I think it very nearly certain that desire and pleasure, in due or moderate degree, are very important aids to, or predisposing causes of, fecundity, not on account of their own proper attractiveness, but on account of some connection between them and the perfection of other parts of the complicated proceedings which result in fecundation. But this is only a firmly held opinion, for I can give no conclusive evidence or proof of it; and this absence of proof diminishes greatly the value of my observations on the absence of desire and pleasure in the sterile. The want most acutely felt here is a knowledge of the state, in this respect, of the fertile. In producing evidence as to the sterile, I shall assume that sexual desire and pleasure are very rarely absent in the fertile. Excess of sexual desire is probably unfavourable to fertility. It is recognised chiefly by excessive indulgence in sexual pleasure, and is observed in the weak and ill-conditioned, in imbeciles and idiots, as it is also in animals under confinement. Excessive indulgence in sexual pleasure is also probably unfavourable to fertility, or a cause of sterility; and it probably is specially influential in the young, as it may also be in prostitutes. In these circumstances, the births of females are, on good grounds, believed to be far above the ordinary average, in proportion to males.

Masturbation in females is an unnatural and generally excessive indulgence in artificial sexual pleasure. It has always appeared to me to affect, especially children and young women of weak mind. I have often been struck by the smallness or imperfect development of the external parts in young women who masturbate, and I have not rarely observed what appeared excessively high development of sexual desire in women who had imperfection or absence of internal genital organs. In one, dissection revealed the presence of ovaries and Fallopian tubes only. Some confirmation of these views may be found in cases such as that of Campbell,* in which a woman addicted to masturbation had never menstruated, and had imperfectly developed genital organs; she had, however, also a dermoid cyst of the ovary. Aran† has a case of what he describes as frightful excess of masturbation in a young woman dying of phthisis, whose uterus and appendages were found to be very imperfectly developed. Kussmaul‡ mentions the concurrence of masurbation and nymphomania with imperfect development of the uterus and the genital organs; and Joulin§ refers to a case of Vaddington's where absence of uterus and exaggerated sexual appetite were combined.

Entire absence of desire and pleasure, or of one of them, or the presence of intense sexual antipathy and dyspareunia, are not necessarily causes of sterility. It is not at all rare for women to be pregnant and bear healthy children who aver in the distinctest manner not only absence of desire and pleasure, but presence of the opposite conditions. But the following statistics make it highly probable that absence of desire and pleasure, and the presence of their opposites, are powerful influences favourable to sterility. The statistics do not indicate what was occasionally found, namely, that desire was present while pleasure was absent; or, in other cases, that desire was absent while pleasure was present. The cases observed were all in women absolutely sterile, of whom the great majority consulted me regarding the sterility. Among 191 sterile wives, desire was absent in 39, or in about 1 in 4. Among 196 of the same sterile wives, pleasure was absent in 61, or in about 1 in 3. The figures show that many sterile wives had desire but no pleasure. They do not show, what nevertheless is true, that some had pleasure who had no desire.

TABLE XXIII.—Case-book Table of Desire and Pleasure in Sterile Women.

Age at Marriage.	Number.	Desire.			Pleasure.		
		Present.	Absent.	No Note.	Present.	Absent.	No Note.
Between to nineteen	59	18	4	37	15	8	36
Twenty to twenty-four	220	78	18	124	69	27	124
Twenty-five to twenty-nine	134	35	12	87	31	18	85
Thirty to thirty-four	54	16	3	40	14	5	40
Thirty-five to thirty-nine	23	3	1	19	8	3	17
Forty to forty-five	9	2	1	6	2	1	6

I have a strong impression, derived from all I know and have observed, which I may express theoretically, that while, in healthy

normal women, there is abundance of sexual or reproductive energy for fertility and all its accompaniments, in many sterile, or relatively sterile, women, there is deficiency which may be exhibited in one or another, or in all the ordinary evidences of reproductive energy, and that excess or deficiency in one department may be associated with deficiency or excess in another. It would seem that, in women of deficient reproductive energy, excess in one department might be compensated by deficiency in another, and *vice versa*, there being only a limited store of the original energy. In illustration, a remarkable class of cases may be cited, which I shall sufficiently describe by stating generally the chief points in one: A robust healthy woman is married at eighteen; she bears three children and has four miscarriages before she has passed twenty-three years of age. Up to the birth of her last child, and for five years subsequently she experiences no sexual desire, and has no pleasure. Five years after her last pregnancy, she almost suddenly comes to have intense desire and pleasure, but remains sterile for four additional years before she seeks a cure for her sterility. Fertility present, while desire and pleasure are absent; sterility present, while desire and pleasure are present.

