rule as regards hypermetropic astigmatism. Nebula up to $\frac{1}{2}$ allowed if other eye is emmetropic. Colour blindness and paralysis of external muscles of eyeball disqualify.

EXAMINATION OF VISION.

The examination of vision is conducted by means of Snellen's types, which are constructed on the principle of a visual angle of 5' for the whole letter and 1' for the strokes or lines forming the letter. The distance in metres at which each letter should be read is affixed to it, and the numerator of the fractions above given indicates 6 metres, or 20 English feet, the distance—a convenient one—at which the examinee is placed from the sheet on which the types are printed. The denominator indicates the particular type which can be read at that distance; $\frac{1}{4}$ indicates that type No. 6 can be read at 6 metres, and $\frac{1}{36}$ that type No. 36, which ought to be visible at 36 metres, is the smallest that can be seen at 6 metres. The fraction therefore indicates the amount of acuteness of form vision possessed by the eye; $\frac{1}{2}$ would indicate $\frac{1}{2}$, or 50 per cent. of normal vision. The examiner should possess some + and - lenses. The perception of colour is generally tested by means of Holmgren's wools, and the candidate should be made to match as well as name the colours. The correct perception of red and green is the most important point. The practitioner should have no difficulty in ascertaining and certifying normal vision; but in cases in which any abnormality of form or colour vision, refractive or otherwise, exists a thorough examination by an expert should be made, and his report, the exact refractive error of either eye and the glasses prescribed, should always be taken to the examining board by the candidate. Each eye ought of course to be separately examined by covering the other. Strong accommodation sometimes conceals a moderate degree of hypermetropia, and when accommodation is weakened by study or sickness defective vision, hitherto unsuspected, is revealed. This circumstance may lead to disappointment, and a special examination for possible hypermetropia by means of atropine or the use of a convex lens should always be made. The convex lens in such cases improves or at any rate does not impair vision.

HEARING.

"Impaired hearing, or discharge from one or both ears, or any disease of the external ear," is the formula used in the naval regulations. The Civil Service rules are almost identical, and the Army requires good hearing. The soundness of this sense is easily ascertained by ordinary conversation. If any doubt arises the hearing of each ear must be tested separately by means of a watch or tuning fork. The use of a second still watch may be advantageous in some cases.

SPEECH.

The importance of strong voice and ready accurate articulation in persons who have to give words of command quickly or under circumstances of emergency is obvious. The Army and Navy regulations, therefore, indicate "impediment of speech" as a cause of rejection. The Civil Service regulations are less absolute. "Stammering is in some respects a question of degree and of the duties of the proposed situation, but any considerable impediment in speech renders a candidate generally liable to rejection." A teacher or lecturer must necessarily possess a sound and ready faculty of speech.

DISCRETION.

Questions of physical fitness for various occupations under varying circumstances cannot all be anticipated or defined by rigid standards. A certain measure of discretion must therefore remain with examining boards and examiners regarding those minor infirmities which may or may not be held to disqualify, and the process of balancing is often a very delicate one. No doubt even as regards these, unwritten rules arise through practice and experience, and some degree of uniformity and continuity results, but a certain elasticity of judgment is inevitable in doubtful cases. Employers likewise reserve for themselves an option to accept or reject, and do not consider themselves necessarily bound by the advice received from medical boards and examiners. Parents and their advisers must be prepared for disappointment under circumstances of doubt.

CONFLICTING INTERESTS.

Official examiners act in the interests of their employers, and naturally incline to give them the benefit of well-founded doubts, while private medical advisers are prone to adopt the view most favourable to their clients; hence arises every now and then unpleasant criticism and antagonism. Official examiners, as far as my experience goes, are always ready and pleased to receive aid in the form of statements and certificates from family doctors and experts, and to give them due consideration and weight. Such statements should be representations of facts and not, as is too often the case, mere expressions of opinion or anticipations of a judgment which it is the business and duty of the examiners to form. The responsibility of advising selection or rejection rests with them, and they are generally in the best position to give due attention to the requirements of the service which they represent. In respect to these they indeed occupy the position of experts.

Differences of opinion between official examiners and private advisers arise not so much or often as regards questions of fact as with respect to the interpretation of, say, a cardiac murmur and the bearing of an admitted abnormality—defect or disease—on the question of future efficiency. When sound subjects can be obtained, it is unreasonable to expect employers to accept the unsound or doubtfully sound, and this consideration becomes of peculiar importance when the employer is the State and the servant a soldier or sailor.

