what we have already found to be the best method of treatment clinically. In pernicious anæmia arsenic is recognised as our most valuable drug, especially when borne in large doses (10 to 15 minims). Now, arsenic lessens the glycogenic function of the liver, and it also hinders the crystallisation of hæmoglobin—that is, it influences in an important way the properties of both the organ and the substance which are in question. It is, therefore, not mere vague theorising to believe that it may aid the liver in hæmogenetic labour.

We also find that the recovery of patients suffering from pernicious anæmia is hastened by the administration of iron. This, also, is what we should expect on my theory. When once the liver has been improved by arsenic, so that it can again perform its hæmogenetic function, then the case becomes one of ordinary anæmia, and iron acts as it does on this latter disease. There is now much evidence and authority in favour of the statement that pernicious anæmia is not necessarily fatal. I saw last summer, in consultation with Dr. Haig of Coventry, an old gentleman whom I was told Dr. Broadbent had previously diagnosed as suffering from pernicious anæmia. You will agree with me that no physician is to be more trusted in such a matter than Dr. Broadbent; yet, when Dr. Haig and I examined the blood, it was normal as far as the corpuscles were concerned, and I have heard quite lately that the patient is much improved in general health, and quite cured of the general eczema for which I saw him.

With regard to the administration of iron in anæmia, it is very rarely that I do not at once begin with it, except in cases of severe gastric disturbance. When the appetite is wanting, and the stomach sensitive with atonic constipation, I order a tonic of nux vomica and soda with peppermint water and tincture of hop, adding to it, if there be much nervous upset, 10 to 15 drops of bromidia. This I give shortly before each meal, and after the meal this pill—*aloes socotrinae*, gr. j; *extr. belladonnae*, gr. ½; *ferri sulph.*, gr. j; *conf. rosæ*, q.s.; giving sufficient of these—one to six daily—to keep the bowels well open. So soon as the digestion has improved I add Bland's pills, one to six a day; and when the appetite becomes good, cease to prescribe the mixture. You perceive that I adhere to the old-fashioned form of iron which is contained in Bland's famous pill. Very rare indeed are the cases of anæmia where I have found it ill borne; then I have met with fair success with Denaeyer's peptonate of iron or liq. *ferri dialysati*, which latter I remember Dr. Wade speaking well of for cases which could not stand the cruder forms. The theoretical advantage of Bland's pill is that in it you prescribe ferrous carbonate in a nascent condition; but the Addendum to the *Pharmacopœia*, 1890, and Mr. Martindale also, state that whereas a 5-grain pill should contain a grain of ferrous carbonate, yet before it can be dried and coated, quite half of this is converted into ferric oxide. If this be so, probably before the uncoated pill is swallowed, very little carbonate remains at all; hence the desirability of coating them, and more especially of the bi-palatinoid disc which prevents the two ingredients mixing till they reach the stomach, and should, therefore, give the full proportion of nascent ferrous carbonate. In private I prescribe these bi-palatinoids, but for hospital practice they are too expensive. Dr. Mackenzie uses *ferri sulph. exsiccatum* with the carbonate of potassium, but Mr. Campbell, our able chemist at the Queen's Hospital, tells me that this salt is nearly always overheated in the process of drying, and an insoluble basic sulphate to some extent produced, which evidently cannot be converted into ferrous carbonate. Whatever form of iron be employed, it must be given in large amounts; many anæmics will take 30 or more grains of Bland's pill daily, and will do much better on this than any less quantity; the iron should be continued, too, for two or three months after the patient feels quite well, and has recovered his proper colour.

FARM LABOUR FOR THE INSANE.—The City of New York maintains a farm for its pauper lunatics. It covers 1,000 acres, and was purchased for 30,000 dollars as an experiment during the mayoralty of Mr. Edsom. Four hundred patients, all men, ranging in age from 35 to 70, live on the farm, being housed in nine one-storey pavilions. They work the farm, and conduct a shoe shop, laundry, and blacksmith's shop. It is anticipated that within a limited time the institution will become self-supporting.

ANÆMIA AS A CAUSE OF PERMANENT HEART LESION.

By HENRY HANDFORD, M.D., M.R.C.P.,
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I HAVE already¹ elsewhere referred to two cases in which anæmic murmurs persisted for over two years. Since then I have been able to add others. From a large experience I believe that in numerous instances of anæmic dilatation apparent recovery is due to compensatory hypertrophy, the cavities remaining permanently more or less dilated. And in a minority of these, especially when complicated by the effects of exertion during the anæmic state, this (permanent) dilatation of the left ventricle is sufficient to cause leakage through the left auriculo-ventricular opening. I have seen nothing to confirm the suggestion thrown out many years ago by Dr. Goodhart that "anæmia, by the dilatation it sets up, and the regurgitation through the mitral valve, may be the etiological factor in the unexplained considerable minority of cases of mitral stenosis in women which cannot be attributed to rheumatism." But to decide this point individual cases must be kept under observation for several years.

