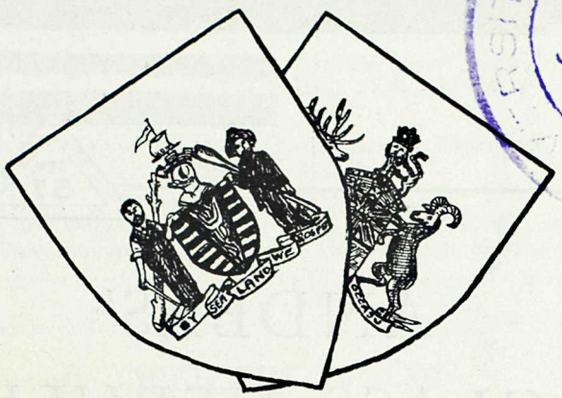
THE BULLETIN



OF THE VANCOUVER MEDICAL ASSOCIATION

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BULLETIN

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Vol. VIII. FEBRUARY, 1932 No. 5

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VANCOUVER HEALTH DEPARTMENT

STATISTICS, DECEMBER, 1931

Total Population—Census, 1931		246,579
Asiatic Population (Estimated)		15,000
	Rate per 1,000 of	Population
Total Deaths	211	10.1
Asiatic Deaths	22	17.3
Deaths—Residents only	187	8.9
Birth Registrations	310	14.8
Male 166		
Female 144		

INFANTILE MORTALITY—

Deaths under one year of age	13
Death Rate—Per 1000 births	41.9
Stillbirths (not included in above)	10

CASES OF CONTAGIOUS DISEASES REPORTED IN CITY

	November, 1931		December, 1931		January 1st to 15th, 1932	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	2	0	0	0	2	0
Scarlet Fever	24	0	20	0	6	0
Diphtheria	15	0	7	1	2	0
Chicken Pox	55	0	38	0	36	0
Measles	68	0	285	0	1277	1
Mumps	24	0	24	0	54	0
Whooping-cough	17	1	21	1	9	1
Typhoid Fever	4	0	1	1	0	0
Paratyphoid	0	0	0	0	0	0
Tuberculosis	97	10	56	21	6	—
Poliomyelitis	1	0	0	0	0	0
Meningitis (Epidemic)	0	0	0	0	1	1
Erysipelas	5	0	3	0	4	0
Encephalitis Lethargica	0	0	0	0	0	0

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VANCOUVER MEDICAL ASSOCIATION

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PROGRAMME OF THE 34th ANNUAL SESSION

GENERAL MEETINGS will be held on the first Tuesday of the month at 8 p.m.

CLINICAL MEETINGS will be held on the third Tuesday of the month at 8 p.m.

Place of meetings will appear on Agenda.

General Meetings will conform to the following order:
8:00 p.m.—Business as per Agenda.
9:00 p.m.—Paper of Evening.

1931.

November 3rd—GENERAL MEETING.
Papers:
Dr. H. A. DesBrisay: "Syphilis in Medical Practice."
Dr. W. T. Lockhart: "Treatment of Syphilis."
D. A. L. CREASE of Essondale will discuss the treatment of Degenerative Types of Neurosyphilis.
Discussion: Dr. J. E. Campbell; Dr. W. L. C. Middleton.

November 17th—CLINICAL MEETING.

December 1st—GENERAL MEETING.
Papers:
Symposium on Fractures to be arranged by Dr. A. B. Schinbein and Dr. D. M. Meekison.
Discussion: Dr. F. P. Patterson; Dr. J. A. West.

December 15th—CLINICAL MEETING.

1932.

January 5th—GENERAL MEETING.
Papers:
Dr. C. S. McKee: "The Interpretation of Blood Pictures."
Dr. Murray McC. Baird: "The Clinical Aspect of Some Blood Diseases."
Discussion: Dr. W. H. Hatfield; Dr. A. Y. McNair.

January 19th—CLINICAL MEETING.

February 2nd—GENERAL MEETING.
Papers:
Dr. H. Dyer: "Tracheotomy in Children."
Dr. C. Graham: "Inflammation of the Accessory Nasal Sinuses in Children."
Dr. E. E. Day: "Indications for Endoscopy."
Discussion: Dr. J. A. Smith; Dr. H. R. Mustard.

February 16th—CLINICAL MEETING.

March 1st—GENERAL MEETING.
The Osler Lecture: Dr. F. P. Patterson.

March 15th—CLINICAL MEETING.

April 5th—GENERAL MEETING
Papers:
Dr. J. W. Thomson: "Emergencies in Abdominal Surgery."
Dr. A. W. Hunter: "Diagnosis and Treatment of Some Urological Emergencies."
Discussion: Dr. G. E. Gillies; Dr. Lee Smith.

April 19th—CLINICAL MEETING.

April 26th—ANNUAL MEETING.

EDITOR'S PAGE

Readers of Kipling's "The Day's Work" will remember one story in this collection called "The Ship that Found Herself". To those who have not read the story, or who, having read, have forgotten it, we commend its perusal. It has a moral for us. The last word in marine architecture. fitted and equipped with all the latest and most expensive gadgets, this ship was yet, at the outset of its first trip, laden, to sea, a mere collection of beams and planks, bolts and rivets—it was not a "ship", with the soul of its own that every lover of ships, or every man who lives and works with them, ascribes to the almost living creature of steel and wood and hemp, that he calls a ship.

And you will remember that its first trip was in midwinter and through howling gales and mountainous seas—and it was all quite new and very distressing to the various *disjecta membra* of the ship, the rivets, and the deckbeams, and the garboard strakes and the capstan and so on—and at first, each one growled at and complained of all the others, for letting the whole load fall on the complainant. Even some of the rivets, finding themselves blamed for all the others' ills, decided they might as well pull out, and give up the battle—but the steam persuaded them to stay. And gradually, and here is the moral, each individual unit found to his pride and exceeding joy, that as long as he did his share loyally, and both supported and trusted the other individual units, everything went beautifully, and the ship rode out the storm and arrived , somewhat dishevelled perhaps, but quite sound, in port. And then the soul of the ship awoke and she was no longer a collection of parts, but a living, breathing whole, one of the community of ships, an entity and a personality, and not merely a machine shop.

Perhaps it needed all the trials and afflictions that have beset us for some time past to awaken us all to the feeling that we are not yet a unit, and that something must be done to make us a single and united body. There is an evident feeling amongst medical men today, that by some means or another, we must in the near future, formulate a plan by which we shall speak with one voice when we meet with our enemy in the gates. There have been too many voices, each one sincere and honest, each one saying what it believes to be true but each with the weakness that comes from a divided allegiance. There has been no authoritative body which could really represent the profession, which could command its undivided loyalty, and speak for it in the full confidence that it really spoke for every one.

And so each part has tended to throw the blame on the other parts. This is not fair, but it is very human. The truth is, that there has been lack of mutual understanding, lack of methods by which we could work smoothly together—overlapping and confusion. Each part has done its best—but was handicapped in the doing. Attempts have been made to work together—but the system is rigid and inelastic and needs recasting .

There is no need for blame. If mistakes have been made they have been honest mistakes, and we all share the responsibility for them. But it is time to reconsider the whole position, and "look to our fences".

To this end, much hard work has been done in the past two or three months, and much more must and will be done. To our readers we would urge that they hold up the hands of those who are working at this, and that they give them their confidence and moral support. So shall we acquire corporate unity, and become a ship that has found herself, and really is a ship. For, to quote the immortal Rudyard once more,

> "The game is more than the players of the game
> And the ship is more than the crew."