Military and naval authorities will not willingly accept weedy, edentulous, or purblind officers or men when they can get robust youths with good instruments of mastication and sight. The best way of avoiding unpleasant conflict in these matters is to take measures that every fact relating to the physique and health of the candidate, whether favourable or unfavourable, shall be frankly and fully placed before the examiners, who are influenced by a generous desire to treat lads fairly and kindly as well as by a conscientious regard to the interests of the service on whose behalf they act. The worst thing that can happen in cases of rejection is the imputation to official examiners of incompetence or partiality—an attitude which is at once unjustified by the fact and inconsistent with that spirit of benevolent altruism which constitutes the ruling motive of the medical profession.

REMARKS

ON

THE NATURE AND TREATMENT OF PERNICIOUS ANÆMIA.

By RALPH STOCKMAN, M.D., F.R.C.P.E.,

Lecturer on Materia Medica and Therapeutics in the School of Medicine, Edinburgh; Assistant Physician to the Royal Infirmary.

(Continued from page 967.)

III.—THE IMMEDIATE CAUSE OF THE CONDITION.

SUCH causes as those mentioned in the first portion of this paper have generally been regarded as remote or predisposing, while the immediate cause has been set down by all writers hitherto, either to deficient blood formation or to excessive blood destruction.

Dr. Bradbury¹ thinks the destruction of red corpuscles so excessive that the blood-forming organs cannot regenerate them in sufficient amount. Pepper² has proposed the name "anæmatosis," for the condition to indicate his opinion that it is due to nonformation of blood, while Quincke is undecided which it may be. Pye-Smith holds it to result from a too rapid and extensive destruction of the red discs; and Mott,³ that it consists essentially in an excess of the normal blood-destroying function of the liver and spleen.

In passing I may protest against the assumption by Dr. Mott and others that the liver and spleen have ordinarily (or even in disease so far as we definitely know) any blood-destroying function. Red blood corpuscles probably disintegrate, just as any other tissue disintegrates, when they become old and effete, and there seems no special reason why they should be actively destroyed by any

other organ in the body. The liver (and also the spleen) is the ordinary storehouse for the reserve physiological iron of the body, and hence iron from the food or from the effete corpuscles is taken up into the liver cells. The same thing is seen when an iron salt is injected directly into the blood, no less than 50 per cent. of it being retained by the liver (Jacobi⁴), and it is also seen when arseniuretted hydrogen, toluylendiamine, or other drugs which weaken and lead to the disintegration of red corpuscles are administered to animals. Possibly the leucocytes do actively destroy damaged or old red corpuscles, but there is no special reason for believing that the liver cells do. Minkowski and Naunyn,⁵ after poisoning with arseniuretted hydrogen, observed in the liver, spleen, and bone marrow large numbers of leucocytes carrying partially broken-down red blood corpuscles, whereas in the blood of other parts such leucocytes were rare. The explanation is that a slowing of the blood-stream is necessary to enable the leucocytes to deal with effete or weakened red corpuscles, and hence although the breaking down occurs chiefly in the portal system, and especially in the liver capillaries, where the circulation is very slow, it occurs through the agency of leucocytes and not of the liver or spleen cells. The products of disintegration are simply stored in the liver, or excreted by it as bile pigment, bile acids, urea, etc. In health the iron from the effete corpuscles may be taken up directly from the blood or indirectly from leucocytes by the liver cells, but exact knowledge on these points is lacking.

In connection with the question of pernicious anæmia and excessive blood destruction, the clinical and experimental researches of Dr. William Hunter require special consideration, as they stand out pre-eminently for originality and completeness. Hunter, while regarding pernicious anæmia as a distinct disease due to excessive blood destruction, holds that the most constant pathological feature is a large excess of iron in the liver, and that this distinguishes it *post mortem* from all other varieties of anæmia. He has advanced the theory that a cadaveric body formed by a special micro-organism in the alimentary canal becomes absorbed, and in the portal vessels, and there only, disintegrates the red blood corpuscles. When these break up their iron is stored in the liver, while their pigment is excreted in the urine and in the bile, both of which secretions assume, therefore, a darker colour. By administering to animals toluylendiamine, pyrogallie acid, and other substances which lead to breaking down of the red corpuscles he was able to produce a condition of anæmia with excess of iron in the liver, and this he regards as strongly corroborating his theory. Dr. Hunter also lays great stress on the varying size of the spleen, the differences in which he attributes to more or less activity of this organ in blood destruction previous to death. Eichhorst, Quincke, and Schauman all state that the spleen is seldom enlarged; and any differences are probably attributable to the degree of fever present during life rather than to special activity in destroying red blood corpuscles.

