effect of the sweating which so generally attends these applications of guaiacol. But since there is no regular relation between the intensity of sweating and the fall in temperature, and since sweating is occasionally absent though the temperature falls, it seems clear that we must look elsewhere for a complete solution of the problem.

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INCOMPETENCE OF THE PULMONARY ARTERY.

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CARDIOGRAPHERS are agreed that diseased conditions of the orifice of the pulmonary artery developed after birth are uncommon. This applies, not only to lesions that narrow the mouth of the vessel, but particularly to those that allow of regurgitation of blood into the right ventricle. Constantin Paul maintains that incompetency of the valves of the pulmonary artery is of all cardiac lesions the rarest. Few authors have written much about reflux through these valves. Huchard and Walshe make no mention of it. Sansom in his classic work gives an interesting summary of the bibliography of the subject, and states that "a murmur of diastolic rhythm having its origin at the orifice of the pulmonary artery is very rare." As a small contribution to the literature of this interesting affection I give in detail the following case:

S. S., aged 60, married, was admitted into the Newcastle Infirmary August 26th, 1890, complaining of shortness of breath and swelling of feet and legs. She has had a comfortable home, but has had to work hard. There is nothing of importance in the family history. She had typhus (?) fever at the age of 9 years. There is no history of rheumatic fever. Patient considers that her illness dates from four years ago, when she caught a severe cold. This was followed by shortness of breath, which has never since entirely left her. She is badly nourished, is sallow and cyanosed. Urine has a specific gravity of 1014, is alkaline, and contains albumen. Tongue moist and coated; bowels are regular. Pulse 76, is rather incompressible; the artery feels hard. The movements of the chest are restrained. There is an increased area of cardiac dullness; it extends from the nipple line to $1\frac{1}{2}$ inch beyond the right border of the sternum. The apex beat is diffused; it is displaced downwards and outwards to the left 2 inches below the nipple. Over the apex a systolic murmur is heard, followed by a loud ringing and accentuated second sound. Down the middle of the sternum and to the left of it a systolic murmur is heard, louder and more grating in character than a similar murmur which can be heard over the aortic area. Along the left border of the sternum a diastolic murmur of fairly long duration is detected. The veins of the neck are extremely distended, and are seen to collapse during deep inspiration. Pressure upwards, with the hand driven firmly but gently under the right costal arch, increases the venous distension in the neck. Under this procedure the jugular veins would almost immediately pass from the size of a penholder to that of one's little finger. There is œdema of feet.

Under rest in bed and treatment patient regained health to such an extent that she was able to return to her duties as an office cleaner. She left the infirmary on October 1st, 1890.

On March 10th of the following year she was readmitted, complaining of a recurrence of her old symptoms, which again subsided under treatment. She left the infirmary, but returned on June 26th, the dyspnoea having become aggravated, cyanosis being more marked, and emaciation more advanced. There was little or no cough. It is noted that there is very extensive pulsation over the cardiac area, and that it affects principally the right side of the heart. The superficial veins in the neck are widely distended. Over the aortic area there is a systolic murmur, followed by a well-marked second sound. Over the xiphoid cartilage is heard a double murmur, whilst over the seat of the pulmonary artery there is a well-marked systolic murmur, followed by a prolonged diastolic. There is a soft mitral systolic at the apex; here the second sound is healthy. Small crepitation, with diminished respiratory murmur, is detected at the base of the left lung, back and front. Pulse varies from 90 to 100; it is small and jerky; the radial arteries are hard and tortuous. Urine, specific gravity 1016, acid and albuminous. Liver dullness is slightly enlarged. By degrees the dullness in the left chest increased, and with it the dyspnoea passed into orthopnoea. The patient is only able to sleep resting on her knees and elbows. The symptoms gradually increased in severity; fluid began to accumulate, too, in the abdomen.

On August 6th we tapped the left chest, drawing off 52 ounces of serum. This gave great relief to her breathing. Patient could now lie in the recumbent position, and she slept comfortably. Gradually, however, the fluid reaccumulated in the pleural cavity; the breathing again became difficult, but nothing would induce her to have the chest tapped. The pulse became irregular, and the quantity of urine diminished to 10 ounces daily. As the illness drew to its close she became rather rambling and delirious, but never acutely so. A very profuse epistaxis

came on on August 18th—over a pint of blood being lost before the bleeding was restrained. Two days subsequently there was again epistaxis. Her pulse now rose to 140, and within forty-eight hours of the second epistaxis she died completely exhausted.

