VOL. IX. SEPTEMBER, 1933 No. 12

THE BULLETIN

of the

Vancouver Medical Association

Anaemia

News and Notes

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THE VANCOUVER MEDICAL ASSOCIATION

BULLETIN

Published Monthly under the Auspices of the Vancouver Medical Association in the Interests of the Medical Profession.

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Vol. IX. SEPTEMBER, 1933. No. 12

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VANCOUVER HEALTH DEPARTMENT

STATISTICS, JULY, 1933

Total Population (Estimated)	247,251
Japanese Population (Estimated)	8,429
Chinese Population (Estimated)	7,759

	Number	Rate per 1,000 Population
Total Deaths	150	9.7
Japanese Deaths	3	4.2
Chinese Deaths	7	10.6
Deaths—Residents only	133	6.3
Birth Registrations	264	12.6
Male 142		
Female 122		

INFANTILE MORTALITY—

Deaths under one year of age	10
Death rate—Per 1,000 births	37.9
Stillbirths (not included in above)	5

CASES OF CONTAGIOUS DISEASES REPORTED IN CITY

	June, 1933		July, 1933		August 1st to 15th, 1933	
	Cases	Deaths	Cases	Deaths	Cases	Deaths
Smallpox	0	0	0	0	0	0
Scarlet Fever	16	1	6	0	6	0
Diphtheria	1	0	2	1	0	0
Diphtheria Carrier	0	0	0	0	0	0
Chicken-pox	204	0	41	0	8	0
Measles	2	0	0	0	0	0
Rubella	0	0	0	0	0	0
Mumps	77	0	7	0	1	0
Whooping-cough	8	0	1	0	3	0
Typhoid Fever	0	0	2	0	1	0
						(Carrier)
Paratyphoid	0	0	0	0	0	0
Poliomyelitis	0	0	0	0	1	0
Tuberculosis	69	18	57	14	45	—
Meningitis (Epidemic)	0	0	0	0	0	0
Erysipelas	6	0	4	0	1	0
Encephalitis Lethargica	0	0	0	0	0	0

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PROPORTION OF DISEASES SPREAD THROUGH MILK

There is considerable interest in knowing how large a proportion of the common diseases are spread through milk. In order to determine this it is necessary to know the total number of diseases and the number of these in which milk is concerned. The difficulty has been to find all of the cases spread through milk. Since Massachusetts has made such a record in locating the outbreaks due to milk the following table shows the percentage of the total reported cases in the State accounted for by the milk borne epidemics:

PER CENT OF TOTAL CASES REPORTED REPRESENTED BY CASES TRACED TO MILK

Period	Septic Sore Throat	Typhoid Fever	Scarlet Fever	Diphtheria
1896—1900	——	2.1	0.0	0.1
1901—1905	——	1.9	0.6	0.0
1906—1910	——	8.1	3.8	0.2
1911—1915	——	5.3	1.2	0.1
1916—1920	75.5*	6.9	0.4	0.1
1921—1925	10.1	6.7	0.2	0.1
1926—1929	58.3	8.3	0.3	0.0
1930—1932	27.6	0.4	0.002	0.0

* Septic Sore Throat made reportable in 1914.

In considering the data given in the above table, it should be remembered that the percentages given are smaller than they would be if all of the cases caused by milk were known.

While Massachusetts offers the longest period of such comparisons some other states furnish higher percentages for short periods. During the fiscal years from July 1st, 1924 to June 30, 1926, in California, there were reported 2,630 cases of typhoid fever, of which 483 cases or 18.3 per cent were traced to infection through milk. In 1898, Whipple in his book dealing with typhoid fever estimated that 25% of the typhoid fever was spread through milk. Likewise in 1906, an attempt was made to trace all cases of typhoid fever in the District of Columbia.

Of the cases where the source of infection could be traced with reasonable certainty, 60.8% were assigned to milk. It should be remembered that these two later percentages were developed before the period when pasteurization had become sufficiently common to influence the figures.

In this connection it should be noted that milk borne undulant fever is probably now producing at least twice as many cases of human disease as all of the epidemic milk borne diseases. In addition milk borne tuberculosis has long been and probably still is our most serious human affliction spread through raw milk.

In the light of these findings it does seem strange that the attempt has been made by some individuals to show that disease spread through milk is too insignificant to be considered a health problem.

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EDITOR'S PAGE

Listening one day recently to a very able speaker and clear thinker, a couple of sentences he uttered struck us very forcibly as especially applicable to the medical profession, though the speaker at the time was making no reference to that section of society.

He said "The inspiration and traditions of the past are sacred, but must not be allowed to usurp the place of the inspiration of the present." Yet it often does—nations have fallen because they trusted to the inspiration and traditions of the past, instead of facing conditions as they were. And a great deal of the trouble of our profession, and a great deal of the grievance that the public feels against us as a profession, is due to the fact that we are still, to a great extent, living in the past, on an outworn tradition, instead of creating new rules of conduct to fit new conditions of social order.

This reluctance to change is not necessarily laziness or selfishness at all—it has, we firmly believe, a much more worthy basis; it is based on traditions which are amongst the loftiest and most generous that any profession has held—but we have reached a point where we should enquire whether these traditions in their present form fit these times— and whether we are being generous and noble, or merely "easy" and foolish. For nobody has any real respect for the man or institution that supinely submits to unfair treatment.

In the formulation of new rules of conduct, which will in their turn become the tradition of our profession, we may, indeed we must, keep the spirit of generosity and self-sacrifice that has made our profession what it is—but this spirit must assume a new form, more closely consonant with conditions as they are. The word "charity" has assumed new meanings, and has become a symbol for a form of socialized and deputized giving, a collective generosity—so that personal and individual charity on our part has become an impossibility, without injury to ourselves and inadequacy as regards the recipients. Free medical service, which once represented our gift to society, is now no longer practicable or of real value—and we must express our liberality in other terms, possibly of a more abstract kind—such as willingness to abandon our individualism to a certain extent, to socialize our service largely, to pool our resources for the common good, and to become to a degree servants of the larger body, the state, rather than entirely personal attendants of the individual person or family. This will require a considerable degree of self-sacrifice on our part, but it would seem to be indicated by circumstances as they are.

Our speaker's other sentence was equally illuminating in this regard. He said "Whenever you find a man harping on the traditions of the past, beware lest he is trying to make a profit out of it in the present." There is very sound philosophy and sense in this, and it certainly is true in our case—everybody, city, province and all sorts of eleemosynary institutions wax really elequent in their panegyrics on the noble traditions of the medical profession, which has done so much for humanity

without any thought of payment. Sitting in at a round-table conference the other day, where some of us were urging the justice of the claim made by certain members of medical staffs, for at least out-of-pocket expenses in return for an immense amount of free work, we were much impressed by the fervour of one lay speaker, who spoke of the splendid work that the doctors of "his staff" had done for years. He thought that it would spoil the whole relationship if money were introduced into it. He told us solemnly that several of his staff had assured him that if money were offered them they would resign.

We took leave to differ from this gentleman, and said we thought he had misunderstood these doctors, that we knew of no doctor who would refuse a fair fee for his work, if it were offered him. But he was a very good example of the truth expressed in the quotation given above.