Having examined the opinions held by others and, to a certain extent, criticised them, I now come to my own views on the nature of pernicious anæmia.

The evidence seems to me convincing that pernicious anæmia follows usually on well-recognised debilitating causes. The cause is sometimes, however, obscure, and is not detected, but we are rapidly reducing this unknown territory, and if it be remembered that only a few years ago all cases of tapeworm-anæmia were regarded as "idiopathic" we may hope in the near future to elucidate the hitherto unrecognised causes. Further, I do not think that a hypothetical destruction of red blood corpuscles by the liver cells or by a ptomaine or ferment can be considered the cause of the condition. I hold rather that anæmia from any cause induces in some persons degenerative changes in the whole vascular system; that these permit the occurrence of numerous minute internal bleedings, more rarely of external ones also, and that a persistent continuance of these leads ultimately to excessive anæmia and death. My reasons for these opinions are as follows:

The earliest observers, Combe, Addison, and Wilks, were all struck by the extremely fatty condition of the heart

muscle as found after death. Their observations have been confirmed by Ponfick⁶ and by all observers without exception. Ponfick extended them by showing that in anæmia following on very numerous conditions (chronic stomach disease, cancer, pregnancy, typhoid, bleeding, etc.) the heart was fatty degenerated, the vessels being also affected. Perl⁷ has shown that by bleeding dogs so as to keep them anæmic, fatty degeneration of the heart muscle can be induced.

In pernicious anæmia degenerative changes occur in the capillaries and small vessels also, although there is some dispute as to their exact nature. Biermer and others have described the change as fatty degeneration, while Müller and Eichhorst think it is a peculiar kind of degeneration of the walls whereby the red corpuscles readily pass through. The former says that it is not visible to the microscope, and suggests that it is simply a nutritive disturbance. Manz⁸ and Eichhorst have figured small capillary aneurysms on the retinal vessels, but they are not always present.

The exact nature of the change need not specially concern us, since all observers are agreed that numerous capillary hæmorrhages take place in consequence, and that bleedings from the nose, bowel, and other mucous surfaces also result. It seems to me that sufficient stress has not hitherto been laid on the important part which these numerous although small hæmorrhages play in producing "progressive pernicious anæmia." Continued small losses of blood lead to much more profound anæmia than a single large bleeding, and the patients gradually accustom themselves to get along with less and less hæmoglobin, so that they only apply for medical advice when very severely anæmic. It is no uncommon thing to find a case of chronic anæmia present itself for the first time with 20 per cent. hæmoglobin and 1,000,000 red corpuscles per cubic millimetre, whereas in a case where I examined the blood two days after an acute hæmorrhage had nearly caused death, I found 60 per cent. hæmoglobin and 3,000,000 red corpuscles. Laker⁹ records a case where a healthy man died next day from hæmorrhage following a stab, his hæmoglobin being then 70 per cent. of the normal. Chronic loss of blood is therefore much better borne than acute loss, and goes on to a much higher degree without causing death.

