In this connection may be cited those animal experiments with anti-platelet sera and capillary poisons which form the double nature of the pathological factor. Injection of both substances is necessary before a red purpura is produced; thus, injection of platelet alone or hemorrhages in produced by injection of the appropriate serum (prepared on platelet basis) and an anti-red cell serum.
Case of Bantu's Disease treated by Splenectomy.

Splenectomy.
Postscript:

On preparing the paper, I have heard that as the early stage of the work, the change is confined to the first 4/3 the small part of the right side of the formula. Experimental work in connexion, showing foreign particles introduced into the Chloe ring, has resulted in confirming the theory. In a paper, the product of research done in the Ohio high school is something, and till its publication, the matter must be kept and printed.
In Bursaphis tophi the tubercle or crust is
larger and the surface more fleshy.
REPORTS OF SURGICAL CASES:

(1) Purpura Haemorrhagica.
(2) Banti's Disease.
(3) Acholuric Jaundice.
   Appendix to cases I-III.
(4) Tuberculous Arthritis of Knee.
(5) Head Injury, with Basal Fracture and Concussion.

Ian G.W. Hill,
15, Spottiswoode Street
EDINBURGH.

June 6th, 1928.
CASE HISTORY:

Name: Madge Buchan.
Age: 14 years.
Address: Edgfield Road, Loanhead.
Occupation: Schoolgirl.
Recommended by: Dr. Hamilton, Loanhead.
Admitted: 15.11.28, (transferred from ward 34).

Complaint: Excessive bleeding on slight injury. Severe loss of blood at first menstrual period.

CASE No 1. MADGE BUCHAN, aet 14 years.

Splenectomy for Purpura Haemorrhagica.

All her life the patient has been troubled with a tendency to bleed. This has always been followed by prolonged haemorrhage, as, for example, the extraction of a tooth. At the time of such operations the blood loss was severe. An operation was performed at the R.H.S.C., and the resulting haemorrhage was so severe that a blood transfusion was necessary. The child's mother acting as donor. Eclampsia has been frequent, and likewise bleeding from the gums. On occasion there has been haematometra. She suffers from extensive bruising on slight injury, and frequently has crops of red spots under the skin.

She was treated in ward 30 in 1920, the diagnosis then being Purpura Sigmoid. Treatment was with grains of calcium salts, horse serum, etc.

All her symptoms had been improving recently, until her first menstrual period began on 7th January, 1923. Prolonged vaginal bleeding set in, which continued till the 29th January she was admitted to Ward 34, where the uterus was packed. This arrested the haemorrhage, but investigation of the case showed that the uterine state of affairs was liable to recur with each period. So surgical advice was sought. She was transferred to Ward 30 on the 10th of February.

Previous Illnesses: Tendency to bleed as noted. Scarlet fever.

Family History: Father and mother alive and well. Two brothers and one sister alive and well. No similar condition in the family.

State on Examination: The patient is a well-developed girl of fourteen, but in fairer health. She is bright and intelligent, and feels perfectly fit. Previous to her admission to ward 14 she was able to get up and attend a concert held in the hospital. There is no jaundice, no evidence of jaundice, but there is definite pallor of the mucous membranes, indicating anaemia.

Examination of Special Systems:
CASE HISTORY:

Name: Madge Buchan.
Age: 14 years.
Address: Edgefield Road, Loanhead.
Occupation: Schoolgirl.
Reconcend by: Dr. Hamilton, Loanhead.
Admitted: 16, II. 28 (transferred from ward 34.)

Complaint: Excessive bleeding on slight injury.
Severe loss of blood at first menstrual period.

History: All her life the patient has been troubled with a tendency to bleed. Any slight injury has always been followed by prolonged haemorrhage, as for example the extraction of a tooth. At the age of seven she had an abscess, which the doctor knowing of this tendency to bleed refused to incise; the operation was performed at the R.H.S.C., and the resulting haemorrhage was so severe that a blood transfusion was necessary, the child's mother acting as donor. Epistaxis has been frequent, and likewise bleeding from the gums. On occasion there has been haematemesis. She suffers from extensive bruising on slight injury, and frequently has crops of red spots under the skin.

She was treated in Ward 30 in 1925, the diagnosis being Purpura Simplex: treatment was by means of calcium salts, horse serum, etc.

All her symptoms had been improving recently, until her first menstrual period began on 7th January, 1928. Prolonged vaginal bleeding set in, which continued till on the 20th January she was admitted to Ward 34, where the uterus was packed. This arrested the haemorrhage, but investigation of the case showed that the same state of affairs was liable to recur with each period, so surgical advice was sought. She was transferred to Ward 14 on the 16th of February.

Previous Illnesses: - Tendency to bleed as noted.
Scarlet fever.

Family History: - Father and mother alive and well
Two brothers and one sister alive and well.
No similar condition in the family.

State on Examination: - The patient is a well developed girl of fourteen, but is rather pale. She is bright and intelligent, and feels perfectly fit. Previous to her admission to ward 14 she was able to get up and attend a concert held in the hospital.

There is no jaundice, no oedema or ascites, but there is definite pallor of the mucous membranes, indicating anaemia.

Examination of Special Systems: -
Examination of Special Systems:

(a). Gynaecological: the operation findings as noted in ward 34 are: the left ovary was cystic, and enlarged to the size of a plum, and the uterine scrapings revealed only blood-clot.

(b). Haemopoietic System: There is no enlargement of lymph glands. The spleen is not palpable, but is slightly enlarged on percussion. There is present a diffuse and widespread eruption, purplish red in colour, not obliterated by pressure: the spots are mainly punctate in size, petechias, with a few larger macules. The girl says that they appear in crops. The application of a Bier's bandage to the left arm resulted in the appearance of a profuse rash below that level.

The following blood examination was carried out:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.B.Cs.</td>
<td>4,400,000</td>
</tr>
<tr>
<td>W.B.Cs.</td>
<td>10,000</td>
</tr>
<tr>
<td>Hb.</td>
<td>50%</td>
</tr>
<tr>
<td>C.I.</td>
<td>0.57</td>
</tr>
<tr>
<td>Platelets</td>
<td>18,000</td>
</tr>
<tr>
<td>Fragility of red corpuscles</td>
<td>normal.</td>
</tr>
<tr>
<td>Coagulation Time</td>
<td>3½ minutes</td>
</tr>
<tr>
<td>Bleeding Time</td>
<td>1½ minutes</td>
</tr>
<tr>
<td>Blood film</td>
<td>Shows small red cells, poorly filled, and marked poikilocytosis: leucytosis moderate in degree, polymorph in character: platelets almost absent.</td>
</tr>
</tbody>
</table>

Blood group tests were carried out on the patient and her relatives with a view to the possibility of a transfusion. Typing with standard sera, (Mosso's classification) and direct tests were carried out. Results as under:

<table>
<thead>
<tr>
<th>Sample</th>
<th>Serotype</th>
<th>Direct Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Madge Buchan</td>
<td>Two</td>
<td>No agglutination.</td>
</tr>
<tr>
<td>Mrs. Buchan</td>
<td>Three</td>
<td>Slight agglutination</td>
</tr>
<tr>
<td>Lizzie Buchan</td>
<td>Three</td>
<td>No agglutination.</td>
</tr>
<tr>
<td>George Buchan</td>
<td>Two</td>
<td></td>
</tr>
</tbody>
</table>

The brother George was therefore selected as the donor, should one be necessary.

Estimation of the serum calcium had been carried out while she was in ward 34........result 9.1 milligrammes %.

(c). Circulatory System: Pulse regular in time and force, rate 80/minute. Nil to note abnormal in the character of the wave: vessel wall not palpable. Blood pressure (Systolic) 110 mm Hg. Heart not enlarged; nothing to note beyond an accentuated second sound in the pulmonary area.

(d). Alimentary System: Tongue slightly furrowed. A few teeth carious, but the majority sound. Abdomen normal on inspection. Moves freely on respiration. No cutaneous hyperaesthesia. No pain, nor tenderness, nor rigidity. Liver not enlarged. Spleen not palpable, but splenic dull
dullness/ slightly increased.

Investigation of Respiratory, Urinary, and Nervous systems purely negative.

PROVISIONAL DIAGNOSIS:— Purpura Haemorrhagica, the "Essential Thrombopenia" of Frank: "Thrombocytolytic purpura" of Kaznelson.

PROGNOSIS:— See commentary.

TREATMENT:— Splenectomy.

Preoperative Treatment: the patient having recovered fairly well from the effects of her haemorrhage, and now being in good condition, little preoperative treatment was necessary. The usual dose of Ol. Ricin was given the day before, and a soap and water enema on the morning of the operation. Atropin Sulph. (gr. 1/100) was given hypodermically half an hour before operation.

Operation: 20. II. 28. Prof. Wilkie.

Ethyl chloride and ether anaesthesia.

A vertical incision about five inches long was made over the upper part of the left rectus muscle, an inch from the mid-line. The incision was carried through the muscle, all bleeding points being caught and ligated: all ooze was carefully arrested before any further steps were taken. The posterior layer of the rectus muscle was divided, and the peritoneum picked up with forceps, nicked with a knife, and pined with scissors in the line of the wound.

The abdomen being thus opened, the spleen was found to be definitely, but not greatly, enlarged (being in fact about the size of an adult organ). It was adherent to the diaphragm at its upper pole. The glands in the gastro-epiploic and lienorenal ligaments were enlarged. The liver was normal: so also the stomach and duodenum.

The adhesion at the upper pole was divided. The gastro-splenic omentum was divided between ligatures. A large vein was seen leaving the upper pole and coursing backwards: it was ligated and cut. The splenic artery was dissected out by blunt dissection, and ligated in its continuity, without the aid of clamps. The rest of the pedicle was then transfixed and ligatured. Preparatory to the ligature of these main vessels, the spleen had been delivered out of the abdomen, mobilising having been effected by division of the adhesion and the accessory veins; in all these operations on the pedicle, there was one of the surgeon's hands behind it to control possible haemorrhage. The pedicle having been securely ligated, clamps were applied distal to the ligatures, in two sets, and the pedicle divided between them. A second series of ligatures was then applied over the clamps.

Though the utmost care had all along been taken not to handle tissues roughly, at the end of the operation it was seen that the omentum and other tissues subjected to handling were bloodstained, and discoloured from haemorrhage into their substance.

The wound was closed with catgut in layers, silkworm gut
being used for the skin incision, and also for the "through-and-through" sutures that held in place an anchored dressing.

While the operation was in progress a blood transfusion was carried out, using the indirect, citrated method. Fourteen ounces of blood were drawn off from the median cubital vein of her brother George by vein puncture. The blood was received into a sterile receptacle containing 40cc. of 3.8% Sodium Citrate. The quantity withdrawn was such as to result in a final concentration of citrate of 0.38%, i.e. the final volume to be 400cc., or approximately 14 oz. The blood as withdrawn was stirred continuously, and kept at blood heat by a water bath. It was injected into the recipient's arm, intravenously, by a funnel and cannula (gravity method). The pulse was watched with care throughout the injection.

The patient on leaving the table was in good condition, and did not appear to be suffering from any degree of shock; her appearance was in fact rather plethoric, the face being flushed and of high colour.

The spleen as removed weighed 3 1/2 oz.

Postoperative Treatment, and Progress notes:

20. II. 28: On returning to the ward the patient was given a dose of 1/12th of a grain of Heroin, just as she was coming out of the anaesthetic. She was also given Glucose salines per rectum, oz. viij, four hourly. A second dose of heroin (1/12th) was given at night. The patient all day was drowsy, and a little sick. Pulse rate varied between 132--136.


22. II. 28: Patient not so well today. Pulse rate reached 146 in the forenoon, but dropped later to 120. Still a good deal of discomfort and pain, the heroin having to be repeated thrice.

23. II. 28: Much better today. The pulse dropped to 108 in the evening. Water was now given by mouth ad lib., and the rectal glucose stopped. The postoperative dose of castor oil was given. Heroin at night.

24. II. 28: Bowels opened by a soap and water enema: return to normal diet started, beginning with milk, toast, and tea.

25. II. 28: et seq.: Patient made a steady and uninterrupted recovery. The stitches were removed on the 12th day, and the patient allowed up on the 13th, and discharged on the 14th day after operation.

Examination of the blood was carried out frequently during this period, special attention being paid to the bleeding time and the platelet count: details will be found on the next page.

A Bier's bandage was applied for a few minutes on 4. III. 28, and resulted in the appearance of a few purpuric spots on the forearm.
Record of Blood Examinations (postoperative):

20.11.28. Operation at noon.
3 p.m. Bleeding Time 5 min 30 sec.
       Platelets ......30,000 plus.
3 p.m. Bleeding Time 5 minutes.

