shows that very well.
COMMENTARY

Time relations of the tonsillitis and the nephritis, and the reaction of the kidney disease following tonsillectomy. Furthermore, although recovery might have been possible following tonsillectomy, this was frustrated by the fact that a new focus then arose (or revealed itself) in the nose and paranasal sinuses, and the exacerbations of infection here produced the same changes in the urine as did the original tonsillitis.

TREATMENT:

Firstly, the attempt to remove the cause of the disease, the septic focus in the tonsils. Tonsillectomy, or any radical treatment of a septic focus, during the acute stage is a bit of a gamble. While it is obviously rational to remove a focus which seems likely to maintain the glomerular inflammation, there are considerable risks. Since exacerbations of the renal disease are common, and these may well precipitate a fatal decompensation of the
COMMENTARY

kidneys. In a case like this, even though the nephritis was severe, and there were reasons for believing that it was likely to become chronic unless the focus was removed, still, the patient was not in immediate danger. The most dangerous period in Acute Nephritis is the first week, when anuria is specially common. To do Nephrectomy is to invite a recrudescence of inflammation of glomeruli, with the risk of anuria developing. Probably exacerbations of some degree will always occur, but one further may consider, that one acute exacerbation, for all its risks, is preferable to the continued insidious assault on the kidneys which the septic focus may provide. In this case, neither was the exacerbation of the nephritis at all severe, nor, unfortunately, was the cause removed, for the new (and never really localized) septic areas which showed themselves in paranasal sinuses, and throat, were probably as active in maintaining the
COMMENTARY.

mephititis as the tonsils had been.

Treatment of this infection with 17.13 643, while reducing the constitutional symptoms, seems to have been quite valueless as far as eradication was aimed at.

SECONDLY, treatment was concerned in lessening the kidneys' task. At the stage at which the patient was admitted, there was not a dangerous degree of oliguria, and there was still some renal concentrating-power. So complete protein starvation was not necessary, and the 60gm. diet was allowed. The patient did not have any desire to drink more fluids than was good for him, i.e. more than the previous days urinary output. No oedema ever developed (though weighing which might have detected occult oedema, was not possible). When the protein intake was later increased to 60gm/daily, there was some anaemia, not marked, and the indications in
favour of a higher protein intake outweighed (as they very often should do) the blood chemistry. There was marked anaemia; marked hypoproteinaemia, both of which might well be benefited. [As important is the fact that a high protein diet both raises the urea clearance, and the renal blood-flow; but this didn't matter here as there was no evidence (hypertension) of severe impairment of renal blood-flow.]

REST IN BED, & WARMTH need no comment.

THIRDLY, SYMPTOMATIC TREATMENTS apart from the use of aperients for constipation, and occasionally Phenobarbitone as a hypnotic the principal symptomatic treatment was the administration of ferrous sulphate for the anaemia. It was not to be expected that an anaemia with a colour index never below 1, and with well filled cells in stained films, would benefit much, but no harm could be done. Probably an increase in dietary protein would be more b
COMMENTARY

The point: The anaemia did not in fact improve, in spite of these measures, but it got no worse.

Clinical Aspects: This has been a fairly orthodox case of a non-resolving glomerular nephritis. Why should some cases resolve, and others become chronic? Probably the initial severity of the acute stage has nothing, or little, to do with the tendency to perpetuation of renal damage. If the acute stage is severe, with oliguria or anuria, marked hypertension, and convulsions, the patient may die initially, but if he does not, he has as good a chance of complete recovery as the mildest case has. Probably the important factor is time; and any degree of renal inflammation is capable of complete resolution if resolution begins soon enough. But if it does not begin, the initial exudative-inflammatory phase (which was capable of resolution) sooner or later
Clinical Aspects

gives place to Degeneration and Fibrosis which, once started, are probably invariably progressive. How long the original stimulus, which caused the inflammatory pathology, must be maintained before producing hyalinization and fibrosis of glomeruli, is unknown and probably variable; but the idea that duration as opposed to severity of the stimulus is the cause of chronicity is now well established.

The continuation of the stimulus to renal inflammation was shown in this case by the smouldering infection of the upper respiratory tract. Nephritis is thought to be a manifestation of infection—Allergy; if this is so, this case illustrates well the fact that Allergy is not proportional to the degree of immunity, for though presumably strongly allergic, this patient's immunity did not enable him to master the respiratory infection.

And so, for longer than was safe, the signs of "acute nephritis"—haematuria, albumin...
Clinical Effects

irreversible renal function etc continued but only when these signs after almost 2 months began to clear up did it become possible to estimate the degree of permanent damage done. That this was considerable became obvious when, with the clearing of the haematuria, the renal function failed to improve. Impairment of renal function, even when severe, occurring in the acute stage, has relatively little influence on the late prognosis; but impaired renal function in the more chronic phase is an index of irreversible damage—glomerular fibrosis, tubular degeneration.

It was only in the last 2 or 3 weeks in hospital that the patient's urine became reasonably free of blood—no macroscopic haematuria, small amounts even microscopically. Perhaps therefore it is rather early to draw useful conclusions from the non-improvement in concentrating power, as revealed by the daily specific gravity of the morning specimen and the urinary outputs. Conceivably maximum resolution of the glomerular inflammatory exudate, both intra- and extra-capillary, may take some time, and, while there is glomerular ischaemia from this cause, tubular concentrating power cannot return. But, if an early improvement in concentration could not be anticipated, at least a larger urinary output might have been. The urinary output, however—around 40 ounces daily, though apparently fairly large, is not so, when
Clinical Notes

taken in conjunction with a concentration never more than two or three points above the isotonicity with the plasma. With a urine undergoing scarcely any physical concentration, a urinary output of only 60 g daily is scarcely enough for safety, and is a measure of the degree of glomerular blockage.

