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.

By T. COLCOTT FOX, M.B.LOND.,

Physician for Diseases of the Skin to the Westminster Hospital, formerly Physician to the Victoria Hospital for Children;

AND

J. B. BALL, M.D.LOND.,

Senior Assistant-Physician to the West London Hospital.

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.

were thin and flabby, wasting somewhat, or rarely distinctly wasted. Nearly all presented various degrees of anæmia, and some such a characteristic pale or chlorotic waxy tint as to make one instantly suspect the existence of an enlarged spleen. Marked anæmia was not, however, invariably present, even when the spleen was considerably enlarged. The liver was almost always abnormally big also, and the external lymphatic glands frequently hard and easily detected in the groins, axillæ, or neck. We have notes of examinations of the blood in 6 cases attended with very marked anæmia. In all there was a considerable diminution in the percentage of red blood corpuscles, and in two cases, towards the close of life, not only a relative increase of white corpuscles, but apparently a positive, though very slight, leucocytosis.

It is only exceptionally that the splenic enlargement leads directly to a fatal issue. In the large majority of cases treatment by cod-liver oil and iron is apparently the cause of a steady improvement in all the symptoms present, with concurrent involution of the spleen, even when very large, the liver, and lymphatic glands, and disappearance of the anæmia. But in a certain small minority (5 out of the 63 cases) the spleen appeared to have received an impulse to hypertrophy which nothing could control, and which was not shared in by the liver or lymphatic glands. Arsenic, phosphorus, quinine, iron, iodide of potassium, mercurials—externally or internally, and all other drugs are alike without effect in such cases; and the child gradually succumbs with the onset of localised oedema, slight albuminuria, and scanty purpura; unless, indeed, some intercurrent lung attack supervenes and ends the scene. In all the spleens we have examined microscopically, or which Dr. Hebb (of the Westminster Hospital) has kindly investigated for us, the enlargement appeared to be purely hypertrophic, with, perhaps, some excessive formation of fibrous tissue. We have never met with the condition described by Sir William Jenner.

To what cause is this splenic hypertrophy to be attributed? From one point of view a satisfactory answer might seem to be easily obtainable, for we could not positively exclude the presence of rickets in a single one of the series of cases. In almost all the presence of rickets was clear and distinct, often very marked, and in only a few doubtful, that is, there was an absence of any definite bone lesions. If, however, we are to include as rickety changes craniotabes and the association of such symptoms as malnutrition, debility, head sweats, and throwing off the bed clothes, then in no case could rickets be excluded. One point, however, is clear, that if an enlarged spleen be a part of the diseased state called rickets, then it is a symptom by no means always present; it may exist without any notable or even definite bone lesions either in the way of softening or enlargement, and may long precede any other sign of rickets. In favour of the rickety nature of the condition may also be adduced the age at which these spleens enlarge, the favourable effects of treatment by cod-liver oil and iron, and the frequency with which the enlarged spleens are met with in rickets. In a series of 105 consecutive cases of rickets showing distinct changes in ribs and long bones we found the spleen enlarged in 25, or rather more than 14 per cent. In another series of 84 very marked cases of rickets the spleen was enlarged in 34, or about 40 per cent.

But the solution of the problem is not so easy; indeed, it is exceedingly complex, owing, first, to the fact that enlargement of the spleen occurs in inherited syphilis, and is indistinguishable, clinically and often histologically, from the hypertrophy met with later on in rickets; and, secondly, to the imperfection of our knowledge concerning the influence of syphilis in causing rickets. The conclusion to which we came, after prolonged and painstaking observations on children recorded in a systematic manner, was that, although rickets cannot rightly be described as a direct outcome of the syphilitic infection, yet in a great number of instances syphilis, by profoundly disturbing the nutrition, indirectly leads to rickets. In addition to the statistical evidence, which is very strong, one of us confesses to an impression received by witnessing the evolution of rickets in all the successive children of two families of rank. The children were brought up, largely in the country, under every advantage, and were born of healthy young mothers, but procreated by fathers who had suffered from syphilis not long before marriage.