CLINICAL LECTURE

ON

ACCENTUATION OF THE PULMONARY SECOND SOUND OF THE HEART.

Delivered in the Queen's Hospital, Birmingham.

By JAMES SAWYER, M.D. Lond., M.R.C.P.,

Senior Physician to the Hospital, and Professor of Therapeutics in Queen's College.

ACCENTUATION of the pulmonary second sound, or, to speak more precisely, accentuation of that portion of the second sound of the heart which is produced at the orifice of the pulmonary artery, and is especially heard in the "pulmonary," as distinguished from the "aortic," area, although discovered and taught by the great Skoda in the earlier days of cardiac auscultation, is not generally recognised, if I may judge from the scanty references to it in text-books, and from my observations of its frequent neglect in the practice of stethoscopists, as one of the most striking and one of the most significant of the physical signs of disturbance in the mechanism and dynamics of the heart. It is a sign which is to be found in association, in causal relations which are tolerably clear, and approximately constant, with the commonest of the organic defects of cardiac orifices and valves, and with the commonest consequences and complications of embarrassed cardiac action. Rightly interpreted, it is a sign which traverses the whole domain of practice, for it conveys reliable indications in the three chief divisions of our relations with a patient, inasmuch as it is significant alike in diagnosis, in prognosis, and in therapeutics. Skoda, with his usual tendency to over-refinement, that frequent fault of physicians, did not grasp the simplicity and singleness of the significance of accentuation of the pulmonary second sound. He observed the physical fact, but he went too far, and in one line in a wrong direction, in his speculations upon its import. He was wrong in his teaching, for example, as Dr. Walshe has pointed out,* that the presence of reinforcement of the second sound in the pulmonary artery will distinguish a systolic murmur at the left apex, caused by mitral regurgitation, from a murmur, of like time and site, caused by friction of the blood against roughnesses on the inner surface of the ventricle. The essence of the matter is this: accentuation of the cardiac second sound, as heard over the origin of the pulmonary artery, is an unfailing indication of increased tension in the blood-current in that vessel. In that it is this, it is a trustworthy sign, which a little consideration will enable you to understand, of a grave pathological condition; it is an unmistakable physical accompaniment of a portentous change in an area of the blood-circulation which is vital, and which is removed beyond the reach of those tactile and metric methods of exploration which are applicable in variations of tension in the systemic arteries.

In health, the aortic portion of the second sound of the heart predominates over that produced at the valves of the pulmonary

* *Memoir on Extra-uterine Gestation*, p. 30.

† *Leçons Cliniques sur les Maladies de l'Uterus*, p. 89.

§ *Der Mensch, etc., der Gebärmutter*, S. 74.

¶ *Accouchements*, p. 138.

* *Diseases of the Heart and Great Vessels*. By W. H. Walshe, M.D., etc. Second Edition, 1882. Page 100.

artery. That is, the second sound is louder in the "aortic" than it is in the "pulmonary" area. By this statement I mean that the second sound is louder close to the right edge of the sternum, over the lower portion of the second right costal interspace, than it is close to the left edge of the sternum, over the upper portion of the second left interspace. The blood-tension may be raised in pathological states, either in the systemic circulation, of which the aorta is at the commencement, or in the lesser circulation, which passes from the right to the left sides of the heart, through the lungs, and at the commencement of which is the pulmonary artery.

Whatever raises the blood-tension in the aorta intensifies the aortic second sound; whatever raises the blood-tension in the pulmonary artery intensifies the pulmonary second sound.