One tends to assume that, with a urine whose concentration is isotonic with that of the glomerular filtrate—1070—renal concentrating power has reached its best test; and that the elimination of a more dilute urine than this indicates no further impairment. But the function of the tubules which is the last to go (if indeed it ever is a lost) is not the power of physical concentration, it is the power of selective chemical concentration. A kidney may still be able to concentrate urea (as the blood range shows) long after it has lost the power of raising the osmotic pressure of the urine; and thus, when this patient for several days ceased to present a morning specimen of 1004, 1008 etc., I did not regard that as a comforting indication that at least he was still capable of active dilution of urine; but rather as a sign (taken in conjunction with the low urinary volume) that concentrating power was so severely impaired that not enough urea etc. could be held in
Clinical Defects

The urine failed to make up for the tubular reabsorption of the high-threshold constituents of the glomerular filtrate.

The plasma proteins, severely reduced by the albuminuria of the acute stage, badly had time to recover at all before the albuminuria began to increase again, as a herald of the "nephrotic stage." On discharge, they had fallen just to what Van Slyke gives as the critical level, below which nephrotic oedema may be expected.

Blood Pressure remained throughout at high normal levels, or only slightly raised. Normal blood pressure in spite of oliguria, nitrogen retention, and similar evidences is of impaired renal function (and, almost certainly, impaired renal blood-flow) is a well-known and inexplicable finding, none the less irritating for its frequency.
PROGNOSIS

Clinically, on discharge, the "nephrotic stage" was just beginning. Classically, this stage is marked by heavy albuminuria, lowered plasma proteins, and oedema, with little impairment of renal function. It is perhaps too early to say whether renal function will improve at all; but since during 3 weeks observation, after the practical cessation of haematuria, there was no improvement of urinary output or concentrating, I am inclined to be pessimistic about the chances of much improvement.

That being so, if the albuminuria becomes so massive as to produce much more hypoproteinaemia, with severe resultant oedema, PRE-RENAL FLUID DEVIATION may contribute to earlier development of azotaemia than would otherwise occur.

In any case, the prognosis is decidedly poor; whether death occurs from oedema or from intercurrent infection is immaterial, it is unlikely to be more than a year or two delayed. The association of hypoproteinaemia and renal insufficiency is a particularly awkward one, for the dietary indications conflict sharply. A low-protein diet will be necessary, for the renal insufficiency is a greater danger than the plasma protein deficiency.
Case IV

Case of
John Donaldson aged 43. Occupation - Coalman.
16 New Street, Coatbridge.
Admitted 6.3.40. Discharged 9.4.40. Recommended by Dr. Black.
To Ward 26.

Complaint: - "Vomiting blood, and stomach trouble."

History: - In the early hours of 3.3.40, the patient woke up with his usual heartburn, unusually severe. As usual he took a powder for it, and got some relief. In a few minutes however, without feeling sick, he suddenly vomited about a pint of dark fluid, with recognisable streaks of blood in it. He felt a little weak afterwards, but not seriously. The doctor was called, and gave patient a pill. He slept that day in bed.

Next morning, at 4 a.m., the patient once again woke up, with some heartburn, and again, without nausea, vomited. This time the vomitus was of about 2 pints volume, and was dark, as before, with some "sticky clots." Later he had vomited he fainted in his cloth's arms.

That morning he was sent in to the R.I.E.

Previous History: -

About 10 years ago he began to have "stomach trouble." This took the form of a "burning pain" in the epigastric area, more marked on the left side, together with a feeling of heartburn in the epigastric area, both coming on about 1½ hours after food. Frequent but not so constantly there was a burning pain beneath the sternum,
which had not the same regular time-relations as the epigastric pain. Flatulence is not a feature except immediately after taking powders.

The gastric symptoms have always been noticeably remittent; and long spells of freedom from symptoms, from a few weeks to a few months' duration, have been enjoyed. These remissions tend to occur particularly in fine weather and in summer. He has for years been taking (t.i.d.) "No. 1 or No. 2 Powders" adjusting the proportions of each to regulate the bowels. Apart from the bowels have always been regular. Once or twice every night, during spells of indigestion, he has to take the powders to relieve pain which wakes him up. No particular food has been noticed to bring on the pain; but for 3 some years he has been adopting a special diet, (later to be described) on medical advice.

Until about 3 years ago, he was a fisherman, and meals tended to be hurried, irregular and ill-cooked. 3 years ago, one night at sea, he felt weak and vomited over the side; the vomiting was followed by much weakness, and a black colour of the stools which lasted some time. He was treated in this ward—they told him that what he had vomited had been blood on the usual lines. He was told he had an ulcer on the stomach.