* * *

As many of our readers know, Mr. W. G. Hunt, Associate Secretary of the Alberta Medical Association, and Assistant to the Registrar of the Alberta Medical Council visited Vancouver during the week of January 9th—16th.

His visit was made at the invitation of the Constitution Committee of the B. C. Medical Association and while here, he addressed the Executive of that body as well at the Council of the College of Physicians and Surgeons and the members of the Vancouver Medical Association Executive who were connected with the work that has been done with a view to amalgamation of the B. C. Medical Association and Vancouver Medical Association.

For some time it has been felt that as far as this last-named plan was concerned, we had about reached an *impasse*. No plan agreeable to all could be formulated and decision was postponed again and again—the real cause being that no decision could be reached.

For some time, too, it has been felt that our methods of handling medical matters in B. C., are not satisfactory—that our relations with the Government are not as amicable and mutually pleasant as they should be—and that our standing with the public is not at all what it ought to be. There are several factors involved and it is possible perhaps to indicate the main ones.

The administration *and enforcement* of the Medical Act are vested in the Council of the College of Physicians and Surgeons. Violation of the Medical Act must be dealt with by them.

This puts the Council, and indirectly the medical profession, in a most invidious and uncomfortable position. Why, one feels like asking, should violations of the law be dealt with by different people for different Acts? All Acts are Acts of Parliament, whose law officer, the Attorney-General and his department, are empowered to see that they are obeyed and violations of them punished. Why should the Medical Act be an exception.

Inevitably, and we must confess we cannot see how it can be otherwise, the public has come to regard the Medical Act as a fence placed around the medical profession to protect this body. Violations are prosecuted by the Council. Of a certainty, the public, not understanding our position at all, sees in this an endeavor on the part of the profession to maintain for itself a special position of privilege. It need hardly be said that the real purpose of a Medical Act is the protection of the public against ill-trained, incompetent practitioners.

Again, as medical men, we have assumed the position of watchdog of the health of the people. We fight the licensing of "cults" of the half-educated—because we feel so strongly that they are a menace to the health of the community. But is this, again one asks, our business? Is it not the business of the legislature to protect the people's health? Most of us are beginning to think it is. When we oppose these people, our motives are misunderstood, we are thought to be afraid of the inroads they may make on our own livelihood, and we are given no credit for our real motive, which is the one set forth above.

The B. C. Medical Council has been unjustly blamed by some for these happenings. It is quite unfair to attach blame to this body. The members have done their honest best for the profession, and if we now feel that the plan was a mistaken one, the mistake is ours as much as theirs. As a matter of fact, they have gone out of their way to consult with the two Associations, and for the past two or three years there has been as close contact as possible.

Again, there is too much cost attached to organized medicine in this province. This makes it very difficult for many who would like to belong to all the bodies concerned—but cannot afford it. Too, it throws an unfair burden of cost on those who do belong. It has been felt for some time that measures should be devised, whereby, in the first place, every man should pay his fair share towards the expense of the necesary work done on behalf of all—and in the second place, this share should be set at a figure which all could afford.

Lastly, it has been appparent for some time that there has been too much duplication of effort—too much overlapping of various bodies —and coincident with these, unnecessary expense—not only unnecessary but useless and inefficient. The effort has been useless, because not possessed of sufficient authority and force. One single voice, speaking for the whole, would impress the hearer—but there are too many voices, none of them backed by the whole strength of the profession.

In Alberta they are, it would appear, in a fair way to solve these problems. They have done it in what would seem an eminently sensible and fair way. Except for local associations, there is only one taxing body, the Council of the College of Physicians and Surgeons. This body collects, compulsorily, a fee adequate to carry on the work of the profession and allots to the Alberta Medical Association

the money necessary for its work. The work of the province is divided between the Council and the Alberta Medical Association and the Council has representation on the Executive of the latter. There is direct and continuous contact between the Council and the rest of the profession. The Medical Act is still nominally under the control of the Council—but practically they have shifted the administration, as far as violations are concerned, to the shoulders that should bear it—those of the Attorney-General. Lobbying in the halls of the legislature, war against cults, etc., are things of the past. But the Council is the body with authority—and it has teeth and can use them.

The Council of the College of Physicians and Surgeons in B. C. is studying this whole matter closely, as are the Executives of the B. C. Medical Association and the Vancouver Medical Association. Our readers will be kept informed of developments. Meanwhile, it will behoove all of us to consider these matters and acquire all the information possible.

MEETINGS

The General meeting of the Association was held in the Medical-Dental Building Auditorium on January 5th, with an attendance of ninety-seven members. Dr. C. W. Prowd, the President, was in the chair. There were two elections to membership—Dr. E. L. Garner, formerly of Duncan, and Dr. G. F. Amyot, of North Vancouver, both of whom were unanimously elected. A resolution was carried that the Vancouver Medical Association does not endorse the Canadian Medical Indemnity and Health Association and that a letter be sent to every member notifying them of this resolution.

The Cancer Committee presented a synopsis of a communication from Dr. J. S. McEachern of Calgary, outlining a plan of development in Cancer Research under the British Empire Cancer Campaign.

The question of the early closing of gas stations was discussed and protests were voiced against any such move unless adequate provision were made for emergency service.

The papers of the evening were presented by Drs. C. S. McKee and Murray Baird. Dr. McKee spoke on "The Interpretation of the Blood Picture" and Dr. Baird spoke on "The Clinical Aspects of Some Blood Diseases". Drs. Hatfield and McNair opened the discussion.

* * *

The monthly meeting of the Cancer Investigation Committee was held in the Auditorium of the Vancouver General Hospital on Monday, January 18th. Dr. D. F. Busteed gave a summary of the cancer cases reported in 1931 in the City of Vancouver and a number of interesting clinical cases were presented. The attendance was good. Dr. J. J. Mason presided.

* * *

The Clinical Section of the Association held its monthly meeting in the Auditorium, Tenth and Willow, on Tuesday, January 19th, through the courtesy of Dr. A. K. Haywood. An excellent programme

was presented, those showing cases being Drs. Frost, Strong, Hatfield, Leeson and Schinbein. 75 members were present.

* * *

ADDITIONS TO THE LIBRARY

Surgical Clinics of North America, June, August, October, December, 1931.
Deafness and Its Alleviation. V. Nesfield, 1930.
History of the War. Closing Volume, 1931.
Medical Clinics of North America, July, September, November, 1931.
Proctoscopic Examination. Buie, 1931
Renal Lesions in Bright's Disease. Addis & Oliver, 1931
Insanity as a Criminal Defence. Meredith, 1931.
System of Bacteriology. Vol. 9. Technique-Methods, 1931.
Transactions American Proctological Society, 1931.
Cancer of the Rectum. Ernest Miles, 1931.
Home Care of Infant and Child. F. Tisdall, 1931.
Harvey Lectures. Volume for 1930.
Quantitative Clinical Chemistry. Van Slyke & Peters, 1931.
Textbook of Surgery. John Homan, 1931.
Poisons & Poisoners. Thompson, 1931.
Recent Advances in Medicine. Beaumont & Dodds, 1931.
Recent Advances in Pulmonary Tuberculosis. Burrell, 1931.
Diagnosis & Treatment of Brain Tumours. Sachs, 1931.
Injuries to Joints. Sir Robert Jones.
Transactions American Association of G. U. Surgeons. 1931.
Intracranial Pyogenic Dieseases. Logan Turner & Reynolds. 1931.
Fractures and Their Complications. G. E. Wilson, 1930.

