which I treated with Iceland-moss poultices applied to the whole thorax, and the internal administration of expectorants with belladonna; stopping inhalations and galvanisation during the bronchial affection. She is at present in a much better state of health than for years, and had no spasmodic attack since January last.

CASE VI.—Mrs. T. of Ashton-on-Lyne, aged 33, widow, consulted me in July 1876. She had a severe attack of capillary bronchitis a year ago, and had since been frequently a sufferer from nervous asthma with bronchial catarrh. She used, under my direction, spray-inhalations of ozonised sea-salt with arseniate of soda, and galvanisation of both nervi vagi, for a fortnight; and she discontinued the treatment as she felt much benefited, and promised to see me again whenever an asthmatic attack might occur, but I had no opportunity of seeing her any more.

CASE VII.—Mrs. E. C. of Altringham, Cheshire, aged 37, married and a mother of two children, had suffered for fourteen years from nervous asthma and chronic bronchial catarrh. Spray-inhalations of chloride of ammonia, arseniate of ammonia, and cherry-laurel water, and galvanisation of both pneumogastric nerves, used during a month, gave her great relief; but I lost sight of her after December 1876, as she left this neighbourhood, and had no opportunity of persevering with the treatment.

REMARKS.—The treatment of nervous asthma by galvanisation of the pneumogastric nerves, and also by spray-inhalations of arsenic, seem to me to be very efficient, as, of seven cases under my observation, five were permanently cured and two greatly benefited. Four of my patients suffered from nervous asthma only, and three had, in addition, bronchial catarrh. One case was also hereditary, her father and sister being subject to asthma nervosum.

Galvanisation has been used with brilliant success for the treatment of two cases of true nervous asthma by Dr. Althaus (*Treatise on Medical Electricity*, second edition, London, 1870, page 522); also by Dr. Benedikt of Vienna, in one case (*Electrotherapie*, Wien, 1868, page 309); and by Dr. Brunner of Warsaw, in four cases lately published (*Sovremennaja Medicina*, Warsaw, 1877, Nos. 1-4).

Successful cures of nervous asthma have been effected with arsenic-inhalations by Dr. Wistinghausen (*Petersburger Medicinische Zeitschrift*, 1862, page 137), and also by Dr. Lewin (*Inhalationstherapie*, Berlin, 1865, pp. 443, 445).

I am of opinion that the two methods of treatment by galvanisation and inhalation may be safely combined, and ensure permanency in their beneficial effects.

NOTE ON FIBROID CHANGES IN THE LUNG AND THEIR RELATION TO SYPHILIS.

By JAMES F. GOODHART, M.D.,

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IN the number of the BRITISH MEDICAL JOURNAL for September 1st, my friend Dr. A. B. Shepherd, in his Further Remarks on the Natural History of Consumption, alluding to syphilitic disease of the lung, says that the cases upon which I based my remarks at the Pathological Society do not agree with the descriptions of the very few cases quoted in his second Goulstonian lecture, and I may add they certainly do not. The subject is one which has lately been fully discussed, and I do not propose to enter upon it again any further than is necessary to explain any discrepancy that may seem to exist between the one class of cases and the other. But to do this is really necessary, as it seems to me; because, if further observations are to be conducted from Dr. Shepherd's standpoint, a large number of cases will receive no consideration, and the main question—Does syphilis affect the lung?—answered in the affirmative by him, is likely to be negated by others on his own cases. For instance, it is surely no unfair argument to say that if, in the hundreds of cases of syphilis that have been carefully observed and of which the *post mortem* appearances are now recorded, only four or five of peculiar lung-change have been found, the peculiarity of the disease is likely enough due to something other than the syphilitic poison. If no other disease than that be syphilitic, then it is, indeed, very doubtful whether that, from its rarity, can be considered syphilitic. But if others, taking a broader view of the matter, say that fibroid changes in large or small quantity are found in the lung in an undue proportion of the cases of lung-disease in syphilitics, Dr. Shepherd's position is thereby strengthened.