21.11.28. 10.45 a.m. Bleeding Time 4½ min.
           Platelets 34,000
8.30 p.m. Bleeding Time 4 min.
           W.B.C. ............36,000

22.11.28. No tests carried out: patient not so well.

23.11.28. 10.45 a.m. Bleeding Time 4½ min.
           W.B.C. ............14,800
3 p.m. W.B.C. ............16,400
       Platelets ...........146,000

24.11.28. Bleeding Time 4 minutes.
           Platelets ......248,000
           R.B.C. ...........4,320,000
           W.B.C. ............18,500

25.11.28. Bleeding Time 3½ minutes.
           Platelets ......230,000

26.11.28. Bleeding Time 3 minutes.

1.11.28. Bleeding Time 4½ minutes.
           Platelets ......230,000
           W.B.C. ............183,00000 21,000

4.11.28. Bleeding Time 4½ minutes.
           Platelets ......196,000
           R.B.C. ...........4,350,000
           Hb. .................55%
           C.I. .................0.63
           W.B.C. ............13?500
           Coagulation Time 3½ minutes.

Reported: 18.11.28.

Bleeding Time 9 minutes.
           Platelets ......92,000
           R.B.C. ...........5,420,000
           Hb. .................80%
           C.I. .................0.74
           W.B.C. ............11,400
           Differential Count: Polymorphs 54%
           Sm. Lymph' tes 30%
           Lg. Lymph' tes 9%
           Transitionals 6%
           Basinop'hils 1%
           Coagulation Time: - 2 minutes.

The application of a Bier's bandage for five minutes resulted in the appearance of 10-15 pinpoint specks near the elbow.
Mama Buchanan: age 47 years

Case of Puerperal Hemorrhage, Treated by Suprarenin

Discharged from Hospital: 5/3/28

White Blood Cells: 38,000
Platelets: 250,000

T.B.: 4,320
Hb.: 50%

Before: Platelets: 35,000

Bleeding time: (Minutes)
<table>
<thead>
<tr>
<th>Platelets</th>
<th>Before</th>
</tr>
</thead>
<tbody>
<tr>
<td>250,000</td>
<td>20</td>
</tr>
<tr>
<td>35,000</td>
<td>5</td>
</tr>
</tbody>
</table>
COMMENTARY:-

The chief points in this case fall naturally under the heads of Pathology, Diagnosis, Treatment, and Prognosis. These will be dealt with serially.

Pathology:—The discovery of the pathology of this condition is a matter of recent years: the advance made has been estimated by a comparison of the old and new terms for the disease..."Morbus maculosus of Werlhof" and "Essential thrombocytolytic purpura haemorrhagica".

It is a matter of clinical observation that in this disease there is a very definite reduction in the numbers of blood platelets, with this reduction to be associated a normal coagulation time, but a prolonged bleeding time, as tested by the usual methods of timing coagulation in fine capillary tubes, and the duration of oozing from a needle puncture respectively. Further though in these cases the blood clots apparently normally in the usual time, the clot is loose and does not contract after its formation, so that sealing of vessels is faulty and natural haemostasis ineffective. This failure to contract is apparently due to the absence of blood platelets. With the normal coagulation time is associated a normal blood calcium content.

The second factor in the disease is some capillary weakness which in conjunction with the poor clotting of the blood allows of the formation of petechiae on alight provocation, as for example after the application of a Bier's bandage to the arm.

As to the cause of the low platelet count, authorities differ. Kaznelson of Prague, the originator of splenectomy for the treatment of the condition, maintained that the spleen was at fault, destroying the platelets, hence his term "thrombocytolytic purpura". Frank also advised this treatment, but because he attributed to the spleen an inhibitory action on the bone marrow...hence his term "essential thrombocytopenia". The normal coagulation time is said to be evidence however that their formation is normal, for it is to the dissolution products of the platelets that the thromboplastic substances of the blood owe their origin. (See the paper by A.W. Spence in the Brit. Jour. of Surgery, 1928, XV; 59; p 466.)

This and other factors favour the view of Kaznelson.

The question of the relation of the platelet count to the coagulation time is dealt with by Crawford thus:—(Lancet, 1924; II; 596)

"In all the cases tested no direct correlation could be found between the platelet count, bleeding time, and coagulation time. The bleeding time was however usually prolonged when the platelets were markedly diminished. The coagulation time appeared to be quite independent of the number of platelets, and would seem to be a very unreliable guide, with regard to tendency to haemorrhage apart from cases of haemophilia, in which bleeding time and platelets are stated to be approximately normal, while the coagulation time is definitely prolonged."

The question of relation of platelet count to the bleeding time is further summarised by Spence thus (loc. cit.)

"The prolongation of the bleeding time in purpura haemorrhagica is probably due more to defective quality of the platelets than to diminished numbers."

This conclusion is drawn from an analysis of a large number of cases in the literature, and points rather to Frank's view that...
that there is defective formation of platelets. As is
so often the case in dealing with subjects of Biological research, the
results of various observers are somewhat in conflict, and judgment is
perhaps best deferred till further facts are available.
The absence of gross changes in the spleen is noteworthy (see path-
ologist's report in case history.)

To summarise, the disease is one due to ineffective haemae-
stasis because of deficiency in the bloodplatelets, which are responsible
for the proper retraction of the clot after the calcium-thrombokinase-
prothrombin-fibrin mechanism has done its work: as such it is in sharp
contrast to haemophilia, where interference is earlier in the process,
due to entirely different aetiology.

Diagnosis: The non-existence of an acute gynecological condition,
e.g. sarcoma butryoides, which had been ruled out by exploratory dilat-
ation and curettage, and the long history of liability to bleeding alike
pointed to this being one of the class of "bleeders" diseases. Of
this class there are two main groups: the true haemophilia on one hand
and that aggregation of odds and ends grouped collectively as 'purpura'
on the other.

True haemophilia is generally accepted as a disease of
the male only, at least in so far as its active manifestations are con-
cerned, though its transmission is by the female. It is familial and
hereditary: and it is characterised clinically by a history of bleedings
external and internal, as into viscera or joints, and by a lengthened
blood coagulation time. Some authors deny that males are exclusive-
ly liable to the disease, but in this case apart from the sex, the coag-
ulation time was normal: we may conclude that here we are not dealing
with a haemophilia.

That Purpura is a symptom and not a disease is a well
worn clinical maxim. Obscure haemorrhages of all sorts are liable to
be grouped as "purpura", and for the casual, or busy, practitioner there
the "diagnosis" rests. On further investigation, however, we are abe
to reduce this chaotic rubbish heap of clinical medicine to something
approaching order. We may recognise purpuras (a) secondary to various
causes...the malignant forms of such fevers as scarlet, measles, cerebro-
spinal meningitis; rheumatism: the cachexias of wasting diseases like
tubercle, or the diarrhoeas of children: deprivation and deficiency
diseases such as scurvy: septic foci here and there in the body, as tonsi-
sils, etc.: blood diseases like leukaemias; and in fact secondary to a
hundred and one causes. (b) we may also recognise a group where
as here, no primary cause can be discovered by systematic clinical ex-
amination...the "idiopathic purpuras".

Too much stress cannot be laid on the exclusion of all
causes of symptomatic or secondary purpura before a diagnosis of primary
or idiopathic purpura is made.

Of this latter group there are generally recognised
three forms: purpura simplex, p. hemorrhagica, and Henoch's purpura.
The first we exclude here, for it is a mild form characterised by the
absence of visceral haemorrhage. The last also we exclude, for it is
accompanied by, in a typical case, gastro-intestinal symptoms of an acute
nature, bleeding into joints, and it may be nephritis, with a severe
general reaction. We are left with one only of this group therefore
Purpura Haemorrhagica, which tallies with our case on rough clinical in-
clinical investigation/

Closer study of the case confirms this diagnosis: the relations of coagulation and bleeding times: the normal blood calcium: the enlarged spleen: and the low blood platelets all fit in with the pathology of the condition as detailed supra. The profuse crop of petechiae on the application of a Bier's bandage is strong corroborative evidence, pointing to the other essential point in the causation: permeability of capillaries.

I would again emphasise the importance of excluding the symptomatic group before coming to a diagnosis of this nature... as for instance the exclusion of leukaemia in this case by the result of a blood-film examination.

Treatment:- "Medical measures in this disease are notoriously ineffective" runs the comment of one physician on this subject. So-called haemostatics... ergot, turpentine, calcium salts, adrenalin, ... have all been tried. Rectal administration of polyvalent antistreptococcal serum has been advocated by some, with claimed good results, and with as blind faith and little basis of reason as in most of the enthusiastic serum and glandular therapy of today! The previous treatment of this case was (as detailed in the history) by means of calcium salts and horse serum.

Our new knowledge of the pathology of the condition has led to the adoption of operative methods, the rationale being easily understood. To Kaznelson of Prague belongs the honour of precedence in advocating and practising splenectomy; he first performed the operation for this condition in 1916. The first cases in this country were those of Sutherland and Williamson, in 1924: (see Lancet, 1925; I; 323.) Since then the operation has been widely practised, an abstract of 101 cases being given by Spence. (loc. cit.) The influence of splenectomy on prognosis is considered infra: here we must deal with points in the actual treatment of such a case as this.

A period of rest in bed is advisable before operation. up to a month or so if possible. An interval of freedom from acute symptoms should be selected, and a time at which the blood shows a good red cell count. Good general condition pre-op. is a good start on the way toward the elimination of operative mortality.

The serum of the patient should as in this case be typed in view of the probability of transfusion being necessary. There is no point in giving calcium salts with a blood calcium at the normal level: nor is hypertonic citrate indicated when the coagulation time is normal.

The points in the actual operation are dealt with sufficiently fully under that heading; but attention should be paid to the necessity for extreme gentleness, and careful haemostasis. This is no operation indeed for the "carnivorous surgeon".

The indications for transfusion, and for the giving of glucose salines post-op. are sufficiently clear.

The results of Splenectomy are dealt with under Prognosis.

The results of Splenectomy in general are summarised ad discussed at the end of case III, q.v.
Prognosis:

Untreated, numbers of these cases die from the effects of haemorrhage. There is a fulminant and rapidly fatal type, but the majority are characterised by chronicity, and by recurrences. In this case there is occurrence of haemorrhage from the uterus of a severe type at the onset of menstruation was a disquieting feature, with the probability that it might recur with each period, that it might complicate a pregnancy, or prove fatal in the postpartum state. The patient's hold on life could not be called secure.

After splenectomy many cases are definitely cured. The blood plates return to normal, the bleeding time falls to reasonable limits, and the outlook is changed entirely. The figures given by Spence are roughly 81% of cures in the chronic form, and a quarter of that figure in the acute form of the disease. The differences are due apparently to differences in the pathology of the condition. The view that the spleen alone was at fault was given under the heading 'pathology' supra. There is much for the argument that the disease is one of the reticulo-endothelial system, of which different portions or the whole may be affected in different cases. It is only in those in which the spleen alone is badly affected that Splenectomy is of avail.

A fall in bleeding time and a rise in platelets is a good omen after operation, indicating that the spleen really has in that case been at fault; but though in this case these changes were found, the results are complicated by reason of the transfusion done at the same time. It is a recognised fact that in these cases a fall in bleeding time and rise in platelets may follow transfusion of citrated blood. The question is of interest as it has been asserted that platelets are destroyed by citrated blood. To quote from the article referred to so often already:

"It seems that transfusion acts as a temporary stimulus to the megakaryocytes of the bone marrow to increased platelet production, but that after three or four days this stimulating effect disappears, with consequent diminution of the platelet count and prolongation of the bleeding time."

To date (28.V.28) the patient has only reported once since discharge. On that occasion there was a disquieting fall in the platelet count as compared with the figure on leaving hospital, and a longer bleeding time. There had not been another menstrual period, which in this case is essentially the crucial test of success. Only time and repeated examinations will serve to prove the success or otherwise of the operation in this case; the justification for the procedure adopted, in view of the high percentage of successes in such cases, was ample.
CASE HISTORY:

Name: Albert Clark.
Age: 20 years.
Address: 10, Parkfield's Green Terrace, Edinburgh.
Occupation: Coal Carrier.
Recommended by: Dr. Gibson, 10, Parkfield's Green Terrace, Edinburgh.
Admitted: 1 III 28 (transferred from Ward 15.)

Complaint: Breathlessness.

CASE No 2. ALBERT CLARK, aged 20 years.

Splenectomy for Banti's Disease.

History: The patient always enjoyed perfect health until he began to notice that he was very tired after meals and in the summer. He became very pale, and noticed that his stools were clay-like. He had no trouble with his stomach.

On admission: On first seeing the patient did not want to work. All went well until about six months ago, when the symptoms of pallor and breathlessness returned. He had a total of 6 days, also very black in colour and after one night's illness, required a cupful of blood. Dark red in colour. He was transferred to Ward 32 on 19 XII 28. He was seen at the time of admission, but has improved steadily since and is at present on the ward. As mentioned above, there were no symptoms whatever in the abdomen, no flatulence or pain, no abnormal masses or tenderness.

Previous Illness:

Family History:

Bedside Examination:

State of Examination: There was no general examination of the heart.