The truth is, we think, that the time is coming when medicine must face the issue of socialisation of medicine to a greater or less degree, and we may as well be frank and candid about it in saying that we think that the sooner this is done the better both for ourselves and the public at large. We feel that only esesntial changes should be made at first—but these things should be done at once, and at our suggestion and under our guidance. The profession is waiting for leadership in this matter and will be prepared, we are sure, to go a long way in helping to bring about the new state of things. Let us make no mistake about it—the changes are coming—and the form they take, whether it be pleasing to us or most unwelcome, will depend upon whether or not we are ready and able to guide them. We believe that political parties would welcome our guidance, if it were offered in concrete and practical suggestions, and if they were convinced, as we could easily convince them, of our sincerity and honesty of purpose. Whilst we may feel we have certain grievances against the public, as represented by various institutions, we must not forget that they too have certain well-grounded causes for criticism of us—and we must be just as anxious to remove these by constructive unselfishness, as we are to improve our own lot, in fact, the latter consideration will fall to the ground completely if we are not successful in the first part of the programme. The day of individualism, with its selfishness and greed and gluttony culminating in the present vast depression and misery, must go—and must be replaced by a collective effort based on a much more unselfish spirit, and we must do our share in bringing about the change.

* * *

NEWS AND NOTES

In our last issue, we "pointed with pride" to the forthcoming investiture of Dr. Dr. R. E. McKechnie with the Good Citizen's Medal. We feel moved to comment again on the proceeding, as reported in the daily press. R. E. in his remarks on the subject, took is as a compliment to his profession, rather than to himself. While appreciating the kindly spirit and loyalty which prompted these remarks, we feel that they did

not accurately represent the true state of affairs, and we are afraid that the responsibility is entirely R. E.'s own. He is merely reaping the reward of a long life of public service, and we desire to inform him that he cannot "pass the buck" to us.

* * *

Dr. Harold Caple was seen last week cavorting over the sands of Savary, in a very becoming sun-tan, and very good spirits.

* * *

Others holidaying at this well-known summer resort were Drs. Harold White, Medical School Officer, Dr. Day-Smith, Dr. F. N. Robertson, Dr. Walker Turnbull, and the Editor.

* * *

Dr. A. P. Proctor Junior is the latest addition (up to the time of writing) to the line that forms "to the right." His marriage with Miss Frances Griffin took place recently, and Dr. Eddie Day officiated as best man. The contagion is apparently one of a long incubating period, but we desire to warn Eddie that it very seldom fails to get its man.

* * *

Again we wish to comment with pleasure on the character of the paper that appears in this issue. Dr. J. E. Walker is to be congratulated on the amount and quality of work he has put into his study of the cases which came under his notice at the Outpatients' Department. The paper will repay a thorough reading, and we regret that it was impossible to publish it in one issue. We could not very well shorten it, and our space is limited.

* * *

Calamus Scriptorious writes: "A prominent lady of this city was given a Baby Shower recently (vide Province of late date). Having in mind the usual meaning of a "shower," one wonders where the babies come from. "Out of the everywhere into here," so to speak, or was it the Maternity Clinic of the V. G. H.? Or—but let us not allow our imagination to rove too far. As one stork remarked to another, "Is my face red!"

* * *

We congratulate Dr. and Mrs. Murray Meekison on the safe arrival of a daughter recently. Mother and child are both doing well.

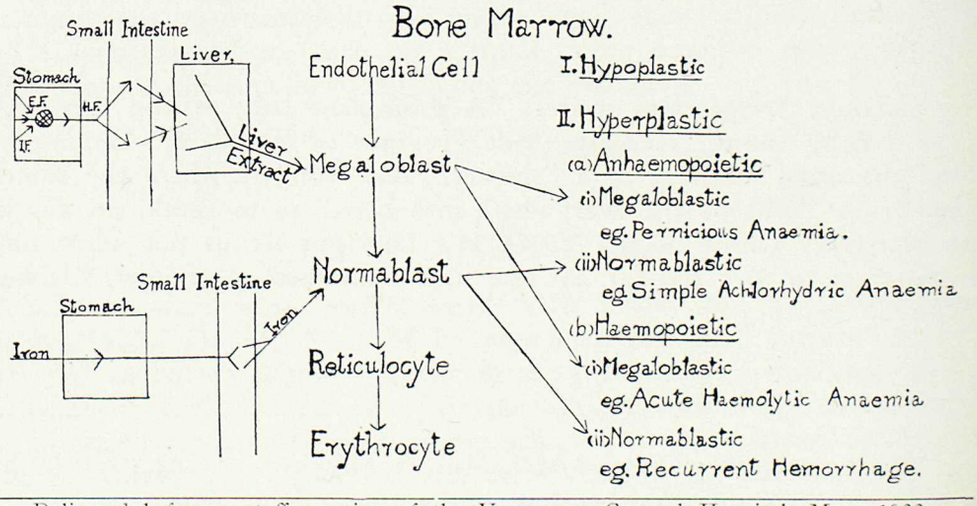
ANAEMIA

JOHN EDEN WALKER, M.B.

This evening we are presenting a number of cases of anaemia, which have been studied in the Outpatients' Department of the Vancouver General Hospital under Dr. S. Sievenpiper and myself during the past year or more, to illustrate especially the treatment of different types of anaemia by means of liver extract and of iron.

In order to understand the action of these substances in the treatment of anaemia, one must first study the modern conception of the pathology of this disease. Before shewing these cases, I should like to spend a few minutes in presenting the theoretical aspect of the subject to your attention. The accompanying diagram deals with the normal physiology of the maturation of the red blood cells, and its relationship to anaemia, based on a classification of this group of disease, from the standpoint of changes as found in the bone marrow. The bone marrow is the source of all the red blood cells in adult life. Before fifteen years of age the marrow of all bones is engaged in cell production, after fifteen only the spongy flat bones of the body and the heads of the femur and humerus produce them. The endothelial cell, lining the intersinusoidal capillaries of the bone marrow, is thought to be the parent cell. These cells produce, by a process of mitosis, an indefinite number of megaloblasts which are larger than the mature erythrocyte and are nucleated. These megaloblasts divide by the same process of mitosis into an indefinite number of normoblasts which are smaller in size and are also nucleated. The normoblast in turn extrudes its nucleus and becomes a reticulocyte. Reticulocytes, when stained with certain vital dyes, show rod-like bodies which are composed of the remains of the basophilic cytoplasm of the normoblast.

The reticulocyte is the final stage of the immature cell. After it reaches maturity, it is known as the erythrocyte. It will be understood that in the normal state, it is necessary for comparatively few endothelial cells to produce megaloblasts, but in pathological conditions where cells are urgently needed, the endothelial cells of the bone marrow serve as a vast potential factory. Governing the maturation of the immature red blood cell into the final product there is a complex mechanism.



Delivered before a staff meeting of the Vancouver General Hospital, May, 1933.