For what is this disease of the lung which is considered as characteristic of syphilis? It is, and I quote from the second Goulstonian lecture of last year (BRITISH MEDICAL JOURNAL, 1876, vol. i, p. 623), "a form very closely resembling that described by Dr. Sutton as

fibroid degeneration of the lung"; and Dr. Sutton closes his article in the *Medico-Chirurgical Transactions* by endorsing the remarks of Dr. Wilks, that this "is merely the extreme development of a morbid condition such as is every day seen in a less degree in phthisical lungs". In this frequently seen gradation between slight and advanced forms of disease, lies the whole strength of my position. The extreme form must have had some beginning; and how else could it begin than by some such changes as those described and exhibited at the Pathological Society? Dr. Shepherd seems to me to take an extreme condition as his type or standard, and to call nothing syphilitic till that extreme is reached. But what was the commencement of this change? Is it any the more syphilitic or characteristic of syphilis in having advanced so far? And should we not expect that, for one such case as this, many others of comparatively trivial disease or of some earlier stage would occur? Again, Dr. Shepherd writes: "The pathological change in this form is quite distinct from that—if I may use the term—accidental occurrence of syphilitic deposits, in which the lung, in common with other organs, reveals the presence of gummata of varying size and shape and in different stages of development." Then, again, referring to my cases, "They include mainly cases of gummata, gangrene, ordinary yellow deposits, with destruction of tissue or partial fibroid change".

But in what, it may be asked, does the difference between the two forms, as thus described, consist? A gumma is a local fibroid change, gangrene is its sequel when it occurs in such cases; and ordinary yellow deposits are found in both forms. Thus, the difference is merely one of degree, not of kind, and this conclusion is further supported by the analogy to be drawn from syphilitic disease in the liver. In the latter, as is of course known to everyone, the usual condition is one of local gummata; but, on examining many of these cases more closely, it is found that there is a spreading interstitial cellular growth (the early stage, in fact, of fibroid disease or cirrhosis) in a large part of the organ. Yet, a true syphilitic cirrhosis is far from common; nay, just as extreme disease of the lung, it is exceedingly rare. But there would, it seems to me, be just as much reason for calling the cirrhosis the essential or specific disease, and the local changes the accident, if we did not by this time know better, as for applying those terms to the changes in the lung. All the extreme changes in such a chronic disease must be rare, because in both cases other viscera become hampered and the patient dies in their course, or (if it be a well founded inference from our present data, that the earlier changes are due to syphilis) because they are arrested by medicines. Moreover, and this does not lessen the force of the evidence which exists in favour of syphilitic disease of the lung, such extreme cases as that recorded by Dr. Pye-Smith, for instance, where one lung was converted into a mass of fibroid tissue and no healthy tissue left, are really not due to the syphilis so much as to the position taken by the disease, and this is determined in most, if not all, cases by local conditions. In Dr. Pye-Smith's case, the general affection of the whole of one lung was due in great part, as I thought when making the *post mortem* examination, to the fact that there was very considerable narrowing of the main bronchus, and which may be presumed to have favoured some collapse. In like manner, other cases may be due to the thickening of the pleura, or effusion, and so on. But these are no characteristics of the syphilis; they are possessed in common by some tumours at the root of the lung and by some old pleurisies; though, if it be essential for its production that complex conditions must concur, the manifestation of a particular form of pathological change must be rare.

It was my great aim at the Pathological Society to strengthen the position of former observers in bringing up further evidence to show that the lung is no exception to the rule observed by syphilis in other viscera; and that in it, as in other parts, nodules, gummata, fibroid changes, cicatrices, or whatever they may be called, were not uncommon. My own sense of right conclusions will be satisfied, even if it be admitted generally, that syphilis modifies other forms of lung-inflammation and tends in them to the production of a fibrous form. Thus, if a syphilitic person get a catarrhal pneumonia or bronchitis from any cause whatever, its course is likely to be modified. I do not know that facts prove more for any other viscus than that. I doubt whether late syphilis has a predilection, so to speak, for any organ any more than I suppose that croupous pneumonia has a predilection for attacking the base of the lung. The outbreak is determined by strictly local conditions, and these are often made more apt by some local cause of inflammation. Thus, one man has his liver affected, another his brain, another his heart or large vessels, another his lungs or skin.