The influence of anæmia in causing fatty degeneration of the heart has been placed beyond dispute. Neither is the common occurrence of dilatation actively disputed now, the only questions remaining being:

1. The proportion of cases of anæmia in which recognisable dilatation is found?
2. The relative affection of the right and left ventricles?
3. Whether anæmia alone is a sufficient cause of cardiac dilatation? And if not, what are the contributing causes?

With regard to the first, I have² already expressed my conviction that cardiac dilatation occurs in the great majority of cases of severe simple anæmia, and it forms by far the most important complication to which they are liable.

And in the second place I have given grounds for believing that the right ventricle is generally more affected than the left. I have no space to pursue either of these points further here.

But the third question requires a fuller answer.

I have referred³ to what I have termed "gastric cases of anæmia," and to others arising from various causes, but entailing confinement to bed from the commencement, in which no murmurs and no dilatation could be detected. The following is a typical example of the latter form. A girl of 14, during a very severe attack of typhoid fever, suffered three very copious hæmorrhages and became in consequence profoundly anæmic. But notwithstanding that her thinness facilitated a very exact examination, no murmurs and no evidence of dilatation were ever detected.

The causes contributing to the dilatation are many, but the two chief are high vascular tension, which may arise from various conditions, and exertions.

Dilatation from over-exertion, whether sudden or long continued, is well recognised; but I do not refer to what would ordinarily be termed over-exertion.

The late Dr. Sibson⁴ showed that heart affections were far more frequent, as complications of acute rheumatism, among young women who had been exposed to the laborious work of domestic service than among females generally of the same age who had not been so exposed. In many of these cases it is probable that the mitral systolic murmur observed was due to dilatation rather than endocarditis.

As the result of much observation, I have become convinced that great care is necessary in protecting the patient against any exertion which causes breathlessness until the anæmia is thoroughly repaired.

If the importance of anæmic fatty degeneration of the heart is recognised, the ease with which very slight exertion may cause dilatation will be understood. And when with the degeneration the general loss of "tone" and contractile power is appreciated, we shall see the unreasonableness of supposing that the dilatation is gone and the heart completely rehabilitated, because after a fortnight or three weeks' treatment by

¹ *International Journal of the Medical Sciences*, December, 1890.

² *Loc. cit.*

³ *Loc. cit.*

⁴ *Collected Works*, vol. iii, p. 259.

iron, etc., the murmurs have disappeared *in the upright position*.

Anæmic dilatation, like all other forms, is due to increased peripheral resistance or diminished contractile power of the heart, or both, together with undue distensibility of the ventricles. In no other form of dilatation is such speedy restitution assumed, and there is no sufficient ground for assuming it in anæmic dilatation.

The persistence of a moderate or slight degree of dilatation is common, and is a frequent cause of the numerous relapses. The heart's apex remains displaced upwards and outwards, and for weeks or months the murmurs can be detected when the patient is recumbent. But occasionally the dilatation is sufficient to cause permanent mitral regurgitation. The mitral systolic is one of the least frequent of the so-called anæmic murmurs, but it is the most important, as it undoubtedly indicates regurgitation through the left auriculo-ventricular orifice. It is conducted towards the axilla like other mitral regurgitant murmurs. And, moreover, it is of all the anæmic murmurs the one most likely to remain permanently. Cases where the right ventricle remains dilated without the left are probably more frequent; but it is unusual for the dilatation to remain of sufficient degree to allow of the production of a regurgitant murmur in the upright position.

From these considerations it follows that rest and heart tonics, as well as aperients and iron, are of the greatest importance in the treatment of anæmia, especially during the first two or three weeks.

The following is a typical case (among several) where a persisting cardiac affection prevented the woman obtaining employment.

CASE.—H. S., aged 22, a very well-developed, strongly-built woman, was born and lived in a country village until four years ago, when she came as a ward-maid to the General Hospital. She had never suffered from rheumatism or any other serious illness, but enjoyed robust health. Her work was heavy, but she attracted the attention of her fellow workers by her physical strength. After about a year she had lost most of her colour, her appetite failed, she became very anæmic, and, as is usual in such cases, took a dislike to meat. In January, 1890, she came under my care on account of a temporary catarrh, and, on examining the chest, I found evidence of cardiac dilatation in increase of dulness and displacement outwards of the apex. In addition to the pulmonary and tricuspid murmurs common in anæmia there was a distinct mitral regurgitant murmur propagated into the axilla, suggesting dilatation of the left ventricle as well as the right, and relative incompetence of the mitral valve. I expressed the opinion at the time that there was considerable probability of the mitral murmur remaining permanent. It is still audible, though much diminished in intensity. A few months after I first saw her, when she had recovered her general health, she became an attendant on probation at the Borough Asylum. But though she looked robust, it was soon noticed that she became breathless on slight exertion. The medical superintendent, Dr. Powell, detected a mitral systolic murmur, and rejected her. She was sent to the seaside for eight weeks, taking iron and tonics all the time, and after that had very light work for two months. She again applied at the asylum, but, the murmur persisting, she was again rejected.