Examination of Special Systems:

(a) Alimentary System:

Apart from haematemesis, no other complaint of the alimentary system as detailed in the history; no slightly forced, there are 3 previous hemorrhages.
CASE HISTORY:

Name: Albert Clark.
Age: 20 years.
Address: 18, Parson's Green Terrace, Edinburgh.
Occupation: Coal Carrier.
Recommended by: Dr. Gibson, 19, Piershill Terrace, Edinburgh.
Admitted: 1. III. 28, (transferred from Ward 32.)

Complaint: Breathlessness.

History: Until eighteen months ago the patient always enjoyed perfect health. About a year and a half ago he began to notice that he was very breathless on exertion. At the same time he became very pale, and noticed that his stools were black like tar. He had no trouble with 'his stomach'. He was admitted to ward 32 after a few months, and improved very much while in the hospital.

On discharge he felt very well, but did not return to work. All went well until about six months ago, when the symptoms of pallor and breathlessness returned. His stools were at this time also very black in colour, and after a month's illness he vomited a cupful of blood, dark red in colour. He was readmitted to Ward 32 on 19. XII. 27. He was extremely weak on admission, but has improved steadily since, and is now feeling very well and quite strong.

As mentioned above, the patient has had no digestive discomfort whatever; he has had no feeling of weight or fullness in the abdomen, no flatulence or pain, no nausea or sickness, except when he had the haematemesi.

The patient often has epistaxis, but has no difficulty in arresting the haemorrhage, and if he cuts himself he has no excessive loss of blood.

Previous Illness: Always well; nil to note.

Family History: Father alive and well, age 51.
Mother died 18 years ago, of pneumonia.
Four brothers all older than the patient all alive and well: none dead. No history of any similar disease in the family.

State on Examination: Though pale the patient looks healthy. The general examination of the case in fact reveals nothing but anaemia... there are no other morbid appearances. He is a well developed, though not muscular, young man, cheerful and intelligent.

Examination of Special Systems:

(a) Alimentary System: There have been no subjective phenomena, apart from haematemesi on one occasion, and the passage of black stools as detailed in the history, q.v. On examination the tongue is slightly furred, there are some carious teeth, and the tonsils are large.
large/

On inspection the abdomen is seen to be a little full; it moves with respiration equally on the two sides. There is no cutaneous hypepaesthesia, no tenderness or rigidity. The spleen is enlarged, as noted below. The liver is not enlarged to palpation, and on percussion the liver dullness is normal. There is no free fluid in the abdomen nor any other abnormality to be made out.

(b) Haemopoietic System:— There are no enlarged glands in the axillae, neck, groin, or elsewhere. The spleen is palpable, greatly enlarged. The right border extends to the midline above the umbilicus, while the lower border lies at the level of the umbilicus. It moves freely with respiration, and is firm in consistence. There is no tenderness or pain, and no friction on auscultating over it.

Blood count results as under:

- R.B.Cs. 5,500,000
- W.B.Cs. 6,500
- Hb. 50%
- C.I. 0.45

Differential White Count:—
- Polymorphs 59%
- L.Lymph'ts. 17%
- S.Lymph'ts. 23%
- Basophils 1%

(c) Circulatory System:— The heart is not enlarged, and both sounds are closed in all areas. The pulse is regular in time and force, rate 80 / minute. Blood pressure (systolic) about 110 mm. Hg. Vessel wall not palpable.

(d) Urinary:— The urine contains no abnormal constituents.

Respiratory and Nervous systems show nil to note.

PROVISIONAL DIAGNOSIS:— Banti's Disease.

PROGNOSIS:— If untreated, see commentary.

TREATMENT:— Splenectomy.

Preoperative Treatment:— The patient was judged to be in good enough condition to stand the operation, and very little preoperative treatment was needed. He got the usual dose of 0.1. in the day before, and a soap and water enema the night before operation. The usual dose of Atropin Sulph. (gr. 1/100th) was given immediately before leaving the ward for the theatre.


Anaesthetic:— Chloroform and ether.

The incision used was the left rectus one described in Case 1. On opening the abdomen the spleen was seen to be very large.
were no adhesions to the diaphragm, but the apex was bound to the posterior abdominal wall by a ligament, making delivery very difficult. Examination of the various viscera was carried out: the liver was rather but showed no definite evidence of cirrhosis. The stomach and duodenum were healthy, and showed no trace of ulceration. There were some enlarged glands along the greater curvature of the stomach.

Further access was obtained by dividing the fibres of the left rectus transversely at the level of the intersection above the umbilicus. The gastro splenic omentum with its contained vessels was ligated and divided. The adhesion of peritoneum on the outer side of the spleen was incised, and the spleen pulled over. One of the surgeon's hands meanwhile controlled the pedicle. The spleen was thus delivered out of the abdomen. The upper smaller portion of the splenic artery was ligated, and the lower and larger portion also ligated without the application of clamps. It was possible in this case to avoid including the tail of the pancreas in the ligature without imperilling its security. Two pairs of forceps were applied beyond the ligatures, and the pedicle cut between them; so the spleen was removed. Further ligatures were applied round the proximal forceps. There was very little oozing applied. The abdomen was closed in layers, and an anchored dressing applied.

The patient was in very good condition at the end of the operation.

The spleen removed weighed 1 lb. 9 1/2 oz.

Postoperative Treatment and Progress Notes:

The patient was put under the influence of Heroin (1/12th of a grain) as he was coming out of the anaesthetic. The sedative was repeated three times that day. He was also given Glucose salines per rectum, four hourly, each of oz. wiij of %.

5.11.28 Patient today not so well. He had a slight rise of temperature, up to 99.2 deg., with a pulse rate of 124, and respiratory ions up to 30 per minute. The cause of this was an attack of coryza a and mild bronchitis...the patient afterwards told us that he had had a slight cold before operation, but had not thought it of any account: it was so slight as to escape the notice of the staff, but had been lit up by the ether anaesthesia.

Heroin was given twice on this day.

7.11.28: The cold still persisted, and cough was severe, but the patient was much easier, and had only heroin on one occasion. The postoperative dose of O1. Ricin was given at night.

8.11.28: Respiration fell this morning from 30 to 22; definite improvement set in. The bowels were opened by a S. and W. enema in the morning. Patient now put onto milk and bread, with a gradual return first to light and later to full diet.

The stitches were taken out on the 12th day after operation, the wound having healed well despite the strain of the coughing in the first few days.

The patient was up on the 13th day, and home on the 14th day after operation.

Details of routine blood counts done after operation are given on the next sheet.
The patient reported on the 24th of April: feeling very well: has a good colour and a well nourished appearance. Red corpuscles over 7 million (?) and whites 5,800.

Pathologist's Report:—On spleen—
"Thickened capsule and marked increase in fibrous tissue stroma. Appearance consistent with diagnosis of Banti's Disease"


Record of Blood Examinations;—

Preoperative: as noted under examination of haemopoietic system.

Postoperative:—5.III.28 (day of operation, which took place at noon)
4 pm. . . . Whites 19,800 per c. mm.
8 pm. . . . Whites 21,600 per c. mm., of which
P-85%; LL-7.5%; SL-5.5%; Tr.-1%

6.III.28: Whites 30,200 per c. mm.
7.III.28: Whites 29,600 per c. mm.
Reds 4,880,000 per c. mm.
Hb 52%; C.I. 0.53

9.III.28: Whites 26,200 per c. mm., of which
P-75%; LL-9%; SL-10%; Tr-5%; E-1%

11.III.28: Whites 15,200 per c. mm., of which
P-71%; LL-13%; SL-10%; Tr-6%

13.III.28: Reds 5,710,000 per c. mm.
Hb 62%; C.I. 0.54.
Differential count as follows:
P-75%; LL-10%; SL-4%; Tr-7%; E-4%

14.III.28: Whites 13,800 per c. mm.
16.III.28: Reds 5,610,000 per c. mm.
Whites 10,700 per c. mm.
Hb. 70%; C.I. 0.63

Reported on 24.IV.28: Reds 7,100,000 per c. mm. (?)
Whites 5,800 per c. mm.

(Routine platelet counts were not carried out on this case, but it was noted in the later films that the thrombocytes were very abundant,

(For the accuracy of these figures, I cannot vouch: there seems to be some doubt as to their value: the observer is not known to me personally.)
abundant/ and apparently increased above normal. In another case of splenectomy for Banti's Disease (D Mackay, operated on in Ward 13 in early February) the platelet count reached 500,000 per c.mm. before discharge.
Pathology and Clinical Features:

There are several conditions of blood disturbance associated with more or less splenic enlargement, and at least four of these were grouped together as splenic anaemia by Griessinger in his original description in 1866. It was nearly twenty years later that Banti isolated from this collection the disease that bears his name. The isolation of this disease as a clinical entity has really been by a process of recognition and elimination if the other members of the original group than by elucidation of the one at issue. The author of one text-book attributes to Mayo the paradox "incomplete knowledge is essential to the diagnosis. If we know the cause of splenic anaemia it is not splenic anaemia." ... from which terse statement we may gather that 'nesio' will figure largely in our description of its pathology.

The disease is one of unknown causation, though as usual chronic infection, located in the spleen, has been cited as the cause. No causal agent has however been demonstrated, whether microbial or toxic. The most interesting and vital point in the pathogenesis from a surgical standpoint is that splenectomy in the early stages completely cures the condition. (See Treatment).

The cardinal points, alike in pathology and symptomatology, are four: splenic enlargement; progressive anaemia; tendency to haemorrhage; and later cirrhosis of the liver.

The splenic enlargement comes without apparent cause, usually in young adult males; it is of fair degree, and spleens weighing up to three kilogrammes have been recorded, though a third of that is a commoner figure. While it may be associated with pain from a peri-splenitis, in most cases it is a painless condition.

On section the spleen shows no gross changes: there is a fibrosis throughout the organ, and a degree of atrophy of the spleen pulp tissue, but no really distinctive changes are found. The organ is commonly bound to the diaphragm by adhesions of peritoneum in which run large venous channels...as big or bigger than the normal splenic vein in some cases. Dilatation is seen in the splenic vein, which is also the seat of atheromatous changes, with it may be calcification; thrombosis may even occur. Likewise the vasa brevia which run from the stomach in the gastro-splenic omentum are dilated...giving rise to one of the dangers of the condition, haemorrhage.

The anaemia present is of a secondary type, not usually very severe, counts ranging up to three millions being common. The haemoglobin is proportionately more reduced, giving a low colour index. Briefly, the anaemia is of a secondary type, and is associated with a definite and very constant leucopenia: Gulland says that in the later stages it is the lymphocytes that suffer especially.

Rolleston leads the school that consider the anaemia as purely secondary to the haemorrhages, but by some it is considered to be independent in causation.

The tendency to haemorrhage is seen especially in the stomach...haematemeses and melena being symptomatic. It is due to
rupture of the dilated vasa brevia referred to above. In the later stages the haemorrhage is due to ruptured oesophageal varices (Osler). Still other factors may be at work, as in the production of the epistaxis in this case.

The late stages of cirrhosis of the liver with ascites are interesting. Apparently the "toxin" from the spleen reaches the liver via the splenic and portal veins, and produces there fibrosis similar to that in the former organ. In view of the opinion that one has recently read that the splenic blood does not mix freely with the rest of the portal blood, but goes to the left lobe of the liver, it would seem that consideration of the distribution of the cirrhosis of Banti's disease might throw light on the truth or otherwise of the statement; or conversely, were the true state of affairs regarding the blood flow known, then we might be able to argue for or against this toxic theory. Be that as it may, the cirrhosis as usually described is universal in distribution and nodular in type (like that of Laennec), often associated with ascites, but seldom with jaundice.

Clinical Course:— The splenic enlargement is frequently primary, and may persist for several years before further symptomatic develop. Sooner or later however there is anaemia, progressive in nature, and associated with haemorrhages. The patient's condition does not steadily go downhill, but the disease tends to ameliorate and to recur in wave-like crises over a period of two or three years or longer...up to 17 years, in one recorded case. In the end the cirrhosis of the liver becomes apparent, ascites develops, and cachexia sets in: the fatal ending draws near.

The immediate cause of death is usually gastrointestinal haemorrhage, probably from the enlarged vasa brevia, but possibly from the varices referred to by Osler. Natural recovery is said to be very rare, if a true Banti's disease ever does recover.

Diagnosis:— This case presents the triad characteristic of the disease: splenic enlargement, the blood picture, and the haemorrhages, all of which are to be found in early cases. In addition there is the recurrent nature of the disease, its progress in wave-like exacerbations.

In spite of the suggestive nature of these symptoms, however, we must not come to a diagnosis of Banti's Disease, which is after all a rare condition, till we have considered all the other possibilities. We must rule out (a) other causes of profuse gastric haemorrhage and of melaena: i.e. in a young man peptic ulcer must be excluded. In the history there is non shred of evidence to suggest such a condition, though of course the acute ulcer seen typically in young women may be "silent" till it produces profuse haematemesis. In this case the leucopenia alone would almost serve to exclude such a simple secondary anaemia as would follow such an accident, for in simple secondary anaemia there is a leucocytosis.