THE CLINICAL ASPECTS OF SOME BLOOD DISEASES

By Dr. Murray McC. Baird

I wish to speak to you for a short time about the clinical side of certain conditions mainly affecting the blood, and shall confine my remarks entirely to anaemia because relative frequency gives it a position of major importance. We may for our purposes define anaemia as a condition in which there is a reduction in the number of red cells or in haemoglobin per unit volume of blood, or both, or a reduction in the total volume of blood. I wish first to mention a few general considerations with regard to the recognition of anaemia, and then to consider some of the main clinical types of the disease, going on to a brief review of recent work on the subject and the modern ideas in treatment.

Delivered at a meeting of the Vancouver Medical Association, January, 1932

It is obvious that upon us as clinicians rests the responsibility of recognising anaemia when it exists and in these days of free use of cosmetics by all classes of society, the task is more difficult than ever before. I have known patients to tint even their ears to avoid the pallor which is so often one of the striking symptoms. We must admit too that many pale patients are not anaemic, and the appearance of the patient is not of great help except when the anaemia is extreme. Anybody can recognise the severe type of case, when the patient walks in complaining of general weakness, dyspnoea, palpitation, and indigestion,and shows on examination pallor, oedema of the ankles and functional heart murmurs. There are many milder cases, however, where the patient comes complaining of one prominent sympton to the exclusion of all others. Such symptoms are varied in the extreme, but the common ones are fatigue, indigestion, headache, giddiness, tinnitus, constipation, or attacks of diarrhoea. In such cases the attention is directed elsewhere, and the diagnosis may be missed because we do not think of the blood as a primary cause. Since the appearance of the patient is often of little help, it is necessary for us all, having thought of anaemia, to have some instrument of precision for estimating haemoglobin at least. Many use the Tallqvist Scale and find it satisfactory. Personally, I do not find it sufficiently accurate. Others use a haemoglobinometer of the acid haematin type. The user should be sure that he is not colour-blind, and that his instrument has been standardised. I was sold a haemoglobinometer in Vancouver which gave for my own blood a reading of 120%, which I knew to be false. So one has to beware of standards that fade. In cases of doubt it is obviously best both for doctor and patient to seek the help of the haematologist.

A clinical fact, which we all realise without giving much thought to it, is that anaemia in general is preponderantly a disease of women. This is so true that if we see a young adult male suffering from any considerable degree of anaemia, it is wise to begin at once a search for some serious cause, such as concealed bleeding ,tuberculosis, or malignant endocarditis. This does not apply to young women, however, who are often anaemic without any very patent reason. We should use this fact in our practice to the extent of thinking of the blood always when we have a female patient, particularly a middle-aged woman whose symptoms appear to be a vague, irrevelant and incoherent jumble. A routine haemoglobin estimation in such cases may often be normal, but now and again it will save us the ignominy of a missed diagnosis and the boredom of listening to an oft-repeated tale. Incidentally, it may save the patient years of ill-health and considerable expense.

Clinical Types

For clinical purposes there are still many excellent reasons for dividing anaemias into two main groups, the primary and the secondary. These terms refer to the blood picture only and not to the ætiology. The terms primary, macrocytic, megalocytic, hyperchromic, Addisonian, and pernicious anaemia, are used rather indiscriminately to describe a blood picture where the cells are large and contain more haemoglobin individually than normal, (hence the colour index is high) and where megalo-

cytes or foetal red cells are found in the bone marrow and sometimes in the blood. The terms secondary, microcytic, hypochromic, and chlorotic are used to describe a blood picture where the cells and haemoglobin are reduced but the haemoglobin is reduced more in proportion than the cells, (hence the colour index is low) and where there is no foetal response on the part of the bone marrow.

Primary or pernicious anaemia is not a disease so much as it is a peculiar kind of blood picture. It is found arising by itself or in association with other ostensibly very far removed conditions, such as:-

1. Carcinoma of the stomach.
2. Sprue,
3. Infestation with intestinal parasites.
4. Syphilis.
5. Surgery of the stomach, notably partial gastrectomy and gastro-jejunostomy.
6. Pregnancy.

The anaemia may be profound with red cells numbering as low as one million, before the patient comes for advice. The symptoms may be merely those of anaemia but there are certain special symptoms and signs which, if present, help greatly to differentiate this type of anaemia from all others. These are as follows:-

1. History of recovery from a similar previous attack.
2. Lemon yellow colour.
3. Symptoms and signs of subacute combined degeneration of the cord.

The first point merely emphasises the strong tendency to remissions and relapses and will not help much if we are seeing the patient in the first attack. The characteristic colour is by no means always present; the patient may be merely waxy pale or show brownish pigmentation of the skin, but if it is present it is a sign of great usefulness, provided we are fairly sure of a normal biliary tract. Definite signs of subacute combined degeneration in a patient of from 40 to 60 may be taken as fairly conclusive evidence of actual or potential pernicious anaemia, at any rate in this country. The common early symptom of this condition is numbness and tingling in the hands and feet, but there is a great variety of symptoms. If the posterior columns are first chiefly affected we may have lightning pains and gastric crises, as in tabes, with Rhombergism and alteration of sensation. Damage to the pyramidal fibres on the other hand may give rise to spasticity and increased jerks. Complicated pictures may arise such as absent knee jerks with extensor plantar responses. The most useful instrument for detecting this degeneration is the tuning fork, because vibration sense is usually affected early. Testing joint sense in the big toe joint will also help one to avoid missing the early case. It is almost unnessary to mention that subacute combined degeneration may arise and be well developed before any signs of anaemia are present, as in the following case.

Woman, age 64, sixteen years ago began to fall down without any apparent reason; fell down stairs several times; if kept talking very long on the telephone, was apt to fall. Complained of "rheumatic" pains in the feet. Ten years ago developed indigestion, and would vomit without cause. Suacute combined degeneration was diagnosed. About six years ago definite pernicious anaemia appeared.

This case also illustrates the fact that gastric or gastro-intestinal symptoms may ante-date the anaemia by several years. Indigestion or diarrhoea are particularly common as symptoms of pernicious as opposed to other kinds of anaemia, but of course are not absolutely characteristic. Paraesthesiae of the extremities may occur in secondary anaemia, but the presence of signs of subacute combined degeneration is probably the best single clinical evidence we have that any case of anaemia is of the pernicious type. (Post mortem evidence of degeneration is found in about 80% of cases.) This applies no matter what the blood picture may be as in true pernicious anaemia it may be of the secondary type at times, particularly during the early stages, during remissions and erythroblastic crises.

Another point often mentioned in the diagnosis of pernicious anaemia is achlorhydria (absence of free HCL) or achylia gastrica (absence of any HCL and ferments in gastric juice) There is certainly a connection between achlorhydria and pernicious anaemia. Wilkinson and Brockbank in a review of the literature find that there have been reported 139 families with two or more members suffering from pernicious anaemia with or without subacute combined degeneration; 59 families in which pernicious anaemia and achlorhydria occurred in different members; 17 families in which achlorhydria occurred without pernicious anaemia. Of 291 relatives of patients suffering from pernicious anaemia 70, or 24.1%, had achlorhydria; about half of these had true achylia. This is vastly more than the normal, which is about 1% to 2%. Several times on routine examination relatives were found to have full-blown pernicious anaemia of which they were unaware. We know however that achlorhydria occurs in normal healthy people; also in people with secondary anaemia. Witts estimates that out of 100 people with achlorhydria, 10 will have pernicious anaemia; 20 will have secondary anaemia, and the rest will be normal. So we must disabuse our minds of the idea that there is anything specific about achlorhydria in pernicious anaemia; also we know now that persons may have an apparently normal acid and pepsin secretion with typical pernicious anaemia.