From the normal stomach mucosa there is created a substance which is called the intrinsic factor of Castle. When this intrinsic factor comes into contact with an extrinsic factor, which is ingested into the stomach in the food, a reaction takes place between the two, producing what is called the haematinic factor. This haematinic factor is absorbed from the small intestine, and stored in the liver, kidney, brain, and other organs. As it is needed, this haematinic factor is carried to the bone marrow, where its presence is required to allow the normal maturation of the red blood cell from the megaloblastic level. In addition to this haematinic factor there are other substances which control the maturation of the RBC from the normoblastic level. The chief of these substances is iron. Other minerals, of which copper is the most important, and vitamin C, are believed to play a part in this control from the normoblastic level onward. These substances are normally ingested into the stomach and absorbed from the small intestine, to be stored in the body, or carried to the bone marrow direct. In the past few years an attempt has been made to classify the various anaemias from the standard of pathology as it is found in the bone marrow. The marrow of the sternum has been found to be the most satisfactory, from the view-point of this study. The classification which I have illustrated above, serves in a broad way to clarify the underlying pathology of the various anaemias. Bone marrow is found to show two outstanding differences, those types of anaemias where there is a decreased production of RBC in the marrow, that is, a condition of hypoplasia, and those cases in which there is an increased production of RBC in the marrow, that is, hyperplasia. Those anaemias which show a hypoplastic bone marrow we will not speak of further. Those types of anaemia which show a hyperplastic bone marrow may be subdivided into anhaemopoietic and haemopoietic. Each of these in turn may be divided into those cases which show a preponderance of immature cells at the megaloblastic level in the bone marrow, and those which show a preponderance of immature cells at the normoblastic level. The anhaemopoietic group: In this group of anaemias there is an increased number of immature red blood cells present in the marrow, but for some reason these are unable to progress through their various stages and enter the blood stream. That is, there is an increased number of immature red blood cells in the marrow but a deficiency of red blood cells in the circulation. In the haemopioetic group there is an increased number of immature red blood cells in the bone marrow, which are able to progress through their various stages and reach the circulating blood, but for some reason the cells, as they reach the blood stream, are destroyed or lost faster than they can be produced.

How may we tell from looking at the blood smear to which type of anaemia the case belongs? Can we tell from looking at the blood smear, what we should expect to find in the bone marrow? We can answer these questions by noting particularly the size of the red blood cells and their haemoglobin content. Various methods of measuring the size of the red blood cell have been devised, which I do not, however, intend to discuss on this occasion. One might make the broad statement that where the average cell is larger than normal and is well filled with hae-

moglobin, thus giving rise to a colour index greater than one, the anaemia is considered to be macrocytic, and the type cell in the bone marrow will be the megaloblast. If the average size of the cells is below normal or poorly filled with haemoglobin, thus giving rise to a colour index below one, the anaemia is considered microcytic, and the type cell in the bone marrow will be the normoblast. These are broad statements and will not cover every case of anaemia but serve as a general working basis. We submit a list of the principal conditions that produce the *anhaemopoietic* type of anaemia (1) with *megaloblastic bone* marrow.

1. Pernicious anaemia.
2. Anaemia of Bothriocephalus latus infestation.
3. Anaemia following resection of small bowel.
4. Anaemia associated with intestinal stenosis.
5. Anaemia of cancer of the stomach.
6. Anaemia of dysentery.
7. Anaemia of T. B. of small intestine.
8. Anaemia following partial gastrectomy.
9. Anaemia following complete gastrectomy.
10. Anaemia of sprue.
11. Anaemia of chylous or fatty diarrhoea.
12. Anaemia of pellagra.

(2) with *normoblastic* type of bone marrow.

1. Simple achlorhydric anaemia.
2. Nutritional anaemias.
3. Scurvy.
4. Tropical nutritional anaemia especially in pregnancy.
5. Fatty diarrhoea.
6. Pellagra.
7. Following resection of small intestine.
8. With intestinal stenosis.

This evening we do not wish to discuss the haemopoietic group any further, but will dismiss it by saying that the pernicious anaemia of pregnancy is believed to be an example of that form of haemopoietic anaemia which shows a megaloblastic bone marrow. Chronic or recurrent haemorrhage is an example of haemopoietic anaemia showing normoblastic bone marrow.

It is the anhaemopoietic group which I wish to take up in more detail. The outstanding example of anhaemopoietic anaemia showing megaloblastic bone marrow, is true pernicious anaemia. An outstanding example of anhaemopoietic anaemia showing normoblastic bone marrow is simple achlorhydric anaemia.

I wish to take up now the subject of pernicious anaemia with the previous statements as an introductory background. The etiology of pernicious anaemia: Pernicious anaemia may be due to one of three possible causes (a) deficiency of intrinsic factor (b) deficiency of extrinsic factor and (c) deficiency of absorption of haematinic principle at any location

from the time it is produced, until the time it reaches the bone marrow. The experiments of Castle have proved that the cause of pernicious anaemia is lack of the intrinsic factor normally manufactured by the stomach mucosa. The other two factors may play a part in some cases; that is, the diet may be extremely deficient in extrinsic factor; or there may be failure of proper absorption of haematinic principle from the small intestine due to disease of this organ. What is the nature of these three factors? We have no idea as to the composition of the intrinsic factor. It would appear to be neither a vitamine nor a hormone, nor a ferment. We have reason to believe that the extrinsic factor so far as blood maturation is concerned is vitamine B2. Extrinsic factor is not present in washed casein, in wheat glutin, in washed nucleo-protein or in yeast nucleic acid, but it is present in beef muscle, and very strongly so in autolyzed yeast. It has no properties which cannot be compared to vitamine B2. The nature of the haematinic factor is also not known, but it may be vitamine B2 altered in some way by the intrinsic factor. It is of course notorious that the blood lesions and the central nervous system lesions of pernicious anaemia may not parallel each other in severity in the same case. This has led to the idea that the factors governing blood maturation and those governing the maintenance of a normal central nervous system may not be exactly the same, although very closely related. It has been suggested that the extrinsic factor concerned in the prevention of central nervous system lesions is probably vitamin B1. It is known that deficiency of vitamin B1 in the disease of beri-beri produces peripheral neuritis. The analogy is obvious. Also it has been shown in experiments on dogs fed upon diets lacking in vitamine B1, that the central nervous system shows lesions similar to those of pernicious anaemia. When we administer liver extract we are really giving haematinic factor that has been absorbed from the intestine and stored in the liver. We have found in our clinic that liver extract given intramuscularly is at least fifty times as potent as when given by mouth. This leads one to think that there must be either a destruction of the principle of liver extract by the gastro-intestinal secretions, or else failure of absorption of this principle from the intestinal tract. It is extremely likely that in pernicious anaemia, there is, in addition to a deficiency of production of the intrinsic factor, also a deficiency of absorption from the intestinal tract.