I never attempted to show that the partial fibroid changes were the same in outward appearance as the few recorded cases which alone Dr. Shepherd would call syphilitic. Rather the contrary; entrenching myself behind Dr. Wilks and Dr. Wilson Fox, I professed to be unable to distinguish decisively between chronic forms of tubercular disease

and these partial fibroid changes. But there are others who would not go thus far with me. Dr. Greenfield thinks that it is possible by histological investigation to say that some of these chronic indurations are syphilitic; and, if he be right, there is an end of the matter. I do, however, think that, in comparing the various chronic destructive diseases of the lung, we can say that some degree of fibroid change in the lung is more common in those lung-diseases associated than in those unassociated with syphilis; and that, without any improbability, we can mentally bridge over the gap between the lesser and the greater, and foreshadow the occasional occurrence of a change such as Dr. Shepherd pictures.

The really important point of this question, however, is this, and so far all must be at one. It is very necessary to recognise clinically as often as possible the form of disease from which a patient is suffering. If we limit our idea of lung-syphilis to that form described by Dr. Bäumlér, Dr. Shepherd, and others, we only find it occasionally, when it is a pathological curiosity, and I may add past cure. But if, on the other hand, we assume—and, in doing so, I repeat we are only assuming what from the course of disease in other viscera is probable, and what there is at all events some evidence to support, if not sufficient to prove—that fibroid changes in the lung are not uncommon in syphilis, we do throw light upon a class of cases where, with some indications of phthisis, the chief symptoms are those of bronchitis with emphysema, and in which, I believe, a great deal of good is occasionally done by iodide of potassium.

CASE OF PULMONARY STENOSIS WITH PATENT FORAMEN OVALE.

By ROBERT SAUNDBY, M.D. Edin.,
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THE patient, a boy, W. W., aged 11 years, was admitted under the care of Dr. Wade on the evening of December 20th, 1876, in a very exhausted condition, with orthopnoea, aphonia, and oedema of the lower limbs. He had a constant teasing laryngeal cough, with mucous expectoration. Physical examination showed redness of the vocal chords; *râles* over both lungs; a double murmur at the base of the heart, heard best in the aortic area; and a systolic murmur at the apex. The urine was scanty, highly albuminous, and contained granular casts. There was no cyanosis; his face was white and puffy. His condition was not favourable for further examination, and he died on the morning of the 24th, less than four days after admission.

I obtained from his mother the following details of his personal and family history. His father is a healthy man, and has had no illness except jaundice. His mother, who follows the laborious occupation of a washerwoman, had rheumatic fever twenty years ago, but has had no symptoms or signs of cardiac affection. She has had several children, of whom none have been blue, but they are delicate. She always thought the deceased to be quite a healthy child up to the age of five years, when suddenly, without previous illness, he became paralysed on his right side, both limbs being affected; but his speech was unimpaired. He was under treatment at the Children's Hospital, and got better of the paralysis, but has ever since been ailing and has had a constant cough. During last year, he was an in-patient of the Homeopathic Hospital in this town, but got worse; and two months since, when he was removed, his legs were swelled. For twenty-four hours before his admission here, he was seized with an attack like croup, when he was seen by Mr. Baker, who advised his removal to this hospital. He had never looked blue at any time.

NECROPSY.—The integuments generally were pale. There was oedema of the lower limbs. The head was not opened. The larynx showed traces of recent catarrhal inflammation. Both lungs were congested, and the lower lobes were hepatized. The liver was congested and fatty. The kidneys were congested; and, under the microscope, the tubules were seen to be stuffed with fatty epithelium. The spleen, stomach, and intestines were healthy. The pericardium was distended with clear serum; the surface of the heart was smooth; the right cavities and the venæ cavæ were distended with recent clot; the aortic and pulmonic valves were tested, and found competent. On opening the right auricle, after removing the clot, it was found to be dilated. The foramen ovale was quite patent, and admitted a cylinder fifteen-sixteenths of an inch in diameter. The Eustachian valve was represented by only a narrow fold. The auricular appendix and the orifice of the coronary sinus were dilated, and the latter contained old clot; the tricuspid valve was healthy; the orifice admitted a cylinder one inch in diameter. The pulmonary orifice was small, the segments being adherent, so as to form a ring admitting a cylinder only six-

sixteenths of an inch in diameter. The valve was very little thickened; above it, in the pulmonary artery, there were numerous warty vegetations on the lining membrane. The ductus arteriosus was, I believe, quite closed; but its condition at the time was not noted. The right ventricle was dilated and hypertrophied. The left auricle and ventricle were empty, and appeared healthy. The mitral orifice admitted a cylinder thirteen-sixteenths of an inch in diameter; and the aortic orifice, measured in the same manner, was nine-sixteenths of an inch in diameter.

