the other tube give way? When we came to examine it, a little pinhole was found at the bottom of the chamber, and through that pinhole the germs got in. Here is a melon infusion; and, in order to show you what would have occurred if the infusions had not been protected from the floating dust of the atmosphere, we have hung beside this case two tubes that have been exposed to the common air and have fallen into a state of utter rottenness. In this way, from the Jodrell Laboratory at Kew, we have had these cases returned with their infusions perfectly intact. Even in our infected atmosphere, when we subject our infusions to experimental conditions sufficiently stringent, we are able entirely to shut out contamination, and to show that spontaneous generation never occurs. When we get clear of our atmosphere altogether, this is a matter of perfect ease and facility; and we find in Kew Gardens that nature runs her normal course.

CASE OF LARGE HYDATID TUMOUR OF THE OMENTUM TREATED SUCCESSFULLY BY A FREE INCISION, WITH ANTISEPTIC PRECAUTIONS.

By THOMAS ANNANDALE, F.R.S.E.,

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IN connection with the most instructive cases recently reported in the JOURNAL by Dr. Bradbury, I trust that a brief account of the following case may not be without interest. To Dr. Stone, my late house-surgeon, I am indebted for the notes.

M. F., aged 32, a married woman and a native of Shetland, was admitted into the Edinburgh Royal Infirmary under my care on the 11th of August last. Her history was that, about eight years before her admission and a few weeks after the birth of her first child, she noticed a small hard swelling at the lower part of the abdomen. This swelling was freely movable, gave her no uneasiness, and remained in much the same condition until about two months ago, when, soon after the birth of her fifth child, it began to increase in size and to cause pain. Her general health, too, began to fail, and she commenced to lose flesh.

When admitted, a tumour was found occupying the lower part of the abdomen. The tumour somewhat resembled in shape and size an ox's kidney, and lay across the abdomen a little below the umbilicus and towards the right side. It was freely movable, fluctuated indistinctly, and appeared to be connected with the omentum. No connection with the liver or other abdominal organs could be detected. The digestive system and general health were in a very unsatisfactory state, and both pulse and temperature were above the normal. After a week of general treatment, I requested Dr. Stone to puncture the tumour with the fine needle of an aspirator, and he accordingly drew off in this way a few ounces of a turbid fluid, which, owing to some mistake of the nurse, was thrown away before being examined. The patient expressed herself relieved by the tapping; but, a week after the operation, the tumour again increased in size and gave her more pain than formerly. The aspirator was, therefore, again used, and a small quantity of the same kind of fluid was drawn off; and, when carefully examined microscopically, hooklets of the "echinococcus" were found in it. After this second tapping, the patient became very ill, and the tumour increased rapidly in size. Her temperature was 103 deg. in the evening, and her pulse was high. She suffered also from rigors; and the tumour became more fixed, and was also more tender to the touch.

On September 19th, the tumour was as large as a child's head; and, from the patient's symptoms, it seemed evident that the tumour had suppurated, and that she must soon die if she were not relieved by the removal of the disease. On September 20th, I made a free incision in the mesial line over the tumour; and, the various layers of the abdominal walls having been divided, a large cyst was exposed. The wall of the cyst was thick and dense, and was found to be firmly adherent to the omentum and abdominal organs behind. In front, it was free. A large trocar and cannula were then passed into the cyst, but, owing to the large number of smaller cysts and their density, its contents would not pass through the cannula. Precautions having been taken to prevent any of the contents of the cyst from passing into the peritoneal cavity, which had been opened into by the incision, the cyst-wall was freely incised, when a large quantity of small hydatid cysts, floating in purulent fluid, escaped. The cyst having been in this way thoroughly emptied, the edges of the incision in the cyst-wall were stitched to those of the external wound, and two large drainage-tubes inserted into the cavity of the cyst. The whole operation was performed under the antiseptic

spray, and the usual antiseptic dressing was applied to the wound afterwards. Her progress after the operation was most satisfactory; and her pulse and temperature at once improved, and in a week were normal. The wound was daily dressed with antiseptic precautions; and the cavity of the cyst gradually contracted, leaving a small sinus, which in time also closed, and the patient was discharged cured on December 17th.