Most from these haematemeses, he has never suffered nausea or vomiting, and his appetite used always to be good. Except after the first haematemesis he has never noticed black stools—only has not been in the habit of making such observations.
Previous History

Such was the history till 2 months ago. But then a certain change appeared in the symptoms. The pain and distension became more severe and more frequent, lost their regular relation to the taking of food, and seemed if anything to come on much sooner after taking food than before. The pain was no longer relieved by the taking of food, and not so completely relieved by taking the powder. The appetite, previously good, became poor. At this time he gave up smoking, but this did not help. He thinks he has lately lost a little weight, but is not sure how much.

Other Illnesses

Remember no childish ailments.

Mastoiditis 15 years ago, operated on in R.I.E. by Dr. Gardner. No trouble since, and hearing good.

Oblique Inguinal Hernia — at the moment he is on Mr. Graham's waiting list for radical cure of this. It began 2 years ago, when lifting a sack of coals. There was a sudden tearing pain, with a sick feeling, and the hernia came down. He saw Mr. Graham who advised the use of a truss pending operation. Without the truss, he cannot get out of bed without the hernia coming down. It has always been easily reducible, and has caused no trouble ever.

Social History

Occupation: Till 3 years ago was a fisherman, which involved hard work, exposure, and irregular indigestible meals. Since his first haematemesis he has been a coal merchant.
Social History

His circumstances are quite comfortable and he has a good house.

Meals: for last 3 years he has adhered to the diet advised by his doctor. Breakfast at 9 - soft boiled egg, toast, tea. 11 a.m. - chicken sandwich. 3 p.m. Chicken Sandwiches. Dinner in the evening - fish, fish, rabbit etc. Before bed, glass of milk and toast. He has adhered to this type of diet strictly.

Alcohol - never touches it. Cigarettes 10-15/day till he gave up smoking 2 months ago.

Family: Married, one son, wife and son alive well. Father died at 45 of bladder trouble; mother at about same age as result of accident. 1 brother died of "bowel obstruction." 1 sister and 2 brothers are alive and well.
EXAMINATION. (4.3.40)

Patient is pale, but not extremely so, with a thin worried face. He is thin, but not emaciated.
Tall, narrow chested, 
A narrow costal angle. He is not in obvious distress, and has no breathlessness as he lies in bed. Skin is cold and damp.


ALIMENTARY System


Examination: Tongue covered by a thick whitish fur. Teeth - upper teeth quite satisfactory denture. Lower teeth - incisor all right, molars rather carious but no obvious sepsis. Tonsils, throat, healthy.

ABDOMEN: Moves fairly freely with respiration.

No hyperaesthesia. Marked deep tenderness in epigastrum especially just to the left of the mid-line. Marked guarding of upper rectus, especially left rectus. No palpable tumour.
Liver not palpable; percussion reveals its lower border just above xiphoid margin of 7th costal cartilage. Spleen not enlarged.
No evidence of wasting.

Left Inguinal Hernia - the hernia is not down as patient lies in bed; it comes down on straining and is easily reducible. The external inguinal ring admits the little finger easily; and the hernia can be demonstrated to descend through it.

ACIMUMARY SYSTEM.

HAEMOPOIETIC SYSTEM: Conjunctivae and lips are well coloured.
Patient is pale* and feels weak, but there is
no dyspnocia, palpitation or “sighing respiration.”

Blood Picture.

4.3.40. RBCs - 3850,000 Haemoglobin (Sahli) 80%. O.I. 1.15.

4.3.40. 12.a.m. WBCs. 13,000. Film cells well filled and normal.

CARDIOVASCULAR System.

Pulse rapid (106) but of good volume and
dwell sustained. Vessel well not palpable.

Blood Pressure. 118/96.

HEART. Apex beat neither visible nor palpable
Percussion reveals cardiac dullness extends 5/½ to
right of sternum in III space. 3¾½" to left of
midline in V space.

Auscultation - sounds rather diminished
in intensity (they got lower a little in a
few days.) Pure and closed in all
areas. No adventitious murmurs.

RESPIRATORY System.

Respiration Rate 20/minute. Chest narrow, poorly covered.
Chest moves well and equally, and the
respirations are of normal depth. V.F normal
Percussion reveals no impairment of note.

Auscultation reveals no abnormality.

Renal System:

Urinal. Trace of Albumin. Orange in colour. S. g. 1.024.

No albuminuria detectable; albuminuria was not seen again.

*. This seems to have been a “constitutional” pallor. It
never changed much.
Examination.

Other Systems — No abnormality detectable.

LABORATORY Investigations etc.

**BLOOD.**

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<th>Date</th>
<th>W.B.C.</th>
<th>Millions of R.B.C.s</th>
<th>Haemoglobin</th>
<th>Correct.</th>
<th>C. I.</th>
<th>Retic.</th>
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<td>13,000</td>
<td>3.85</td>
<td>80% S. Hb.</td>
<td>73</td>
<td>1.15</td>
<td>(correct)</td>
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<td>5-3-40</td>
<td>8,000</td>
<td>4.14</td>
<td>83% (Hct. 48)</td>
<td>0.98</td>
<td>&lt;1%</td>
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<td>6-3-40</td>
<td>3,99</td>
<td>4.99</td>
<td>78% S. Hb.</td>
<td>9.2</td>
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<td>72%</td>
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<td>82%</td>
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Films: On admission 4-3-40 I stained a film; and the appearances were in every way normal. Cells were well filled; and by no means obviously macrocytic, whatever the blood-counts might say.

On 10-3-40, the appearances were similar; the cells were apparently quite normochromic, but there seemed to be rather more anisocytosis though this was just an impression (I had left the first film). There were a few normoblasts, about 1 in 2 fields.