In pernicious anæmia the internal hæmorrhages are of the size of a pin-head to a linseed, or may be small or very large ecchymoses, and they are found in every organ of the body. I have several times examined them; but perhaps it will be most satisfactory to quote what a few writers of the widest experience in pernicious anæmia say about them. Biermer, who first described them in the retina, lays such special stress on their presence in other parts of the body that he includes their occurrence in his definition of "progressive pernicious anæmia." One patient he describes as dying in consequence of the enormous number of such capillary bleedings in the brain. Numerous other authors have described more or less severe nervous symptoms following on such bleedings into the brain and cord, namely, paralysis or paresis, sensory disturbances, twitchings of groups of muscles, epileptiform attacks, and loss of sight, smell, or hearing. Eichhorst says: "The bleedings are almost always of a capillary or petechial character. There is scarcely an organ which is not affected by them; on the skin, mucous membranes, in the brain, on the organs of the thorax and abdomen they are frequent and almost regular appearances." Müller gives a very full description of them, more especially in the brain and its membranes. He describes that a false membrane about the thickness of the retina is generally found on the under surface of the dura mater, and that this is yellow in colour, and is formed from the enormous number of capillary hæmorrhages which have taken place there. "Hundreds of capillary hæmorrhages may be distributed throughout the brain substance, and give to the wax-pale brain a red punctated appearance, so that one may justifiably use the comparison of Biermer and speak of purpura hæmorrhagica of the brain." They resemble small cut vessels, but the blood cannot be removed by slight pressure with the flat of the knife, and they are thereby distinguished from the infrequent red vascular points. Müller says that they may easily pass unobserved, as happened in all his earliest cases.

Pye-Smith says: "This hæmorrhage is as constant, or

almost as constant, a *post-mortem* occurrence as fatty degeneration. It is sometimes extensively distributed, as much as in purpura or malignant fevers, in variola or pyæmia, but it is rarely large."

These bleedings being so small disappear very rapidly and completely, this being assisted by the well-known tendency of the blood to resist coagulation in extreme anæmia (Eichhorst). They usually produce no special symptoms (unless they occur in the central nervous system or organs of special sense, and even in those very rarely), but if they do cause paresis or other symptom, this passes off in a very few days, and no trace of bleeding may be found *post mortem* owing to rapid absorption of the small extravasations. In some cases which I have examined after death the hæmorrhages have been so few that I have doubted very seriously whether they could ever have been very numerous, but taking into account their rapid absorption and the probability that with extreme anæmia the number must diminish just before death, I believe that in such cases they had previously been much more numerous.

Further, bleeding from the nasal mucous membrane is fairly common, and hæmatemesis, melæna, and hæmoptysis not infrequent. After death no local lesion may be found to account for these hæmorrhages.

The importance of these hæmorrhages must be considerable, because the effect of even a small loss of blood in an anæmic person is very striking. For example, in a woman, aged 41, whom I was treating for severe anæmia, supposed to be pernicious, and in whom menstruation had been absent for some months, the red corpuscles equalled 3,000,000 and the hæmoglobin 36 per cent. She began to menstruate and continued for six days, losing, according to her own account, very little blood. At the end of her period, although the corpuscles still numbered nearly 3,000,000, the hæmoglobin had fallen to 30 per cent.—a loss which she very slowly and with great difficulty made good.

These bleedings are, to judge from the exacerbations and partial remissions of symptoms, more numerous at one time than at another, and according to the amount of blood lost the case progresses rapidly or slowly.

The hypertrophy of the bone marrow, which has been so often described as a characteristic anatomical feature of pernicious anæmia, is not infrequently present also in cancer and other conditions, and is now generally, if not universally, regarded as an attempt on the part of the great red-corpuscle-forming organ of the body to cope with its excess of work and make up the blood loss. The same hypertrophy can be induced by bleeding dogs to anæmia, and is seen after chronic bleeding in man (Neumann).¹⁰ In some cases, however, the red marrow is not increased in amount, and this may depend on its not having had time to hypertrophy before death overtakes the patient. I believe that in many cases the red marrow gets exhausted from overwork, and there is no doubt that as age advances the blood is regenerated with much greater difficulty and slowness, this probably accounting to a large extent for most cases of pernicious anæmia occurring in middle life or later. I have been led to this conclusion partly from observations on anæmic people at different ages, but also from observation of two typical "bleeders." The one was a girl, aged 20, with a family history of hæmophilia, and subject to epistaxis, menorrhagia, and hæmatemesis. In two years I attended her during five attacks of anæmia, when she presented all the characteristic symptoms of extreme chlorosis, and each time she recovered readily under treatment with rest and iron. The other was a man, aged 45 when I first saw him, and at this time he presented all the most typical features of pernicious anæmia. He told me he had often been bloodless previously after bleeding, but had always responded to treatment with iron. This time iron and every other method of treatment failed; during a year he made practically no progress towards recovery, and died suddenly after hæmorrhages from the nose and bowel. As the first patient reaches middle life she will probably also have more difficulty in regenerating the lost blood, and will lapse into a condition of "pernicious anæmia."