What, then, is the clinical import of the variety of abnormal loudness of the second sound of the heart, to which I am directing your attention? Answering the question broadly, I say, it is beyond dispute that an increased intensity of the pulmonary second sound is due to an increase in the blood-tension in the pulmonary artery, and that this heightened tension is due to some obstruction in the pulmonary or lesser circulation. The sign is clinically associated with organic and permanent lesions of the mitral valves and of the mitral orifice. Either insufficiency of the mitral valves, or narrowing of the mitral orifice, adds a distinct and new physical obstacle to the flow of blood through the lesser circulation. In so far as such an obstacle elicits increased force in the contraction of the right ventricle, by so much does it raise blood-tension in the pulmonary artery, and consequently accentuate the pulmonary second sound. But, while this statement is strictly true as a generalisation, you must remember certain qualifying circumstances which may hold good in particular instances. Advanced mitral regurgitation, or advanced mitral stenosis, or both, may be present, and the pulmonary second sound may not be accentuated, but may even be less loud and clear than in health. This may arise from one of two causes, or from a frequent combination of them, namely, from failure in the power of the right ventricle, or from the appearance of tricuspid regurgitation. In the course of mitral disease, when the force of the right ventricle at last fails to compensate for the obstacle on the left side of the heart, the blood-tension in the pulmonary artery inevitably falls and falls, and with it the loudness of the pulmonary sound inevitably declines and disappears. When, also, in the course of mitral disease, the tricuspid valves, as so often happens near the end, give way, the pulmonary tension is at once lowered, and its physical sign disappears. Let me emphasise these important points by quoting some words of Rosenstein. He writes—"When the tension decreases in the pulmonary artery, the intensity of the second sound ceases; this takes place either when the right ventricle's force has been impaired by disease in the performance of its increased work, or when the right side of the heart is so filled by the increased stagnation that the ring of insertion of the tricuspid valve is widened, and the valve is no longer able to close the orifice.* I must also point out to you that, in comparing the pulmonary second sound with the aortic sound in cases of mitral disease, you must remember that the aortic second sound is likely to be relatively weakened by reason of the reduced systemic tension which mitral defects entail. As Dr. Walshe points out, there is a "pseudo-accentuation of the pulmonary second sound, from real weakening of the aortic second sound, through the lessened current and diminished calibre of that vessel, that follow on long continued mitral regurgitation."† You must not fall into the error of mistaking a pulmonary second sound of normal loudness for an accentuated sound, because it coexists with a feeble aortic sound. On this point, which undoubtedly is sometimes a difficult one in practice, you must look to an extended experience of cardiac auscultation to aid you. The recognition of variations in the tone and loudness of the heart's sounds is a refinement of stethoscopy, which only long practice can develop. It is only when, by patient clinical work, you have acquired in your minds a sure standard of the characters of cardiac sounds, that you can readily detect deviations from their normal intensity.

So far as I have been able to judge from my own observations at the bedside, the presence or the absence of accentuation of the pulmonary second sound, or the presence of a high or of a low degree of such accentuation, is valueless as a differential sign in itself in the diagnosis of mitral stenosis from mitral insufficiency. I know this statement is opposed to the teaching of some physicians and of

some writers of acknowledged authority in cardiac diagnosis. Both varieties of mitral disease, whether they exist singly and purely, or however they may be combined, impose a morbid obstacle to the passage of blood from the right to the left side of the heart, and tend, *pro tanto*, to increase the blood-tension in the pulmonary artery. So long as this obstacle is met by a compensating increase of force in the contraction of the right ventricle, so long is the pulmonary second sound of more intensity than in health. The presence of such accentuation is not a sign which distinguishes one form of mitral disease from another, but it is a sign common to mitral lesions in general, which rises and falls in direct proportion to the vigour of the right ventricular systole. When, in the backward march of the results of a mitral lesion, the saving force of the right ventricle becomes impaired, by dilatation of its cavity or by degeneration of its walls, the pulmonary second sound loses its accentuation and may become almost or quite inaudible.