Our cases in the Out Patients' Department have all been treated with liver extract, but preparations of hog's stomach, in which the mucosa and the muscular layers have been ground together and extracted, have been found equally potent, or more so. During the past two years we have averaged fifteen cases constantly in attendance, of which about three quarters have been given liver extract intramuscularly, for the past year, at weekly intervals. Some of these cases had previously received liver extract in varying lengths of time, from one to three years, by mouth. The intramuscular injection has been given into the buttock or into the arm, whichever location the patient prefers. The majority of our patients have experienced both forms of treatment, and state that they prefer to take liver extract intramuscularly. We are still maintaining several patients on liver extract by

mouth for various reasons. In some cases the site of injection is painful for only a few minutes; others have experienced pain for from two to three hours, or from two to three days. The size of the dose up to a certain point, does not seem to influence the degree of discomfort following the injection. We make a practice before starting a patient on intramuscular treatment, of doing a skin test, particularly when he has been taking liver by mouth previously. In one patient who showed quite a marked cutaneous reaction we started to desensitize him, but with the first injection he collapsed for a few minutes. That is the only case which has shown any reaction to intramuscular liver therapy. We have been giving our intramuscular cases Lederle's liver extract. Three cc. of this purified extract is derived from 100 grammes of liver. It has been estimated that in oral preparations of liver extract, the extract is only equivalent to about 65% of the whole liver from which it is made. Whether the purified liver extract used for intramuscular injection is effective in the same ratio, I cannot say. In cases of relapse with low red blood count and haemoglobin, using Lederle's preparation, it has been the practice in this Hospital to give 5 cc every day for several days, increasing the interval to about twice a week and finally once a week. In patients whose blood has been brought to normal, one must find what dosage is necessary to maintain the patient at his optimum level. In our series, the patients have been on doses varying from 2 cc a week to 6 cc a week. The average dose has been around 3 to 4 cc. We started giving regular intramuscular treatment a year ago last April. Last fall we tried to cut down the dose from 1 to 2 cc in each case but found that the patients were not getting along so well and in the last three or four months we have tried the opposite course and have put them back on doses even higher than those they were previously getting.

I would like to say a word about the cost of liver therapy. The cost to the Hospital of 3 cc of Lederle's liver extract is $1.17. The patient on this dose would therefore have to pay $1.17 per week. The same patient taking liver extract by mouth would require about three vials per day, at approximately 25c per vial, which would amount to $5.25 per week; more than four times as much as on the intramuscular method.

During the last couple of months we have been trying new cases of pernicious anaemia on the Connaught Laboratories preparation of liver extract for intramuscular use. This extract is not as concentrated as Lederle's, in that 100 grammes of liver is represented by 10 cc of extract. If the potency of all extracts for intramuscular use derived from 100 grammes of liver is the same, then we would have to give 10 cc of intramuscular Connaught Laboratories extract to get the same effect as from 3 cc of Lederle's. From the few new cases that we had in the last two months, we would think that the Connaught Laboratories preparation for intramuscular use is more potent than Lederle's preparation, using as a basis of comparison the extract from 100 grammes of liver. The cost of the Connaught Laboratories preparation to the Hospital is 45c for 10 cc. The question of expense in the treatment of pernicious anaemia is a very important one.

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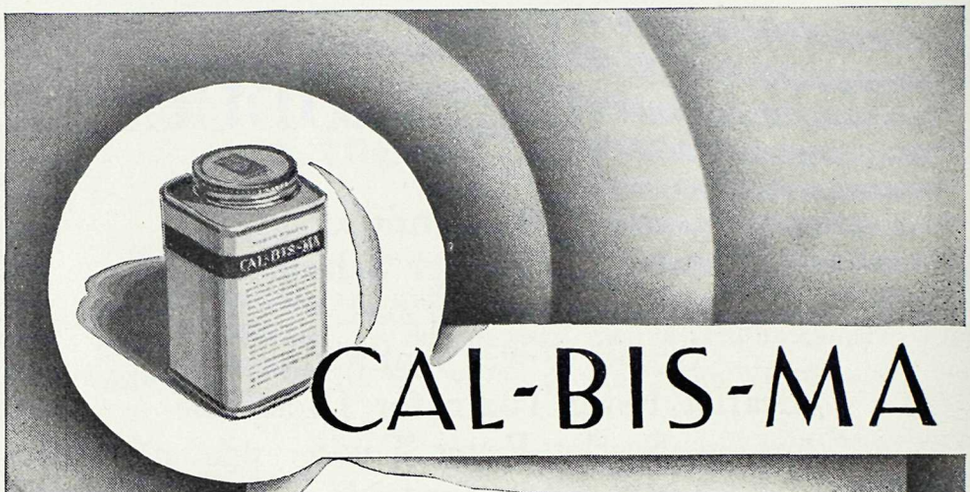

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There must be a difference in the potency of the different extracts which are on the market. Some authorities in England claim that the extract from 15 grammes per week of liver is sufficient to maintain the average patient. We can contrast this statement with our experience in Vancouver, where the extract from 3 to 6000 grammes of liver has been found to be necessary.

It may be asked why we should have started giving liver extract intramuscularly when the extract is potent when given by mouth. The reasons for this are twofold; first on the ground of economy; secondly on the ground of better control of the patient. On the whole we are more satisfied with the results of intramuscular therapy than when given by mouth. We think that if sufficient quantities of liver extract are given by mouth or intramuscularly the result should be the same, but we have found that patients do not always take the full dose they are told to take; that when they are feeling beter, or have felt better for some time, they begin to omit a dose or take a smaller dose. On the other hand when giving the extract by the intramuscular method, we know that the patient is getting what he is supposed to be getting. Those patients who have not been taking liver intramuscularly have been taking the Connaught Laboratories extract for oral use.

The treatment of pernicious anaemia cases does not consist wholly in administering liver extract, though this is by far the most important factor. Foci of infection should be eliminated. Mental hygiene is often necessary. Iron therapy is sometimes indicated and complications if present should be dealt with. Patients should be on a simple balanced diet. We think that if they are getting a satisfactory dosage of liver extract, the so-called anaemic diet is not necessary as long as the diet is balanced. We feel that this factor of diet has not been entirely satisfactory in the Out Patients' Department in all cases; many of them are on City Relief and the rest have very limited incomes. We make a practice of giving dilute hydrochloric acid in doses of one drachm t.i.d. in a glass of water to counterbalance the deficiency practically always present in the stomach. All authorities do not agree on the necessity of this practice. Certain principles must be followed in the administration of liver extract. Treatment must be continued indefinitely and in optimum doses. Patients in whom liver is discontinued will always show a relapse in time. When we say an optimum dose, we do not mean one in which sufficient liver extract is given only for the patient to hold his own; his blood picture should be absolutely normal; his nervous signs and symptoms should improve or at least show no retrogression, and his general constitutional symptoms should be satisfactory. One should never treat a patient with his blood count or haemoglobin as the only guides. If a patient is allowed to lapse either through discontinuing liver extract, or through insufficient liver extract being administered, on recommencing treatment it will often be found that larger doses than before are required to maintain him. There are two factors which definitely inhibit the action of liver extract; these are the presence of foci of infection, and of arterio-sclerosis. Some workers have divided pernicious anae-

mia into groups according to the amount of liver extract necessary to maintain them. Those requiring the smallest doses were found to be younger in age, having a shorter duration of the disease, showing less chronic infection, and little arterio-sclerosis, and with less involvement of the nervous system. Those requiring larger doses showed the reverse of the above factors.

Most of our cases come under the second heading. We have fixed the arbitrary interval of one week for administration of liver extract intramuscularly, but we think that if one could give in one dose, as much extract as would be taken daily, or weekly, by a pernicious anaemic, for say, one month, his condition would show no deterioration. It would seem that the liver extract is not destroyed or excreted by the body, but is stored in the reservoirs of the body, of which the liver, kidney, brain are the chief, and is released when required. In ordinary treatment one should aim not only at meeting the daily requirements of the bone marrow, but in filling up the reservoirs of the body as well, so that any extra strain on the marrow may be adequately handled.