Let us now consider for a short time some types of secondary anaemia. Haemorrhage, either acute and massive, or chronic and slight, may cause profound anaemia. There are a certain number of cases of chronic anaemia which seem to date from a single large haemorrhage. It must be remembered too that a patient may bleed because of anaemia or because of some other blood condition which is producing the anaemia, such as leukaemia. In these cases a complete blood examination is the only thing to do. Chronic post-haemorrhagic anaemia is most commonly associated with gastric and duodenal ulcers, bleeding haemorrhoids, and

menorrhagia, all of which must be looked for in any case as soon as anaemia is diagnosed. Patients with chronic anaemia and gastro-intestinal symptoms are relatively common, and it may be very difficult to determine the primary factor clinically. In this case the test for occult blood in the stool is of great value, particularly if it gives negative results. This test is probably not sufficiently used.

All acute infections are inclined to produce an anaemia of some degree, the most notable example of course being frank septicaemia. In acute cases, sometimes called acute septic anaemia, there may be increased bilirubin in the blood with mild clinical jaundice and also, of course, fever. Chronic infections or foci of infection are a much more doubtful cause. We are beginning to realise that in anaemia just as in rheumatoid arthritis, we have in the past under-emphasized the fact that secondary infective complications may be due to the primary condition. Thus pyorrhoea or infected tonsils may be due to chronic anaemia, and it is improbable that any infection which gives no other signs or symptoms is causing anaemia.

The conditions associated with secondary anaemia being too numerous to specify in detail, let us pass on to consider more obscure cases which are often labelled "Idiopathic." Witts has recently described a form, occurring chiefly in women between 40 and 50, which he calls chronic microcytic anaemia. The colour index is low and the Van den Bergh reaction negative. Patients often complain of dysphagia, and about 50% of his series had glossitis. Achlorhydria was present in 81%, and one-third had enlarged spleen. Witts considers this type of anaemia to be allied to the Plummer-Vinson syndrome in which we have a secondary anaemia associated with glossitis, dysphagia and enlarged spleen. Glossitis as a symptom was first popularised in connection with pernicious anaemia by William Hunter. Clinically there may be complaint of sore tongue with nothing to see through all the stages of fissure and superficial ulceration up to a smooth, bald tongue originally described. Sore tongue in the absence of local causes should lead to blood examination, but is not at all pathognomonic of pernicious anaemia any more than is enlarged spleen. Witts emphasises the fact that this type of anaemia occurs in middle-aged females who are often full of complaints, chiefly abdominal, who often go undiagnosed through years of ill-health, but who can be restored to usefulness by adequate diagnosis and treatment. Mills describes idiopathic hypochromaemia occurring in females aged 30 to 40, with low gastric acid or achlorhydria, low colour index, chronic course, and sometimes glossitis and paraesthesiae of the extremities. Dameshek describes what he considers a new syndrome called *primary hypochromic anaemia*, occurring mostly in middle-aged women who have all the symptoms of pernicious anaemia except jaundice and subacute combined degeneration. The similarity of these descriptions is striking, as is also the fact that there are included many symptoms and signs which we are rather inclined to regard as typical of pernicious anaemia. The fact is that, clinically, there is no very hard and fast distinction in some cases. The colour index is not always reliable. As before stated, the best clinical evidence that an anaemia is primary is the presence of subacute combined degeneration. If this is absent, we may have recourse to the best haematological evidence, which is the

average diameter of the red cells. In England Price-Jones has plotted the diameters of the red cells on a curve called the Price-Jones curve. Five hundred cells are actually measured, and the results plotted with numbers of cells as ordinates and the abscissae marked out in microns. In pernicious anaemia the curve is shifted to the right and the base usually broadened.

Murphy has recently evolved another method which he thinks is even better both diagnostically and as a guide in treatment. It is called *the iron index*. It is a figure given by dividing the Fe in milligrams per 100 cc. of blood by the red cells in millions.

Normal figures would be $42/5 = 8.4$. In pernicious anaemia this index is increased; in secondary types it is diminished. It may also be used as a measure of effectiveness in treatment.

Another type of secondary anaemia which we must consider here is *splenic anaemia*. The original description of this form included any kind of chronic anaemia with enlarged spleen, from which clear entities such as familial acholuric jaundice, primary splenic tuberculosis, thrombosis of the splenic vein and neoplasms have been split off. There remains a group of cases consisting of young adults who suffer from a secondary anaemia, enlarged spleen and leucopenia with relative lymphocytosis. The white cells usually number less than 4000. The anaemia is subject to febrile exacerbations, and haematemesis is an early symptom. The spleen may gradually enlarge to an enormous size. It is generally believed that Banti's Disease is a late stage of splenic anaemia. After a period of years the liver enlarges, then shrinks again and becomes cirrhotic, with consequent ascites, jaundice and haematemesis from oesophageal varices. It is not known whether this is a primary disease of the spleen or, as some argue, merely a stage of cirrhosis of the liver. At any rate it is a form of anaemia which should be recognised early if possible. There are several good grounds for separating this type from other secondary anaemias:—

1. The incidence in young adults of both sexes.
2. The marked leucopenia, which does not occur, for example, in Witts' chronic microcytic anaemia.
3. Late development of hepatic cirrhosis.
4. Ordinary treatment for secondary anaemia ineffective.
5. It is one of three blood conditions in which splenectomy has been shown to be effective treatment, the other two being chronic thrombocytopenic purpura and familial acholuric jaundice.

One should refer here to another rare type of anaemia which occurs almost exclusively in young adults. It is called *aplastic anaemia,* and the picture is that which we should expect to find if we were to imagine that all the blood-forming elements had become senile and ceased to function. Thus there is a progressive decrease in red cells, white cells and platelets. The duration of the disease is usually a matter of months.

It may follow poisoning with benzol, particularly novarsenobenzol; it occurs sometimes in phthisis, in the course of pernicious anaemia, or following excessive doses of radium or X-rays. These patients tend to bleed in a very persistent way from the mucous membranes, a phenomenon possibly associated with the low platelet count. The diagnosis is made by the exclusion of other types of anaemia, by the blood picture and finally by examination of the bone-marrow at post-mortem.

There is nothing known at present which will stimulate the formation of blood cells in these cases. Liver, liver extract, stomach, iron and arsenic have all been tried and found wanting. Transfusion is the only measure which is of any value and its effects are only temporary. In a case recently described by Stewart Harrison 109 blood transfusions of a full pint had been employed over a period of four years without any signs of blood regeneration.