REMARKS.—The stenosis of the pulmonary orifice was considerable. According to Dr. Wilks's tables, the pulmonary orifice should be one-eighth larger than the aortic opening; but the measurements given show it to have been one-third less. Moreover, the foramen ovale was so large, and the tension in the pulmonary veins so diminished, that blood, in all probability, passed through in considerable quantities. Much unoxidised blood, no doubt, passed into the systemic circulation; yet none of these coincident phenomena at any time caused cyanosis. It is now generally admitted that simple admixture of venous with arterial blood will not produce cyanosis. Breschet's case, quoted by Rokitsansky (Sydenham Society, vol. iv, p. 248), of the subclavian artery of the left side arising from the pulmonary artery without causing anomalous colouration of the limb, is sufficient to disprove the hypothesis; and the case reported by Dr. Peacock (*Med.-Chir. Trans.*, 1849), in which a perforated septum divided the conus arteriosus from the right ventricle and caused considerable obstruction and cyanosis, although the auricular and ventricular septa were intact, shows that such admixture is not even a necessary factor in the production of this phenomenon. According to Rokitsansky (*ibid.*, p. 343), cyanosis "depends upon an obstruction in the passage of the venous blood into the heart, and upon an overcharging of the venous system, which is either transient or habitual, according to the circumstances of the case, and induces a corresponding repletion of the capillaries". Thus stated, the proposition is too general, and lacks the definiteness requisite for its application to those cases, which are called cyanosis proper. Rheumatic affections of the auriculo-ventricular valves never produce cyanosis to such an extent as to deserve the name of morbus ceruleus. Niemeyer justly observes in a note (*Text-Book of Pract. Med.*, vol. i, p. 369), "that the great cyanosis of persons with congenital malformation of the heart is due to an especial cause, becomes evident from the fact that individuals with congenital cyanosis do not become dropsical nearly so soon as those suffering from acquired cyanosis. This would not be the case if the cyanosis were due to venous engorgement". He finds this especial cause in the mixture of the blood and its consequent dark colour, or in the dark colour it acquires by the extreme retardation of the circulation, added to the venous obstruction. But the occurrence of cyanosis does not keep a definite relation to these causes; for mixture of the blood, slowness of the circulation, and venous obstruction may occur singly, or all combined, without producing cyanosis. Here, as a loophole for escape, we are told that cyanosis never occurs in anæmic or thin-blooded subjects. Certainly the children described by Dr. B. W. Foster (*Clin. Med.*, p. 35) as suffering from diarrhoea, ill fed and ill cared for, and of whom he says "the extreme poverty of the parents rendered the supply of the bare necessities of life often precarious", were, in all probability, anæmic and "thin-blooded"; yet they were marked examples of cyanosis.

The absence of cyanosis in the great majority of cases of venous obstruction compels us to seek for some more precise expression of the cause than the words quoted from Rokitsansky. As a rule, venous obstruction causes, not cyanosis, but oedema; and the exceptions are to be found in those cases where the obstruction is gradual and the vessels can accommodate themselves by dilatation and by gaining support from the condensation of their surrounding tissues. In the disease called cyanosis, the blueness is always most visible in those parts which, from their size, position, or anatomical peculiarities, are prone to turn blue under the influence of external cold. The vessels in these parts are easily dilated; the blood-current, from some cause, is naturally feeble, and stagnation easily occurs. We must bear in mind that blueness in these parts is not under any circumstances a true indication of the general condition of the capillary circulation. Most, if not all, of these cases of cardiac defect with cyanosis are congenital or acquired in early childhood, when the modifiability of structure is very great. The appearance of cyanosis depends upon the compensation being sufficient to allow the venous obstruction to do no more than cause dilatation and retardation of the current in the capillaries of certain peripheral parts. If the compensation be greater, no symptoms may be present until they are developed by the strains on the circulation entailed by the growth of the body or other causes. If the compensation be less, death ensues, or oedema occurs and the cyanosis is