In handling patients in the Out Patients' Department we have a limited staff, and a great many patients to see, so that we have tried to develop an economy of effort in examining and treating those patients. On admission we do a complete physical examination and complete laboratory survey. In following their course afterwards we try to note especially the following features; the condition of the tongue, foci of infection, condition of the circulatory system, especially the presence of arterio-sclerosis, an dthe condition of the nervous system. During the past we have taken, at least once a month, red blood count, haemoglobin, colour index and smear. Lately we have modified this to some extent and are taking blood counts less frequently. We are depending more on the examination of the smear by means of the microscope and by the eryometer. This throws much less work on the laboratory and we feel gives us the essential information.

Introduction to Presentation of Cases

In presenting some of the cases which have been attending the O.P.D. during the past couple of years, we do not intend to show each case in its entirety. We propose merely to show cases to illustrate certain principles, or to bring out certain points that may be of interest. These cases have all had full laboratory tests carried out on them, so that in these cases where a full record of laboratory tests is not recorded, it may be taken for granted that the necessary tests have been done; also in the physical examination, negative tests are not recorded. Some of these cases did not attend the clinic for anaemias for varying lengths of time after their admission to the O.P.D., so that we have not been familiar with all cases at their onset.

I. A case showing *presence of normal amount of free HCL in stomach contents.* This is the only case we had attending the O.P.D. which does not show achlorhydria.

J.P. male, age 63 years. In August 1930 he was admitted to the General Hospital for the period of a month. He gave a history of about one and a half years duration: difficulty in walking, tingling and cold sensations in the hands and feet, great loss of strength, mental irritability and poor memory. His nervous symptoms had become so pronounced that he could hardly walk with the aid of two canes. On examination the chief findings were in the C.N.S.; he had a staggering spastic gait; Romberg's sign very marked, Babinski (R & L) positive. Knee jerks were plus plus, other reflexes not recorded. Vibration sense absent in both extremities. Laboratory work. R.B.C. 2,900,000, haemoglobin 75%, colour index 1.3, macrocytes plus 3, WBC 6000; poly's 57%, lymph's 41, stomach conditions free HCL 0,34,14,0.

Blood and spinal Kahn, negative. At this time there was much doubt by the attending physicians as to whether this was a case of pernicious anaemia or not, on account of finding free HCL. However, patient was started on liver therapy by mouth, ½ ampoule liver extract, (Connaught Laboratories) t.i.d. He continued on this dose from October 1930 to August 1932, during which time his blood counts could be considered fairly satisfactory. He was able to walk a little better than on admission but still had to use two canes; on the whole, he was a little better, but not a great deal, as far as his nervous symptoms were concerned. His oral dose of liver extract was apparently enough to keep his blood fairly normal, but was not sufficient to correct his nervous symptoms.

In September 1932 he first attended the clinic for anaemias. He was started on Lederle's extract, 3 cc (100 grammes of liver) intramuscularly, once a week. By November he was able to discard one cane, and by December he was able to walk a short distance with no assistance. About this time he also noticed a great improvement in the proprioceptive sensations in hands and feet. His tongue which had been sore from the start, improved a great deal; the same may be said of his general strength. On examination of his nervous symptoms February 1933, reflexes were about the same, Romberg's sign much improved, he could walk a straight line. His vibration sense was still absent in lower extremities but present in upper. His blood picture has been normal since starting the liver extract intramuscularly. During the last two months we have increased his weekly dose to 6 cc, and he says that his walking ability and general well being are improving all the time. Stomach contents in August 1932 and February 1933 still showed free HCL present. We think that this case is one of pernicious anaemia in spite of free HCL in the stomach, because of his blood picture 2½ years ago and the great improvement in his nervous symptoms on adequate liver therapy.

II. Case showing *combined sclerosis but blood picture not typical of pernicious anaemia.*

J.S. male, age 54 years. About 4½ years ago he was feeling in his usual good health. One winter morning he awoke and found that his legs were weak and that he had no sense of balance. He had a general feeling of weakness and later developed tingling in hands and feet, For about 1½

years he had various treatments but made practically no progress. During this time he lost 20 lbs. in weight, and could walk only with great difficulty. In December 1930 he had his blood examined by his family doctor for the first time, RBC, 3,200,000, haemoglobin 55%, colour index 84. WBC 3,200. stomach analysis, no free HCL Blood Kahn, negative.

At a later date a spinal puncture was done with entirely negative findings. There is no record of physical examination at this time, except that a note was made to the effect that the spleen was palpable. He was put on ventriculin at this time and at once started to show improvement. In May, 1931 he first came to the O.P.D. at which time his RBC were 4,450,000 haemoglobin 87%, colour index .96, leucopenia with relative lymphocytosis. In September 1931, he first reported to the clinic for anaemias. The symptoms at this time were numbness, tingling and feeling of coldness in both extremities, weakness, and great difficulty in walking. Examination at this time showed his knee jerks plus 4, Romberg marked, gait spastic, with inability to walk a straight line. Vibration sense impaired over both lower extremities. He was started on liver extract (C.L.) per os, ampoules. 1—t.i.d. In December 1931 he reported that he felt stronger, gait much improved, and tingling of hands improved. At this time his dose was increased to 1½ ampules t.i.d. (approximately 1200 grammes of liver) on which dose he continued until April 1932, showing, during this period, a definite improvement. In April 1932, he started on 3 cc of Lederle's extract intramuscularly, which was later stepped up to 4½ and then to 5 cc. At the present time symptoms have all improved greatly; he can now walk a straight line, Romberg's sign not marked, knee jerks are not nearly so exaggerated. Physical examination otherwise the same as a year ago. During all the time he has been attending the O.P.D. his blood picture has never looked like pernicious anaemia, except for an almost constant leucopenia. We consider this to be a case of pernicious anaemia in spite of the absence of a characteristic blood picture, and in spite of the sudden onset of his disease, which apparently came on over night,—because he shows no free HCL in stomach contents and because he has a combined sclerosis which has improved since he has been receiving liver therapy, previous to which treatment there had been no changes in his symptoms for 1½ years. It is probably one of those cases in which the factor governing the maintenance of the normal central nervous system has been very markedly upset while the factor governing the normal maturation of RBC has not been upset to an appreciable degree.

III. Case showing *elevated blood pressure*. Osler writing before the institution of liver therapy, states that a low blood pressure is characteristic of pernicious anaemia and that high blood pressure is never found. That is true, no doubt, in cases showing relapse; but as the following cases show, is not true of those cases on adequate liver therapy.

(a) J. F. male, age 63 years. This patient was admitted to the O.P.D. March 1930. History extended back to 1918 when he was confined to bed for about six months, complaining of general weakness, numbness and tingling of hands and feet, and attacks of vomiting. He improved

from this attack and was fairly well until 1927 when he was again in bed for three months with the same complaints; he recovered from this attack with liver therapy. Since that attack in 1927 has been in bed off and on and has never been really well. On coming to the O.P.D. in March 1930 his RBC were, 2,528,000, haemoglobin 75%; colour index 1.5; macrocytes plus 2, WBC 5,450 P. 70; L. 24. Stomach contents showed no free HCL. Physical examination showed some degree of combined sclerosis. March 1930 to April 1932 given liver extract by mouth and from that time on liver extract intramuscularly. The item of interest in this case is however, his cardio-vascular system. This patient's blood pressure while on liver extract intramuscularly varied between 170/90 to 210/110, the last reading being 170/100. His electrocardigram showed left ventricular preponderance and ventricular extrasystoles. X-ray of heart showed marked dilatation of aorta with heart not grossly enlarged. There is quite a marked general arterio-sclerosis. His eye grounds a year ago showed minute haemorrhage in right side. NPN normal.