5-3-40. Urea Nitrogen in Blood. 57 mg. %

... Chlorides in Blood. 49.6 mg. %

Blood Pressure

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<td>120/94</td>
</tr>
<tr>
<td>13-3-40</td>
<td>105/70</td>
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**Urine Day. Volume**

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<tr>
<td>3rd</td>
<td>40 g.</td>
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<tr>
<td>4th</td>
<td>46 g.</td>
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**Stools**

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Stools showed obvious melaena through this period.

**Fractional Test Meal**

6.4.40.

Fasting Juice - 40 cc. contained Mucus + Tracey Blood.

A rather high acid curve, of normal configuration, was obtained. After 1 hour, no starch remained in the stomach. No mucus after fasting specimen. No bile.

**Barium Meal**

5.4.40.

"Oesophagus negative. Four ounces residue 12 hours after Barium meal. Pylorus wide and atonic due to scarring of Duodenum towards apex of Cardia. No obstruction to passage of fluid, but channel is narrow."

**Wasserman** (4.3.40) Negative
TREATMENT.

There was no marked degree of shock on admission; so, apart from keeping the patient quiet in bed with hot-water bottles, there was no special restorative treatment. There was little restlessness or anxiety, so morphine was not needed. Luminal was given that night to ensure sleep, and thereafter gr. iii three daily, as the patient was of a restless worrying temperament.

DIET. For the first 10 days, including the day of admission, a Stage I Ulcer Diet was given; consisting of Activated milk 3 V 3 hourly, with towards the end of this period milk puddings as well. On 14.3.40, a Stage II Diet was begun, with the gradual addition of eggs, minced meat, and chicken. Paraffin was given thrice daily throughout, and secured one or two satisfactory motions each day.

IRON. Iron and Ammonium Citrate gr. 5 t.i.d. was given for the anaemia from the third to the sixth day. It was then increased to gr. XXX t.i.d.

On 3.4.40, patient was allowed up for an hour or so in the evening, and thereafter he was allowed up for much of the day. He remained in the ward till 9.4.40, and was then sent home, to continue with the 2nd Stage Ulcer Diet, the iron, and the Luminal in the same dosage.
Progress.

Progress was uneventful and satisfactory throughout the month he was in Ward 26. The pain and heartburn had disappeared in two days from admission. The deep epigastric tenderness remained for a week or so, but the muscular guarding had only disappeared a day or two before discharge. The tarry stools continued for between 2-3 weeks; after 3 weeks the stools were bencidure negative.

The Pulse Rate was 120-100 for the first 3 days, then fell quickly to between 60 and 40, which after the first week or so, was its constant level.

The feeling of weakness, which had been the only symptom directly referable to the blood loss, disappeared in about a week.

After the first week, in short, the patient was symptom-free.

Diagnosis:

Haematemesis from a Chronic Duodenal Ulcer.

Discussion:

Differential Diagnosis: Differential diagnosis of haematemesis begins, traditionally, with an attempt to exclude Haemoptysis. Apart from tradition, there was little cause to think of Haemoptysis here; there had been no respiratory symptoms at all; there was a clear history suggestive of alimentary disease; and the patient was clear that the blood had been vomited, was dark, and mixed with food. Melena, although it may be the result of swallowed blood from a haemoptysis, was too massive and prolonged for that in this case.
Discussion - Differential Diagnosis

CAUSES OF HAEAMATEMESIS:

The absence of demonstrable splenic enlargement and change of the size of the liver, and the absence of any suggestion of raised portal pressure, made splenic anaemia or cirrhosis of the liver unlikely causes. No history of bleeding and nothing in the blood picture suggested thrombocytopenia. The obvious suggestion was Peptic Ulceration; and certain aspects of the history suggested the possibility of malignant degeneration of a gastric ulcer, or carcinoma arising apart from such ulceration.

Peptic Ulceration: Without the evidence of the Barium meal, one would be inclined to plump for a chronic gastric ulcer, from the history, and the physical examination. Time of onset of pain - 3 hours after meals, and lately less - is suggestive of gastric rather than duodenal ulceration. The fact of Haematemesis, which is much commoner in gastric ulcers, is further evidence. The site of maximal pain and tenderness, the guarding which was definitely more marked in the Left Rectus - these are all typical of gastric ulceration. The only thing about the history which tends the other way is the absence of vomiting. The test meal showed an acid curve unlike the "heath climbing curve" of Duodenal Ulcers. Unfortunately, the X-ray showed distinct evidence of ulcer-deformity of the Duodenum, and in view of this one can't argue.

There has been a Duodenal Ulcer; could there have been a gastric one too? Theoretically there is no reason why, in a person with a constitutional diathesis to hyperchlorhydria, attacks of gastritis should always cause ulceration in the
DISCUSSION - Diagnosis

same place. If a Duodenal Ulcer is present, and perhaps heals, there is no particular reason whether a new attack of gastritis should not cause Gastric Ulceration. Hurst says that negative X-ray findings exclude a chronic gastric ulcer; I have heard other opinions, but even if the former is correct, X-rays cannot exclude acute gastric ulceration. On the whole, I am inclined to think that the recent alteration in the character, regularity and time of onset of the pain may quite possibly have been due to a recurrence of the "gastritis", or "gastroduodenitis", which affected the stomach most, perhaps producing acute ulceration. It is possible, at any rate.