Recent Work

Modern ideas on anaemia originated in the experiments of Whipple and others on the post-haemorrhagic anaemia of dogs, in which it was found that liver by mouth was very effective treatment. Minot and Murphy, applying this to man, discovered that large amounts of liver would produce a reticulocyte response and a remission in cases of pernicious anaemia. It was subsequently found that kidney and whole stomach given by mouth had the same power. These organs, then, evidently contain some factor which, on absorption by the patient, enables his haematopoietic organs to function normally. Castle and his co-workers, investigating further, found that there is absent in the gastric secretion of patients with pernicious anaemia something which interacts with ordinary food stuffs to produce an anti-anaemia substance. The evidence for this can be condensed as follows:—

1. Shredded beef muscle given in large quantity to a pernicious anaemia patient will not produce a remission.
2. Normal gastric juice, given by stomach tube to a pernicious anaemia patient will not produce a remission.
3. Shredded beef muscle and normal gastric juice, incubated together and then fed by tube to a pernicious anaemia patient, will produce a typical reticulocyte response and a remission.
4. Shredded beef muscle, incubated with gastric juice from a pernicious anaemia patient, and then fed to another pernicious anaemia patient, will not produce a remission.

The conclusion follows that there is, in normal gastric secretion, an "intrinsic" factor, and in food an "extrinsic" factor, probably of protein nature, which react together to form a "Substance X," which on absorption brings about normal blood formation. Castle, Heath and Strauss, in a recent paper, show that the intrinsic factor was absent in the otherwise normal gastric juice of two patients with pernicious anaemia who afterwards reacted to liver, and was present in the achylic gastric contents of one patient with no anaemia, and three patients with

hypochromic anaemia. Possession of this intrinsic factor seems then to prevent the development of the pernicious anaemia picture while its absence is a *sinqua non*. Pernicious anaemia thus becomes a nutritionly disease in that it is intimately associated with an inadequate digestion of food-stuffs probably of a protein nature. The part played by liver in producing a remission in its relation to this gastric deficiency has not yet been worked out.

Secondary anaemia is often associated as we have seen with achlorhydria and gastro-intestinal disorders. There is a good deal of evidence that many of these cases are related to a poorly balanced diet, or to faulty digestion and assimilation. The practical disappearance of chlorosis is evidence that better hygienic conditions and a higher standard of living amongst the submerged tenth has a good influence in preventing secondary anaemia. The diets usually recommended as containing most anti-anaemic factor are based on the work of Whipple on anaemia due to haemorrhage in dogs, where liver is very effective. This work cannot be applied in its entirety to man under similar conditions. We seem at the present moment to have more information about the factors concerned in the production of pernicious anaemia than in the case of the more obscure forms of secondary anaemia.

Treatment

In general it may be said that in the treatment of any severe anaemia the importance of *bed rest* cannot be over-estimated. This was emphasised by many of the older physicians, who found that in pernicious anaemia rest in bed was often effective after other treatment had failed. Since the oxygen-carrying power of the blood is reduced the heart must send out an increased minute-volume in order to supply the needs of the tissues. This is the reason for the increased pulse rate so often seen, and rest has a large part in reducing this effect on the cardiovascular system. All patients with severe anaemia then should be put to bed and kept there until they are able to be up and about with a normal pulse rate.

There is now a good deal of evidence that in all kinds of anaemia diet is also of very great importance, and this applies particularly to anaemia of the pernicious type. Here we should probably follow the principles laid down by Minot and Murphy. The main outlines of their diet for pernicious anaemia are as follows:

1. At least half pound of liver per day.
2. About 100 grammes beef or mutton in addition.
3. Fruits such as peaches, apricots, prunes, strawberries, oranges and grapefruit.
4. Enough butter and cream to make the diet palatable.
5. Enough carbohydrate to make the diet up to 2000 or 3000 calories.

For those who cannot eat so much liver there are potent extracts such as that made by the Connaught Laboratories or whole stomach

preparations such as Ventriculin. There are now available also liver extracts which can be injected intramuscularly or intravenously. The effect of these is little short of dramatic, and they are especially useful in very ill patients who would otherwise require transfusion. The maintenance dosage is that required to keep the blood count normal in the particular case, and as far as we know these patients should continue the liver diet indefinitely in order to avoid relapse. It should be remembered that some cases may not react to liver or liver extract, and in these stomach preparations may be effective. Another point is that in the presence of infection the dose of liver must be increased. For example, I am now treating a patient whose effective maintenance dose was three vials of Connaught liver extract a day. Two months ago she caught influenza and developed pneumonia at the right base. Her dosage of liver was not increased at that time, with the result that now she is suffering from a relapse of her anaemia with the typical clinical picture. Adequate liver diet at once controls such symptoms as sore tongue, vomiting and diarrhoea. Appetite comes back, and the patient is able to eat a proper diet. The return of strength and a feeling of well-being is very rapid. As far as the nerve involvement is concerned, cases have been reported showing marked improvement after liver therapy. This seems to be particularly common in early cases where the nerve symptoms are of recent onset. It is probable that, as in pellagra, pernicious anaemia patients often have an associated peripheral neuritis, and it is the symptoms and signs of this condition which clear up. There is no good evidence that cases of long standing with well-marked degeneration show any improvement even after massive doses of liver. In early cases, even in the absence of anaemia, liver treatment should be pushed in the hope of preventing extension of the process.

In secondary anaemia, an adequate good mixed diet is undoubtedly of importance, but it is doubtful as yet whether there is any more specific factor involved as in the case of pernicious anaemia. Whipple has worked out a table showing the comparative values of different foods in producing haemoglobin formation in dogs, and has recently isolated an anti-secondary-anaemia substance from liver. This substance has not yet been shown to be effective in man. There are of course many cases of mild secondary anaemia which react quite favourably to quite small doses of iron, but we have learned recently that in many chronic cases we must push the dosage. Witts regards 90 grains a day of a scale preparation as a minimum dose for cases such as he describes under the title chronic microcytic anaemia, and states that, with improvement in the blood, symptoms such as sore tongue, dysphagia, dyspepsia and enlarged spleen disappear. The medication must be kept up for a long time, and both Witts and Dameshek stress the fact that these patients are difficult to cure and very liable to relapse. Mills has recently published evidence that small doses of copper are very effective in such cases. Out of 22 patients, 19 improved at once and remained well on Blaud's gr. xxx with copper carbonate gr. 1/48 t.i.d. Some of these patients were previously resistant to large doses of iron alone. There is other evidence that copper may be of use, based on experimental work and also on the fact that stores of copper in the livers of infants on milk diet become depleted just as do the stores of iron. Lewis has

recently published evidence that in anaemia in infants small doses of copper with iron are very effectual. I should say that at present copper therapy does not rest on any known scientific foundation, but it is certainly worth trying in a case of chronic secondary anaemia resistant to large doses of iron.

In conclusion, we have seen that anaemia is a condition which when well advanced presents obvious symptoms and is easily recognized. On the other hand there are many milder cases in which the symptoms are vague and referred to other parts of the body, and in these the diagnosis may easily be missed because the blood is not thought of.

Pernicious anaemia is a nutritional disorder associated with a constant defect in gastric secretion. It has certain clinical characteristics which mark it out from other kinds of anaemia, of which the most conclusive is subacute combined degeneration. As a result of recent observations it can be treated effectively over long periods.

Secondary anaemia is predominantly a disease of women, and may be due to many causes, but there are many chronic cases of which we do not know the cause. The most important point in the diagnosis is to think of anaemia as a cause of the symptoms, which often appear irrevelevant, and the next is to have some instrument of precision to check the diagnosis in the suspicious case. Energetic and persistent treatment will result in marked benefit to many of these patients.