Studying now the possible influence of syphilis more closely, we find that in our 63 cases of enlarged spleen 26, or about 41 per cent., were undoubtedly the subjects of inherited syphilis. As for the rest, the presence of syphilis could neither be excluded nor affirmed positively, though in a few its existence appeared most improbable. Everyone who has attempted a similar inquiry will bear witness to the difficulty of arriving at a decision possessing anything like scientific accuracy. In several instances, where we were inclined to conclude that syphilis was absent, we found, on subsequent inquiry, that the father had suffered from syphilis, or it happened that the next succeeding child was unquestionably syphilitic. On the other hand, in a large proportion of cases, a history of some eruption, often of short duration, about the buttocks in early infancy, or of snuffles, more or less chronic or recurrent, was obtained, but no one with experience will affirm positively the presence of syphilis from such a history alone. We have seen most extensive and typical syphilitic bone lesions occur in an infant who never presented any eruption, and anyone who has observed the successive infants of a syphilitic family will appreciate the difficulties attending an inquiry instituted at a later stage of their lives.

Let us now see what occurs in inherited syphilis; and for this purpose we refer to systematic notes of 155 cases, about which there can be no question. Many of these cases we followed up over a series of years. In 75, or about 48.4 per cent., the spleen was found by palpation to be enlarged at some time or other, and this result agrees fairly well with Dr. Gee's conclusions. Dr. Barlow¹ thought the percentage of enlarged spleens in inherited syphilis still greater, and we also, from experience of necropsies, have some grounds for thinking this probable. The percentage arrived at by various observers will probably vary, because the splenic enlargement is frequently not great; it is necessary to examine and re-examine the child at favourable opportunities, and experience shows that a certain training is necessary for correct observation in this matter. Parrot affirmed that, of all the viscera, the spleen is most frequently affected in inherited syphilis, and he ascribed it to retardation of the blood circulation in the liver.

There is now a good deal of literature dealing with the influence of syphilis, whether inherited or acquired, in producing enlargement of the spleen, but we need only quote here Professor Haslund's observations,² kindly epitomised by Dr. Rasch, of Copenhagen. In 154 necropsies of children dying with inherited syphilis in the Municipal Hospital of Copenhagen in the years from 1866 to 1880 the spleen was found without pathological alteration 96 times, and diseased in 58 cases, or nearly 38 per cent. Of the latter, there was simple hyperplasia in 14 cases, hyperplasia with induration in 31, soft hyperplasia in 10, infarct in 1, fibrinous membranes in the serosa in 1, and in 1 thickening of the capsule with adhesions to the surrounding organs. Among the cases of hyperplasia, fibrinous membranes existed in 10 on the surface of the organ, and in 4 local thickening of the capsule. Tuberculosis of the spleen occurred in 8 cases, always connected with tuberculosis of other organs, and twice with gummata of various organs, but in none of the cases were gummata or amyloid degeneration of the spleen detected. We do not possess information about the ages of these children, and think it probable that the necropsies were not confined to infants, which would account for the percentage of diseased spleens not being still larger. It is interesting, and important for our argument, to compare these results with those obtained by Professor Haslund at the necropsies of 44 adult syphilitics. In 14 cases the spleen was normal, in 27 there was hyperplasia (11 times the soft, in 16 the indurated form), in 2 thickening of the capsule, in 4 adhesions to the surrounding organs. From these and other observations it is established that, though the spleen is not very often the seat of gummata, yet it undergoes enlargement in both acquired and inherited syphilis with great frequency.

The next point is to determine the period in the evolution of syphilis when the enlargement occurs. Weber,³ for instance, states that in acquired syphilis in adults 75 per cent. have an enlargement of the spleen which may be detected

¹ *Path. Soc. Trans.*, vol. xxviii.

² *Miltens Forhold under Syphilis, Syphilitis Tidende*, 1882.