Possibility of malignancy: this was suggested by the recent sudden change in symptoms; particularly the change in the regularity of pain, the anorexia which developed, and the fact that the patient believes he lost quite a lot of weight recently. But the marked improvement on treatment; the absence of blood, and the absence of pus and organic acids from the test meal; the blood picture and the X-ray findings all combine to exclude the possibility.

In view of the severity of the Heartburn, especially recently, one had to remember the possibility of Peptic Ulcer of the Oesophagus, which is frequently productive of haematemesis. X-rays showed no evidence of this, but as with the infinitely commoner acute gastric ulcer, they couldn't exclude acute oesophageal ulcer.
Discussion - Etiology & Pathogenesis

In this case, neither could we establish the presence of a strong constitutional factor, nor was there anything in the history to suggest that the usually accepted proximate causes of peptic ulceration had been active recently. The patient was by no means of hypertonic type; if anything his physique suggested the hypertonic, with rather narrow costal angle, narrow chest, and only moderate muscular development. There was no obvious focal sepsis; in recent years he had been treating his stomach very kindly from a dietetic point of view, eating small meals at regular intervals, of bland foods. Certainly, until 2 months ago he had been smoking moderately, but except for that, the usual causes of gastritis do not seem to have been operative, and the continuance (or renewal) of the ulceration, if his history was honest, must have been in the class of acts of God. In view of the good response of symptoms to dietary treatment however, one is inclined to think that he may have been slack in the observance of his régime than he admits. At the time when the ulceration presumably started, of course, his meals were irregular and unsuitable, and he smoked more, so the original onset is quite understandable.

Clinical Aspects.

As far as the ulcer symptoms were concerned, treatment proved immediately and characteristically effective; and the pain, heartburn and loss of appetite vanished, so much so that after 2 or 3 days the patient was complaining of the severity of the dietary restrictions
Discussion - Clinical Aspects

The behaviour of the blood picture after acute haemorrhage is always interesting. Not for about 24 hours at least is the blood loss made good by absorption of tissue fluids into the bloodstream, so that in the first 24 hours, the sooner after the haemorrhage is the blood count done, the less abnormal will be the findings. In this case, there had elapsed 36 hours since the first haemorrhage and less than 12 hours since the second when the count was done, so the findings were not very abnormal, and the film was quite normal. There was the characteristic mild leucocytosis, which always follows haemorrhage in the first 24 hours.

Next day the leucocytosis had declined; and the haemoglobin had also fallen sharply, since it was only 83% Haldane - (93% actual) against 86% (Sahli) - (93% actual) on the previous day.

The next day the haemoglobin rose, inexplicably, the count had been done by a different person, and the apparent rapidity of recovery must be ascribed to synchrony of the counter, I suspect.

From then on the haemoglobin behaviour was erratic, 5 days after admission they reached their lowest level - 69% Sahli, and thereafter put on 16% in 15 days steady progression - a rate of about 1% a day which is a good average recovery rate. At the figure then reached the haemoglobin became steady - (83% Sahli) and the increase stopped.

With the blood loss never having been very severe, it was not to be expected that marked hypochromia would appear in the film, and in fact a film made on the day when the haemoglobin
DISCUSSION.

was lowest, the film showed no obvious deficiency in the filling of the cells, but there were normoblasts present now, to provide evidence of rapid blood regeneration. The Reticuloocytes rose, reaching 3.7% by the 5th day, which was about the expected time of maximal response, and about the expected peak, considering the anaemia had never been severe.

The continuation of the decline in cells and haemoglobin for 5 days after admission is inconsistent with the idea of a simple acute haemorrhage; depletion of the blood after such a haemorrhage should have been complete in 25 hours; so presumably there was a continuation of occult bleeding for a few days. The melena was considerable, and "fresh" in appearance throughout the first week, which squares with this idea.

The extraordinary character of the colour index can only be explained by technical errors; the possibility of a "shift to the left" in erythropoiesis with the production of a macrocytic blood picture, is remote after all but the most severe haemorrhages. And in fact the stained film showed no apparent macrocytosis.

The observed blood-pressure readings which have remained very constant, show that not even on admission was there any noticeable degree of shock. No doubt the blood loss was compensated for by reconstitution in the skin etc., which produced the cold damp pale skin of the first 24 hours.
DISCUSSION

The hyperchloaemia and azotaemia the day after the haemorrhage may be of pre-renal causation. The urinary output on the first day was not recorded, but does not seem to have been excessively small, according to the patient. The changes in blood chemistry may have been due to failure of renal circulation due to the blood losses, or it may have been due to the tissue fluids mobilised into the bloodstream after the haemorrhage, having a higher chloride - N.P.N. content than the blood.

TREATMENT. Nothing in the treatment requires particular comment. TRANSFUSION was looked like being required, but the usual preparations were made in case another haemorrhage should occur, and necessitate it. The small doses of iron employed in the first few days were no doubt prompted by fear of causing undue gastric irritation before the gastric condition had settled down under treatment.

Prognosis: the prognosis for recovery from the haemorrhage was good from the start, and anxiety was never felt.