THE SUPRARENAL CAPSULE AND LYMPHATIC GROWTHS.

By P. H. PYE-SMITH, M.D., F.R.C.P.,

Assistant-Physician to Guy's Hospital, etc.

IN commenting on the remarkable case of lympho-sarcoma of the thorax, which Dr. Byrom Bramwell published in the JOURNAL of January 6th, he writes as follows: "The condition of the (left) suprarenal capsule was a remarkable feature of the case, and tends to show the close physiological relation which these organs have to the lymphatic glandular system. The absence of pigmentation of the skin proves that all forms of disease of the capsules are not necessarily attended by bronzing."

If Dr. Bramwell will refer to Dr. Wilks's classical paper on Morbus Addisonii in the *Guy's Hospital Reports* for 1862, he will see that the last conclusion was even then fully established, and it has since been abundantly confirmed by the exhaustive analysis of cases of disease of the adrenals, on which Dr. Greenhow founded his well-known Croonian Lectures, which appeared in the JOURNAL during March 1875. It is only the peculiar quasi-tubercular change in both organs which produces the symptoms of Addison's disease. Primary or secondary tumours, whether cancerous or sarcomatous, hæmorrhage, hydatid, amyloid degeneration, miliary tubercle, can no more produce bronzed skin, muscular weakness, feeble pulse, nausea and cerebral symptoms, than cancer, or hydatid, or calculus of one kidney can produce the symptoms of Bright's disease. True carcinoma, in the histological sense, probably never affects the adrenals (or any other organ without an epithelial surface) except as a secondary disease. But among the twenty-three cases in Groups VIII and X of Dr. Greenhow's list (*On Addison's Disease*, 1875, pp. 208, 210) there are several of various forms of sarcoma which in their clinical features were, like Dr. Bramwell's, malignant, and almost always secondary.

Almost all cases of primary intrathoracic growths are only clinically cancerous, and the majority are, like Dr. Bramwell's, lympho-sarcomata. This is the conclusion from Dr. Risdon Bennett's cases in his monograph on *Intrathoracic Growths* (1872), as well as from the numerous cases recorded in the *Pathological Transactions*. At one end, the series of these lympho-sarcomata approaches cancer, but, at the other, they become indistinguishable from the lymph-adenomata of Cornil and Ranvier. These again pass into the multiple glandular hypertrophy of Hodgkin and Wilks, and so by anæmia lymphatica to anæmia splenica, or true leucæmia. Dr. Bramwell does not state what were the microscopical characters of the blood in his case, during life or after death. Probably there was only anæmia without the increase of leucocytes, since even in the most "constitutional" and least "cancerous", i.e., the most multiple and least destructive form of Hodgkin's disease, it is doubtful whether true leucæmia has ever been observed.

A recent writer in another medical journal described a typical case of anæmia lymphatica as something new in this country, and announced the discovery of a similar condition, under the name "adénie", by Trousseau. The late eloquent professor at the Hôtel-Dieu made no such pretension. He says: "D'abord, messieurs, qu'il soit bien entendu que je n'ai rien découvert, que la description de la maladie a été assez bien faite avant moi; en Angleterre par M. Hodgkin (1832); en France, par un de mes élèves, M. Bonfils (1856); et en Suisse, par M. Cossy (1861)". (*Clin. Méd.*, tome iii, p. 569.) He afterwards mentions Dr. Wilks's name, who in the *Guy's Hospital Reports* for 1856, had accurately described the disease under its most appropriate title, "anæmia lymphatica". See also Virchow's comments (*Krankhafte Geschwülste*, Bd. ii, S. 619). I must not trespass on space by further references, but it is worth while recalling the labours of our own pathologists when they are so soon forgotten. In a late discussion on the subject, not only was the anæmia lymphatica of Hodgkin and Wilks confused by some speakers with the allied leucæmia splenica discovered by Virchow, but two other entirely unconnected diseases were brought in as allies—viz., idiopathic anæmia, first and completely described by Addison (*Works*, p. 212), since rediscovered and named "pernicious" by Biermer and others, and the totally distinct disease which is more generally connected with the same physician's name as morbus Addisonii. In a recent work on Medicine, it is stated that in the latter disease the white blood-corpuscles are sometimes increased

but I know of no evidence for this statement, and it is contrary to the very numerous observations which are already on record.