Post mortem a considerable quantity of fluid was found in the left pleural cavity. Beyond being compressed, the left lung presented nothing abnormal. The right lung was congested and œdematous. The heart was enlarged to almost twice its normal size, the enlargement affecting particularly the right side. A branch of the left coronary artery in the interventricular groove was atheromatous, and a portion of it was seen to be firmly plugged by a white fibrinous thrombus. The aortic valve was competent, and its segments were healthy seen from above. The pulmonary artery was incompetent. The myocardium appeared to be healthy except at the apex, where the wall was very thin. Lying firmly attached to it internally was an old laminated greyish-white clot. The aorta was slightly dilated. The pulmonary artery was widely dilated; it was from one-fourth to one-third as wide again as it ought to be. The aortic valves seen from below were quite healthy. Those of the pulmonary artery, when again tested with water, did not fall closely together, but allowed the liquid to escape. On closer examination the defect was observed to be due to the presence of thickened patches on two of the segments, which had caused them to shrivel. Mitral and tricuspid valves were healthy. A fairly deep ulcer, the size of a threepenny piece, was found at the beginning of the descending aorta. On the surface of the liver was a large patch of thickened capsule, the size of a five-shilling piece; it had all the characters of cicatricial tissue (? specific). The liver on section was nutmeggy. The kidneys were enlarged; in the left was a cyst, the size of a small nut, with gelatinous contents. Both kidneys were irregular in shape, and granular. The capsule was adherent, and the cortex was diminished. The other abdominal viscera were healthy.

Apart from congenital heart disease, lesions of the orifice of the pulmonary artery are extremely rare. Now and again they are met with in the course of ulcerative endocarditis. Under most circumstances the diagnosis of pulmonary insufficiency is a matter of difficulty. In the case reported it was suspected on the grounds that the diastolic murmur was located over the pulmonary artery, and conveyed down the left side of the sternum to the apex of the right ventricle; that the second sound heard over the aortic area was healthy and quite distinct; that the pulse was hard and incompressible, and not the collapsing pulse of aortic regurgitation. There was just some doubt, however, in my own mind in regard to making the diagnosis of pulmonary regurgitation absolute, and it arose from the possibility of there being some cardiac adhesions which might have given a twist to the heart in its long axis, thus altering the positions of the orifices of the large vessels arising from the base of the heart relative to their normally corresponding points on the surface.

It is noted in my case that the pulmonary artery was widely dilated. Hope, in his *Diseases of the Heart*, reports a similar case. There was no stenosis of the orifice, a point of some interest when viewed in the light of a strong statement by Dr. Michel Peter of Paris. In the brief paragraph devoted to lesions of the pulmonary artery in his *Maladies du Cœur* he says "we have seen *à propos* of lesions of the aortic orifice that the most common lesion was insufficiency, and that stenosis indicated a stage further advanced in the evolution of chronic endocarditis. It seems, on the contrary, that insufficiency of the pulmonary orifice is only an exaggerated product of the lesion which has caused narrowing of this orifice." As a clinical and anatomical entity Peter says stenosis of the pulmonary artery is well known, but there are very few examples of insufficiency without stenosis. I am using the word "stenosis" here in the sense of very distinct narrowing, such as would cause obstruction to the onward flow of the blood. With this meaning attached to the word there was in the above case no stenosis. The diastolic murmur was well heard over the seat of the pulmonary artery and down the left of the sternum, a circumstance I again allude to, for it must be admitted that during the course of many cases of chronic endocarditis there must occur pulmonary regurgitation so slight as not to give rise to murmur. In fact, this is one of the points that Duroziez lays considerable stress upon, and which he regards as one of the reasons why pulmonary incompetence is not recognised during life. It is the absence of a diastolic murmur. As this remark applies more or less to all forms of valvular disease, we cannot assign to it the importance claimed for it by Duroziez. Bramwell, whilst admitting the rarity of pulmonary incompetence, gives an excellent summary of the symptomatology of the disease. My own case fully confirms what he says of the seat and convection of the diastolic murmur, and of the effect of the lesion upon the right side of the heart and the venous system. The right ventricle was dilated and hyper-

trophied, and during life the veins in the neck would occasionally stand out like tubes the thickness of one's little finger. The pulse at first was rather hard and incompressible, later on small and jerky, whilst towards the end of life it became very rapid and small, but never to any extent irregular. Taken in conjunction with my remarks upon the distended right chambers of the heart and the venous system generally, the two attacks of profuse epistaxis are worthy of notice.

NOTE ON THE STAINING AND MOUNTING OF TUBE CASTS AND OTHER ORGANIC URINARY DEPOSITS.

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THE interesting paper by Dr. Harris in the BRITISH MEDICAL JOURNAL of June 23rd leads me to publish the following note, which was written several years ago, on the staining and mounting of tube casts and other organic urinary deposits. It will be seen that the method is simple, and that no apparatus, other than the ordinary clinical appliances in everyday use, is required. The results are eminently satisfactory, and the preparations permanent.

THE STAINING OF TUBE CASTS.