We must also rule out (b) other splenic enlargements that are associated with anaemia, as for example leukaemias or pernicious anaemia: excluded by the blood picture alone. Lymphadenoma, excluded by the absence of glandular enlargement, though the blood picture is very similar; scholastic jaundice, excluded by the lack of jaundice, and by the much too gross enlargement of the spleen: Gaucher's disease/
Gaucher's Disease: excluded by the wrong age and sex of the case, the lack of familial history, the absence of gross liver enlargement, and the absence of the earthy skin tint so often seen in these cases.

Further we must consider the group of splenic enlargements (c)...due to infection, with concurrent anaemia: the common ones, tubercle, syphilis, and malaria are easily ruled out: the others such as Kala-Azar are of merely academic interest in this country.

(d)...There is no suggestion of the form associated with chronic suppuration...waxy disease.

(e)...The duration is argument enough against the case being one of sarcoma primary in the spleen.

To quote again, this time from Moynihan, "What remains? Only the forms of splenic anaemia, the cause of which is unknown."...

In other words the diagnosis of Banti's Disease must stand.

Treatment and Prognosis:-

"As is our pathology, so is our practice"...so runs one of the numerous dicta of the 'father of modern medicine', and here its truth is abundantly in evidence.

Did we but know exactly the cause of Banti's disease our therapeutic measures would tend toward its elimination or coagulation without damage to the tissues of the patient. But as our knowledge only runs to an experimental observation that excision of the spleen cuts short the disease, we are forced to practise that rather heroic measure on all our cases.

Till such time as more exact pathological knowledge is ours, so long must our treatment be in a sense a mutilating operation...involving the loss of a presumably useful organ. (The results of splenectomy in normal people are dealt with after Case III.)

Whatever may be our hopes for the future, we can claim that in splenectomy we have a cure for all reasonably early cases of the condition; should liver involvement be extreme it is hopeless to expect removal of the causal agent to undo the fibrosed work of years of irritation, any more than strict abstinence would avail the hapless victim of 'gin-drinker's liver'. In cases with moderate or early liver change, however, the operation should be attempted, for that organ shows in the condition as in others its wondrous power of regeneration.

The operation itself is not without danger, especially if attempted in a case that is run down through severe haemorrhages. An operative mortality of 20% is quoted by some of the less successful writers. On what lines must one proceed to reduce this?

In the pathology of the disease stress was laid on the adhesions that exist between the spleen and the diaphragm, and the large veins that run therein. Here then is one source of danger...the division or tearing of an apparently innocent adhesion without preliminary ligation may result in rapidly fatal haemorrhage: size of vessel, and difficulty of access account for the seriousness of the accident.

Mention was also made supra of the atheromatous condition of the splenic vessels, a condition which renders them friable and makes a tight ligature liable to cut through. It is for this reason among others that the operator in this case preferred to ligate the vessels in their continuity, without the use of clamps.
Postoperative disasters may be due to (a) shock: the routine administration of rectal glucose-salines helps to minimise this risk; so also does careful operative methods. (b) to thrombosis, especially in the mesenteric vessels. This is due to increased coagulability of the blood, due in turn to the increase in the blood-platelets after the removal of the destructive spleen. This is a very real risk, and one that is not easy to combat. It will be noted that such an increase in the platelets was noted in another case of this sort, and also was observed to exist here. (vide p.5, top.) (c) to haemorrhage, probably due to hurried and inefficient haemostasis at operation.

To summarise, a careful, unhurried technique is the first step toward the reduction of the operative mortality: with that combined of course, attention to the above points.

The prognosis, once the operation is safely passed, is uniformly good: in a successful case the complete arrest and permanent cure of the condition is to be expected.

In this particular case, the rise of the haemoglobin as seen in the chart immediately after operation, and well maintained till discharge, is significant. A corresponding rise in the red cells would hardly have been expected, since their figure was normal to start with, but apparently, from the figures obtained on reporting, there has been some increase in their numbers also.
CASE

CASE No 3.

MARY TAYLOR, aged 28 years.

Splenectomy for acholuric jaundice.

Mary Taylor.

28 years.

1 Mary's Row, Main Stree, Kelty.

Used to work in a shop.

Dr. Keay, Rosssan, Kelty.

15. III. 28, (transferred from the medical aid).

For the past nine months, pain and excessive loss during micturition, associated with weakness.

For four years, pain with micturition.

It varies greatly in degree from time to time, and on occasion is very pronounced; at these times she is apt to feel tired and sick. She never has any discomfort associated with it, nor any pain in the upper abdomen; she has no indication of flatulence. In spite of this tinged of yellow in her skin, the patient has always considered herself a normal person. It was only about a year ago that it was suggested to her that she was in any unusual condition.

She has had no bleeding from the nose or mucous membranes (except as noted under menorrhagia) but has always noted a tendency to bleed excessively from any small cuts.

For the last four years the patient has had pain when time to time at the end of micturition; this is seldom very severe. She has not noticed any change in the appearance of her urine.

The complaint for which she eventually sought hospital treatment only arose on nine months ago. Two months before her attack of acute bronchitis, which left her weak and anemic.

About last July she began to have excessive loss at her menses period, accompanied by severe dysmenorrhea, pain felt an ache of the abdomen, low down, during the first two days of each period. These symptoms have persisted ever since, and in consequence she has found it impossible to work through weakness. During this time she has suffered from headaches, sleeplessness, and giddiness; these have come on at any time, and are quite independent of her periods.

She was admitted to Ward to an account of her gynaecological complaints, but was transferred to Prof. Gulland's ward without any operation being performed. She has been treated as in bed for four weeks, but does not feel any better.

Menstrual History:

The menarche was established at eighteen years.

As a young child she was often troubled with
CASE HISTORY:

Name:-
Age:-
Address:-
Occupation:-
Recommended by:-
Admitted:-

Complaint:-
For the past nine months, pain and excessive loss during menstruation, associated with weakness.
For four years, pain with micturition.
All her life, slight jaundice.

History:
The patient says that all her life she has been a little jaundiced. It varies greatly in degree from time to time, and on occasion is very pronounced; at these times she is apt to feel tired and sick. She never has any discomfort associated with it, nor any pain in the upper abdomen; she has no indigestion or flatulence. In spite of this tinge of yellow in her skin, the patient has always considered herself a normal person. It was only about a year ago that it was suggested to her that she was in any way abnormal.

She has had no bleeding from the nose or mucous membranes (except as noted under menorrhagia) but has always noted a tendency to bleed excessively from any small cuts.

For the last four years the patient has had pain from time to time at the end of micturition; this is seldom very severe. She has not noticed any change in the appearance of her urine.

The complaint for which she eventually sought hospital treatment only came on nine months ago. Two months before she had had an attack of acute bronchitis, which left her weak and run down. About last July she began to have excessive loss at her monthly periods, accompanied by severe dysmenorrhea... pain felt on both sides of the abdomen, low down, during the first two days of each period. These symptoms have persisted ever since, and in consequence she has been unable to work through weakness. During this time too the patient has suffered from headache, sleeplessness, and giddiness; these symptoms come on at any time, and are quite independent of her periods.

She was admitted to Ward 35 on account of her gynaecological complaints, but was transferred to Prof. Gulland's ward without any operation being performed. She has been treated in bed for four weeks, but does not feel any better.

Previous History:
The menarche was established at eighteen years. Prior to this illness there has never been menorrhagia. Nothing to note in the gynaecological history.

Eight years ago patient had a "chill on the kidneys".

As a very young girl she was often troubled with
troubled with sick turns.

Family History: -

Father and mother alive and well.
Two sisters and one brother alive and well.
No member of the family has had any similar trouble.

State on Examination: -

The patient is a cheerful and intelligent young woman. She has rosy cheeks and a high colour, but the brow and neck show a distinct yellow tinge. The skin of the body likewise shows a slight degree of jaundice. She has the look of health, for the jaundice is not very deep, and is easily overlooked. There is no other morbid appearance: no fever; no loss of weight or emaciation; nor is there anaemia.

Examination of Special Systems: -

(a). Gynaecologically there is nothing to note.

(b). Haemopoetic System: - There is no enlargement of lymph-glands. The spleen is much enlarged, and reaches to within one inch of the umbilicus: it is easily palpable, and recognisable by its notch: there is no tympanic band on percussion across the front of the tumour. It is not tender on palpation, and is firm in consistency. On auscultation over it there is no evidence of perisplenitis.

Blood examination: - (17. III. 28).

<table>
<thead>
<tr>
<th>Component</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.B.Cs.</td>
<td>4,230,000</td>
</tr>
<tr>
<td>W.B.Cs.</td>
<td>7,188</td>
</tr>
<tr>
<td>Hb.</td>
<td>80%</td>
</tr>
<tr>
<td>C.I.</td>
<td>.0 95</td>
</tr>
<tr>
<td>Platelets</td>
<td>141,000</td>
</tr>
</tbody>
</table>

Differential White Count:

- Polymorphs: 69%
- Large lymphocytes: 19%
- Small lymphocytes: 9%
- Eosinophils: 3%

Fragility of red cells: - The red cells are haemolysed by a dilution of 0.5% saline.

The patient's blood was 'typed' in the event of a transfusion being necessary. Her blood is Type 1 (Mosso's Classification) Her Brother's (Robt. Taylor) is group 2, but agglutinates with the direct test. Her friend, Mr. Joe Anthony's blood is group 4, and the direct test shows no agglutination.

The patient's bleeding time, as tested by a needle puncture in the lobe of the ear, and observations made at 15 second intervals, is 2½ minutes.

The coagulation time, as tested by means of capillary tubes kept at blood temperature and tested at 15 second intervals, is 1 minute 45 seconds.

(c). Cardiovascular System: - Radial pulse regular in time and force: rate 80/minute: of good tension: radial wall not palpable. Examination of the heart shows some enlargement, the apex being in the
in the midclavicular line in the 5th space. There is a soft systolic murmur in the mitral area. Other sounds all closed.

(d). Respiratory System: There is no cough or other symptom referable to this system. Examination of the lungs purely negative.

(e). Alimentary System: No history of indigestion; no flatulence. Appetite good. On examination, the tongue is clean and moist. The teeth are artificial. The abdomen is well covered with subcutaneous fat; moves easily and regularly; no cutaneous hyperesthesia, or rigidity. There is slight tenderness over the caecum, and also over the 'gall-bladder point', midway between the umbilicus and the tip of the ninth costal cartilage. The spleen is definitely enlarged as noted supra. The color of the stools is normal.


(g). Nervous System: Nothing to note.

PROVISIONAL DIAGNOSIS: Acholuric Jaundice: probably the congenital form of Minkowski.

Treatment: SPLENECTOMY.

Operation by Prof. Wilkie, 19.III.28.

The left rectus incision was used; length about 7 inches, the lower end being at the level of the umbilicus. The vessels cut were caught and tied immediately. The intercostal nerve and the muscles to the left of the incision were infiltrated with % novocain. The muscle was split, the posterior wall of the rectus sheath divided, and the peritoneum picked up and incised between forceps, the incision being extended with scissors.

The abdomen having thus been opened, the spleen was found to be very large. There were no adhesions, but the superior pole was held down by undue shortness of the lienorenal ligament. The gastro-splenic omentum was ligated and divided, and also the lienorenal ligament, in which the splenic artery was taken separately. It was found that about 2 inches of the tail end of the pancreas ran up to the hilum of the spleen in the lienorenal ligament, and this had to be separated before the splenic veins were ligated. There were two accessory spleniculi, which were removed: a lymph gland was also removed for culture.

The gall bladder was examined, and was found to be normal...no thickening, and no stones. Stomach, duodenum, and appendix all healthy. Caecum fixed. All pelvic organs normal. The kidneys were mobile but not enlarged. The liver showed some mottling, but no gross abnormality.

The wound was closed in layers in the usual way, using catgut sutures, a "through and through" series of silk worm gut sutures having first been passed and left untied till the skin was closed. The deep sutures were then tied over a roll of gauze...an anchored dressing.
4.
The spleen as removed weighed 1 lb. 6½ oz. Portions were sent for examination, for animal experiments, and the gland was sent for culture. See page 6.

Pre- and Post-operative Treatment: Progress Notes:

In accordance with modern practice the preoperative treatment was simple...a dose of castor oil the day before operation, and a soap and water enema during the night before. Immediately before operation 1/100th of a grain of atropin sulphate was given hypodermically. The question of a possible blood transfusion had been foreseen, and the patient's blood typed and a suitable donor found.

On returning to bed after the operation the patient was in very good condition: there had been little haemorrhage, and there was no apparent shock. A hypodermic injection of heroin (1/12th.) was given just as she was coming out of the anaesthetic. This was repeated in the evening, at midnight, and early in the morning.

20. III. 28: Patient was very uncomfortable, and had a good deal of sickness. A flatus enema was given (Glycerin and Mag. Sulph. aq. j in water, oz. vj.), and the heroin repeated at night. Food nil: water in sips as required.


22. III. 28: Much better. S. & W. enema in the morning. Food: a gradual return to normal diet now started, beginning with milk, tea, and bread.

