moulder, aged 49, who, on admission on March 16th, complained chiefly of cough, dyspnoea, and inability to speak above a whisper, the symptoms being of six months' duration. His family history was good, he had always previously enjoyed good health, was temperate in his habits, and had never suffered from venereal disease. About a year and a-half before admission, as the result, he thought, of exposure to cold and wet, he became slightly hoarse, and in a day or two this was accompanied by some cough, while he indicated the seat of irritation by pointing to his larynx. The hoarseness gradually became more pronounced, until he found it impossible to speak above a whisper; and two months before I saw him the cough became more severe, and was accompanied by slight mucous expectoration. Latterly he complained of weakness and loss of flesh, and exertion of any kind produced severe dyspnoea. He then consulted the late Dr. Foulis, who treated him by means of the induced current applied to the interior of the larynx, but without material benefit.

A laryngoscopic examination showed—what is probably the rarest form of laryngeal paralysis—bilateral paralysis of both abductors and adductors, the cords remaining in the "cadaveric position," that is, midway between complete closure and complete opening of the glottis. The heart was displaced downwards and to the left, the apex beat being $3\frac{1}{4}$ inches below and 2 inches to the left of the nipple line, and the veins on the left side of the neck and infraclavicular region were a little fuller than those on the right. The carotids were equal, but the pulse at the left wrist was much feebler than at the right, while pulsation was felt in the suprasternal notch. Slight musical rhonchi were heard at times over the greater portion of the chest on both sides, while the breath sounds were much more feeble over the left than over the right lung. It was therefore concluded that there was a tumour within the chest which had dislocated the heart, interfered with the free return of venous blood from the veins of the left side of the neck and the free passage of blood to the left upper extremity, and which pressed upon the left bronchus as well as upon the recurrent nerve. I came to the conclusion that the tumour was aneurysmal, and probably springing from the posterior wall of the left part of the aortic arch, thus accounting for the total absence of direct symptoms, with the exception of the slight pulsation in the suprasternal fossa. The patient's age (49) was quite in accordance with the aneurysmal theory, while there was no trace of the cachexia accompanying malignant disease, which is the most common form of solid tumour within the chest.

The sequel of the case was this: Nineteen days after admission (on the morning of April 4th), while sitting up in bed, this patient was suddenly seized with violent coughing and breathlessness, and, when the house-surgeon arrived, he found him semiconscious, livid, and spitting bright red blood. About 2 ounces had been ejected, when he became too weak to expectorate, and in about half an hour death closed the scene.

On *post-mortem* examination by Dr. Coats, an aneurysm about the size of an orange was found springing from the posterior wall of the whole of the transverse part of the arch, the great vessels springing from the aorta lying quite in front of it. It is doubtful whether the right recurrent nerve can have been pressed upon during life, but the left was subjected to very great pressure as it wound round behind the tumour, being, indeed, incorporated with it. The posterior wall of the aneurysm was very thin, and adherent to the oesophagus and trachea, which were flattened out and lying almost side by side. On opening the trachea, the left bronchus was found to be diminished in calibre, and, at the bifurcation of the trachea, there were two small openings communicating with the aneurysm, which accounted for the hæmorrhage at the last.

PROFESSOR KLEBS has declined the Chair of Pathology at Chicago, which was recently offered to him, and intends to settle at Karlsruhe.

FRENCH CONGRESS OF SURGERY.—The questions proposed for discussion at the next meeting of the French Congress of Surgery in Easter week, 1893, are the following: 1. Fibrous Tumours of the Uterus; 2. Surgical Treatment of Tuberculous Affections of the Foot.

TWO CASES OF SMALL OVARIAN TUMOURS SIMULATING UTERINE FIBROID: REMOVAL: RECOVERY.

By ALBAN DORAN, F.R.C.S.,

Surgeon to the Samaritan Free Hospital; Vice-President Obstetrical Society of London.

[WITH A LITHOGRAPHIC ILLUSTRATION.]

THE two cases which are described in this paper are of considerable clinical and surgical importance. In each, the tumour was small and closely connected with the uterus, so that uterus and tumour moved together. So far, the tumour bore the characters of a uterine fibroid. In each, however, the age of the patient and the relatively rapid growth of the tumour were in favour of ovarian disease, although fibroids may appear in young subjects and grow with rapidity, whilst patients are often mistaken about the rate of growth of tumours. The doubtful nature of the tumour rendered an immediate exploratory operation justifiable. The result proved that, had I waited longer, the risks of operating would have greatly increased. An ovarian tumour cannot be removed too early, and this rule is most important when bimanual exploration and the sound prove that the tumour is closely connected with the uterus. The removal of a large ovarian tumour with a very short pedicle, or no pedicle, is a formidable operation.