You will now be able to appreciate the help which may be gained in the diagnosis, prognosis, and treatment of a given case of organic disease of the mitral orifice or valves, from observation of the condition of the second sound in the pulmonary artery. Stating the case broadly, it may be said mitral valvular defects are generally the practically immediate and permanent results of acute endocarditis. Once established, the affection of the valves or orifice becomes a permanent defect, which never grows less, but which rather tends, by the organisation and contraction of inflammatory exudations, and by other well-known consecutive changes, to become more and more pronounced as time goes on. From the date of the endocarditis which first damaged the heart, there occurs a variable period of practically good health, or of quasi-health, but slightly impaired by certain of the less pressing signs of cardiac embarrassment. This period may vary in length from a few weeks or months to a few or many years, being determined by a variety of variously combined circumstances, such as the extent of original mitral damage, and the degree of subsequent compensation, and the age, mode of life, social position, and habits, of the patient. But, whether this period be short or long, there surely comes, sooner or later, an ultimate or penultimate stage, marked by failure of compensation, and by dropsical complications, leading on to death.

Accepting this brief outline as a rapid sketch of the usual progress of mitral affections, let us answer this question: What is the usual state of the pulmonary second sound in the progress of such a case? When the mitral disease arises, that is, from the time acute endocarditis so affects the mitral orifice or valves as to set up a physical obstacle there to the normal progress of blood through the heart, the pulmonary second sound becomes accentuated, but only slightly so, for the most part, at this early stage. During the second period of quasi-health, that is to say, from the time of convalescence from the acute endocarditis until the onset of the later secondary complications consecutive to the mitral defect, the pulmonary second sound remains only slightly reinforced. You will generally find it as loud as the aortic sound, or a little louder, but not very markedly intensified. But towards the end of this second period, when the pulmonary tension is nearing the point when it shall overcome the compensating force of the right ventricle, the pulmonary second sound becomes very distinctly accentuated, and attains its maximum development. The sign is at this time of grave portent, for it is the sure index of an extremely heightened tension in the pulmonary circulation, which is not likely to be borne long; it is an unfailing sign that the pulmonary circulation is only maintained by an increased expenditure of force by the right ventricle, which cannot long be kept up. At this point, a straw breaks the back of the labouring camel. A little added difficulty to the circulation through the lungs, which usually comes as a bronchial catarrh, which would be trivial under some other circumstances, and the next, the ultimate or penultimate, stage of mitral troubles is ushered in. Compensation fails, and with it falls, *pari passu*, the accentuation of the pulmonary arterial sound. With failing compensation, viscera and surface become engorged with blood, anasarca gradually develops, and dropsical exudations begin to gather in the serous cavities.

This is the stage at which you often see patients admitted to my wards. With rest, good and carefully adjusted food, evacuations, and, above all, with digitalis, our great heart-restorer, many improve, lose the later complications of their mitral disease, revert to the second stage of quasi-health which I have been describing, and return to their occupations. As they improve, as rest, suitable food, evacuations, and digitalis, do good, you may notice the pulmonary second sound, which had waned before, wax strong again, surely marking the recovery of compensation in the propulsive power of

* Rosenstein. *Ziemssen's Cyclopædia of Medicine*. English translation. Vol. vi, page 129.

† *Op. cit.*, page 100.

the right ventricle, which is the essential factor in the patients' relief. Here observation of the pulmonary second sound is of inestimable service. With a rising sound, our treatment is doing good, and our patient is making progress towards recovery. But the complications of this later stage of mitral disease, unhappily, cannot always be removed even once; and if removed once, or twice, or thrice, or oftener, there surely comes a time when all our remedies are at last of little or no avail. Be our treatment never so patient and skilful, the patient's condition remains stationary, or goes on from bad to worse. Here the compensating power of the right ventricle is finally and irretrievably exhausted; it is past all repair. Here the pulmonary second sound never rises under our treatment, but remains feeble to the end. Its continued feebleness, in the presence of dropsical complications, and in spite of our best therapeutic efforts, is a sure sign that the end is not far off, and that the patient is suffering his last illness.

ABSTRACT OF TWO CLINICAL LECTURES

ON THE

PLEURITIC EFFUSIONS AND PARACENTESIS.*

By W. H. A. JACOBSON, B.A.Oxon., F.R.C.S.,

Assistant-Surgeon to Guy's Hospital; Surgeon to the Royal Hospital for Women and Children; Examiner in Surgery at the University of Oxford.