³ *Deutsch. Archiv f. klin. Med.*, 1879.

eight to twelve weeks after infection, shortly after the appearance of general symptoms, and that it usually continues for four to eight weeks. Parrot again asserts that an enlarged spleen is nearly constant in, and one of the best signs of the malady in infants attacked with, inherited syphilis not having passed one month, but that after three months and especially six months this hypertrophy becomes exceptional. This point is obviously one of much importance in our inquiry, and our observations, as well as those of Barlow and others, are directly at variance with Parrot's conclusions with respect to the frequency of an enlarged spleen in inherited syphilis after three months. Haslund's observations also seem opposed to Parrot's in this matter. Both clinical and pathological evidence would appear to demonstrate that this splenic enlargement is often a chronic process.

We next present a table (as simple as possible to economise space) showing the ages at which the spleen was found enlarged in our cases of inherited syphilis. It is necessary to bear in mind the age at which the symptoms of syphilis usually evolve, and to note that many of the older infants had apparently never been treated previously. To all grey powder was administered, to some for considerable periods, to others only intermittently according to circumstances of their attendance, etc.

Ages at which the Spleen was observed to be Enlarged in Cases of Inherited Syphilis.—1 at 5, 6, and 6½ weeks respectively; 1 at 7 weeks (early R at 11 months); 7 at 2 months (in 1 almost, and 1 quite, to umbilicus, 1 R at 15 months); 1 at 8½ weeks (R at 14 months); 4 at 9 weeks (1 R at 8 months); 2 at 10 weeks (1 R at 9 months); 1 at 11 weeks (spleen to iliac crest); 10 at 3 months (in 3 R developed); 3 at 3½ months (in 1 R developed); 10 at 4 months (in 1 spleen to umbilicus, in 4 R developed); 7 at 5 months (in 2 spleen very large, in 2 R developed); 5 at 6 months (in 1 R); 4 at 7 months; 3 at 8 months, all rickety; 3 at 10 months, all rickety; 2 at 11 months (both rickety); 3 at 15 months, all rickety; 3 at 16 months, all rickety; 1 at 19 months, rickety; 2 at 2 years, both rickety. (The letter R stands for bone rickets, excluding craniotabes).

Of these 75 cases of syphilitic infants with enlarged spleen, all, with three exceptions only, of those who were kept under observation during the period when rickety bone changes become evident, developed rickets; and of those not having an appreciably enlarged spleen who were similarly observed, only about the same proportion failed to develop distinct rickety bone changes. We are thus led to confirm the opinions of Kassowitz and Fournier that rickets is observed with an incontestable degree of frequency in subjects attacked with inherited syphilis. In only one of our cases did the spleen begin to enlarge after the rickety bone changes became obvious, although in several the spleen increased notably in size as the rickets developed. Antisyphilitic treatment, with cod-liver oil and iron administered from the time the infants first came under observation, was undoubtedly followed by an early diminution in the size of the spleen in many cases, but in some this organ long remained stationary or slowly increased in bulk in spite of similar treatment.

To sum up: In 63 cases of splenic enlargement in infants we could not positively exclude the presence of rickets in a single one, and in almost all the rickets was clear and distinct. In 41 per cent. of the 63 cases inherited syphilis was undoubted, and in the remainder syphilis could neither be positively affirmed nor excluded. Of 155 cases of inherited syphilis the spleen was found by palpation to be enlarged at some period in 48.4 per cent., and it is universally accepted that enlargement of the spleen is of excessive frequency in inherited syphilis—at any rate in the earliest stages—and we assert that it may persist to later stages also. This enlargement is apparently often indistinguishable histologically from that met with in cases of rickets. Our observations confirm the opinion of Kassowitz and Fournier that rickets occurs with an incontestable degree of frequency in subjects attacked with inherited syphilis. In rickets the spleen is found enlarged in perhaps 25 per cent. of the cases. From these facts—and we present our observations with some confidence, as having been checked by two observers—we do not wish to draw any dogmatic conclusions. Bone changes constitute only one of the symptoms of rickets, and are not the earliest, so that, allowing for the frequency of enlarged spleens in early syphilis, the bulk of our cases may quite possibly be due to rickets, either primarily or engrafted upon syphilis. We think, however, that we are justified in saying that in every case of enlarged spleen in an infant, such as we have been discussing, there is reasonable ground for suspecting syphilis. We are not aware of any observations on the viscera and lymphatic

glands in cases of rickets in animals, but such an inquiry seems to be very desirable as an aid to the solution of the vexed question under discussion.