23. III. 28: Steady improvement; colour better. From now on, cascara given p.n.r.

27. III. 28: Jaundice now hardly perceptible.

30. III. 28: Stitches out.

31. III. 28: Patient got up.

2. IV. 28: Discharged from hospital.

It was noted that the temperature after operation reached a maximum of 99.6 deg. The recovery was uninterrupted.

Details of the blood changes after operation will be found on p. 5.

The patient reported on 2. V. 28: She has no jaundice, and feels very well. A fragility test was done, with a result showing greatly increased resistance...no haemolysis above 0.3%
### Record of Blood counts after operation:

<table>
<thead>
<tr>
<th>Date</th>
<th>W.B.C.</th>
<th>Platelets</th>
<th>Bleeding Time</th>
<th>Coagulation Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>20.III.28</td>
<td>13,590</td>
<td>218,900</td>
<td>2 min</td>
<td></td>
</tr>
<tr>
<td>22.III.28</td>
<td>14,140</td>
<td>170,000</td>
<td>2 min</td>
<td>1 min 45 sec</td>
</tr>
<tr>
<td>24.III.28</td>
<td>3,360</td>
<td>12,000</td>
<td>2 min</td>
<td></td>
</tr>
<tr>
<td>27.III.28</td>
<td>9,400</td>
<td>212,000</td>
<td>1 min 50 sec</td>
<td>1 min 30 sec</td>
</tr>
<tr>
<td>29.III.28</td>
<td>4,200</td>
<td>15,600</td>
<td>2 min</td>
<td></td>
</tr>
<tr>
<td>2.IV.28</td>
<td>4,120</td>
<td>15,000</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Differential count:
- Polymorphs: 70%
- Sm. Lymph's: 14%
- Lg. Lymph's: 15%
- Eosinophils: 1%
- Basophils: 1%

Fragility test: Haemolysis occurred with 0.5% saline. Two controls were done, and in these haemolysis occurred between 0.5% and 0.55%.

Reported: Fragility test showed no haemolysis above 0.3% of saline.
Biochemist's Report:

A duodenal tube was passed in the usual way... lubricated with glycerin, swallowed by the patient, passed to the stomach and left there for some hours till it passed into the duodenum; the patient lay on her right side throughout to facilitate the passage; the fact that the duodenum had been reached was tested by the aspiration of alkaline fluid. Irrigation with 20% Mag. Sulph. produced a profuse flow of bile: a sample was sent for analysis. The report was to the effect that Urobilin and Urobilinogen were both absent to spectroscopic tests while the presence of both Bilirubin and Biliverdin was detected by various qualitative tests. Analyst: Mr. C.P. Stewart.

Pathologist's Report:

"Bacteriologically: no growth from the splenic gland.

Histologically: (spleen)... capsule slightly thickened. Marked congestion of the splenic tissue. No gross changes seen".

Sgd. K. Patterson Brown.

Animal Experiments:

It was noted supra that in intimate relation with the excise spleen there were two spleniculi. One of these was used as splenic tissue in the following experiments.

The spleen tissue was minced and made into an emulsion with saline.

Mr. Illingworth then performed the following operation on two rabbits anaesthetised with ether. (The skin of the abdomen had previously been shaved and painted with iodine). The abdomen was opened, and the spleen excised. An injection was then given into the spleen of the emulsion of human spleen from the case M.T., the dose for each being equivalent to 0.02 grammes of splenic tissue. The animal's abdomen was then closed.

Two series of tubes had been prepared, containing saline in different dilutions. Blood was obtained from each rabbit by cardiac puncture, and two drops placed in each tube of the series, i.e. an ordinary fragility test was carried out on each animal.

The results were read 24 hours later... haemolysis in each case with saline of concentration of 0.5%.

The fragility test was repeated from time to time, as under:

26. VII. 28: Both series, haemolysis with 0.5%
2. IV. 28: Both series, haemolysis with 0.5%
8. V. 28: No 1... haemolysis with 0.6%, and trace with 0.7%.
No 2... haemolysis with 0.5%.
4. V. 28: No 1... haemolysis with 0.5%
No 2... no haemolysis with concentration above 0.5%.
March 19 20 21 22 23 24 25 26 27 28 29 30 31

Operation

Case of Acute Jaundice, healed by Splenectomy

Blood Platelets
Thousands per c.mm.

Red Blood Corpuscles
per c.mm.

White Blood Corpuscles
per c.mm.

Mary Taylor aged 28

Discharged from Hospital

R.B.C. W.B.C.
Pathology:—So far as our methods of investigation carry us, the central feature of this disease is the increased fragility of the red blood corpuscles, as tested by their resistance to the osmotic effects of saline solutions of varying concentrations. As to the cause of this fragility, opinions differ.

In one view the fragility is regarded as primary; there is congenital (or acquired) malformation of the red cells, which are therefore destroyed in great numbers by an organ whose normal function it is so to eliminate all damaged or imperfect cells. In support of this view we have the statement that the gall stones so frequently accompanying the condition are often totally devoid of cholesterol. Arguing perhaps on slender grounds, some pathological authorities attribute the fragility to a lack of cholesterol in the red cell envelope.

That the primary fault lies at the door of the spleen, on the other hand, has been asserted by some pathologists, Minkowski in particular: the changes he says are due to increased or exaggerated haemolytic activity on its part, a condition analogous to the increased platelet destruction that results in purpura haemorrhagica in modern conceptions. In this view the lowered resistance of the cells is difficult to explain. It is true that in cases after splenectomy for other conditions the resistance of the red cells rises; in animals splenectomised experimentally the action of known haemolytic substances is subsequently more difficult to elicit. In this way we are led to assume a sensitising action of the organ, rendering the red cells liable to an unduly early advent of the doom that normally is reserved for the effete members of the circulating host.

Discussion of the fragility and its causation does not by any means exhaust the difficulties in the pathology of the condition. Anaemia, secondary in type of blood picture...microcytic, with low colour index and poor haemoglobin...is easy to understand, in view of the foregoing paragraphs. Excessive destruction implies excessive, hasty, imperfect formation. But the jaundice is not quite so simple.

In the generally accepted view, the jaundice is due to loading of the portal blood with more blood-derived pigments than the liver can deal with adequately: the passage of the excess into the circulating blood results in jaundice; there being no gross obstruction to the flow of bile into the intestine there is no lack of bile pigment in the faeces: and the normal colouration of the urine is preserved, probably a matter of concentration, since the pigments are in the blood and presumably excreted by the healthy kidneys.

The results of the van den Bergh's reaction and the modern conceptions of hepatic physiology and pathology render the problem more complicated. The essence of the reaction is that bilirubin in serum exists in a different state when its presence is due to excessive breakdown of red blood cells than when it is due to obstruction. It would point to the existence of a true haematogenous jaundice without liver intervention, a possibility by many denied.

The conception held by Eppinger and others as to the
to the

physiology of the liver cell is that the Kupffer cells elaborate the pigments, which are turned over to the liver cells proper for excretion into the ducts: the bilirubin formed by the Kupffer cell is altered in its passage through the liver cell in some way. Hence we must differentiate the types of bilirubin that are responsible for the two types of reaction in the van den Bergh test: one that is due to bilirubin reabsorbed into the circulation (probably via the lymph vessels and the thoracic duct) after passage through the liver cell to an obstructed duct; the other due to non-absorption of pigment circulating in the portal blood and elaborated presumably by other parts of the reticulo-endothelial system as well as the Kupffer cells, but which never enters the true hepatic cells...an anhepatogenous jaundice.

Stadelmann, quoted by MacCallum and by van Noorden, produced experimental icterus in animals by injection of haemolytic substances. He found that not only was haemolysis produced, but that there was copious secretion of bile thick enough to plug fine ducts; Eppinger in similar work found that minute thrombi plugged the actual bile-capillaries.

A biphasic result as is seen in toxic cases or infective cases would be explicable on the grounds that the bilirubin was partly that which had passed through the liver cells, been excreted, and reabsorbed from such a plugged duct, and partly that which had never been dealt with at all by the poisoned liver cells.

The question opens up the whole of the debatable grounds of liver function in health and disease. An attempt to trace some sort of order in the apparent chaos proved abortive. It was no encouragement to depend on the accuracy of one's sources of information, for example, to find the following passage in van Noorden: "Urobiligenin is lacking in the bile only after complete closure of the ductus choledochus or hepaticus, or after profuse diarrhoea." Reference to page 5 of this case reveals that it was absent here, even on spectroscopic analysis. Reconciliation of the facts of the case with the dogmatic statements of varying authors seems hopeless, and confusing in the extreme.

In discussing the pathology we must recognize the two clinical types: the chronic, mild, congenital, familial and hereditary form described by Minkowski, and the acquired, rather more acute form of Hayem and Widal. Whatever may be their differences clinically, they are alike in this, that removal of the spleen results in complete cure.

In this place too should mention be made of the animal experiments performed in connection with the case. These are still in progress, of course, so that results are not complete, and as yet I am not prepared to draw any conclusions, save the obvious one that as is to be expected in a condition of this indolent, chronic type, the results of animal inoculation as here performed, if they are going eventually to be positive, are in no hurry to do so. The object of the research was to find whether the condition was due to some toxic or irritating substance resident in the affected spleen, and capable of reproducing the disease in animals: or alternatively of producing a reaction in the way of increased resistance in the selected animal...an immune reaction.
Immune reaction. In this connection too must be considered the results of histological examination of the spleen, which failed to reveal any recognisable morbid process, and bacteriological examination of the splenic gland, which was also negative. (See reports, p.6.)

Clinical Features and Diagnosis:— Elaboration in detail of the clinical features of this disease would entail repetition of most of the preceding section on its pathology. These remarks will therefore be restricted to a comparison of the two clinical types mentioned above.

"In the congenital form the patient is icteric rather than ill" is one apt statement on the matter. It is said to be characteristic that advice should be sought on totally different matters... in this case it was for her dysmenorrhoea. Most patients, if they do notice their colour, put it down to an inborn characteristic as unalterable as the colour of their hair, and are said to regard it with as little interest! This congenital form is essentially chronic, and does not apparently shorten life or produce debility: Gulland and Goodall state that several female cases have been the mothers of large families.

In the acquired form the course is more acute, and the outlook less hopeful. Setting in with some severity, often accompanied by fever, the disease progresses in attacks or waves over a series of years, and may end fatally: the patient is really ill, not merely icteric. In each attack there is marked anaemia and jaundice; an increase in the size of liver and spleen; some pyrexia; deeper pigmentation of faeces; and a heavy deposit of urates in the urine.

The differential diagnosis is from... (a) Obstructive jaundice of any type accompanied by anaemia. The acholuric type of the icterus is against this, likewise the normal coloration of the stools... Further no cause of such a jaundice was found.

(b) Pernicious Anaemia, stated by Gulland to be accompanied by definite jaundice in the acuter cases starting about puberty (rare). The microcytic type of the blood, the absence of leucopenia, and the mild nature of the condition suffice to rule out this possibility.

(c) Banti's Disease: here we would have jaundice only in the late stages, along with ascites; there would be extreme anaemia in such a stage, plus a marked leucopenia, and a history of recurrent haemorrhages.

(d) Gaucher's Disease: the patient is rather too old, and there are no liver changes here.

(e) Rare conditions that from the nature of the case are absurd here, though in some cases of the disease they may have to be excluded: such as Congenital Obliteration of the Bile-ducts, seen only in infants, no recorded true case having survived beyond the first year (Thomson). So also Congenital syphilis in infants with jaundice, by no means rare, but usually fatal in infancy.

The undue fragility of the red corpuscles, plus the enlarged spleen and the acholuric icterus put the diagnosis beyond all doubt.

As to the exact type of the disease here, though no familial history is available, the duration of the case from early life, and the quiet non-virulent nature of the condition render it
render it/ probable that it belongs to the type of Minkowski...congenital.

Prognosis: If untreated, a persistence of the condition much as it stands at present throughout life. This form is not a life-shortening affection. The accompanying gallstones, if present, may add their peculiar risks to the prognosis.

Treatment: Splenectomy, as already stated, definitely results in clinical cure. By some it is stated that the corpuscles regain their normal resistance (as here), by others this is denied: but in all cases the jaundice clears up within a week, permanently. The result is as good in the acquired as in the congenital form.

The operation is not complicated by the risks that accompany that for Banti's Disease, and operative mortality is so very low as to justify splenectomy in all cases, even for merely cosmetic reasons.
APPENDIX TO CASES I-III.