E. L., aged 30, a tall, strong woman, married eighteen months previously, but a widow for six months, was admitted into my ward at the Samaritan Hospital in December, 1891. She was a patient of my friend Dr. Chill, of Tollington Park. Two months previously she had noticed a tumour in the hypogastrium towards the right iliac fossa. It grew very rapidly, and for the fortnight preceding admission the patient was troubled by a continual desire for micturition. A firm, spherical, apparently solid mass occupied the lower part of the abdomen, reaching half way up to the umbilicus. The uterus was pushed to the left, the cervix was short, and the sound passed 3 inches upwards and to the left. The tumour extended into the right fornix, and to a less degree into Douglas's pouch and the left fornix. On placing the patient on her left side, the lower part of the tumour could be pushed up, the right side being much the more movable. The movements of the tumour were communicated to the sound. There was a tender body, evidently the left ovary, under the part of the tumour which pressed downwards in the left fornix. The patient had never been pregnant.

I could not feel certain as to whether this tumour was ovarian or uterine; its rapid growth was in favour of ovarian tumour, but a fibroid sometimes grows quickly. The close connection with the uterus and the length of the cavity of that organ gave all who examined the case a strong suspicion that the growth was uterine. I determined to explore. On December 21st, 1891, I operated, assisted by Mr. Butler-Smythe. Some slightly adherent omentum was detached from the surface of the tumour, which was found to be ovarian. On aspiration, a little ovarian fluid escaped, but I had to break down the semisolid interior of the tumour, and as it remained fixed I enlarged the incision and passed my hand behind the tumour, pushing it out of the pelvis. It was a semisolid tumour of the right ovary, with a broad, short pedicle. I secured the vessels on the outer border of the pedicle and then, after applying a large pressure forceps, transfixed the pedicle and tied it, finally cutting away the tumour.

The left ovary was then drawn up; it burst as I lifted it over the pelvic brim. The pedicle was very short. I applied a ligature by transfixion and cut away the ovary. The peritoneum was freely flushed with hot water. There was great vascularity of the pelvic organs. Uterine hæmorrhage had set in two days before the operation and continued for three days afterwards. The period was due a week later. A drainage tube was inserted into the wound. It was removed in twenty-three hours. The patient made a very good recovery and was discharged on January 18th in perfect health.

The right ovary (Fig. 1), weighed one pound and a-half after much of its contents had been removed during the operation.

FIG. I.—Tumour. Case of E. L. The numerous areolar spaces were filled with colloid material. Size about half of nature.

FIG. II.—Microscopic section of a portion of the tumour, Fig. I., showing the columnar epithelium investing the walls of the areolar spaces and undergoing the usual degeneration, so as to form the so-called "colloid" material.

FIG. III.—Tumour. Case of K. K. The dilated Fallopian tube is seen cut across. A rod has been passed into each segment, and the course of the tube on the outside of the cyst is indicated by dotted lines. On the left the tube is seen opening into the upper cystic cavity of the tumour. Size one-third of nature.

FIG. IV.—Microscopic section from a papillomatous mass growing from the inner wall of the lower cystic cavity of the

Its interior consisted of trabeculae enclosing areolar spaces stuffed with "colloid" material.

The left ovary measured 2 inches by 1½ inch, after soaking in spirit. On the site of a kind of cyst which had burst during extraction was a large clot, fading off into the ovarian tissue. There were a few small follicles near the surface, but the inner and middle part of the ovary consisted of a uniform, rather tough stroma. The tube had thickened walls, the ostium was patulous, the fimbriae well developed.

The microscopic examination of the substance of the tumour into which the right ovary was converted displayed areolar spaces lined with perfect columnar epithelium. The cells were being shed into the interior of the spaces so as to form the "colloid" substance (Fig. 2).

The pathological characters of the tumour have been given in full, for they show how formidable the operation would have proved had it been delayed a few months later. A large areolar cyst, full of actively secreting epithelial elements, and partly burrowing in the pelvis, gives great trouble to the surgeon, especially if adhesions be multiple—and they were beginning to form in this case, although the tumour was still small. In the next case the morbid growth was of a yet more formidable class.