The prognosis for the underlying peptic ulceration is not so good if it be true that the patient had been strictly observing the appropriate régime, without benefit. But in view of the excellent response to hospital treatment, I think there must be room for improvement in the patient's habits, and if this is so the prognosis is reasonably good. But if intractable dyspepsia continued in spite of a strictly observed régime, operation will have to be considered reluctantly, for "failure to respond to medical treatment" is not a promising indication, by itself, for surgical treatment.
Case of
Andrew Pearson  act. 29. Railwayman

Admitted to Ward 26  5.2.0 G.  Recommended - 17.0.0.
Discharged.  19.2.0 to Convalescent Home.

Complaint - "Pain in side for the last 5 days which is worse on deep breathing." Breathlessness.

Examined 6.2.0.

HISTORY From about the 26th January, 10 days before admission, patient had "a cold in the head" which soon involved the chest too. At first the symptoms were not severe, but in a day or two a feeling of great "seediness" developed together with aching in all the muscles of his body. He felt he should be in bed, but stuck to his work for a day or two longer, though the work was exposed and the weather bitter. But after 5 days, when he got home one evening he noticed that the general myalgic had disappeared "leaving behind" a severe burning pain in the lower part of the left thorax. This was made worse by deep breathing, and aggravated by coughing, which brought to his attention the fact that he had now acquired a short "catering cough from the chest." With this cough a scantly tenacious sputum was brought up; its appearance was not remarked. The patient thought the chest pain was the result of a strain, for he had been doing very heavy lifting that day. He felt feverish that evening.

All that night the pain and cough prevented sleep; in the morning he felt "very bad" and a little breathless, so sent
for his doctor. (2. 2. 40)

He says the doctor "didn't do anything," but as he appears to be of a queasy and critical habit, this statement is possibly not accurate.

The next 6 days, the illness was "no better and no worse" though his appetite quite disappeared, breathing was considerable by day and night, and the listlessness was perhaps a little worse. The cough bitterly became a little loose. "No sleep" was obtained at nights because of pain.

On Feb 5. he was admitted to Ward 26 and last night he got his first good sleep (aided by Atwood Brains - Nefenthe mixture)

There was never any rigor.

Previous Illnesses.

"Children's diseases" - no details remembered.

Diphtheria.

Septic Hand - 6 years ago - treated for 6 months in SOPD. No residual incapacity.

Social History


Family History

Father died "over 60" of "heart failure" following "Bronchitis."

Mother, brothers and sisters all alive and well.

No TB in family.
EXAMINATION: 6-2-40.

Patient is a well-built young man, sallow but with a marked malar flush; the face and body are clam with sweat. He talks without much effort and moves about easily in bed. The tongue is coated with a thick white film; there is no herpes on the lip. Respiration are laboured and rather rapid and shallow; the pulse nasi are not working.

P. 120 yesterday
P. 98 today.

T. 101.6°
T. 96.5°

R. 36
R. 28

Urine (morning specimen) Spec. Grav. 1028, Orange, Acid, Chlorides-trace.

RESPIRATORY SYSTEM.

Symptoms: Pain in left chest, worse a deep inspiration, but constantly there. Breathlessness, not severe. Feeling of "wheezing" in the lower part of the left chest. Cough, at first dry, now looser; sputum usually yellow occasionally streaked with pink.

Signs: Inspection reveals a well-developed chest. There is no bulging of intercostal spaces, or obvious bulging of the chest in any area. Movements greatly impaired at the left base, and only very slight movement detectable on palpation. The movement of the rest of the chest is good. No friction pectori.

Vocal fremitus absent at left base, unusually marked on the right; no difference in fremitus between the right and left sides. The vocal resonance is affected similarly.

Percussion: On the left side the note is stony dull over the base, and up to the 6th rib in the scapular line. It is impaired for an inch or
RESPIRATORY SYSTEM - Signs

so above the 6th rib, and the apices give a normal percussion note.

On the right side the note is unimpaired in all areas: No Gruela's Triangle of dullness.

Auscultation. At the left base, the air entry was weak, and the breath sounds very faint, and apparently medium-pitched bronchial in character, with prolonged expiration. The character of the breath sounds were best heard just above the stony dull area: there they were clearly medium-pitched bronchial with prolonged expiration. A few medium crepitations were heard. Over a circular area about 4" in diameter below the inferior angle of the scapula, a coarse friction rub was heard, unchangeable by coughing.

The breath sounds were normal at both apices, there were no adventitious sounds.

At the right base, breath sounds were of a harsh vesicular character; no adventitious sounds.

Sputum. The sputum may contained a little tenacious yellowish sputum.

Sputum 5.2.40 - contained Pneumococci and Streptococcus Haemolyticus.

X-Ray of Chest 5.4.40. showed "the appearance of pleural effusion on the left side. No cardiac displacement was present."

Parecentesis was performed on 5.2.40. A needle inserted in the VI space in the Posterior Axillary Line tailed a thin yellowish seropurulent fluid. Enough for bacteriological examination was withdrawn. The bacteriological report said that the fluid contained "numerous lymphocytes and polymorphs. No organisms in films or culture."
EXAMINATION

CARDIOVASCULAR SYSTEM: No referable symptoms
Pulse: Rate 98, regular in time and force, volume good, vessel well palpable.
Blood: Pressure 124/65.
Heart: Apex beat soft, in 1/3 space, 4" from midline. [Mid-clavicular line 4½" from midline]
Percussion reveals the Rt. Border of Cardiac dullness 1/4" outside the Rt. border of sternum. - no displacement. Heart sounds pure and closed, and of normal intensity in all areas.