There are clinical varieties of anaemia known as splenic and aplastic anaemia, the causes of which are unknown and which are more difficult to treat.

In spite of much progress in the last five or six years we still know very little about the gastro-intestinal tract, the liver, spleen and bone-marrow in their relations to blood formation, and it is to be hoped that continued combined study by clinician, haematologist and biochemist will gradually elucidate these mysteries, and so place more and more of the obscure cases within the reach of practical therapeutics.

"DISEASED" BUILDINGS

Several articles have appeared recently in medical journals calling attention to inaccuracies often noted in scientific terminology, or nomenclature, not only in secular periodicals and newspapers but in technical bulletins as well.

A well-known tuberculosis laboratory has issued a circular announcing that a "Tubercular Building" for children was to be erected. This is a misnomer commonly used even among medical workers. Those who know have repeatedly pointed out that a diseased organ may be "tubercular," but the patient is "tuberculous." It is to be hoped that the building in question will not suffer from this type of organic disorder, at any rate not for some years to come.

In the field of mental hygiene it has been necessary to explain to the uninitiated the difference between "mental defect" and "mental disease." But we too have been careless with our psychiatric vocabulary. Why the "Psychopathic Hospital"? There may be "psychopathic social workers" but the state hospitals and mental hygiene clinics try as far as possible to employ safe and sane "psychiatric" social workers. Facetiously, and for the sake of brevity, professional workers have referred to students of mental deficiency as the "feebleminded group."

"Insane" is a good old fashioned word, try as we might to discard it as a medical term, but why announce, as does a current bulletin that the foundations have been completed for two "disturbed buildings" and two "epileptic buildings" for the blank "insane hospital"? Have you ever seen a "nervous hospital"? But even the purist is stumped at "mental institutions," the phrase has come into such general use. The technologists have given us the televox, the electric man and the robot, but it takes a psychiatrist to endow a hospital for the insane with mind.

(*Mental Hygiene Bulletin*)

THE MODERN MANAGEMENT OF SOME COMMON FRACTURES—Continued

Dr. Murray Meekison

Spine Fractures.

A few high lights in the management of certain spine fractures.

1. Those of the body of a vertebra. Here one should always have a lateral radiograph made where the spine has been injured. Indeed compression fracture of a body is usually missed and the importance of diagnosis and adequate treatment will be realized when you have seen such cases a year or so after injury, and have tried to do something for the disorganized mechanics of the unfortunate victim's back. One can almost positively state that in early, uncomplicated cases adequate non-operative methods may be expected to restore full wage-earning capacity. The ideals of treatment are simple. 1. To reduce the fracture and 2. adequate protection until the injured area can take the strain of weight-bearing. I would like to describe to you the device first reported by Rogers in Surgery, Gynaecology and Obstetrics Vol. 50, p. 101, 1930, which has met with widespread apporoval. He devised a frame, essentially a Bradford frame with flexible sides and a cross-bar capable of being elevated which can be moved to any point under the frame. The patient lies in the dorsal decubitus on the tight canvas cover and the spine is gradually hyperextended by cranking up the crosspiece, correction of the fracture being gained by means of the action of gravity on the spine above and below. Maximum correction is usually obtained in about ten days. The patient is then placed on a Whitman or bent Bradford frame and kept there for an additional eight weeks. He then wears an ambulatory plaster jacket for several months. There is grave doubt as to the value of forcible reduction of these fractures but I can speak for the efficiency of this conservative method. Let me again, before leaving this type of fracture, urge prolonged treatment. In frac-

tures of the transverse processes, particularly lumbar, which are by no means uncommon, I am beginning to think that the disability is mainly mental. The most important part of the whole treatment is to refrain from telling the patient that he has a broken back. I have stopped using body casts on these patients, and now simply put them to bed for a month of physiotherapy and exercises. The majority of the patients should be back at work well within three months and those disabilities lasting longer than six months are very much out of the ordinary. The continuance of symptoms is due to the associated sprain or contusion resulting in scarring, and not to the fracture itself. The most important factor in treatment, I reiterate, is to impress upon the patient how negligible the injury is. Tell him he has a broken back and he may be a cripple for life purely by alteration of his mental point of view.

Fractures of the Pelvis.

There is nothing new to report. The treatment of complications would require a long paper in itself, while the usual uncomplicated fractured pelvis is easily handled and if properly reduced and held for three or four weeks there is no residual disability. Extension, as usual, with counter-traction is probably the best method in displaced fractures while some men prefer manipulation under anaesthesia followed by plaster. In those where there is no displacement, a stout binder, adhesive plaster or a pair of plaster tights may be used.

Fractures of the Neck of the Femur.

Let us now dogmatize for a moment on fracture of the neck of the femur. This is a condition about which much has been written. There is an old and established treatment that yields a high percentage of excellent results and, moreover, is fairly simple to carry out. It is most satisfactory for the handling of any fracture in the region of the head of the femur, and if it fails to accomplish the purpose, one would be wise to try and pass the case on to someone else. Briefly outlined, it is as follows. Use the Hawley Table if it is available and prepare to apply a double hip spica with the plaster extending well up on the chest. Short anaesthesia is necessary, preferably gas in the aged, or spinal if one is so inclined. With counter traction by the use of the post and applied to the sound leg in medium position, the surgeon lifts the injured hip forward, rotates it inward, extends it with a good steady pull and carries it outward to an abducted position of about 45 degrees. Here it is held while a plaster spica is applied around the pelvis and down to the toes on the affected side with the knee slightly flexed and the foot at right angles. I believe it is sound practice to include the opposite thigh down to the knee. The plaster is carried up to about the level of the nipples and is generously cut out in front to aid respiration and eating. One is usually gratified to find that the hip is in good position and may be left to unite. The cast is left on for three months and frequent changes of the patient's position are strongly indicated. At the end of this time, the plaster is removed and for another month the patient is kept in bed during which period massage and exercises are carried out. X-rays, of course, are taken from time to time to determine the stage of union.

When they indicate bony union, the patient is allowed up on crutches or a walking caliper, preferably the latter, but in no case is he allowed to bear weight until six months have elapsed from the time of injury. Then it is assumed gradually. After another six months, the average case has recovered to an approximately normal state. Let me quote a few reviews before I pass from this fracture. Using the Whitman method Campbell reports 95 per cent good results in 70 cases; Anderson 95 per cent in 200 cases; 93 per cent in 16 cases. Even discounting these figures, I think we have the answer to the question of how to treat a fractured hip.

Fractures of the Femur-shaft.

I will now briefly review the treatment of fractures of the shaft of the femur. For this purpose they may be conveniently grouped into three classes which we will take in order.

1. *Up to age* 5. I believe in these, the best and easiest treatment is overhead suspension to a gallows frame of both legs using Buck's extension. The usual 3 inch wide strips of adhesive plaster are applied to the inner and outer sides of both legs from high in the thigh to the malleoli and bound in position with bias-cut flannelette bandages. The cord from each spreader is passed through its own pulley fixed to the frame and then they join. On the connecting cord rides another pulley to which are added sufficient weights to keep the buttocks free of the bed. The child is quite happy in this position, strange to say, and the extensions are adjusted from time to time. In five or six weeks union is sound, in most cases in excellent position, and the child may walk after another week or so in bed.