K. K., aged 24, married 6 years, first came under my care in November, 1891. She was short and rather corpulent. She had never borne children nor miscarried; the period was regular with scanty show and little pain. About a year and a-half previously she noticed a swelling just above the pubes. It began to grow shortly before her first visit, and for two months it had caused great pain and prevented her from working. The lower part of the abdomen was filled by (apparently) two firm lobular growths reaching close to the umbilicus. The right, or rather the central, growth was spherical and very movable. The left was of an oblong oval form, lying above the left crural arch. As the abdominal walls were thick with fat and the tumours firm no distinct evidence of fluid could be ascertained manually. The cervix was long and free from any growth, the sound passed 2½ inches forwards; all movements of the sound were communicated to the left tumour.

On January 9th, 1892, I operated with the assistance of Mr. Butler-Smythe. The abdominal walls were very vascular, as is the rule in fibroid disease of the uterus, the exception in typical ovarian cystoma. The omentum had to be detached from the fundus of the uterus and bladder. A tumour was then exposed, which, from its glossy silvery surface on the left side, was evidently ovarian. The two swellings, apparently separate on external examination, were simply lobes of the same tumour. Over the right lobe part of the broad ligament was reflected. It was tapped with the aspirator, but the fluid contents were too thick for removal by that instrument. I had to enlarge the incision, then, on passing my hand behind the tumour, I lifted it out of the brim of the pelvis in which it was engaged. A few pints of thick glairy fluid escaped. The tumour was sessile, and burrowed under the peritoneum, touching the vermiform appendix. I transfixed the tissues at the outer border of the peritoneal connection of the tumour with the pelvis, and tied with No. 2 ligature silk; then I transfixed the base of the broad ligament, whence the tumour had just been enucleated, and tied with No. 3 silk, cutting the tumour away. On the left side was a dilated tube. I detached it from very firm adhesions to the pelvic peritoneum, and drew up the ovary; the appendages were removed, the pedicle being transfixed with No. 3 silk. The long thickened omentum was tied in four places and partly cut away. There was very free hemorrhage from the pelvis. The peritoneum was flushed with hot water. I then found that the ligature in the right broad ligament had slipped. I transfixed and tied the inner part, including the stump of the tube, placed a large elbowed pressure forceps on the outer part of the pseudo-pedicle, including the No. 2 silk ligature on the outer border, and transfixed and tied again. There was much oozing from the torn adhesions on the left side. The peritoneum was freely flushed with hot water once more. The drainage tube was applied, and the abdominal wound closed.

This patient made a rapid recovery. The vaginal temperature never rose above 100°. During the first twelve hours the drainage tube was emptied every two hours, several drachms

of deep red fluid coming away on each occasion. The tube was removed forty-seven hours after the operation. Eight hours later the period commenced, about four days before it was due. As is often the case when enucleation is performed and a broad false pedicle ligatured, there was considerable pain for the first two days. When the menstrual "show" appeared the pain subsided. The period ceased in four days. On February 4th she was discharged. I saw her on May 24th. She was in very good health, but complained of frequent flushing in the cheeks, a symptom so often seen during the menopause.

The importance of securing separately the large vessels in the outer border of the broad ligament was manifest in this case. Had it not been done, the slipping of the ligature would have caused very serious hemorrhage. When a tumour is sessile and touches uterine tissue, it is advisable to secure separately the inner border of the broad ligament, including the tube as well. For fatal hemorrhage may follow should the inner part of the ligature transfixing the broad ligament happen to slip after the operation.

The right ovary weighed 1½ ounces. It was converted into a tumour with a very irregular surface. It was distinctly, though not deeply, divided into two lobes. The broad ligament invested its outer half. A large piece of omentum and two appendices epiploicae adhered to the surface. The Fallopian tube, elongated and dilated, lay quite sessile on the surface of the cyst. It was divided in opening the cyst; its course is indicated in the drawing (Fig. 3) by two bent rods. The uterine end looked like a small oval excrescence on the tumour. This excrescence was formed by a pale fleshy substance with a few bright yellow specks, which infiltrated the tubal walls. A piece was preserved for the microscope. The tortuous channel of the dilated tube, about 4 inches in length, was lined with plicae, and opened into the cyst by a wide orifice guarded by a crescentic valve, which was infested with papillomatous growths (Fig. 3).

The interior of the tumour was divided into two almost equal parts by a broad septum of dense fibrous tissue, which included a large clot of the size of an almond. The one half of the tumour was a single cyst, bearing abundant papillomatous growths on its inner wall. The other half, placed uppermost in the drawing, was multilocular. The loculi near the orifice of the tube bore papillomata.