Notes on the normal splenic function, and the results of splenectomy:

The functions of the spleen are not yet beyond dispute, but the generally accepted views are (1) that the destruction of effete red blood corpuscles is a normal function liable to pathological exaggeration, as in splenic anaemia, pernicious anaemia, or scholuric jaundice. Normally it merely disintegrates the cells, and they are then dealt with more completely in the liver, by the Kupffer cells there, (See case III, page 3.) (2) It acts as a reservoir for red blood cells and blood. Under the old views (see Burton-Opitz) the smooth muscle in the organ is innervated by fibres coming from the coeliac ganglion, and stimulation of these causes reduction in the size of the spleen. The fibres lie in close association with the splenic artery. More recently there have been performed the experiments of Barcroft, on dogs, where the spleen was transplanted into the subcutaneous tissues without interfering with its pedicle. The effects of exercise or rest on the size of the organ could then be watched with ease. It was found that resting the organ was large, but under exercise it rapidly diminished in size: This is now said to be under the control of the phrenic nerve, Tait of Montreal having demonstrated the fibres of the left phrenic running to the splenic vein, and stimulation of these resulting in contraction of the segment of vein. (3) Phagocytosis of foreign bodies and of bacteria is a function, as shown by injecting an animal with Indian ink till it is black all over; then minutes it is clear again and the liver and spleen are loaded with pigment. (4) It is engaged also in the destruction of the blood platelets, as shown by their increase after removal, with special interest in Pernicious Anaemia...see Case I. Other functions have to do with (5) the maintenance of resistance to infections, probably linked up with phagocytosis, or with lymphocyte production. (6) Sensitisation of the red cells to haemolysis, hence an influence on their fragility...See Case III. (7) Formation of uric acid, detected in the spleen after removal of both kidneys. (8) The formation of white blood cells...in normal cases the splenic blood is said to be richer in white cells than the general circulation: It may have an influence in the production of lymphocytes, but not of polymorph cells in normal cases. In leukaemias stimulation of the spleen results in the liberation of white cells into the circulation, but this is probably analogous to the release of red cells on stimulation, though these are not formed there. (9) In foetal life it is the important haematopoietic organ.

After excision of the organ these functions are of course lost, but other organs may take over such as they are fitted for: e.g., the rest of the reticulo-endothelial system takes over phagocytosis and red cell destruction: the splanchic blood bed is quite an effective blood reservoir. In the healthy animal the effects are in fact trivial. In addition to the blood changes discussed below, there is a lowered resistance to infection, which ought to be kept in mind.

The nature of the blood changes is not beyond dispute. In general there is supposed to be a transient anaemia following the operation, the red count falling to about three millions: there is greater resistance to haemolytic agents, with an increased cholesterol content of the blood: and a post-operative leucocytosis of 30,000 or so, which falls
Appendix 2.

which falls/
in a day or two to 20,000, though a definite polymorph leucocytosis persists for four months. Noguchi is quoted as the authority for a statement that the character of the leucocytosis is lymphocytic for a year, then eosinophilic, and later still normal, but Spence (Brit. Jour. Surg., 1928, XV, 59, 476.) was unable to trace this in the case records at his disposal. He states:
"Splenectomy is followed by an immediate rise of erythrocytes and a "leucocytosis with normal proportion of cells, followed in most "cases by a very gradual fall."

From a comparison of the charts given with these cases here surveyed, which were of course in no case associated with a "normal" spleen, we see the definite postoperative leucocytosis which in one case reached /30,000 but fell to 10,000 in a fortnight, and to under 6000 in three weeks...not a "very gradual fall." As to the character of the leucocytosis, it was in all cases polymorph in character. Further counts over a period of months would be interesting.

In no case was there a reduction in the red count after operation to the 3,000,000 quoted by Noguchi: there was always a slight preliminary fall, never serious, and then a steadily rising count till discharge.

The increase in platelets has already been noted (Cases I and II) and occurred also in another case of splenectomy for Banti's disease, where it reached 500,000 per c.mm.

Notes on a case of Splenectomy for Traumatic Rupture:-

A youth aged 20 was admitted as an emergency case in February 1928, to Ward 13. He was suffering from concussion sustained through a 20-foot fall from some scaffolding. In the course of the evening, 10 hours after the accident, he recovered consciousness, and complained of acute spasmodic pain in the tip of the left shoulder, the diaphragmatic type. Pulse and temperature were fairly satisfactory, and not like those of bad internal haemorrhage, but in view of the persistence of the paroxysms laparotomy was performed. The spleen was found to be torn, and there had been an escape of blood into the abdominal cavity. As sutures consistently cut through the soft splenic tissue, excision was done. The patient did not take the anaesthetic well. Some days later, for purposes of having a normal "control" in this series his white count was done, and was found to be nearly 40,000; in view of this and of the clinical picture an intercurrent complication was suspected, which was in fact the case, and which in conjunction with the head injury (found p.m. to be fracture of the orbital plate with laceration of the brain) proved fatal.

The case is quoted (a)...to exemplify one of the indications for splenectomy. (b)...to draw attention to the type of pain that occurs in these cases, and also in some cases of splenic infarct. It is due either to diaphragmatic irritation, or to irritation of those twigs of the phrenic nerve that run to the splenic vein. In either case the pain is referred by the phrenic to the fourth cervical segment, and so to the twigs of the supraclavicular nerves that run to the skin over the shoulder.
This added indication for the operation serves as a link to connect with a list of the indications for splenectomy: these are:

1) Cases of Banti's Disease, and von Jakisch's Disease.
2) Gaucher's Disease.
3) Acholuric Jaundice.
4) Purpura Haemorrhagica.
5) Traumatic rupture (partial excision the best practice)
6) Rarely, for caseating tuberculous disease.
7) Rarely, for syphilis resistant to treatment.
8) Rarely, for pernicious anaemia in young cases: not routine treatment.
CASE No 4.

ROBERT DUNCAN, aged 41 years.

EXCISION OF THE KNEE for TUBERCULOUS DISEASE.

CASE HISTORY:

Name: Robert Duncan.
Age: 41 years.
Address: 29, Alexander Street, Kirkaldy.
Occupation: Confectioner.
Recommended by: Dr. Maxwell, Kirkaldy.
Admitted: 28th Jan.

Complaint: "Weakness in the left knee", duration ten months.

About ten months ago the patient noticed that on running upstairs he had a slight pain just above the left knee. Later on he noticed a low state of health at this time, as he became rapidly worse so that in a fortnight he had to consult his doctor.

At this time the pain was pretty severe, and the joint was swollen. He was treated for "synovitis" with local applications, etc., but did not clear up as he was ordered, believing he "could not spare the time".

Three months later he went to a "bone-setter" who stated that the knee-cap was out of place, and was subjected to forcible manipulations which were very painful both at the time and later.

As the condition was steadily getting worse the advice of his doctor and Mr. Jardine at Kirkaldy, the joint was X-rayed, and immobilization in plaster suggested as the best treatment. This was done.

He took a holiday for a week hoping to derive some benefit, but little good resulted. He then consulted another confectioner who treated him with massage and electricity for three months, but was not helped by the forcible manipulations of the above. No improvement resulted, and eventually the knee "refused to carry him". All this time it is to be noted he had been going about his work as usual, and was at that which necessitated a good deal of standing.

He consulted another doctor; the application of "Ice" and blisters, and immobilization in plaster. He was seen by Prof. Gollan, and seen as an outpatient by Prof. Valkie, and admitted 28th Jan., January, 1923, to Ward 19.

Patient had noticed that the pain was always worse just after resting, say in a chair. It was worse after getting up from a rest before he was able to use the limb freely. Lately, too, the pain had become troublesome at night, especially just after he had fallen asleep. It used to wake him then with a sudden start.

Apart from the local condition in the knee, he has felt in every way fit for work, and complains of no other symptoms.

Previous Illnesses: Until four years ago the patient had always been in healthy man; at that time he had a bad attack of pneumonia, diabetes, complications and sequelae. He had swelling of the body and legs for eight months after this attack, and was treated in the R.I.E.

There is an history of previous pneumonia; of illness at the age of 10.

He suffered a severe attack in his back, which left him with no further symptoms.
History:-

About ten months ago the patient noticed that on running upstairs or on similar exertion he had a slight pain just above both knees. He attributed this to his low state of health at the time, as he was still convalescing from a serious illness (vide infra). However, while his right knee speedily returned to normal, his left became rapidly worse, so that in a fortnight he had to consult his doctor. At this time the pain was pretty severe, and the joint much swollen. He was treated for "synovitis" with iodine applications, etc., but did not rest as he was ordered, believing he "could not spare the time".

Three months later he went to a "bone-setter" who stated that the knee-cap was out of place, and who subjected the joint to forcible manipulations which were very painful both at the time and later.

As the condition was steadily getting worse the patient on the advice of his doctor saw Mr. Jardine at Kirkaldy: the joint was X-rayed, and immobilisation in a plaster case suggested as the best treatment. This advice was refused.

He took a holiday for a week, hoping to derive some benefit, but little good resulted. He then consulted another bonesetter, who treated him with massage and electricity for three months, but who did not resort to the forcible manipulations of the other. No improvement resulted, and eventually the knee "refused to carry him". All this time it is to be noted, he had been going about his work as usual, and work at that which necessitated a good deal of standing.

He called in another doctor, who advised rest, the application of 'Iodex' and blisters, and immobilisation in plaster. He was sent to the R.I.E., and seen as an outpatient by Professor Wilkie, and admitted on 25th, January, 1928, to Ward 13.

Patient had noticed that the pain was always worst just after resting, say in a chair. It was some time after getting up from such a rest before he could use the limb freely. Lately, too, the pain had become to trouble him more at night, especially just after he had fallen asleep. It used to wake him then with a sudden start.

Apart from the local condition in the knee, he has felt in every way fit for work, and complains of no other symptoms.

Previous Illnesses:-

Up till four years ago the patient had always been a healthy man; at that time he had a bad attack of pneumonia, with it seems complications and sequelae. He had swelling of the body and legs for eight months after this attack, and was treated in the R.I.E. by Prof. Gulland, and Mr. Wade, in 1925.

There is no history of previous pleurisy: of "chest troubles"; or of glandular swellings.

He suffered a severe wrench to his back, which might also
might also have affected his legs, just before he took the pneumonia.

Family History:-- The patient is a married man, with four children: all, he says, are alive and healthy. Wise healthy.

State on Examination:-- Patient is a fairly healthy looking man of middle age: rather thin and of poor colour, but with no marked wasting or cachexia. There is no jaundice, ascites, or oedema: a slight degree of anaemia is present.

Height 5' 4½": weight about 9 stones.

Local Examination:-- The left knee is seen to be held rigid and very slightly flexed. The joint and the tissues around it appear to be swollen, and this is accentuated by the marked wasting of the extensors of the thigh on that side. The skin over the joint is glazed and dry, but does not show reddening, nor any breach in its surface. On palpation the temperature of the skin over the joint is distinctly higher than that of the rest of the limb. The periarticular tissues are felt to be of a doughy consistence, and the synovial membrane of the joint is distinctly thickened. There is but little fluid in the joint. There is great tenderness on palpating all along the line of the joint.

Movements:-- the patient will not voluntarily perform any movement with the joint, and passive movements are not possible on account of the extreme pain when these are attempted. Any jarring of the limb causes extreme pain.

Examination of the hip joint revealed nothing abnormal there.


Examination of Special Systems:--

(a). Respiratory System:-- Careful examination revealed no evidences of pulmonary disease.
(b). Circulatory System:-- pulse regular in time and force: rate 80/min: radial wall not palpable: blood pressure not increased.
(c). Urinary System:-- Physical examination negative: urine contains no albumen or sugar.
(d). Alimentary and Nervous Systems:-- N.A.D.

DIAGNOSIS:-- Tuberculous arthritis of the left knee, of the type known clinically as white swelling. It is probable that some ulceration of cartilage has occurred. The patient is otherwise in very satisfactory condition.

PROGNOSIS:-- See commentary.

TREATMENT:-- Excision of the joint.

Preoperative treatment:--
Preoperative Treatment:—

**General...** Ol. Ricin., oz. jss, the day before operation, and a soap and water enema the night before operation. Atropin. Sulphate, gr. 1/100th., half an hour before anaesthesia was commenced.

**Local...** The limb was shaved from ankle to hip two nights before operation: the skin was cleansed with spirit, and ether, and dressed with tincture of iodine (2½ %). The limb was wrapped in a sterile square and bandaged. The preparation with ether and iodine was repeated the night before operation.

**Operation:** (27.1.28). Surgeon: Mr. Cochrane.

Anaesthesia was induced with ethyl chloride, and maintained with ether. The operation was performed on a Halyfax table, to permit of easy application of the plaster casing at the end of the operation without having to move the patient about and so risk displacement of the bones.

The curved U-type Textor incision was used with its limbs over the femoral condyles and the convexity over the patellar ligament about its midpoint. Ready access was obtained by dissecting up the upper skin flap, and dividing the ligamentum patellae. The ligament and the patella were retracted upwards, and the joint cavity laid open for inspection. There was evident synovitis of the joint, but little fluid was present. The synovial membrane was thickened and hypertrophied, and studded with little tubercules. The synovial membrane was spreading as a pannus over the edges of the articular cartilages, and below this pannus there was molecular destruction of the cartilage...commencing ulceration.