(b) Mrs. B age 55. Patient has had symptoms of pernicious anaemia for several years and had a typical blood picture on admisison. Her blood pressure stays around 150/74.

(c) Mrs. R. Age 53. Has been on liver extract by mouth for the past three years at least, and showed blood pressure between 150/90 to 170/90. We have two or three other cases showing blood pressure slightly elevated; the remainder in our series either have normal blood pressure or a little lower than normal while on liver therapy, but none of them so far as we can find, have blood pressures below 100 systolic.

IV. Case *receiving iron therapy as well as liver therapy.*

J.F. This case we reviewed under III. This man was put on Frosst's Ferro-catalytic capsule 1, t.i.d. January 12th, 1933. At this time his blood count was as follows: RBC 4,600,000, haemoglobin 93%, colour index 1, smear normal. He stated that he did not feel well. This was not surprising perhaps, in view of the fact that he had a cardiovascular condition. We thought we would try him with some iron as well as liver extract. Ever since starting the iron he says that he has felt very much better and has more energy. During the last few months various authorities have stated that they do not consider liver extract alone a sufficient treatment for pericious anaemia. They think that either whole liver or iron should be administered as well. This idea sounds reasonable because in view of the theory of the etiology of pernicious anaemia, one would think that the people are more susceptible to failure of iron physiology than normal individuals.

V. A case that *has always received liver extract intramuscularly.*

Mr. T.S. Age 52 years, was admitted to the V. H. G. Feb. 23rd, 1932 discharged April 18th, 1932. On admission his symptoms were as follows. Indigestion 4 years, weakness 5 months, shortness of breadth, numbness and tingling of hands, 3 weeks. A review of his past illnessess showed

that he had typhoid fever in 1900, jaundice age 30, dysentery age 30. It will be noted that these diseases are all diseases involving the gastro-intestinal tract, and very probably have some bearing on his present condition. On physical examination the tongue was found to be smooth, red and cracked, teeth showed marked caries, nervous system showed absent ankle jerks, stomach contents no free HCL, platelets 55,000 per c.m. RBC 1,270,000 haemoglobin 22%; colour index 1.1; microcytes plus 2, macrocytes plus 2. He was given Lederle's liver extract intramuscularly 5 cc Feb. 29th, March 2nd, March 3rd, and 4th at which date his reticulocyte count was 50%. Following this the intervals between injections were gradually lengthened to an interval of 5 days. By March 27th his reticulocyte count was 1%. On discharge from the Hospital his RBC were 4,920,000; haemoglobin 80%; colour index .9. On coming to the O.P.D. after discharge from the Hospital he was given three cc once a week. RBC have never been below 4½ million and haemoglobin stayed between 90 and 100%; colour index usually slightly below 1.

He can now work in his garden for 6 hours a day, numbness and tingling very much improved and his ankle jerks which on admisison to Hospital were absent, are now obtainable.

VI. *Cases showing slight neurological signs.*

We find it impossible to find any case of pernicious anaemia in our series that does not show neurological signs. Most of them show marked signs. We have, however, picked two cases to show this part of the picture of pernicious anaemia to a comparatively slight degree.

(a) Mr. C. age 46. Admission January 1933. He states that he was in excellent health until about three or four years ago. At this time he noticed that he was becoming tired, weak and pale. For the past two years has had a sore tongue and for the past year has had praecordial pain and shortness of breath on exertion. For the past two or three months, numbness and tingling of the fingers, but no other symptoms of nervous origin. Has had indigestion for past two months. On physical examination his colour is pale lemon tint, marked pyorrhoea of lower teeth, nervous system negative, except that tuning fork sensation is slightly diminished over both upper and lower extremities. RBC 2,620,000; haemoglobin 80%; colour index 1.5; microcytes plus 4, WCB 4,500, poly. 71; lymph; 26, Van den Bergh .55 mgms., blood Kahn negative, barium series negative. Fluoroscopic and radiographic examination of oesophagus and mediastinum negative. Oesophagoscopy negative. These examinations were made because the patient has never been able to swallow a tube for stomach analysis. He was started on Lederle's extract 3 cc once a week and increased to 6 cc. Symptoms and blood picture improved satisfactorily. This is a case where the patient has evidently had pernicious anaemia for at least three years, and yet has shown very little involvement of the C.N.S.

(b) Mrs. B. age 55. Was admitted to O.P.D. Feb. 6th, 1933, with history that she has been anaemic for 30 years. She had been told

that she had pernicious anaemia 16 years ago while in a Hospital for a nervous breakdown. In the past 16 years has had to stop work at least 15 times on account of weakness. For the past 7 or 8 years had had numbness and tingling of feet. Three years ago she started taking liver therapy but in the last year has had no treatment. Physical examination shows a pale lemonish colour, palpable spleen. Vibration sense absent in lower extermities, central nervous system otherwise negative. Laboratory work RBC 1,580,000 haemoglobin 46%; colour index 1.5 microcytes plus 4, WBC 7,700; poly 73; lymph 24. Stomach analysis—no free HCL. Was admitted to the hospital and given Lederle's extract intramuscularly, showing maximum reticulocyte response of 39%. She returned to the O.P.D. March 23rd, 1933 with RBC of 4,850,000; haemoglobin 81%, colour index .84. She has been kept on 4 cc of Lederle's extract intramuscularly weekly since March 23rd. States she has not felt so well for years. This patient has had a history of anaemia for a great many years, possibly 16, and yet has very little evidence of nervous system involvement.

IX. *Cases showing a palpable spleen.*

We do not think the presence of a palpable spleen is of much importance in diagnosis. Many blood dyscrasias show a palpable spleen so that it is of very little use in differential diagnosis. We do not think that the percentage of cases of pernicious anaemia that show a palpable spleen is as great as is commonly thought. In cases on maintenance doses we never palpate the spleen, and in cases of relapse the percentage does not appear to be very high, probably less than 50%.

X. *Cases showing reaction of WBC to liver extract therapy.*

(A) Mr. C. On admission Feb. 2nd, 1933, RBC 2,600,000; haemoglobin 75%; colour index 1.4; WBC 4.500; Poly. 71 Lymph 26. April 18th, 1933 RBC 4,670,000; haemoglobin 90%, colour index .9; WBC 8,850; poly 66; lymph 30. In this case, as his red blood cells became normal so did his white blood cells become normal.

(B) Mrs. B. April 7th, 1933. RBC 4,500,000; Haemoglobin 85%; colour index .94; WBC 9,825. One week later blood picture approximately the same; WBC 5,600 Poly. 47; Lymph; 51. We did not know why her white blood count should have fallen without apparent reason.