There is certainly no pathological connection of the suprarenal capsules with the lymph-glandular system. No less certainly is there no physiological connection between them. The function of lymph-glands with the other cytogenic structures (spleen and thymus, tonsils and Peyer's patches) is to make leucocytes; the function of the adrenals is unknown, if they have any in the adult, which in a recent number of the JOURNAL (December 9th, 1876, p. 741) I have given reasons for doubting. But the structure of the adrenals is totally different from that of cytogenic organs—as different as from that of true (secreting) glands. The absurd name, “ductless gland”, and the ambiguous terms, “glandular” and “adenoid”, have led to endless confusion in histology as well as in physiology, by connecting together such absolutely different organs as the suprarenal capsules, the thyroid, and the true lymphatic or cytogenic structures. That the confusion may not spread to pathology, is my apology for these remarks.

REPLIES TO DR. BROADBENT'S QUESTIONS AND OBJECTIONS IN THE MATTER OF THE SOUNDS OF THE HEART.

By ARTHUR LEARED, M.D., F.R.C.P., etc.,
Senior Physician to the Great Northern Hospital, etc.

THE tone of my friend Dr. Broadbent's communication in the JOURNAL of December 23rd reminds me of that of certain puritan pamphlets still extant. These productions bear such titles as “A Ready Method with Unbelievers”, “The Sinner convinced against his Will”, etc. There really seems to be a resemblance between the polemic effects of theology and those of controversy about the cause of the heart's sounds.

“In contradiction,” says Dr. Broadbent, “to Dr. Leared's statement that it is absolutely impossible to distinguish the sound caused by friction of the heart's surface against the end of the stethoscope from any allied sound—i. e., in this case muscular sound—I maintain, from a large experience of auscultation of the naked heart when assisting Dr. Sibson in his experiments, that it is both possible and easy.” The argument, of course, refers only to the contracting empty heart, because, so long as the organ is acting upon its contained blood, several other alleged causes of sound are in operation. From a large experience of the same kind, I can fearlessly meet one flat contradiction by another equally flat, or, in other words by maintaining my statement as above quoted. Now, the matter in question is stated to be possible and easy. A German investigator evidently did not regard it in this light when he took the precaution of causing the heart to act while surrounded by water. But I do not admit that the faint sound alleged to have been transmitted by the fluid medium was necessarily muscular. The experiment was also exposed to a fallacy arising out of the sound-waves formed by commotion in the water caused by the moving heart.

Let us now see whether Dr. Broadbent's contradiction cannot be met by one of at least equal force and free from all taint of partisanship with my views. Dr. Halford, now Professor of Anatomy in the Melbourne University, is well known to have performed many vivisections in reference to the question of the causes of the heart-sounds; and the conclusions arrived at were, in the main, not in accordance with mine; but he positively affirms that, when the supply of blood was cut off from the acting heart, “neither first nor second sound was heard”.

Dr. Broadbent's reply to my question, Why is it that in anæmia *the first sound of the heart only, and the second never, becomes replaced by a murmur*?—is “by disputing the fact. The first sound is not replaced under these circumstances, *but accompanied by a murmur*”. If Dr. Broadbent will modify his statement—and it will then equally answer its purpose—by inserting the word “always” before “replaced”, and the words “is sometimes” before “accompanied”, I will at once admit its truth. But how does this “take much of the ground from under” me? My contention is, that the body or main portion of the first sound is constantly transformed, under the influence of the change from normally viscid blood to abnormally thin blood, from a shock sound to a murmur. The accompanying feeble shock-sound heard in certain instances over the aorta may have various origins. “It may,” says Dr. Broadbent, “save trouble to say that I am aware that there are two first sounds, and that I speak distinctly of the co-existence of a murmur and sound both of left ventricular origin.” Now, it may also save trouble to remind the reader that so closely placed are all the apertures of the heart, that the bell of a very small

stethoscope would cover a portion of all the valves which close these apertures.