The periarticular tissues and the synovial membrane were carefully dissected away, beginning with those most easily reached from the front of the joint...the lig. mucosum, the retro-patellar pad of fat, and the suprapatellar pouch. The anterior attachments of the menisci and the attachments of the synovial membrane to the head of the tibia were next cleared, an assistant meanwhile holding the joint in the attitude of moderate flexion. The cruciate ligaments having been divided, the joint was fully flexed, and the periarticular tissues cleared from the bones in the region of the joint. The saw was then used to remove the articular surfaces from the femur and the tibia, along with about a quarter of an inch of the adjoining bone: the precautions referred to in the summary were carefully observed (q.v.). The articular surface was likewise removed from the patella, with saw and gouge. The limb was straightened, and the raw surfaces of femur and tibia apposed, to make sure that the true axis of the limb had been maintained.

The periarticular tissues in the posterior aspect of the joint were then removed, the joint being fully flexed, and the lower end of the femur pulled as far as possible away from the tibia by an assistant to give adequate access.

The whole of the extent of the synovial membrane having been carefully dissected away, and the periarticular tissues removed with special care on the posterior and less easily accessible parts of the wound, the leg was straightened and the raw surfaces again apposed. The alignment of the limb was given special attention, and this having been secured an assistant was detailed to maintain it. Two short vertical incisions were made in the skin over the head of the tibia, about three inches from the sawn edge, on the anterolateral aspects of the bone. Two long excision pins were then driven in with gentle taps of a mallet in a direction upward and backward, to reach the lower end of the lemur, the axis of the limb being still carefully maintained.
The quadriceps tendon was sutured down into place again with catgut, and then the skin wound closed with interrupted S.W.G. sutures. It will be noted that no drain was inserted.

Antiseptic dry dressing was applied, and the leg wrapped in strips of boric lint, extending from foot to hip, and also over the abdomen as far up as the costal margin. Plaster of Paris bandages were then applied wet to the whole leg, and to the hip as a spica. The axis of the leg was always carefully preserved. The final position was full extension at the knee, and moderate abduction at the hip.

Postoperative Treatment and Progress Notes:

On returning to bed the limb was placed on a mackintosh sheet, and left exposed to the air to dry. Heroin was given, gr.1/12h as the patient showed signs of coming out of the anaesthetic.

The general lines of the postoperative treatment were much as usual...heroin as required; postoperative dose on the third day; etc. The only point of note was the amount of pain suffered during the first twenty-four hours or so, which necessitated heroin to be given five times in the first two days.

Progress thereafter was satisfactory, till on 18.II.28: a window was cut in the plaster, the stitches taken out, and the excision pins removed. These latter came out quite easily and with little or no pain, being by this time fairly loose owing to the absorption of the surrounding bone. 20.II.28: The patient was discharged from hospital on this, the twenty-fifth day after operation.


Reported on 18.IV.28: feeling very well: the plaster was removed, and a light posterior splint was applied. He was provided with suitable crutches, and allowed to use the limb for weight-bearing. Arrangements were made for him to have daily massage at home.
There are many points of interest in this case, which I think will be best considered under the heads of (a), Pathology, (b), Diagnosis, (c) the lines of possible Treatment, (d) the special points in the operation that was done here, and (e) the after-treatment and prognosis of such a case. Before taking up these in detail, however, I think that there is a point in the history worthy of at least passing reference. I refer to the peculiar mental attitude displayed by the patient, who refused treatment by a skilled medical man on the grounds of expediency, and yet submitted first to the none too gentle 'manipulations' of one professed bonesetter, and later to three months tedious treatment from another of that ilk. Not the least of the difficulties that confront the orthopaedist is this disinclination or refusal of many laymen to endure at the hands of a skilled surgeon what they submit to with alacrity from persons of to say the least doubtful ability.

Pathology:—This is a case of articular tuberculosis primary in the synovial membrane. It will be noted that no bony focus was discovered at operation, nor was any suggested in the X-rays. In a person of this age of course we expect a primary joint affection, since the vascular distribution is different from that in the child. Further, in the knee the primary synovial form is the commoner, as contrasted with the hip for example, and this is probably due in turn to the difference in the relation of the joint capsule to the metaphysis in the two joints. In the hip Babcock's triangle is a fertile primary source of bone tubercle, which is very liable to spread to the adjacent joint cavity, in the lower end of the femur disease starting in the metaphysis is more likely to erupt on the posterior surface of the bone, away from the synovial membrane, than on the anterior where it might infect the suprapatellar pouch, owing to the greater thickness of the bone on the anterior aspect. In this case the sole bone change demonstrable was in fact a sclerosis of the lower end of the femur, a reaction to the adjacent irritant.

The clinical type of the disease was that of white swelling: pathologically this is characterised by a myxomatous degeneration of the structures surrounding the affected joint. Ligaments and other tissues around the joint are swollen and largely replaced by a low grade connective tissue. The synovial membrane is also swollen and likewise largely replaced by tuberculous granulation tissue, in which characteristic tubercles are found. The tendency of the exuberant synovial membrane to spread over the peripheral parts of the articular cartilage is noteworthy, for this it is that leads to the occurrence of 'perichondral ulceration'. This is quite distinct from the other exfoliative type of ulceration of the cartilage seen in acute cases, where large areas of the central parts of the cartilage are undermined and cut off from their nourishment, and die en masse under the action of the toxins from the local seat of disease.

It will be noted that, as usual in this type, there was but little fluid in the joint, but the cavity was filled by exuberant granulation tissue, pale, unhealthy, tuberculous.

The changes in the bones in this case are solely due to toxic effects....an osteoporosis, which may have been intensified by prolonged disuse of the limb. The bones showed no actual foci of the...
of the disease.

The muscles acting on the joint are also wasted, more than can be accounted for by mere disease; it is due also to toxic action: actual foci in the muscles are of course excessively rare in tuberculosis.

There was no caseation in any part of the diseased area in this case. Such a case left untreated would probably go on to extensive destruction of cartilage, caseation, and the formation of an abscess containing not true pus but cheesy, caseating material. Such an abscess pointing and bursting through the skin would of a certainty become infected with secondary invaders, and the chronic suppurative ensuing would lead to grave toxic effects throughout the body, and even to amyloid disease. At the best, should the disease quieten down under the effects of rest or improved hygienic conditions, the end result would be a fibrous ankylosis of the joint, a smouldering volcano always ready to erupt when bodily resistance was low.

**Differential Diagnosis:**

In the first place the movements of the hip joint were tested, and disease of that joint excluded. It is a well-recognised condition to have pain and disability due to hip disease referred to either knee of spine by the patient.

The chronicity of the affection serves at once to rule out such acute affections as arthritis of pyogenic, rheumatic, acute gonococcal, or similar origin. Purely degenerative lesions like Charcot's joint could similarly at once be excluded.

The conditions remaining to be excluded are roughly five in number:

1. A possible internal derangement of the knee: this is excluded by the negative history as to trauma, locking, localised tenderness, and by the absence of hydrops. Nor does one get such changes in the periarticular tissues (apart from wasting of the quadriceps) in that condition.

2. Rheumatoid Arthritis, i.e., arthritis deformans, of the atrophic type, is usual multiple and symmetrical in its lesions (as this seemed to be in onset). But it is associated with special constitutional symptoms, with hydrops, and with a tendency to progress by exacerbations.

3. Osteoarthritis, or arthritis deformans of the proliferative type though monoarticular and associated with much muscle wasting is associated with hypertrophic bony changes that are quite absent here. Further, movement through a limited range is possible till very late in the disease, while ankylosis and fixation by taut muscles are not gross features if the condition.

4. Gonococcal arthritis: of the more chronic type is fairly common: but there is no history here of the early polyarticular involvement that usually precedes the affection of one large joint.

5. Tertiary syphilitic arthritis of the synovial form is rare, but is clinically indistinguishable, practically speaking: in France it is known as "syphilitic white swelling." Its rarity, and the all too common incidence of the tuberculous type, would almost be enough to rule it out of count in clinical, as apart from academic, work.

So much for purely negative points: as opposed to these we have the fact that the clinical features fit in exactly with those of tuberculous disease, of the particular type specified.
Lines of possible Treatment: - In this, as in any case, there are two. The pros and cons of each will now be discussed.

Conservative treatment means at least two years immobilisation, with at the end of this period only a problematical fibrous ankylosis. The optimum result that could be hoped for is a stiff joint in good position. Less successful treatment would fail to secure the good position. And there are the added risks of recurrence in the future of concurrent complications such as abscess, and sinus formation, or even of such general conditions as amyloid disease, generalised tuberculosis, or tuberculous meningitis.

Operative treatment at the best means eradication of the disease at once: bony ankylosis in 3-6 months, and functional use of the limb in nine months. It too can only result in a permanently stiff joint. Its disadvantages are...if badly done the disease may not fully be eradicated; the limb may not be put up in good position; there is inevitably ⅛" of shortening, which is really no disadvantage with a stiff knee joint and healthy pelvic articulations; and there are the usual operation and anaesthetic risks. All, or practically all, of these possible undesirable eventuations are under the direct control of the operating surgeon, and it is on his skill and care that the end-result will ultimately depend.

In this case the patient is well over the optimum age at which conservative treatment yields its best results. It is a clinical maxim that over forty conservative treatment is seldom satisfactory. Further he is anxious to get back to work as soon as possible: and he is in quite good general condition, and well able to withstand the radical operation of excision. The line of treatment is therefore clear.

Special Points in the Operation: - In the actual technique of the operation there are about five points that require special attention if a satisfactory result is to be obtained from the curative and from the functional points of view. But as a first requisite to the success of the operation the intelligent cooperation of at least two assistants with the operating surgeon is essential. Their help is needed in the achievement of the "team-work" that can alone give consistently good results.

The special points on which emphasis should be laid are:

(1). The complete eradication of the diseased tissue; this is especially important and difficult posteriorly, around the tendon of the popliteus, and in the recesses of the joint-cavity.

(2). Complete arrest of all haemorrhage is a sine qua non to permit of closing the wound without drainage.

(3). Careful section of the bones, so that the axis of the leg is preserved.

(4). Maintenance of the axis of the leg during subsequent manipulations such as the insertion of pins, or application of plaster.

(5). Care to immobilise the hip, pelvis, knee, and ankle joints in the plaster casing as applied.

After Treatment: - Preparatory: - After treatment an extended under "co-operation" treatment is required. Even a satisfactory result will not mean a full return to activity. Patience limits in
CASE NO. 5.

JOHN RIORDAN, aet 35 years.

CASE OF HEAD INJURY, WITH BASAL FRACTURE AND CONCUSSION.
CASE HISTORY:

Name: John Riordan.
Age: 35 years.
Address: 15a, Buccleuch Place, Edinburgh.
Occupation: Postman.
Recommended by: S.O.P.D.
Admitted: 10.1.28.

History: The patient was picked up in the street after being knocked down by a tramcar; was brought to the Infirmary in an ambulance; and after examination was admitted to Ward 13.

Previous Medical History: (Elicited later from relatives). When in the Navy he was treated at Chatham for "neurasthenia". He is not a confirmed alcoholic, but drinks a little. Otherwise nil to note.

State on Examination: The patient was unconscious. He was bleeding freely from the nose, and there were clots in the left external auditory meatus. The right eye and the surrounding tissues were swollen and discoloured by effused blood. By palpation no fracture of the vault of the skull was detected. No other bones were broken and as far as could be elicited by examination of an unconscious patient, there was no paralysis. The plantar reflex was extensor on both sides, but the other reflexes were not affected. The pupils were small, equal, and reacting. It was found that the patient was quite incontinent.

Examination of the chest and abdomen showed nothing of note. There was no abnormal dullness, nor did the general condition of the patient suggest internal haemorrhage.

Blood pressure and pulse readings were taken on admission, and half-hourly thereafter. The first two readings were:
- On admission: B.P. 120/90. Pulse rate 62/minute.
- 30 mins later: B.P. 125/80. Pulse rate 70/minute.

Further readings showed no evidences of rising B.P. or falling pulse rate. There was no starter of respirations on admission or in the first few hours thereafter.

Provisional Diagnosis: Head injury: Concussion: and Fracture of the base of the skull, probably through the left middle fossa and right anterior fossa.

Treatment and Progress Notes: 10.1.28. The patient was admitted about eleven p.m., and was put to bed, the head of the bed being raised on low blocks (4""). He was left as quiet as possible, but became restless and vomited a good deal of material that smelt strongly of alcohol. 11.1.28. Next day the patient was very restless still, and had to be given three doses of morphine (gr. ½) hypodermically. He vomited several times. He was given calomel, gr. iii, and a soap and water enema. A dose of 1500 units of anti-tetanic serum was given intra-muscularly.
Intramuscularly as a prophylactic.

The patient was still only semiconscious, but resisted violently when touched. His pulse to-day was much faster, up to 108/minute.

12.1.28. Condition to-day much worse: he lay on his side, in an attitude of flexion, and was extremely irritable. His pulse at 10 a.m. was charted as "160" and was very difficult to count because of its running nature. In the course of the forenoon he became very pale, and his breathing changed to the Cheyne-Stokes type. His temperature rose to 101.2deg. Unconsciousness persistent. No sedatives given to-day.