2. *Age 5 to 15 or later.* Again Buck's extension applied as before but this time in conjunction with a Thomas splint. The extension is fastened to the splint and the splint fastened to the end of the bed. The foot of the bed is then elevated on blocks for counter-traction, and the fracture just naturally reduces itself. Both this and the fracture in younger children can be reduced without anaesthesia. A grain of codeia by hypo or an eighth of morphia is useful as a preliminary. With the usual atention to keep the apparatus tidy, the extension is left on for four weeks. Then the patient is taken up to the O. R. where a single short hip spica is applied without anaesthesia from the level of the crests of the ilia down to and including the foot. This is left on for four or five weeks and after another week in bed the patient is allowed to walk. This, I may say, was the routine treatment at the Children's Hospital in Toronto, for fracture of the shaft of the femur, and a common fracture it was.

3. *After age* 15 Here the controversy starts, and here the multiplicity of treatment commences. I belive that many can be treated successfully by means of the Buck's extension and Thomas splint with the addition of overhead suspension and counter-traction by means of pulleys and weights. I have not found manipulation and plaster an adequate treatment, although in the hands of some men it yields excellent results.

I believe that, taking results all around, (and this fracture is almost as much written about as that of the neck of the femur) the best and most accepted form of treatment at the present time is direct skeletal extension combined with the Thomas splint and overhead suspension. Extreme care must be used in employing skeletal, as the fear of infection is always with us. The surest preventive of infection is to *leave the dressing alone,* once the traction is applied. Fiddling around with the dressings is almost bound to lead to trouble. There are several devices for obtaining skeletal traction, and they all have their advocates, Pearson's icecaliper tongs or some modification, Steinman pins or nails. They are used above the condyles of the femur, above the malleoli of the lower leg or above the os calcis. I prefer a modified Pearson tong used just above the condyles of the femur. The knee is flexed and the pull is arranged in the direction of the upper fragment. I will omit the details of fixing the tongs as these are readily available. Less weights are required than when using skin traction, and when measurements show that the leg is out to length, the weights may be cut down to the point where they are just sufficient to hold the fractured ends in position. The calipers may be left in for five or six weeks, if they are left alone, and following their removal the leg may be left in the bent Thomas splint until X-ray indicates sufficient solid callus to allow it to be removed. The patient is then kept in bed for a further two weeks, while movements are encouraged and massage is carried out. Special attention should be paid to the knee throughout the treatment, and it may be moved from time to time while the calipers are in position. Finally when the patient is allowed up, a good walking caliper is an excellent device to use for a month. Many a good result has been ruined by allowing weight bearing too early.

Fractures of Both Bones of the Leg.

My paper is beginning to assume untoward proportions so I will hurriedly run through one or two ideas or devices that I have found of value in the treatment of fractures of both bones of the lower third of the leg and ankle. Firstly in speaking of those of the lower third of the leg we all know how difficult it is to reduce, and hold reduced the fracture of the tibia, without anterior angulation. In George Wilson's book, he makes a suggestion that is contrary to all accepted teaching flexion. Along with this idea he uses the two stage plaster, and puts the knee at the angle something between 45 and 90 degrees. First of all the upper portion of the cast is applied from well up on the thigh down to within 3 inches of the fracture. Then the foot is encased to the toes and up to within 3 inches of the fracture line on its distal side. The fracture is then reduced by extension and hyperangulation if necessary, and the gap is bridged. One finds in most cases excellent reduction. This plaster is left in position for about three weeks or a little longer. At the end of this period, it is removed and the knee extended to a slightly flexed position and the foot gently brought to a right angle and slightly inverted position. This is left on for a further six or eight weeks when it is bivalved and physiotherapy commenced. I am rather in favor of gentle attempts at active movement and gentle massage as early as seven weeks from the time of the injury and in some cases

even six weeks. At all events it is important to start it early. I have used this method of reduction and retention exclusively for the past year and have been extremely gratified with the results. I might add that I believe it is good surgery to keep the foot strapped in inversion, for at least three months after weight-bearing is commenced. Further, the patient should never be allowed to commence weight-bearing in a slipper. Nothing less than a boot is adequate support.

Just one point in connection with Pott's fracture before I pass on. This is a trick in its reduction. I do not know who told me about it or where I saw it, but wherever it was it has stuck in my mind and I have always used it. The patient lies, of course, on his back anaesthetized, with the injured leg slightly flexed at the knee over a sandbag and the lower half of the leg extending beyond the end of the table. A loop made of factory cotton bandage is hung over the leg above the ankle and it is made long enough to allow the surgeon to place his right foot in its lower end. With the right hand grasping the ball and the left holding the anterior part of the foot, you will find that you can hold the leg immobile with counter traction by shoving down with your right foot, and can place the patient's foot in the properly reduced position with the greatest of ease. Try it sometime.

Fractures of the Os Calcis.

I was going to speak about fractures of the os calcis, but the subject is much too controversial and certainly there has not yet been a sound and universally successful treatment devised. The continuous traction method is not without danger. The method of reduction by remodelling either with thumb and forefinger, or a wrench is uncertain. I have never been able to understand the wisdom of pounding the os calcis with a mallet. It seems to me that it adds insult to injury. Reconstruction operations seem to be of doubtful value. Subastragalar arthrodesis is not always indicated, but I feel that in old cases with persistent pain this is the only treatment that, so far, is worth considering. I think that it is best to leave the treatment of this particularly obnoxious fracture to the dictates of your own minds or hearts until something really good is produced. All our results are for the most part bad, and I am sure that I have nothing startling to offer.

Fractures Not Dealt With.

In this mad gallop through fracture treatment I have omitted several. Fractures of the *carpals* are controversial as regards treatment and are unsatisfactory in anyone's hands. Fractures of the *skull* forms a subject by itself. There is nothing new in the handling of *ribs or sternum* fractures. Some fractures always require open operation and these I will but enumerate:—

1. *Head of the Radius*—if the joint surface is injured or whole fragment is displaced.
2. *Olecranon*—if any displacement.

3. *Exeternal condyle* or epicondyle of the humerus.

4. *Patella.*

5. Many *joint fractures.*

In closing, I would advise you to read an article by Hey-Groves in the Lancet of Jan. 28th, 1928. The title of this article was striking and productive of much food for thought. It was called "Damages to Bones and Reputations." He summarizes the histories of the last 100 patients seen by him for residual disability following fractures.
Summing up he says:

1. Make a radiological examination of every injury.

2. Secure good reduction.

3. Prove that after reduction, the alignment is satisfactory.

4. Use simple traction, and in case of failure, resort to open operation and impact the fragments.

5. Do not plate unless you can plate efficiently.

6. Never plate an open fracture.

7. Do not comment on another physician's treatment.

Discussion

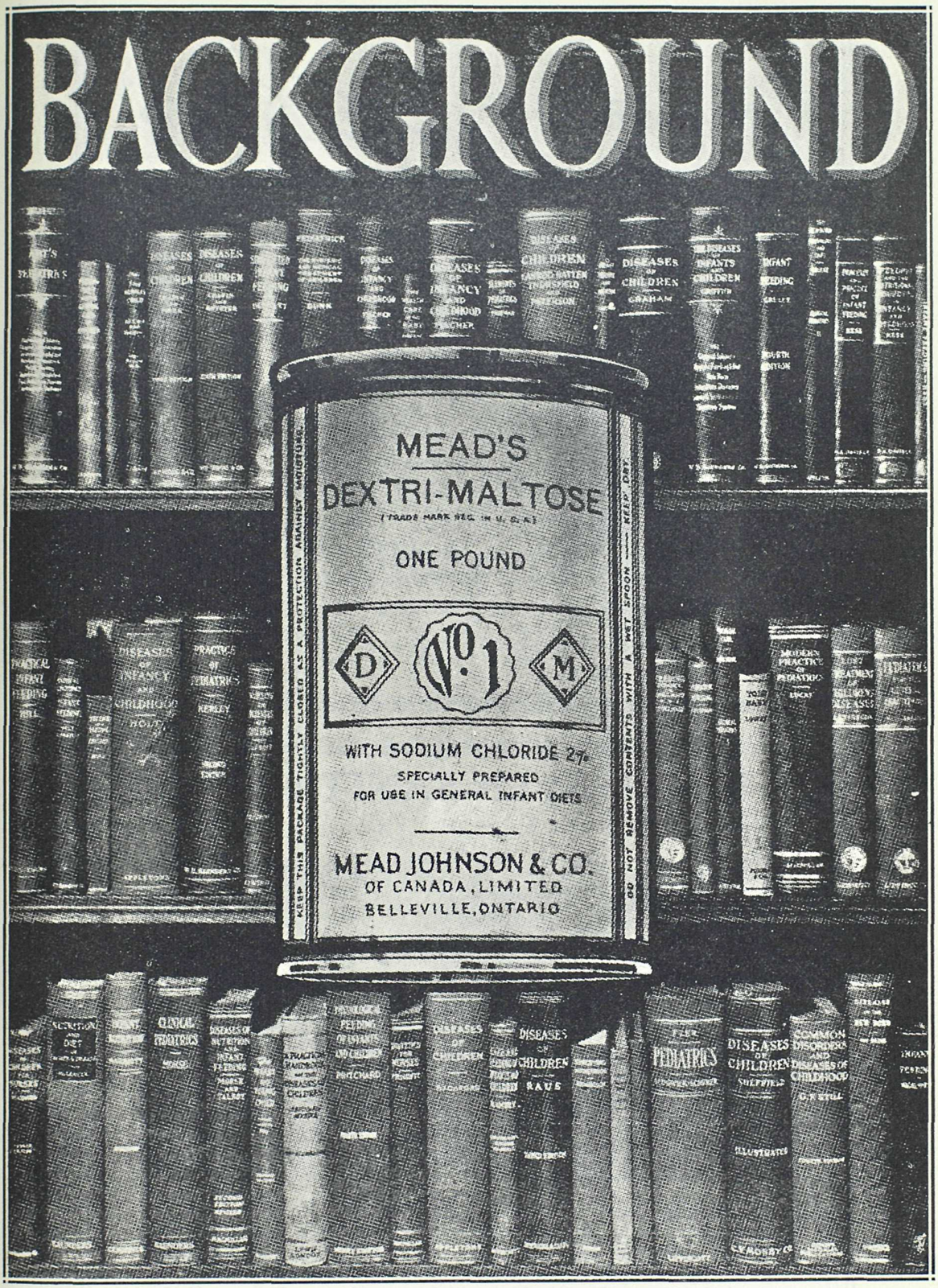
These papers were followed by a very full discussion in which Dr. F. P. Patterson took the leading part. He dealt with the matter of time loss due to fractures and emphasized the fact that this was largely due to the following causes: (1(Interference with joint motion where arthritis ensued. This is increasing, especially in the Lower Mainland. The anatomy of the joints concerned must be borne in mind here and the limb so placed when reduction is effected that if arthritis should ensue we shall obtain a favourable position for function. Arthritis must be guarded against by a suitably long period of immobilisation and avoidance of early weight-bearing. (2) Improper alignment, producing incorrect joint planes and causing strain. Proper alignment Dr. Patterson considered most essential, angulation giving a particularly bad functional result. This can be prevented by traction which the speaker considered was of supreme importance in the treatment of fractures. Another point Dr. Patterson emphasized was that a fracture is an emergency case which should be treated at once. We should not wait till the swelling goes down, and muscle spasm is best overcome by traction. To obtain correct anatomical position is more important in adults than in children, with a view to subsequent function.

As regards treatment, Dr. Patterson emphasized the following points: In industrial fractures we should not be in too great a hurry to get the

patient back to work—too early weight bearing is bad and will produce arthritis owing to the abnormal strain on the joint structures. Indications of delayed union are (1) excess of callus, (2) tenderness and heat at the site of fracture and as long as these two conditions are found we must avoid weight-bearing.

Dr. Patterson dealt with the question of physiotherapy, which he considered of very doubtful value in the treatment of fractures. Massage in the later stages may be of value in restoring muscle tissue, but if in doubt do not massage was the rule he gave. Too early and too rough movement in fractures around joints will inevitably produce bad results.

The speaker drew attention to two conditions often overlooked in the treatment of fractures. These conditions cannot be discovered by X-ray but must be borne in mind if we are to avoid the charge of malpractice: (1) nerve lesions. In every fracture this possibility should be borne in mind and paralyses or nerve injuries should be looked for at the time the fracture is first seen and before it is reduced. (2) Vascular damage. This is frequently overlooked, especially in fractures around the elbow joint and ischaemias may result which are no fault of the surgeon but may be attributed to his supposed carelessness. Large haematomata or interrupted circulation of the extremities give the clue and must be looked for.



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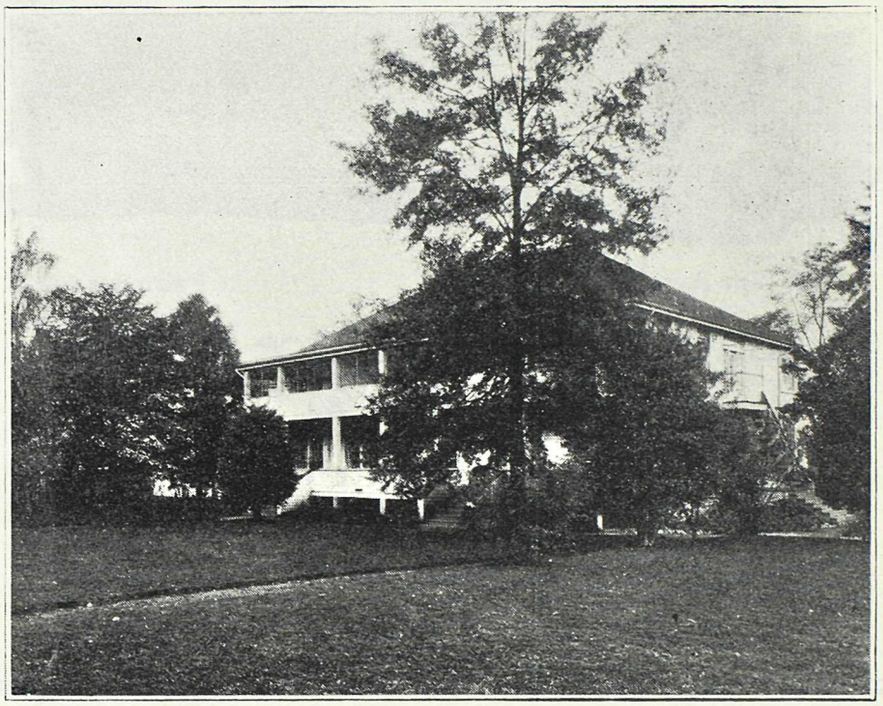

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