I now come to an important part of the argument, namely, the endeavour to prove that these bleedings account satisfactorily for the characteristic symptoms of "pernicious

anæmia" as usually distinguished from chlorosis and so-called "simple anæmias." These symptoms are: (1) The extreme anæmia, along with a usually fat and well nourished appearance. (2) The yellow colour of the skin, blood serum, and fat. (3) The excess of iron in the liver and other organs. (4) The richness of the red blood corpuscles in hæmoglobin. (5) The excess of urobilin, and probably of other pigments in the urine. (6) The fever.

1. *The Extreme Anæmia.*—As has been previously pointed out, much more profound anæmia is brought on by chronic small bleedings than by one or more large losses. In the latter case the patient is very apt to die, whereas in the former the body has time to accustom itself to the altered conditions, and does so to an extraordinary degree. The large amount of fat is due to its not being oxidised owing to poverty of hæmoglobin, for it is well known that cattle can be fattened more rapidly if kept anæmic by bleeding. But further, if the diet be good and there is no organic lesion of the digestive organs, the blood plasma will not be deteriorated, and will afford sufficient nourishment to all the tissues. The great debility is doubtless due to fatty degeneration of the heart and (though to a much less degree) of the other muscles, which results from lack of oxygen rather than from lack of nutriment. It is really a want of hæmoglobin and consequent non-oxidation which induces the stoutness. Some cases are, however, thin and emaciated.

2. *The Yellow Colour of the Skin, Blood Serum and Fat.*—The more or less yellow colour of the skin and fat is due to altered hæmoglobin dissolved in the blood serum. So far as my own observations go the blood is generally more yellow than normal, and this is not due to the fewness of red corpuscles depriving it of its purplish hue, because the oedematous and serous exudations have also in some cases an unusually deep tint. Schauman, on examining the serum from a blister, found it of a deep yellow colour, but giving no characteristic spectrum, and Broadbent,¹¹ in one case, observed that the blood serum stained his hands of a colour which in its intensity and difficulty of removal reminded him of a nitric acid stain.

The pigment of effused blood is very apt to undergo this yellow change, the most familiar example being an ordinary bruise. In rabbits in which I had made subcutaneous hæmorrhages by cutting veins, after waiting until the clot had been apparently all absorbed, I found the connective tissue in the neighbourhood stained of a fine yellow colour. This yellow pigment gave an intense iron reaction, showing that it still contained part at least of the iron of the hæmoglobin. My friend, Dr. Berry Hart, also informs me that after a large hæmatoma of the broad ligament patients always become yellowish in a few days, the coloration not being due to jaundice. There can be no reasonable doubt that here, and in pernicious anæmia, the yellow pigment is derived from altered hæmoglobin, and that it circulates in the blood stream. Probably other derivatives of hæmoglobin also occur but with less frequency, as Gusserow records that in two of his cases the blood serum was brownish like thin coffee. The yellow tint varies a good deal in intensity, and its presence in any case justifies us in assuming that internal hæmorrhages are occurring, the depth of it giving us some sort of gauge of their extent.

3. *The Excess of Iron in the Liver and other Organs.*—Hunter, Mott, and others have laid great stress on the presence of excess of iron in the liver, and this has been advanced as a proof that the liver cells actually break down red corpuscles and absorb their iron. But iron, although usually too abundant in the liver in most cases of pernicious anæmia, is also found in great excess in other organs. Thus Grohe¹² describes it as present in the liver, spleen, kidney, pancreas, and surface of brain; Quinke in the liver, pancreas, and kidney; Mott in the liver, spleen, pancreas, and kidney. It is also found not only in the liver cells, but in the hepatic capillaries and connective tissue, as well as in the bone marrow.

Whatever opinion may be held regarding the liver and spleen as possible blood-destroying organs, it is hardly imaginable that the surface of the brain or the kidneys have any special function of the kind. The true explanation seems rather to be that the hæmoglobin of the numerous minute hæmorrhages becomes altered, and thus liberates in

the circulation a large amount of iron-containing pigment. This is deposited in the liver and other organs; if the bleedings be numerous the pigment is taken up by many organs, if few chiefly (but in my experience never exclusively) by the liver, which is a principal storehouse for iron in the body. Sometimes when the internal bleedings have been very numerous (or perhaps always) the red blood corpuscles are partly absorbed by the lymphatic vessels, and are found both in them and in the glands (Weigert¹³).