OUTLINE OF CASE; VARIETIES OF PLEURISY.—I. *Dry*.—Common in phthisis. Pleuritic adhesions usually corresponding to site of lung-mischief. Purpose. Site, cap-like over apex.—II. *Acute Fibrino-Serous Effusions*. Usually of an actively inflammatory kind. Onset of attack. Pyrexia. Rigors. Pain. Character of respiration. Side early fixed. Course. Prognosis good; little tendency to pus. Treatment local and general. Medical usually sufficient, as these effusions quickly reach their height and subside. Morphia, subcutaneously. Fomentation. Leeches. Drugs, simple febrifuges, hyd. cum creta and Pulv. Doveri. No cupping or blisters at this stage. Surgical treatment only when patient becomes uneasy, and the entrance of blood into the right heart is impeded. Paracentesis trocar not too small. Large needles of aspirator. Little danger if air do enter (C. Allbutt),† as these fibrinous effusions, poor in cells, do not tend to suppurate? Two varieties of inflammatory effusions (Paget).‡ (a). *Fibrinous*.—(b). *Corpuscular*.—Existence of one or another of these determined by three conditions. 1. Condition of the blood; e.g., effect of blisters. "The highest health is marked by an exudation containing the most perfect and unmixed fibrine; the lowest, by the production of the most abundant corpuscles." 2. Site of inflammation; e.g., skin, exudation corpuscular, as in herpes, eczema; mucous surface, exudation corpuscular, e.g., gonorrhoea; serous membrane, exudation fibrinous. 3. Degree of the inflammation. The less the inflammation, the more the exudation resembles that natural to the part.—III. *Quiet Serous Effusions*.—Onset. Insidiousness. Symptoms little marked. Dyspnoea. Character often slight and thus misleading. Slight cough. Temperature perhaps a slight evening rise. These cases liable to be taken for debility and anæmia. (C. Allbutt)§. Occasionally Secondly; e.g., after typhoid fever convalescence may be prolonged or irregular, c. dyspnoea, etc. Such effusions often corpuscular.—IV. *Intermediate Forms between II and III*.—V. *Empyema*.—VI. *Dropsy of Pleura-Hydrothorax*. Often double. Cardiac or renal disease. No surgical treatment unless great urgency be present.

DIAGNOSIS.—Twofold. I. Diagnosis of fluid. II. Diagnosis between non-purulent and purulent effusions. I. *Diagnosis of fluid*. a. *Exploratory puncture*.—A matter of routine in all doubtful cases. Hypodermic syringe; never grooved needle. Precautions as to needle; clean, pervious, not too flexible, length two inches, and stouter than the ordinary ones. b. *Percussion*.—Character of dullness. Varies occasionally with position. Fallacies. Lung more or less solid and adherent. Pleura much thickened. Chronic strumous deposits in mediastina. Adhesions. Fluid may be localised by adhesions, or too thick to move freely. New growths. c. *Diminished movement and expansion*. d. *Breath-sounds feeble or absent*.—Fallacies. Lung partly solidified, and adherent with very thickened pleura. Bronchial breathing is often heard over effusions,

especially in children. Sounds may be conducted from opposite side, whose action is increased. e. *Absence of tactile or vocal vibrations*.—Fallacies. 1. This may be present over collections of fluid when strong adhesions run between the lung and chest-wall, and when the lung is not extremely compressed. 2. It may be absent when the voice is weak, or absent, especially in children, or where tubes are at all occluded. f. *Enlargement of chest*.—Fallacies. Only in very large effusions, and in young subjects. In long-standing cases, affected side may be smaller from shrinking, or from opposite side being overdistended from increased action. g. *Obiteration of spaces*.—Fallacies. Only in large effusions and in young subjects. h. *Displacement of viscera*: e.g., heart to right; liver downwards.—Fallacies. 1. Displacement of liver may occur in lobar pneumonia or new growths in chest. 2. Where effusion is undoubtedly present, heart may not be displaced, owing to adhesions or to shrinking or collapse of lung. ii. *Diagnosis of pus from serum*.—Importance. Pus is very rarely absorbed; may burst into lung, cause hectic, etc. a. *Needle*. b. *Hætic*.—Not always reliable. May be little marked or absent in empyema; may be present in serous effusions; e.g., in these, evening temperature may reach 101°. Explanation of absence of hectic in empyema; perhaps alteration or tension of pleura prevents absorption. c. *Aspect of Patient*.—Anæmic, earthy tint, clubbing of finger-ends. Dr. Barlow's dictum: "If a child be seen with general pallor and finger-clubbing, one ought to think of empyema rather than of the other causes of clubbing—viz., chronic bone-disease, bronchiectasis, and congenital heart-disease." d. *Age*.—Common in children and young adults. In children, pleura seems to have a tendency to form pus (Goodhart).† e. *Rigors*.—Slight, irregular, especially towards night. Often absent in children. f. *Preceding disease*.—Measles. Scarlet fever. Childbirth. Pyæmia. Small-pox. g. *Power of transmitting vibrations*.—Professor Bacelli's dictum that a thin homogeneous fluid (e.g., simple serous effusion) conducts sounds more intensely, though more slowly, than a more compound fluid;‡ (e.g., serous with flakes of lymph, or especially an empyema). h. *Edema*.—Often absent.