ALIMENTARY System
Patient is habitually constipated and has to take laxatives. Bowels moved only about 3 times in the ten days of illness before admission. First bowel movement in hospital occurred 4 days after admission. Appetite has been very poor in the last week; usually it is excellent. No other symptoms.

Tongue: dry, white, furred. Breath: not unpleasant.
Teeth: All Rt. lower molars & premolars have been extracted. Remaining teeth are all carious, no obvious sepsis.
Throat: normal in appearance.

HAEMOPOIEIC SYSTEM:

HAEMOPOIEIC System: 6.2.40. W.B.C. 14,000. Hb. 65%.
8.2.40. W.B.C. 11,000. R.B.C. 4,210,000. Hb. 90%.
12.2.40. W.B.C. 8,520. R.B.C. 4,800,000. Hb. 85%.
EXAMINATION.

OTHER SYSTEMS. No abnormality discovered.

DIAGNOSIS: Resolving Pneumonia (Lobar) with Empyema.

TREATMENT, Progress Notes

On admission, patient was put to bed, beside an open window with the usual measures to keep him from chilling. Diet was of the type usually given in febrile conditions, namely, fluid, low calorie, high carbohydrate. Fruit drinks with glucose added formed the major part of it. Fluids were encouraged. After the pyrexia settled in a few days, additions were made - custard, fish, eggs etc. to bring up the protein and caloric value. Hypnosis: on the first night when the pain and restlessness were preventing sleep, a mixture containing Chloral gr. XV
Pot. Bromide gr. XV and Nepthe gr. XV was given at midnight and produced a deep and restful sleep. Next morning, patient's condition was better. Pulse temperature and respiration rate were all better. Temperature at 96.2° was subnormal. The pulse and respiration rates had not yet reached normality, but, at 98 and 28 respectively, they showed clear improvement. The patient was bated in sweat, but said he felt much better; though the pain in his chest was no less he was "not so seedy," and he was very grateful for his sleep. That evening, the temperature rose to 99.2°; the pulse and respiration rates remained about the same.
TREATMENT, Progress Notes

6.2.40. The same day, the second in hospital, Codliver Oil and malt, and vegetable laxatives were begun, and their administration was continued throughout.

7.2.40. On the 3rd Day, the subjective improvement continued. The pain and sweating were less, there was no breathlessness now, and the sputum was loose, more plentiful, with a reddish tinge, and was being brought up in greater quantity. Temperature at 97.2° was now normal, and remained so from now on. Pulse had fallen to 90, Respiration to 20. Physical signs as before.

8.2.40. On the 4th day, after the presence of pus in the pleural sac had been established, 11.13.4 5 gm. 1/2 hourly, during the day, was begun, and continued for 4 more days. There were no toxic symptoms.

Improvement continued steadily. On the 5th day, the bowels moved for the first time, and the specific gravity of the morning specimen of urine, at 10.23, showed that the patient had more fluid to spare. Chlorides were now present in normal concentration in the urine. Pulse 84, Resp. 22. The pain had now gone, and pleural friction was no longer audible. No change as yet in the level of the dullness. Above the effusion, the breath sounds were still medium pitched bronchial; and there were now numerous coarse ronchiations.

9.2.40. From now on, pulse temperature and respiration were all normal; and the patient always "felt fine" on inquiry. Today, the 6th day in hospital, the upper limit of bronchial dullness was 1" lower, and the breath sounds were better heard. Voice conduction over the dull area was better.
TREATMENT, Progress Notes.

12. 2. 40.
On the 8th day, the stony dullness had disappeared; there was only a slight impairment of percussion note at the left base, and the breath sounds were now louder than on the right, harsh vesicular, with increased voice conduction was increased at the left base.
The friction rub had never reappeared after its first disappearance.

X-ray showed "complete resolution of the pleuropulmonary inflammation."

13. 2. 40.
But till the 11th day, the 15th, there were still occasional moist medium rales at the rt. base. On the 15th, curiously enough, a few scattered sonorous rhonchi appeared in both lungs; the first time that any abnormality had been noticed on the right side. But these were not present thereafter.
Expectoration of brownish thick sputum continued for a few days more; will its cessation, the signs of respiratory infection had all cleared up.

19. 2. 40.
On 19. 2. 40, the patient was discharged to the convalescent home; where the usual building-up regime will be employed; and breathing exercises prescribed to promote full expansion of the lungs and prevent fibrosis.
COMMENTARY

DIAGNOSIS

It may be as well, first of all, to remark that I am not very sure where the boundary-line between "Pleurisy with Effusion" and "Empyema" should be drawn. But I have chosen to regard Empyema as any condition in which there is purulent fluid in the pleural cavity. Even that is not altogether satisfactory, because the differentiation between pus and an inflammatory exudate is difficult to make. I suppose pus is an inflammatory exudate in which cells are so numerous as to render the fluid turbid. If so, it was pus which we found in this case, in the pleural sac, and therefore it was an Empyema, though a mild one.

The symptoms and history were those of disease of the respiratory tract, which appeared to have begun, as the patient himself stated, from the patient's story, with the symptoms characteristic of the INFLUENZA which was present at that time, and had been characterised by general muscle pains, upper respiratory tract involvement and severe malaise. The patient had had such symptoms for about 5 days; at a time when severe weather, and exhausting out-door work were no doubt lowering his resistance still further. Then, he developed the chest pain, of a cutting, burning character, with dyspnoea, and harsh cough, and feverishness — which was worse that night, he felt, than it had been during the "flu."