That the hæmorrhages are the real origin of the excess of iron in the liver is borne out, I think, by the following proofs. After an internal hæmorrhage there is found a large excess of iron in the liver, spleen, and other organs, which is derived from the effused blood. This is readily enough shown by experiments on animals, of which one may be quoted. A rabbit during ten days had four subcutaneous bleedings made, two about the size of a pigeon's egg, the others smaller. It was killed three weeks after the last, when the liver and spleen were found to contain a large excess of iron, and stained deeply with ammonium sulphide and other reagents. Microscopically, after staining for iron, stained granules, just as in pernicious anæmia, were seen abundantly in the liver and spleen cells. But after blood effusions in man the same thing has been observed. Thus Tillmanns,¹⁴ in a case of fracture of the pelvis with extravasation of blood where death occurred two months afterwards, found an excessive amount of iron in the liver, spleen, and lymph glands.

Purpura hæmorrhagica also furnishes us with an analogous condition. In one case Hindenlang¹⁵ found large quantities of iron in the liver, pancreas, and lymph glands, the dried liver containing no less than 1.246 g. Fe per 100 parts. In a case of purpura which I myself examined the liver contained 0.37 g. Fe per 100 parts dried and the spleen 0.29 g. Fe per 100 parts dried, amounts much beyond the normal, which is about 0.08 for the liver and 0.15 for the spleen. In malaria also, where the corpuscles are broken up by the specific organisms, a very large amount of iron-containing pigment is found in the liver, and in fact all over the body nearly. The evidence seems to me conclusive that in "pernicious anæmia" also the large excess of iron found in the liver and elsewhere is derived from the hæmoglobin of the internal hæmorrhages.

4. *Richness in Hæmoglobin of the Red Blood Corpuscles.*—Laache first pointed out that in pernicious anæmia the individual corpuscles have a normal amount of hæmoglobin, and that the hæmoglobin is diminished in the same ratio as the number of the red discs. This holds good for most cases, but not for all, as I shall presently show. Since the time of Duncan¹⁶ it has been known, on the other hand, that in chlorosis the hæmoglobin is diminished out of all proportion to the fall in red corpuscles, which is sometimes extremely slight or even nil. The explanation of the maintenance of the hæmoglobin value in pernicious anæmia is simple enough, namely, that the blood serum, liver, bone marrow, and other organs contain excess of iron, and hence there is plenty to draw on for the formation of new red corpuscles. In a case of purpura—in which disease, as I have previously pointed out, there is also in the viscera an excess of iron derived from the effused blood—Laache has carefully recorded the blood condition, and I find that in his case the hæmoglobin was in equal proportion to the number of corpuscles, and remained so during the regeneration of the blood. Here also, just as in pernicious anæmia, there was plenty of iron available in the liver and other organs for the manufacture of new red corpuscles.

5. *The Excess of Urobilin and other Pigments in the Urine.*—The urine is usually light in colour, sometimes however it is dark, and Hunter and Mott have shown that this is due to excess of urobilin. Urobilin (hydrobilirubin) is a derivative of hæmoglobin, and these authors regard its excess in the urine as a proof of the active destruction of red blood corpuscles in the liver or portal system. It is present in all normal urines, and in some cases it has been observed to form only after exposure to the air, being derived therefore from some pre-existing yellow pigment. Neubauer and Vogel¹⁷ state that it is present in excess in fevers, in many other conditions, and in scurvy, while Hecht¹⁸ found it in the urine of patients with blood extravasations. As it is

present in excess during scurvy and blood extravasation there is no doubt that it can be formed from internal hæmorrhages, and this I take to be its origin in pernicious anæmia.

The increase of the biliary pigments can be accounted for in the same way. The excess of indican in the urine of some cases, as described by Senator¹⁹ and by Müller, has a different origin. This pigment is a derivative of indol, and excess of it is found in the urine when an abnormal amount of putrefactive change is going on the intestinal canal. Its occurrence is, therefore, simply an outcome of the mal-digestion so common in extreme anæmia. The increase of iron in the urine found by Dr. Emerson Reynolds and by Damaskin is probably due to one of these yellow iron-containing pigments derived from hæmoglobin being excreted through the kidneys.