The detection of tube casts when the urine is copious, the deposit scanty, and the tube casts few and far between is, as everyone knows, a matter of considerable difficulty, and one which involves (when the ordinary method of examination is employed) the expenditure of considerable time and trouble. And yet it is just in those cases in which the detection of the casts is most difficult (cases of polyuria, with scanty deposit and a small amount of albumen), that the presence or absence of tube casts is of the greatest importance for the purposes of diagnosis.

The recognition of tube casts under such circumstances is greatly facilitated by the use of staining reagents. Picro-carminic is the stain which I chiefly use. The method which I adopt is as follows:

1. An ordinary conical urine glass is filled with equal parts of urine and an aqueous solution of boracic acid and set aside until the deposit settles.

2. The deposit is then drawn off by means of a pipette and transferred to an ordinary test tube in which a small quantity (half a drachm is quite sufficient) of picro-carminic solution has been previously placed.

3. The urine and staining fluid are then thoroughly mixed by inverting the test tube two or three times, the end being, of course, closed by the thumb.

4. The test tube containing the urine and staining fluid is then set aside to stand for twenty-four hours.

5. The deposit, which has by that time settled at the bottom of the test tube, is then drawn off by a very fine-mouthed pipette (most of the pipettes used for clinical purposes are far too large in the mouth),¹ placed on a slide, covered, and examined under a low power.

If any tube casts are present they are very easily detected by this method.

When a cast is detected with the low power, it should be carefully brought to the centre of the field; the high power should then be applied, and the structure of the cast more carefully studied.

If there is reason to suspect the presence of amyloid degeneration of the kidney, a solution of methyl violet may be used instead of picro-carminic; for in some cases of waxy disease of the kidney, the tube casts give, with methyl violet, the characteristic rose-pink reaction.

PERMANENT PREPARATIONS.

In order to bring out the fine details of tube casts, which have been stained in the manner described above, and in

¹ The best pipette is, I find, a narrow glass tube which has been drawn out to a fine point; the end then broken off and heated in the flame of a spirit lamp so as to round off the edges. A pipette with as small an orifice as may be desired can be made by anyone for himself in this way in the course of a few minutes.

order to preserve them as permanent preparations, they may be mounted in Farrant's solution.

The procedure which I adopt is as follows:—

1. A small test tube—one of the small glass tubes, in which Messrs. Burroughs and Wellcome pack their specimen tabloids, answers the purpose admirably—is filled three-quarters full with Farrant's solution.

2. The stained deposit from the test tube containing the mixture of urine and picro-carminic solution is drawn off by means of a fine-mouthed pipette, and transferred to the small test tube which contains the Farrant's solution.

3. The small test tube is then securely corked, inverted two or three times in order that the coloured urine containing the stained deposit and the Farrant's solution may be thoroughly mixed, and set aside to stand until the tube casts or other urinary deposits have settled to the bottom.

4. In the course of three or four days a minute drop of the deposit, which is of course mixed with a certain amount of Farrant's solution, is drawn from the bottom of the test tube by means of a very fine-mouthed pipette, placed on a slide, covered and examined by the microscope.

5. The preparation may, in the course of a few days, be sealed in the ordinary manner, if it is desired to preserve it as a permanent preparation.

If the preparation mounted in this manner is overstained with the picro-carminic solution (that is, when the mounting fluid as well as the deposit is coloured) the deposit which has collected at the bottom of the small tube should be drawn into the pipette and transferred to a second small tube containing fresh Farrant's solution. The deposit which settles in this second small test tube is, after it has settled, then drawn off and mounted in the ordinary manner. The tube casts stained in this way and mounted in a perfectly transparent and unstained medium make beautiful microscopical preparations, their minute characters can be studied with the highest powers of the microscope.

Any organic urinary deposits may of course be stained, mounted, and preserved in the same manner.

HEREDITARY MALFORMATION OF THE HANDS AND FEET; WITH OPERATION ON ONE SUBJECT.

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In April, 1894, a boy, aged 15 years, with malformed hands and feet, was brought by his father to Dr. Smith, on account of pain and difficulty in walking, due to the malformation of the feet. The case was of more than merely passing interest on account of the persistency and consistency with which the malformation had affected several generations. It will be well to begin by giving an account of the structure of the boy's hands, then to compare them with the father's hands; afterwards to describe the malformation in the feet, and to give the history of these malformations so far as it can be traced through various generations.

CONDITION OF THE BOY'S HANDS.

The malformation in the boy's hands affects the middle and ring fingers. The right hand shows these fingers webbed to the tip. The conjoined nails exhibit a slightly elevated ridge running from the lunule and ending at a notch in front; and at the tip of the conjoined fingers there is a small linear depression running from the nail notch to the palmar surface of the tip. These are the only indications of separation of the digits. Each digit has the usual number of phalanges. The ring finger has its first phalanx rather shorter than normal; the second phalanx is articulated to it at an obtuse angle, the distal end of the first phalanx forming thus a