With such a history and the symptoms present on admission, it was reasonable to
**DIAGNOSIS.**

Suffice that he probably had Lobar Pneumonia, though it might have been a "Primary" Pleurisy, or an Influenzal Bronchopneumonia with pleural involvement.

Examination revealed unmistakable signs of left sided pleural effusion. The right chest was uninvolved, which excluded Influenzal Bronchopneumonia. The stony dullness, weak breath sounds, impaired voice conduction at the left base, were all clearly indicative of pleural effusion; and the character of the breath sounds, medium pitched bronchial, with medium crepitations showed that there was consolidation of the left lower lobe. The pleural friction confirmed the pleural involvement.

It is unlikely that mere collapse of the lung secondary to pleural effusion would have produced the auscultatory findings noted here; besides, now that I think of it, if there had been any collapse of the upper zone of the lung, there could have been no pleural friction; and since it was in the upper zone that the auscultatory findings were most characteristic, that disposes of that.

The diagnosis therefore was one of Emphysema, consequent upon a Lobar Pneumonia.

**Etiology and Pathogenesis.**

The causative organism, the Pneumococcus, was found in the sputum, along with haemolytic streptococci. There appears to have been a well-defined set of predisposing factors in this case; the exposed, fatiguing work, the bitter weather, and perhaps an associated or preceding influenzal infection which was epidemic at the time, and...
Commentary - Etiology and Pathogenesis

Took a form which produced great lowering of vitality without any well-marked respiratory symptoms. The pneumonia was at least 5 days old on admission, but the pleural effusion was well-developed then too, so if it was an empyema it was not characterized as "meta-pneumonic type;" but in fact meta-pneumonic empyema is probably merely late-diagnosed empyema.

The development of empyema is probably not an index of unusual virulence of the organism, or severely lowered resistance in the patient, in this case at least. For the organism cannot have been especially virulent if the empyema fluid was sterile; and the leukocytic response and the fact that the patient never seemed to be critically ill are evidence that the "soil" was not a particularly suitable one. Probably therefore the empyema was more of an unlucky accident than a sign of severity of the pneumonia.

Clinical Features.

On what, from the history, seems to have been the 4th day - the traditional day for the crisis to occur - the crisis actually came. Preceded by deep sleep, and accompanied by profuse perspiration, the temperature fell to a subnormal level, and the cough loosened, and the general condition improved. And the signs in the chest that morning were those of resolution. Unfortunately, I didn't see the patient on the preceding day so I don't know when the resolution had begun. The urine of that morning showed only an opalescence in the test for chlorides; next morning there was a
heavy precipitate, which tends to confirm that the crisis occurred in the course of that night, the first in hospital.

The leucocytes of 19,000 immediately after the crisis is rather less than one expects in a "stenic" case of pneumonia, no doubt there had been a considerable fall in the white count already. Two days later it was 12,000 only; and at the end of the week it was normal.

The blood pressure was always satisfactory, and the systolic pressure was always safely above the pulse rate—though I wonder if it would have been much above if it had been taken on admission when the pulse rate (at 124) was rather high for comfort.

The Empyema, or Pleural effusion, or what you will, later began to clear—4 days after the pulmonary consolidation began to resolve. What was surprising was the speed with which the effusion was absorbed, once absorption started. 48 hours after the stony dullness had first been noticed to have fallen, 1" the stony dullness had disappeared altogether, and movement of that side of the chest had quite largely returned. I think that the apparent rate of absorption represented by this was perhaps exaggerated in the following way: when first seen, the lung was presumably firm and consolidated, less easily collapsed by the pressure of a pleural effusion; on the 10th when the level of stony dullness began to sink, there must have been considerable resolution in the lung, which may have allowed a certain amount of
Discussion, Clinical Aspects

collapse, with consequent increase in the potential space at the base, to which the effusion could gravitate, so producing a dramatic fall in the arterial level of the signs of effusion.

TREATMENT: Perhaps the only credit due to medical treatment in securing recovery here falls to the account of the hypnotics given on the first night. Naturally the other measures played their part, but here it was the securing of sleep which most obviously helped in the favourable outcome. It may have been co-incidental; but the patient himself felt that it was the sleep which made him feel so much better on the second day. When the temperature remained down, and the general condition was obviously improving, there was no immediate indication for pleural aspiration or drainage; and these conservative tactics were justified by the spontaneous absorption of the pleural fluid. The administration of 17.13.693 was begun on the 8th; cleaning up of the pleural condition began on the 10th; but I imagine it was a case of post, not propter hor.

Treatment in the convalescent home will be directed to restoring the patient's general health, and resistance, with rest, and a graduated return to activity, nourishing diet rich in "protective foods", Tonics, and as much open air as the weather will permit. Secondly, measures to prevent residual pulmonary injury - such as fibrosis, pleural adhesions etc. - will be instituted, mainly in the form of breathing exercises.
Prognosis is of course excellent; the risk of fibrosis of the lungs after lobes pneumonia is almost negligible, particularly when convalescence is not hurried. The only thing to trouble the prognosis really is the increased liability to other attacks of Pneumonia which the disease itself produces. I don't suppose it involves a serious insurance risk.