About two years after I first saw her I had the opportunity of thoroughly examining her chest. There was now no appearance of anæmia. She considered herself perfectly well, and all her functions were regular; but it was noticed that she still became breathless on exertion. In the recumbent position the apex was in the fourth space, $4\frac{1}{2}$ inches from midsternum, and in the nipple line. There was also pulsation in the second and third left spaces, close to the sternum, and in the episternal notch. The latter was venous, as is common, and was due to slight upward displacement of the left innominate vein. The upper limit of deep cardiac dulness reached the middle of the second left space. This, with the position of the apex in the fourth left space, indicated some upward as well as outward displacement of the heart. The pulse was 88, regular, small, and of moderate tension. There was a systolic murmur at the apex, propagated 2 inches towards the axilla. Over the right ventricle a loud systolic murmur (indicative of tricuspid regurgitation) was audible, of lower pitch, and having two points of nearly equal inten-

sity, one over the fourth left cartilage, and the other in the third space $3\frac{1}{4}$ inches from midsternum. Between either of these points and the left apex under the nipple the murmur was almost entirely lost. On auscultating further up the chest on the left side another loud systolic murmur of distinctly higher pitch than the tricuspid was detected, with the point of maximum intensity in the second left interspace, but audible from the third costal cartilage to the left sterno-clavicular articulation. The pulmonary second sound was accentuated. On the patient taking a deep inspiration and holding the breath, the pulmonary murmur became scarcely audible, while the tricuspid one was made decidedly louder. In the aortic area a faint systolic murmur was audible from the second right space to the right sterno-clavicular articulation. The second sound was normal.

On the patient standing the pulse rose to 96. The systolic murmurs in the pulmonary, aortic, and tricuspid areas disappeared to such an extent that they were not at first detected by one of the house-physicians to whom I was demonstrating the case. At the apex the systolic murmur was distinct, and propagated for 2 inches into the axilla, but was not quite so loud as in the recumbent position. The upper limit of cardiac dulness had fallen to the upper border of the third rib, and the apex was beating in the fifth space in the nipple line. This movement of the heart, upwards or downwards to the extent of $\frac{1}{2}$ to 1 inch or more, according to the position of the body, is common, and is one of the chief causes of the increase of the pulmonary murmur in the recumbent position as I pointed out in 1890⁵. The pulmonary artery, often dilated, is pressed against the unyielding aorta. The next most important cause is the rise of vascular tension in the recumbent position, as indicated by touch and the slowing of the pulse increasing the work of the ventricles.

ON HYPERTROPHY OF THE SPLEEN IN INFANTS.

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AND

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SOME years ago we systematically took notes of all the young children presenting notably enlarged spleens who came under our observation at the Victoria Hospital for Children. In a recent discussion of a paper by Dr. Walter Carr at the Medical Society of London, one of us gave a short summary of our results, and we now venture to present our observations in more detail.

During the course of two or three years we collected 68 cases of obvious enlargement of the spleen, but 5 of these were instances of early general tuberculosis in which there was a special incidence of that disease on the spleen, just as there may be occasionally on groups of the external lymphatic glands simulating lymphadenoma. In these cases of tuberculous spleen the viscus may reach to the level of the umbilicus or beyond, whilst as yet the symptoms of a more generalised tuberculosis require careful investigation for their recognition. The remaining 63 cases all bear on the discussion initiated by Dr. Carr, for in none could the enlargement be attributed to new growths, parasites, gummata, amyloid degeneration, leukaemia, malaria, circulatory disturbance arising from heart disease, or any other of the well known and definite causes of increase of size. The ages of the children, it is important to notice, ranged from 4 months to 2½ years, and nearly all the cases occurred in the period when rickety bone changes set in and progress. Several times we noted that where one child of a family was affected the next one born became similarly diseased. The spleen varied in size from a distinctly felt projection below the ribs to a tumour reaching to the iliac crest, or pelvis, or across the mid-line into the right half of the abdomen. In the majority the spleen reached half way to or beyond the umbilical level, and in a good many further still. Nearly all the children

⁵ Loc. cit.