NOTE.—Since this paper was written Mr. Bland Sutton has been kind enough to inform us that the spleen and liver are often, though not constantly, enlarged in rickety monkeys, especially in the very young ones.

ON SOME SYMPTOMS ASSOCIATED WITH THE URIC ACID DIATHESIS IN CHILDREN.

By G. A. SUTHERLAND, M.B.ED., M.R.C.P.LOND.,

Physician to the North London Consumption Hospital; Registrar and Clinical Assistant Paddington Green Children's Hospital.

THE presence of uric acid, or oxalates, in the urine of children is such a familiar sight that by many it is regarded as the normal condition, or, at most, as the evidence of a slight stomachic derangement. The conditions of occurrence of uric acid and oxalates are so closely allied that, for clinical purposes, they may be taken together. These substances may be present in the system and in the urine for a considerable time without producing any symptoms to attract the notice of the patient's friends, and it is only on the occurrence of hæmaturia or severe abdominal pain that the physician is called in.

From a clinical standpoint, the symptoms may be classified as follows:—

1. *Symptoms Due to the Presence of Uric Acid in the System.*—The subjects of the diathesis are often easily recognised. They have keen precocious minds, and small restless bodies; they are excitable, nervous, bright and amusing at one time, and greatly depressed at another; they do not readily fall asleep at night, often talk in their sleep, and have a habit of awaking in the very early morning; and they are dainty feeders, with a taste for everything that is bad from a nutritious point of view. The children are often described by their parents as being very subject to colds, and a chill in some form or another is the usual precursor of an acute attack. Along with this, and acting possibly as the cause, is a tendency to profuse sweating on moderate heat or exertion. Cold hands and feet are very frequently complained of—a symptom which Dr. Haig ascribes to uric acid in the blood, and which, he suggests, may be found in a more advanced condition as the local asphyxia of Raynaud's disease. During the course of the affection acute attacks occur, which are recurrent in type and usually of short duration, especially if the child is kept in bed. The pharynx is often relaxed and irritable, causing a loud barking cough, most marked when the child goes to bed, and which may be accompanied by some bronchial inflammation. The tonsils and adenoid tissue of the nasopharynx are liable to acute attacks, which lead to chronic thickening and enlargement. Frontal headache and symptoms of intestinal catarrh, with a furred tongue and foul breath are common. As regards the heart, a slight irregularity is occasionally found, and the pulse is often small, weak, and irregular. The liver and spleen may be enlarged. In some cases abdominal pain is the only complaint, and this may sometimes be found to be localised in the right iliac fossa.

2. *Symptoms due to the Excretion of Uric Acid from the System.*—Pain is one of the most prominent symptoms. Dr. Goodhart,¹ in the course of some remarks on the uric acid diathesis of childhood, says "renal colic is far commoner in children than is usually supposed, but it passes for stomach-ache pure and simple, and goes unrecognised." The pain may be present in any part of the urinary tract, from the kidney downwards. If the irritation is in one or both kidneys, pain is referred to the region affected, and is described as passing downwards and forwards in the line of the ureter, or it may be localised entirely at the umbilicus. The pain is intermittent in character, and is often so intense that the child will scream loudly, especially in the middle of the night. Hæmaturia (renal) is frequently the first symptom to cause alarm, and there may be more or less shivering, nausea, and sickness present during an attack. When the bladder is irritated and inflamed by the crystals and the ex-

¹ BRITISH MEDICAL JOURNAL, 1891, ii, p. 251.