QUESTION OF TREATMENT OF NON-PURULENT, ESPECIALLY THE SEROUS, EFFUSIONS.—As a rule, treatment medical. "Mortality in uncomplicated pleurisy is exceedingly small" (Watson). These effusions are not like ascites, requiring mechanical aid; they are more or less inflammatory, and tend to be reabsorbed. Drugs. Iodide of potassium, syrup of iodide of iron, etc. Laxatives. Dry, nutritious diet.

QUESTION OF OPERATION.—If the above treatment fail, two questions arise. A. What is the danger of leaving the fluid? B. What is the risk of paracentesis?

A. *Danger of Leaving the Effusion*.—1. Risk of sudden death in large quiet effusions with persistence. Risk greatest in left-sided effusions, which displace the heart, and cause twisting of the inferior vena cava. Effect of sudden exertion. Risk of formation of thrombi. 2. Risk of lung being more and more tied down by adhesions. Not probable in sero-fibrinous effusions. 3. Risk of tuberculosis. Exaggerated in sero-fibrinous effusions. 4. Risk of slow purulent transformation. Only in a limited number of cases. If patient be weakly or pulled down by previous illness. Effect of chill.

B. *Danger of Paracentesis*.—1. Shock. 2. Syncope from alteration of position of heart or large vessels, by removal of supporting fluid. 3. Embolism from detachment of clots from pulmonary veins. To prevent 2 and 3, draw off slowly, and not all the fluid. 4. Oedema of lungs; an undoubted risk. Symptoms. Shortly after tapping (usually large effusions), urgent dyspnoea comes on, with frothy serous expectoration rich in albumen. Death often in 24-48 hours. Probable explanation. Compressed lung after removal of a large effusion = limb after use of Esmarch's bandage, i.e., vaso-motor nerves are paralysed, with expansion of long sudden stress thrown on toneless vessels; and hence the transudation of sero-albuminous fluids which = oozing after removal of the bandage (Dr. Duffin, BRITISH MEDICAL JOURNAL, 1874, vol. ii, p. 372).

INDICATIONS FOR PARACENTESIS IN NON-PURULENT EFFUSIONS.—1. Threatened failure of cardiac action. Signs. Pulse extremities, etc. Overloading of right heart. Signs. 2. In all cases, and at any date, when the fluid is so copious as to compress the opposite lung. Dangers. Signs. 3. In all cases where, with a large effusion, there have been one or more fits of orthopnoea. Emphatically required in some cases. Age. Coexisting lung-trouble. Distance of patient from medical man. 4. In all cases where a pleuritic effu-

* These abstracts were given out previously to the Lecture. See the remarks in the JOURNAL for January 20th, 1883.

† BRIT. MED. JOURN., 1877, vol. i, p. 729.

‡ Surg. Pathology, p. 245.

§ Loc. supra cit., and Med. Times and Gaz., 1874, vol. i, p. 497.

* BRITISH MEDICAL JOURNAL, 1877, vol. i, p. 759.

† Id., p. 797.

‡ Medical Times and Gazette, 1876, vol. i, p. 306.