It is certainly possible by certain manœuvres to distinguish the sounds of the heart in relation to the separate sides from which they spring; but I maintain that, when the normal aortic sound is replaced by one of entirely different character, it is not always possible to guard against error arising from conduction of sound. If the chest-wall did not intervene, the difficulty would be much lessened. Now, an instance in which this difficulty did not exist presented itself to a very competent authority—namely, Cruveilhier—in the case of an infant born with the heart exposed. He found that the maximum intensity of both sounds was at the same place—namely, the base. And he adds explicitly: “Hence it is that at the base of the organ we are to look for the cause of these sounds.” Thus it comes to pass that, although “a systolic murmur and a first sound may coexist”, and is admitted by me, I am not conscious of any insecurity of “the ground”.

The undisputed facts are that, in anæmia without valvular lesion, the first sound of the heart, examined over its left side, is sometimes entirely replaced by a murmur, while at others the murmur is accompanied by a sound more or less resembling the normal first sound. The second sound at the same time never undergoes the like change. My explanation of this is, that in the one case the unnaturally thin blood allows the formation of a blowing sound instead of a shock sound, on known physical principles; while in the other case the intervention of the valve prevents the formation of the blowing sound on the same principles. When, on the other hand, a murmur replaces the second sound owing to incompetency of the valve close to which it is formed, more or less of the normal second sound may sometimes be heard coincidentally with the murmur. Dr. Broadbent would perhaps assign the normal portion of the sound to the vibration of the healthy part of the valve. This, for reasons too long to discuss, I would deny, and would assign simply to the same cause as I have already done the sound heard with the anæmic murmur on which Dr. Broadbent lays so much stress—namely, to conduction. There is, indeed, only one argument that can be brought to bear with effect against my view that sounds are always caused at the outlets of a heart acting with ordinary force, and that the aortic murmur of anæmia is nothing but a modified, or, as I would term it, a degenerated shock sound. That argument has been afforded by Dr. G. Balfour, who, in his lately published work on *Diseases of the Heart*, maintains that the anæmic cardiac murmur is not of hæmic origin at all, and is not formed in the great outlet-vessels. Founding his argument on the circumstance that, according to his experience, the murmur is most distinct just over the left auricular appendix, he ingeniously maintains that it is really mitral and regurgitant. Here the difficult question of conduction again meets us; and there are besides so many reasons for upholding the long established blood-theory, that I do not think he will find many supporters.

Dr. Broadbent ends his paper with a series of questions, which, in order to be answered, must be reproduced; but I shall abbreviate them when it can be done without injury to their meaning.

1. A sound due to collision between blood issuing from the ventricles with stationary blood in the great vessels must be produced at the aortic and pulmonic orifices. How is it, then, that the first sound is better heard over the ventricles, and even at the apex, than at the base and over the great vessels? If the first and second sounds be originated at the same point, they ought to be heard over the same area. Here the difficult question of the propagation and conduction of the first sound again presents itself. When the ventricle contracts upon its contents and the sound is formed at its outlet, it is probable that, owing to forcible contact with the stationary blood, sound-vibrations in the blood are propagated backwards and conducted by the walls of the ventricle, so as to make the first sound most audible over the ventricle and its apex. Such vibrations are prevented in case of the second sound by the closure of the valve, and therefore the second sound is not heard so distinctly over the ventricles. I put this explanation forward hypothetically. But I beg to draw Dr. Broadbent's special attention to the fact that there are exceptions to his statement. In some instances, the first sound is as loud or even louder at the base than at the apex. Moreover, how is he to explain the positive statement of Cruveilhier already mentioned in this connection? His objections are, I think, fairly balanced until these points are set aside.

2. In cases of high arterial tension, with its accompanying increased propulsive power in the left ventricle and consequent increase in the violence of the collision between the ventricular and aortic blood, “the first sound near the seat of this collision, its supposed cause, is enfeebled. Will Dr. Leared explain this?” He will, and, he believes, satisfactorily. From my various experiments on the production of