(C) J. F. This man has been attending the clinic for three years, and during that time has had only one white count which approached normal, in spite of RBC never being below 4½ million and haemoglobin between 90% and 100%.

(D) Other cases show a normal white count with relative lymphocytosis in spite of normal red counts. We do not know what the significance of these white counts is, but we believe that sufficient liver extract should be given to make the blood picture normal. These readings may mean that we are not giving sufficient dosage.

XII. Cases showing that *normal colour index and smear are more important than high RBC count and haemoglobin.*

(A) Mr. H. October 1932. Two weeks after starting treatment, RBC 3,990,000; haemoglobin 100%; colour index 1.25; smear-macrocytes plus 3. March 1933 RBC 4,620,000; haemoglobin 90%; colour index .98; smear is normal.

In the first count this patient had haemoglobin of 100% but his smear showed marked pathology. In the second count haemoglobin was lower than on the previous occasion, but smear was normal. A single haemoglobin estimation in this case would have been most misleading.

(B) Mr. S. January 1933. RBC 5,240,000 haemoglobin106% colour index 1.02; smear shews occasional macrocytes, slight irregularity in size and shape of cells. April 1933, RBC 4,700,000; haemoglobin 90%; colour index .96; smear normal. First estimation was taken after the patient had been put on reduced doses of intramuscular liver. The rising colour index and slight pathology as shown in the smear were, in our opinion, sufficient reason to increase his dose of liver again. After stepping up his dose we found the second estimation normal and well balanced.

(C) Mr. J. D. On admisison to O.P.D. September 1932, RBC 4,550,000; haemoglobin 108%; colour index 1.2; macrocytes plus 2. After treatment RBC 5,300.000; haemoglobin 105%; colour index .98; smear normal. At the time the first count was taken this man showed very marked involvement of the central nervous system. If one had taken merely the haemoglobin estimation one would have said that he showed no signs of anaemia. Practically speaking, this man did not show anaemia, but his smear showed marked signs of cellular pathology. The second reading showed very little change in haemoglobin but there are now no findings of cellular pathology. These cases serve to emphasize that the examination of the blood smear is more important than the individual RBC or haemoglobin estimation. It is for this reason that we have been relying upon the appearance of the smear while keeping our cases on their maintenance doses.

XIII. Cases *showing comparison of effectiveness of liver per os and liver extract intramuscularly.*

Mr. F. M. Age 58. This patient was admitted to the V. G. H. on August 14th, 1930, with history of numbness of hands and feet, with difficulty in walking, of about 3½ years duration. For a year previous to admission had been on liver treatment but noticed that his weakness and nervous symptoms were getting progressively worse. On examination, he was of a pale lemon tint, atrophic changes in the tongue, moderate oedema of the feet. Reflexes of the lower extremities were unobtainable and his vibration sense was diminished in the legs. RBC on admission 1,070,000; haemoglobin 24%; colour index 1.14; WBC 4000; stomach contents, no free HCL. On discharge from the Hospital, RBC 4,580,000; haemoglobin 96%; colour index 1.05. While in the Hospital he was getting two and then four vials of liver extract per day. In November 1930 to April 1932 he was kept on vials 3 to 4 per day. During that time his blood was never really normal. His haemoglobin was always well

up; but his colour index was generally higher than 1. He was undoubtedly getting an inadequate dose of liver, though three to four vials a day is a fairly good dose (750 to 1000 gramnes of liver per day) In April 1932 he was started on 4 cc of Lederle's and then 5cc per week. After a month of starting Lederle's extract intramuscularly RBC 4,510,000; haemoglobin 96%; colour index 1. Blood has remained normal on 5 cc for the past year. With regard to the nervous system there has been marked improvement. Numbness and tingling improved a great deal and he feels much stronger. He can walk a straight line very much better. His knee jerks can be obtained with difficulty, whereas they were absent on admission to the Hospital. His ankle jerks are still absent.

XIV. *Cases showing marked central nervous system lesions.*

The majority of our cases showed marked involvement of the central nervous system. Some of them were so severe, that before starting treatment they were able to walk only with the help of two canes, and then some of them could hardly navigate. As a general rule, it takes longer to improve the nervous symptoms than to improve the blood picture. When one finds patients with absent or exaggerated reflexes, in many cases these do not improve, but we have several cases where they have shown improvement. One finds the greatest improvement in gait and sensory disturbances though there have been cases where numbness and tingling have been very obstinate. The improvement in gait may be astonishing. We have patients who could hardly walk with the aid of two canes before starting treatment, but can now walk a short distance with no help whatever. As we have said before, the severity of the blood changes may or may not parallel the severity of the nervous changes.

(A) Mr. J.D. age 59. This patient was in apparent good health until March 1932, when he noticed difficulty in walking, sense of girdle constriction, numb feeling from the waist down, tingling in fingers and marked weakness. These symptoms continued to get worse "in spite of chiropractic treatment," until at the time of admission to O.P.D. September 1932, he could hardly walk with two canes and a second person's assistance.

On examination at that time his triceps and biceps reflexes were equal and active plus 1; knee jerks plus plus plus; ankle jerks plus plus; ankle clonus plus; Babinski positive; Romberg sign positive; vibration sense absent in the lower extremities; RBC 4,550,000; haemoglobin 108%, colour index 1.2 stomach contents—no free HCL. He was put on Lederle's extract intramuscularly, 5cc a week. On April 20th, 1933, 7 months later, he states that he is feeling better. Last week he walked 38 blocks and worked all day in his garden. He can walk a short distance with no assistance. He still has a great deal of numbness and tingling. In spite of this marked improvement in his symptoms his reflexes and vibration sense remained unaltered, except that ankle clonus was not so apparent. His blood picture has become normal.

(B) Mr. F. B. Age 60 years. This man first came to the V. G. H. June 1930 with history dating back to 1925. At the time of admission he was practically bed-ridden. On examination his knee and ankle jerks were absent on both sides; biceps and triceps equal and active; Babinski

suggestive. RBC 1,600,000; haemoglobin 38%; colour index 1.2. He remained in the Hospital from June 1930 to January 1931. From January 1931 to April 1932 he attended the O.P.D. and was on large doses of liver extract by mouth, 4 to 5 ampoules per day. On this dose he did not show much improvement in his nervous symptoms. In April 1932 he was started on intramuscular injections of liver extract, 3 cc and later worked up to 5 cc. At the present time he says that numbness and tingling is not much better but his gait is much improved; it is better than at any time in the past three years. He can walk without canes or assistance, whereas formerly he needed two canes. On examination of his nervous symptoms there is practically no change in reflexes and vibration sense, but there is a wonderful improvement in his gait.

We would like to say a few words on the inadvisability of trying to cut down maintenance doses.

As we have indicated elsewhere, one must be very cautious about cutting down the maintenance dose. If too little extract is being given the poor results may become evident almost immediately, or not for several months. For this reason we may give inadequate dosage for a long time before this becomes evident. Last fall we tried cutting down the dose on some of our patients, and we soon got the impression that they were not doing so well. In giving liver extract (C.L.) by mouth, the dose in milder cases may be only the extract from 250 grammes of liver per day, but in several cases larger quantities of extract, up to that from 1200 gramnes of liver per day are necessary. Most of our cases are from 50 to 70 years of age, with marked nervous lesions, and we find in some of these, that the equivalent of 1000 grammes of liver per day by mouth does not hold them any too well. Practically all of them received or are receiving the extract from at least 750 grammes of liver per day. Since giving liver extract (L) by the injection method, the dose has ranged from the extract from 100 grammes of liver to the extract from 200 grammes of liver per week. Where patients do not respond as they should to liver extract, one should keep in mind this question: Is our diagnosis correct, or is the extract we are using not potent?