At 2.30 p.m. his pulse was 130, his blood-pressure 110/60. 30 c.c. of a 15% hypertonic Sodium Chloride solution were given intravenously very slowly, at 2.30 p.m. The condition of the pulse was carefully watched during the injection, which took about fifteen minutes.

At 3 p.m. his pulse was still 130, his blood-pressure 108/60.

His breathing was much better, his pulse better, and he was much quieter. His pulse rate was 120, and his blood-pressure 98/65. His unconsciousness was not so deep, for he knew when he was being attended to or changed by the nursing staff.

13.1.28. Able to speak a few words more or less coherently to-day. Still incontinent however, and very restless at night. Appears to suffer from persistent headache.

14.1.28. At 2.30 p.m. lumbar puncture was performed. The fluid was under slight tension. The first ten ccs. showed no trace of blood; then a streak of red appeared, and the next five ccs. were collected separately, being very distinctly bloodstained. After about 15 ccs. in all had been withdrawn the fluid became clear again. On allowing to stand for some time the blood gravitated to the bottom of the tubes, and the supernatant fluid was seen to be clear—but of a distinctly yellow tint.

After the lumbar puncture the patient was kept with the head low for some time. He was still very irritable, however, and extremely restless. His condition was not materially altered either way by the operation.

15.1.28. Condition little changed. The patient is now very thin, having lost a great deal of weight during the ten days since admission.

The hypertonic saline was repeated to-day, 30 ccs. of 15% Sod. Chlor. being given intravenously at 3 p.m. In the evening his condition had improved noticeably. He was then able to drink from a feeding cup, but was still very restless. Sedatives discontinued.

20.1.28. Condition little changed. The patient showed slow improvement.

Tonight he answered questions fairly intelligibly, though still somewhat confused.

Intravenous saline (30 ccs. of 15% Sod. Chlor.)
was given at 3.45 p.m.

Pulse rate 74.

At 3.30 p.m. Blood-pressure was 110, systolic.

At 9 p.m. the blood-pressure was 108 systolic.

Pulse rate 72.

24.1.28., et seq. The patient's physical condition continued to show slight improvement, and the danger to life was apparently over. His temperature was now consistently about 97-98 deg.F., his pulse rate normal, and his respirations about 18-20/minute. As the bruising around his eye cleared up, he was seen to have a subconjunctival haemorrhage on the right side, spreading in fan-shaped fashion from the lateral aspect. He is very thin and the skin of the back shows signs of impending bedsores from the continual irritation, the result of his restlessness.

His condition being no longer in the strict sense of the term 'surgical', he was transferred to Ward 28 (Dr. Chalmers Watson) on the medical side on 31.1.28.

Throughout his stay in Ward 13 the patient has been very constipated and has required a daily dose of calomel, Henry's solution, or an enema.

His diet has been 'light', or indeed anything he could be induced to swallow, for feeding him has been difficult.

AFTER HISTORY:- On March 2nd the patient was still an inmate of Ward 28. Physically he is very emaciated, but he has no palsy or other physical trace of his injury beyond some irregularity of the left optic disc, and an extensor Babinski response on both sides. His treatment has been along purely general lines.... diet, aperients, etc,

His mental condition is very interesting however. He is confused as to time, place, and person. His conduct has failed, and many of his acts are those of a lunatic. He has intervals during which he recognises people as for example the house surgeon who was in charge of his case in Ward 13, but general speaking he is quite confused. The present diagnosis is Traumatic Insanity, and he is to be certified, and transferred to Bangour.

The PROGNOSIS is very guarded in view of the history of previous mental instability, and the probability of an alcoholic background.
COMMENTARY ON THE CASE:-

Under this heading I will discuss briefly the differential diagnosis, the probable pathology of the condition, the indications for operation, the rationale of the therapeutic method employed here, and in a few words the possible sequelae of head injury.

Differential Diagnosis:- Under this head must be considered the various conditions that might be present in the case of men brought to hospital having been picked up unconscious in the street. The simple procedure of testing the urine will help to exclude such conditions as Uraemia and Diabetes which may have gone on to coma. Alcoholic poisoning must always be kept in mind, but even in a patient smelling strongly of alcohol one must be chary to diagnose it as the cause of the condition. Opium poisoning likewise should be remembered though rarer. Space forbids the differentiation of these conditions by means of their symptoms and signs: they are mentioned merely because they should flash through the mind of the house surgeon called on to see such a case as this.

Whereas here the man was seen to be knocked down by a tramcar, head injury is of course suggested as the cause of the unconsciousness. We must remember that a cerebral haemorrhage might cause a man to fall in front of a car; an epileptic fit might likewise cause such a fall succeeded by coma. It is important to remember that a trivial knock on the head in a person with a glioma may cause a haemorrhage into the substance of the tumour, with sudden onset of symptoms: such cases are frequently met with in medico-legal practice.

In an unconscious man with supposed head injury, we examine the pupils; the reflexes: the tone of the muscles of the limbs: to rule out paralysis we look for haemorrhage from the eye, ear, or nose, indicating fracture of the base: and we palpate the skull for fracture of the vault. The other bones are examined to exclude fractures, especially perhaps the ribs, where fracture may not be very obvious, and may yet be serious from damage to the underlying lung. The chest and abdomen are examined...abnormal or shifting dullness on percussion suggests free blood in the pleura or peritoneal cavity, from say ruptured lung, spleen, or kidney: diminished liver dullness with a tympanitis note suggests the presence of gas from a ruptured stomach. The general condition of the patient as to pulse, pallor, temperature, etc., helps to rule out or to suggest internal haemorrhage.

The diagnosis of the particular type of head injury, concussion as opposed to cerebral compression, laceration of the brain, subdural haemorrhage etc., rests on the symptoms of the case. In this case the blood-pressure and pulse did not suggest 'cerebral compression' had the pulse fallen in rate and become full and bounding, the respiration become stertorous, or the B.P. risen markedly, the onset of this condition would have been diagnosed. Nor were there the signs of spreading localised compression, progressive and successive irritation and paralysis, to indicate a local haemorrhage. The absence of the characteristic 'lucid interval' ruled out extra-dural haemorrhage. The irritable, half-conscious condition characteristic of compression affecting a fairly large area outside the motor zone was not present till some days later when it's onset probably was not due to haemorrhage but to oedema. There was therefore no clinical ground for diagnosing the case as other than one of concussion. There was superadded of course/
course a fracture of the base, but this could for all practical purposes be neglected, as the prognosis in these cases is that of the brain condition, and not that of the fracture.

Pathologically:- The primary condition of the brain at the time of the accident was one of momentary intense cerebral anaemia, producing unconsciousness and the symptoms of cerebral shock or concussion. Minute punctate haemorrhages were probably caused throughout the brain at the same time. Later reactionary oedema set in, and the subsequent rise of intracranial tension caused symptoms of generalised irritation to set in which fitted in exactly with Erichsen’s description as quoted by Thomson and Miles. (See progress notes, 12th January).

The cranium being a rigid closed box, whose contents are tissues, blood, C.S.F., and ‘tissue-fluid’, the equivalent of lymph elsewhere, it is obvious that any increase in one of these contents must be at the expense of the others... as seen in cases of cerebral tumour, haemorrhage, hydrocephalus and oedema.

Up to a point displacement of C.S.F. can compensate for increase in say the ‘tissue-fluid’ in oedema: after this limit is passed the thin-walled veins are compressed, and irritative symptoms develop characteristic of the area of brain involved. Later still complete capillary anaemia with loss of function (unconsciousness or paralysis) occurs.

An understanding of these phenomena is essential to the proper appreciation of the pathology outlined above. It will be noted that the old Edinburgh idea, world wide in its adoption though initiated by Monro Secundus, that the volume of blood in the cranium was always constant, has now been entirely given up.

Many theories are current as to the cause of the fall in B.P. and the other symptoms of concussion. The view of Duret is that wave disturbances of the cerebrospinal fluid are set up by the force of the blow, and undue stimulation of the vital centres in the medulla results.

Treatment:- The question of operation is at once raised. This is called for in all cases of (1) compound or depressed fractures, excluding fractures of the base implicating as here the ear or nose. (2) Cases with signs of localised compression, with the irritative and paralytic signs referred to above. (3) Cases with the early signs of bulbar compression, as evidenced by the pulse etc. (4) Cases with cerebral irritation with high intracranial tension. In clinical terms we may summarise the cases as (a) those with subnormal temperature, rapid pulse, and shallow respirations. (b) Those with temperature that rises steadily, the pulse bounding and slowing, and the breathing stertorous, and (c) those like the last but not so severe. It is a good rule never to operate except on the last type.

On admission therefore there was no indication for operation. The question only arose on the second day, when the symptoms of generalised cerebral irritation were in evidence. Was a decompression advisable?

The patient being literally in extremis, and a most unfavourable subject for a major operation, the administration of hypertonic saline was tried. Its result was dramatic: it acts as a "bloodless/
"bloodless decompression", and in this case at any rate it turned the scale in favour of the patient. Its use rests upon the experimental work of Weed and McKibben of Baltimore on "The Experimental Alteration of the Brain Bulk". (Am. Jour. Phys., 1919, 48, 351.) Reference to their paper revealed that using cats as experimental animals, and 30% Sod. Chlor., or saturated Sod. Bicarb. as hypertonic solutions, they were found that the intravenous injection of strongly hypertonic solutions, which has been found to cause a profound lowering of the pressure of the cerebrospinal fluid, has been observed to produce also a decrease in the bulk of the brain... A marked individual variation to reaction has been found to exist. A change in the distribution of the C.S.F. was demonstrated by these workers by injecting such a hypertonic solution intravenously and then injecting the subarachnoid space with 2 or 3 cc. of an iron solution (Sod. Ferrocyanide and Iron Alum Citrate) Subsequent killing of the animal and fixation of the brain in formalin acidified with HCl resulted in the demonstration of Prussian blue not only in the subarachnoid space but along the perivasculars into the brain substance, reaching to the interfibrous spaces in white, and the pericellular spaces in the grey matter. In subsequent papers, which I have not been able personally to consult, Weed showed that the C.S.F. is absorbed after such injections by the ependyma lining the ventricle. Foley, in the Arch. Surg. for 1923 confirms this, and states that the currents in the C.S.F. are reversed, so that fluid flows from the subarachnoid space into the ventricular system. Experiments with the opened skull in the living animal show that the injection causes the brain to shrink from three to four millimetres below the level of the skull, the maximum effect occurring in from 10 to 30 minutes.

These results are, of course, made use of in cerebral surgery by Harvey Cushing and others: their application to this case is obvious. Such a condition of generalised oedema would seem of all causes of high intracranial tension to be that which would yield most readily to their therapeutic application.

It will be noted from the record of the case that in the time at which the effect on the intracranial tension was at its maximum (i.e. 30 minutes from the time of injection) there was no appreciable effect on pulse or blood-pressure. The fall in both later, as well as the change in respiration is to be attributed to secondary effects from the lowered pressure within the skull. As with many forms of intravenous medication, however, there is a distinct risk of collapse etc., I believe, should the injection be given too rapidly: no toxic effects were noted in this case.

As a therapeutic measure in this case lumbar puncture was not a success, and was not repeated. From the fluid withdrawn we gleaned some confirmation of our diagnosis...it was at first clear, then bloodstained: on settling the supernatant fluid was yellow: these facts lead us to believe that the source of the blood was high up in the cerebrospinal system, probably torn pial vessels in the vicinity of the basal fracture. Had the blood been from a punctured vessel at the site of L.P. the fluid would have been quite colourless on settling.

The rationale of the other details of the treatment are easily disposed of...the head of the bed is kept raised to lessen cerebral congestion which succeeds the initial anaemia, and causes the oedema; the use of morphine needs no explanation; nor does that of aperients
In all cases of concussion, however slight they may seem, it is advocated by Thomson and Miles that the patient be confined to bed for 14-21 days.

Sequelae:- Death may of course occur at the moment of injury, or later from 'compression'. It is with cases that survive that I would deal here.

Persistent headaches for a variable time after the injury are a common sequel. An analogous condition to "railway spine" may develop, affecting higher mental faculties, which must be classed with the neurasthenias.

Epilepsy of the Jacksonian type is a well recognised complication most commonly after depressed fracture, or haemorrhage followed by cyst formation or sclerosis.

The sequel in this case has been Traumatic Insanity. According to Clouston, cases of this disease amount to only 1/3% of all cases of insanity admitted to the Edinburgh Asylum, so it cannot be a common sequel. The prognosis varies with the time that elapses after the injury before the condition develops...the longer the interval, the worse the outlook. In this case, as already discussed, the prognosis is complicated by the neurotic taint, and by the alcoholism.

Meningitis etc., are hardly complications of the brain injury, but of associated skull or scalp lesions. Yet it is worth noting in conclusion that active treatment such as syringing clots out of the meatus is to be avoided in suspected basal fracture, lest infection be conveyed to the cranial contents.