6. *Fever.*—The fever has been ascribed by several authors to irritation of the heat centres in the nervous system by the small hæmorrhages. Into this, however, I have made no special inquiry and am unable to give an opinion. I may point out that feverish attacks are extremely common in chlorosis also, although they are seldom so severe as in pernicious anæmia, and hence the high temperature may depend in some way on the blood conditions. It has been shown that animals rendered anæmic by bleeding are subject to feverish attacks, but the explanation of this has never been definitely given.

Having shown, therefore, that the ordinary phenomena of "pernicious anæmia," as distinguished from other anæmias, are explainable on the theory that it is due to excessive loss of blood by internal capillary bleedings, sometimes aided by external hæmorrhage, I wish to point out, and this I think further confirms my views of its causation, that certain cases do not present some of what we have been asked to regard as the distinctive pathological changes. I refer especially to the presence of large amounts of iron in the liver and to the corpuscular richness in hæmoglobin.

To begin with the liver. In the liver in health is largely stored the iron needed for physiological purposes, which is present in various organic combinations resembling hæmoglobin in constitution, but more simple in structure (Zaleski²⁰). It has been shown by Schmiedeberg,²¹ that after external hæmorrhage this iron may wholly disappear, having been used to form new corpuscles, and I have confirmed this in dogs and in rabbits. The animals were bled from a vein repeatedly until there was a very distinct fall (20 to 50 per cent.) in the corpuscles and hæmoglobin. The livers when treated with ammonium sulphide gave no iron reaction, and on incineration a mere trace. They gave up no iron compounds to Bunge's fluid (10 parts of 25 per cent. hydrochloric acid, and 90 parts of strong alcohol).

In some cases of pernicious anæmia, brought on and maintained by external hæmorrhages chiefly, little or no iron is to be found in the liver. Even if internal bleedings are pretty numerous in such cases, the iron resulting from them is so soon used up in making new red corpuscles that the liver may contain little or none after death. I have met with one case of this kind in a woman, aged 51, who presented the clinical features of pernicious anæmia, including the yellow colour and stoutness. The anæmia was due to a bleeding uterine polyp; the hæmoglobin amounted to only 10 per cent., and the red corpuscles to 1,000,000 per c.mm., with marked poikilocytosis and numerous nucleated red corpuscles. *Post mortem* the liver and spleen contained the merest traces of iron (probably fully accounted for by the blood in them), and the bone marrow was in a typical condition of hypertrophy.

Quincke²² describes a man, aged 45, as a case of pernicious anæmia due to severe and repeated epistaxis, lasting for eighteen months. After death no iron was found in the liver.

But the most striking evidence is to be found in cases of pernicious anæmia due to the *ankylostoma duodenale*, which are caused by external blood loss, the parasites sucking blood from the vessels in the intestinal wall of their host. That is to say, the anæmia is due chiefly to external blood loss, although internal capillary hæmorrhages also occur from changes in the walls of the vessels consequent on anæmia. Dr. Beaven Rake estimated the iron in the liver in five cases

one gave a trace of iron, two others gave 0.0123 g. and 0.0228 g. per 100 parts of dried liver respectively; the other two gave 0.26 g. and 0.20 g., these two last being slightly over the normal, the others being very much under. He concludes that the internal anæmia must be due to blood loss, and not to blood destruction. If it were the latter, iron would be found in the liver in great excess (say 0.6 g. or more in 100 parts).

But external blood loss of this kind affects the relation between the number of red corpuscles and their individual richness in hæmoglobin, because the iron of the hæmoglobin is lost to the body, and is not stored in it as occurs when the bleeding is internal. Hence in my case previously cited, where the pernicious anæmia was due to uterine bleeding, the hæmoglobin was reduced to one-tenth and the corpuscles only to one-fifth of the normal. Lappert²³ states also that in anæmia from ankylostoma the hæmoglobin is more diminished than the red discs, and this he regards as very remarkable, because, "as it is a secondary anæmia, the contrary would be expected." Leichtenstern²⁴ confirms this, as he found the corpuscles usually reduced to 25 or 30 per cent. of the normal, while the hæmoglobin was reduced in one such case to 16 per cent. In these cases, however, the simultaneous occurrence of both external and internal bleedings in varying proportions and amounts must complicate such relationships very considerably.