The fleshy substance in the tubal wall consisted of several layers of plain muscle cells in an almost transparent stroma. There were numerous spaces with sinuous margins lined with cubical epithelium. The appearances probably represented old inflammatory infiltration of the tubal walls (interstitial salpingitis), including some of the diverticula from the mucous membrane (Landau, Rheinstein, and Whitridge Williams), or the tubular structures of uncertain nature described by Drs. Ballantyne and J. D. Williams.¹

Fig. 4 represents a microscopic section of one of the papillomatous growths from the cyst wall. The drawing, like the others illustrating this paper, was made by Mr. Lewin. It shows an abundance of the most perfect papillary growths, as seen under a low power. The papillae bore cylindrical epithelium, on which I failed to distinguish cilia.

The left ovary weighed 1½ ounce. There was a large, perfectly fresh corpus luteum full of soft dark clot, and half an inch in diameter, close to the ovarian ligament. A similar corpus luteum lay external to the first. The tube was dilated, the ostium effaced; it coiled round the outer part of the ovary, reaching the under side of that organ, as often happens when the tube has been long dilated and adherent. The mucous membrane of the outer surface of the tube lay adherent, to the extent of an inch, to the surface of the ovary, which contained no cysts. Had a cyst existed, it must ultimately have communicated with the cavity of the dilated tube, then the condition would have corresponded to that observed on the right side as above described.

I believe that the disease consisted of papillomatous degeneration of the right ovary after long standing tubo-ovarian inflammation. The left side showed an earlier stage, the tube but not the ovary being cystic. There was no evidence of "ovarian hydrocele," a congenital condition described by Mr. Bland Sutton. The case bore a close re-

¹ Histology and Pathology of the Fallopian Tubes, BRITISH MEDICAL JOURNAL, vol. 1, 1891, p. 109.

semblance to another which I described and figured in the *Transactions of the Pathological Society*, vol. xxxix, 1888, p. 200. In that instance inflammatory changes in the appendages had set in seven years previously.

It is evident that an operation was even more urgent in this case than in the first. I have seen neglected cases where the papillomatous masses have passed beyond the limits of the cyst and invaded the pelvic viscera, so as to be irremovable. These cases ended fatally, whilst by timely interference in the case here described the patient was restored to perfect health.

ON THE ACTION OF DIGITALIS IN CASES OF AORTIC REGURGITATION.

By GEORGE W. BALFOUR, M.D., LL.D.,
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FROM Corrigan down to the present day many practitioners and authors have objected to the use of digitalis in cases of patency of the aortic valve from a theoretical idea that this drug, by prolonging the diastolic pause, favours the regurgitation of the blood into the ventricle, and so tends to fatally embarrass the heart's action. It is, however, nearly fifty years (1844) since the late Professor Easton, of Glasgow, pointed out that when the heart beats less frequently there is less opportunity for regurgitation, and not greater.¹ This is obvious enough: when a heart beats forty times a minute there are only forty regurgitations per minute; when the heart beats ninety times a minute there must be of course also ninety regurgitations. But it is argued that though the regurgitations are fewer, they are more prolonged, therefore greater in amount and more dangerous to the maintenance of the integrity of the circulation. But a prolonged diastole does not necessarily signify an increased reflux. Within the vascular system there is neither air nor a vacuum, but one unbroken column of blood extends from any given point within it back to itself again. The heart is the only part of this system endowed with the power of active movement, and upon it the circulation of the blood depends. When the left ventricle of the heart contracts it propels its contents into the aorta, that vessel—already full of blood—by virtue of its elasticity expands and receives this further supply thrown into it. When the systole of the ventricle is completed and its diastole commencing there is developed "a negative pressure in the rear of the column of blood driven from the ventricle into the aorta with great suddenness and rapidity."² This negative pressure follows the blood past the semilunar valves, and in the normal state it in part at least determines the closure of these valves. This negative pressure, which is greatest within the ventricle, is largely due to the return of the ventricle from its contracted to its normal form. At the end of the ventricular systole the segments of the mitral valve form a depending cone with its apex directed to the apex of the ventricle. This cone is full of blood in free communication with the auricle now fully distended,³ and at the moment of diastole the sucking influence of the negative pressure within the ventricle opens up its apex, the blood closely follows the expanding wall of the ventricle, and gently floats up and into apposition the segments of the mitral valve. At the moment of the ventricular diastole the systole of the aorta takes place, this vessel returns to its normal dimensions. In the normal man the systolic compression of the blood within the aorta helps to close the semilunar valves, but the whole amount received from the heart is propelled onwards. When the aortic valve is incompetent, the systole of the aorta, coupled with the negative pressure within the ventricle, determines a certain amount of regurgitation which can never be equal to the amount propelled, otherwise the circulation would at once come to a standstill. This reflux has been calculated as amounting to one-third of the whole ventricular contents,⁴ and is probably usually considerably

less than this. Under the influence of the aortic systole and of the intraventricular negative pressure the blood from the aorta and from the auricle closely follows the wall of the ventricle during its expansion, fills up its cavity, and floats up and into apposition the segments of the mitral valve. So soon as the ventricle has returned to what Foster calls its normal condition its active expansion ceases, and a period of rest or quiescence sets in, during which the blood within its cavity, in the aorta, and in the auricle comes under the influence of hydrostatic laws only.