Another factor that has been discussed lately, is that of the need of iron or whole liver in these cases.

It is possible that in some of these patients their iron reservoir may become depleted, especially in relapse, and that more cases need iron therapy in addition to liver therapy than is commonly supposed.

Since this paper was written we have started those patients shewing marked central nervous system lesions on massive doses of iron.

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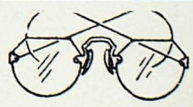

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Preventing NUTRITIONAL ANEMIA *in Infants through a Normal* DIETARY REGIMEN

NUTRITIONAL anemia was present in 45% of the breast-fed and 51% of the bottle-fed in a group of more than 1,000 infants studied by Mackay.[1] Although this anemia was of mild degree, *it was sufficient approximately to double the morbidity among the artificially fed.*

Anemia Prevalent

Commenting on this work, the British Advisory Committee on Nutrition writes, "This form of anaemia is prevalent among infants, especially those living under conditions of city life, and is attributed to a deficiency of available iron and possibly also of copper. Its most important feature is susceptibility to infection, particularly a liability to colds, otorrhoea, bronchitis, and enteritis, and a tendency for infections to become chronic."[2]

	IRON	COPPER
Cow's Milk, 14 oz.	1.01 mg.	0.166 mg.
Dextri-Maltose with Vitamin B, 1 oz.	2.40	0.570
	3.41	0.736
Daily Requirement *	3.11	"traces"

It is generally agreed that breast milk and particularly cow's milk are markedly deficient in iron and copper. But when 1 oz. of Dextri-Maltose with Vitamin B is added to 14 oz. cow's milk, properly diluted (as at 1 month), the above increase in iron and copper results.

Iron, incorporated in powdered milk, should be given as a routine to bottle-fed infants, according to the recommendations of this committee in a report to the Ministry of Health.

Milk Deficient in Iron

Stored in the liver of the full-term infant is a supply of iron and copper theoretically sufficient for the first six months of life. But actually the reserve is subject to wide variation,[1] probably because of variations in the iron content of the mother's diet during pregnancy. Hill, for example, says, "If the mother is anemic herself, or if she has eaten little iron-containing food during the last months of pregnancy, her offspring is born with an insufficient iron deposit. . . ."[3]

	IRON	COPPER
Cow's Milk, 20 oz.	1.44 mg.	0.24 mg.
Dextri-Maltose with Vitamin B, 1½ oz.	3.60	0.855
Mead's Cereal (dry), ¼ oz. or Pablum	1.70	0.09
	6.74	1.185
Daily Requirement*	4.18	"traces"

When ¼ oz. of Pablum is fed to the 3-months-old infant receiving 20 oz. cow's milk and 1½ oz. Dextri-Maltose with Vitamin B, a significant increase in iron and copper takes place.

The trend is also toward the introduction of iron-rich solid foods at an early age. The iron content of many foods is variable, however. Leichsenring and Flor[4] found that children's diets planned to contain 5 and 8.5 mg. iron actually contained only 3.25 and 6.5 mg., respectively. Pablum, higher than most foods in iron and containing standardized amounts of this mineral can be administered as early as the third month. Clinical studies by Summerfeldt[5] show that Mead's Cereal (of which Pablum is the pre-cooked form) is capable of increasing the hemoglobin percentage of growing children.

* The desirable iron intake for children, according to Rose *et al*, is 0.76 mg. per 100 calories. Infant of 1 month (8¼ lb.) and infant of 3 months (11¼ lb.), both require 50 calories per lb.[6]

[1-6] *Bibliography on request.*

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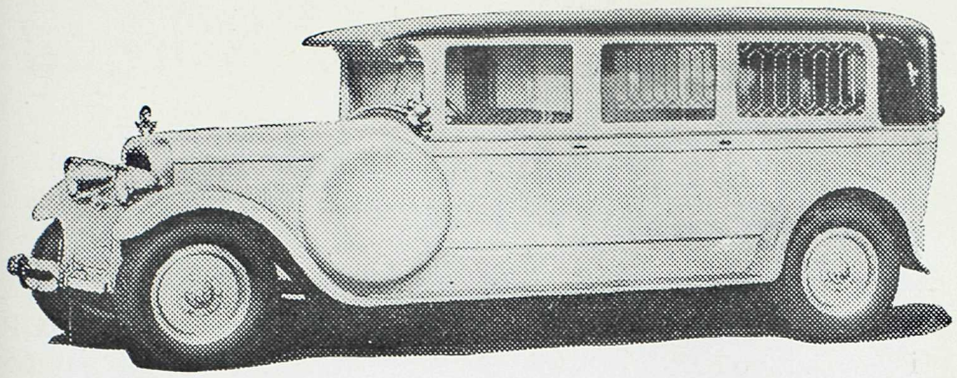

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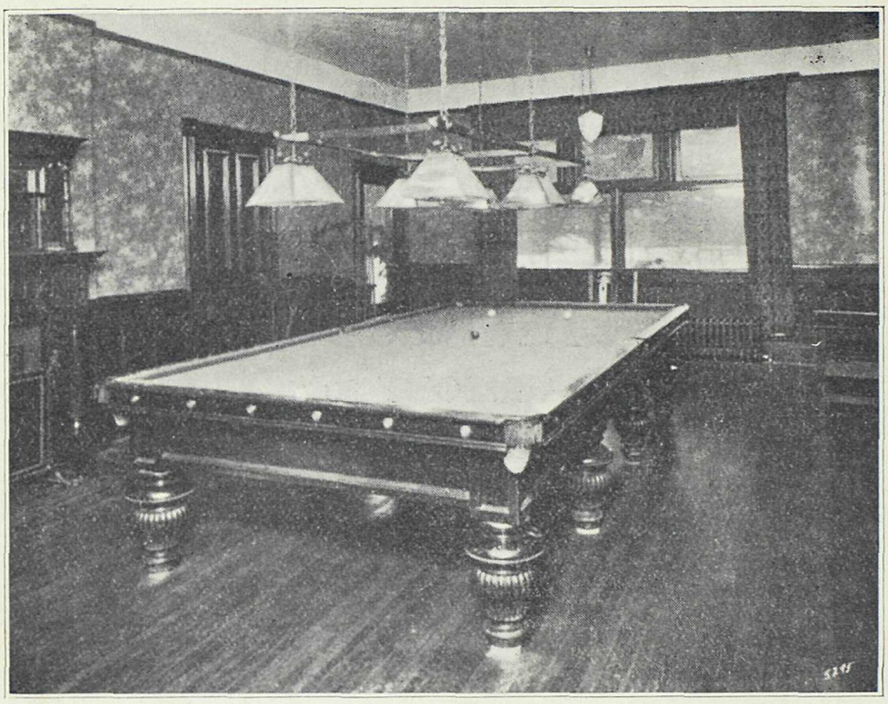

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