It is evident, however, that in some cases of undoubted "pernicious anæmia" there may be little or no iron in the liver, and the corpuscles may be individually very poor in hæmoglobin. These features, like all other individual features of this condition, are therefore not pathognomonic.

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(To be continued.)

ON THE TREATMENT OF ACUTE PNEUMONIA BY ICE CRADLING.¹

By P. BLAIKIE SMITH, M.D.,

Physician and Lecturer on Clinical Medicine, Aberdeen Royal Infirmary.

THE subject of ice cradling first attracted my attention about two years ago,² when Dr. W. Soltau Fenwick reported most encouraging results following the adoption of certain antipyretic proceedings in cases of acute sthenic pneumonia. Out of 108 cases treated by sponging or ice cradling, the mortality amounted to 10 per cent., while the death-rate in 552 cases treated in the ordinary way reached 23 per cent.

During the past two months I have had under my care in the Aberdeen Royal Infirmary, five cases of acute pneumonia, and all of these have been treated, almost exclusively, on the lines recommended by Dr. Fenwick. All the patients recovered.

The mode of carrying out the treatment which I adopted was practically identical with that described by Dr. Fenwick. The patient, clothed in a cotton nightdress, is placed in bed with a sheet covering the mattress. Over the body and legs are arranged two large cradles, extending from the shoulders to the feet. To the arches of the cradles are attached six or eight small pails filled with ice; a thermometer is suspended from the centre of the upper cradle, and both the cradles are covered, first with a blanket, second with a waterproof, and lastly with the ordinary coverlet. The ice pails are recharged as the ice melts (usually about every two or three hours). The temperature of the air under the cradles, as well

as that of the patient, is ascertained at regular intervals, and the treatment is maintained until the temperature becomes normal.

All the cases treated on the above plan were examples of acute sthenic pneumonia occurring in men whose ages ranged from 30 to 40; the whole of them were complicated with pleurisy, and in one instance the pneumonia was double.

With the view of avoiding wearisome repetition I shall relate only two of these cases. In every instance I kept a record of three temperatures as the disease progressed: the temperature of the ward, the temperature of the air under the ice cradles, and the patient's body temperature every four hours. I also determined the temperature of the air under the blankets before the cradles were placed in position, and after they were withdrawn at the termination of the case, that is, the temperature of the bed when prepared in the ordinary way, and, finally, I ascertained the temperature under the cradles before the ice pails were suspended, and I observed what effect was produced on this temperature after ice cooling had been fairly established.

The first case is that of a man aged 30, who was admitted into the infirmary on the fifth day of his illness. He was suffering from inflammation of the whole of the left lung complicated with pleurisy. His pulse was 106, his respirations 41, and his temperature almost reached 103° F. In this case, before antipyretic treatment was commenced the temperature of the air under the blankets was 88° F. After four hours' simple cradling it fell to 72° F., after four hours' ice cradling it went ten degrees lower, and thereafter during the maintenance of the treatment it oscillated between 60° and 70° F. until the eighth day, when crisis occurred, and the treatment was discontinued. Soon after the removal of the cradles the bed temperature rose to 81 F. With reference to the effect of the treatment on the body heat, I cannot say that any marked depression was observed. The temperature gradually, and in a somewhat fluctuating manner, declined, the fall becoming more rapid as the crisis approached. The pulse and respiration rates, and their ratio to each other, seemed practically unaffected by the treatment.

x, temperature under bed clothes before cradling; x x, temperature under bed clothes after four hours' simple cradling; x x x, temperature under bed clothes after four hours' ice cradling; a, ice pails removed; b, cradles removed; c, temperature under bed clothes.

The second case is also that of a man who was admitted on the fourth day of pneumonia of the apex of the right lung complicated with pleurisy. At the commencement of this case the bed temperature was unfortunately not ascertained, but after the cradles had been in operation for two hours (without ice) the bed temperature stood at 80° F., the body temperature being 105.4°. The pails were then filled with ice, and in two hours more the bed temperature had fallen to 73° F., but there was no material alteration in the body temperature. For the next few days—until the eighth day, when

¹ Read before the Aberdeen, Banff, and Kincardine Branch of the British Medical Association.

² Lancet of January 31st and February 7th, 1891.