The most important of these laws in regard to aortic regurgitation, is that to which the name of its earliest enunciator, Pascal, has been given. "Pressure exerted anywhere upon a mass of liquid is transmitted undiminished in all directions, and acts with the same force on all equal surfaces, and in a direction at right angles to those surfaces." This law has a probable influence in promoting the gradual dilatation of the left ventricle when the aortic valve is incompetent, but it acts very slowly, as one of the corollaries of Pascal's law is that what is gained in power is lost in velocity. There is also another and from the present point of view a more important corollary to Pascal's law, and that is that the pressure transmitted by a liquid is proportional to the extent of its surface. Now the area of the mitral opening may be taken at 1.25 square inches, and the area of the abnormal aortic orifice at 0.25 of a square inch, never more, often very much less. The ratio of the respective surfaces of these openings is thus as 5:1.⁵ From the mere surface point of view, therefore, the blood column in the auricle is four equivalents in excess of what is necessary to counterbalance the blood column in the aorta, and it acts perfectly favourably because there is a third corollary from Pascal's law which states that the upward pressure of liquids at any point is governed by the same laws as the downward pressure. We may, however, suppose the pressure of the aortic blood column to be equivalent to the blood pressure on the sound aortic valve which the ventricle has to overcome before the segments of that valve are thrown open and the blood projected into the aorta, equal therefore to a pressure of 250 m.m. Hg., or 3 metres of blood. But then we must also remember that from the absence of capillary resistance and from the non-existence of vasomotor nerves within the lungs, the mechanical effect of the systole of the right ventricle makes itself felt from pulmonary artery to left auricle, and this has been calculated at one-fourth of the force of the left ventricle, 60 m.m. Hg., or $\frac{1}{4}$ of a metre of blood. These figures multiplied by five to bring them to the proper ratio give more than enough of upward pressure—300 m.m. Hg., or $\frac{3}{4}$ metres of blood—to prevent any regurgitation from the aortic orifice. But if we consider that the filling up of the ventricle detracts somewhat from the intrapulmonary pressure, and suppose that the blood passes through the mitral orifice at the pressure of only 6 inches of blood, as Davies has done,⁶ then we must also allow for the reduction of blood pressure by regurgitation from the aortic column, and this coupled with the patency of the valves, would seem to reduce the pressure in that column to that of about 18 inches of blood or the height of a column of blood reaching from the base of the heart to the top of the head. But the pressure at the mitral orifice—6 inches—multiplied by five, its proportional area, gives a relative pressure of 30 inches at the mitral orifice, much more than sufficient to prevent all regurgitation through the aortic orifice during the quiescent period of the diastole.

A slow pulse, therefore, not only gives fewer opportunities for regurgitation, but though it is indubitably accompanied by a prolonged diastole the regurgitation is not thereby increased, the reflux is actually diminished by a slowing of the heart's action, and the balance of the circulation made more equitable. The action of digitalis therefore in slowing the heart is not hurtful in aortic regurgitation but beneficial. But apart from its action in slowing the heart—which is a mere accidental or secondary effect—digitalis possesses pre-eminently the properties belonging to what is called the digitalis group of remedies. It increases the elasticity of the muscular tissue so that this extends and contracts more completely and perfectly, and as all the blood passes more frequently through the heart than through any other muscle,

¹ *Monthly Medical Journal*, July, 1844, p. 634. Easton goes fully into the question, and his paper is well worth studying.

² Foster's *Physiology*, 6th ed., p. 248.

³ Pettigrew, On the Relations, Structure, and Functions of the Valves in the Vascular System of the Vertebrata, *Transactions of the Royal Society of Edinburgh*, vol. xxiii, p. 799.

⁴ Davies, *The Mechanism of the Circulation through Diseased Hearts*, London, 1889, p. 51.

⁵ Davies, *op. cit.*, p. 48.

⁶ *Loc. cit.* The calculations in this book are by Dr. Haughton, of Trinity College, Dublin.