PART IV.

MEDICAL MISCELLANY.

Reports, Transactions, and Scientific Intelligence.

ACADEMY OF MEDICINE IN IRELAND.

President—ROBERT M'DONNELL, M.D., F.R.S.
General Secretary—W. THOMSON, M.D.

PATHOLOGICAL SECTION.

President—WALTER G. SMITH, M.D.
Sectional Secretary—J. B. STORY, M.B.

Friday, May 13, 1887.

The PRESIDENT in the Chair.

Malformation of the Shoulder-Joint.

DR. E. H. BENNETT exhibited a specimen of malformation of the shoulder-joint. It was a form of malformation or disease that he had never seen before; and the only way in which he could read it was that it was not congenital, nor the result of a fracture or dislocation, but a condition brought on by the action of a burn, the cicatrices of which existed on the limb and near the joint. The limb belonged to a slender man of middle age. It was dissected in the School of Physic, and had previously attracted attention in consequence of deformities that appeared in the fingers, the result of burn contractions. The little finger was burned towards the ulnar side, and its extremity had been lost; and the finger next it had suffered similarly, but to a lesser degree. The cicatrices were not such as to produce large webs or great deformities, but the end of one finger was completely destroyed, and there were marks upon the limb of burning of the skin. These were the only circumstances that threw light on the question of the cause of the deformity. It was noticed before the dissection was made that the shoulder-joint, although not completely fixed, was limited in its range of motion. There was nothing abnormal in the muscles, except that those of the dorsum scapulæ were

to some extent wasted; all the muscles were, however, otherwise good. The condition of the shoulder-joint was curious. On clearing away the soft parts on the outside of the joint, he found what at first sight appeared to be the head of the humerus directly underneath the muscles, without any capsule, while the upper part of the humerus was distorted and bent. There was no disease of the scapula or glenoid cavity; on the contrary, everything there was normal. The length of fibrous capsule was very small. The appearance, as of a head of the humerus, which the specimen presented, looking at it from the outer side, was fallacious. On turning the specimen it was seen that the head of the humerus was half buried in an ossified capsule; while the remaining half of the capsule was free, soft, and flexible, and received the attachments of the capsular muscles normally on the axillary side. There were no deep cicatrices involving the muscles; and the tendons and joint had been in no way affected except by the skin contraction. It was hard to account for this condition of the capsule and the extraordinary limitation of the ossification of it to the outer side. On the inside the cartilage of the joint was perfectly normal. He had not made any section through it, because he did not think it would reveal much. The head of the bone was greatly distorted in position. He did not think there had been any fracture of the bone, but believed that the flexion of the upper part of the bone and distortion of the head of it had been produced by the limited range of movement resulting from the superficial cicatrices.

The PRESIDENT asked was there not some deformity of the shaft of the bone?

DR. BENNETT.—It is twisted and bent.

DR. HEUSTON said it appeared to him that there was a line of fracture running towards the head on the inner side and proceeding through the epiphysary line.

DR. BENNETT, in reply, said the only fracture which produced a deformity in any way similar was that of an impacted fracture external to the capsule; but he did not know of any other specimen that presented a similar feature as regarded the capsular ligaments. He had thought it better to show the specimen first without making any section; but he would make a section; and if it should reveal anything in the nature of a fracture, the fact could be recorded in the report. It was like the deformity that resulted from chronic inflammation; but there was no evidence here of chronic inflammation beyond the capsular ossification. If a fracture existed it would give additional interest to the specimen; but the remarkable thing was the association of the deformity with the cicatricial destruction on the ulnar border of the hand, and along the forearm; as the marks of the burn—though it was only of the skin—reached as high as the axilla. The deformities must have existed for a great number of years, if not from childhood.

Capillary Transudation.

Mr. Story exhibited an apparatus intended to illustrate an experiment of Körner upon capillary transudation. It had been made by Mr. Yeates, of Grafton-street, and he had to express his thanks to Dr. M'Kee, Sir Charles Cameron, and Dr. Piel for assistance in bringing it before the Section. The experiment of Körner was known to him (Dr. Story) only second-hand through an article on the pathology of glaucoma written by Birnbacher and Czermak in von Graefe's Archives. In glaucoma the increase of tension of the eyeball can be produced only by an increase of the fluid that goes into the eye, or by a diminution of the fluid coming out of the eye, or possibly by the joint operation of both of those causes. Before proceeding to hypothesise as to the production of glaucoma, those two authors thought it necessary to find out what were the physical laws governing the passage of fluids through tubes which, in part of their course, had walls that were permeable to fluids and movable. The blood went into the eye through arteries and came out through veins. Between the arteries and the veins it passed through capillaries which had permeable and distensible walls. By the experiment of Körner—which the apparatus represented—fluid was passed through a tube formed partly of India-rubber and partly of brass, to which two manometers were attached. Another portion of the tube, which was enclosed in a glass cylinder, was formed of a membrane that admitted of filtration—namely, the ileum of a monkey, off which the peritoneal coat had been stripped. When water was allowed to flow freely through the system, a filtration of the fluid took place through the membranous part of the tube into the glass cylinder, which cylinder might be termed the tissue chamber, and the filtration went on steadily until the pressure in this chamber became higher than the pressure on the exit tube, and higher than the pressure in the portion of the membranous tube next to the outflow. He found that no matter what was the pressure applied—whether there was a restricted arterial current or a restricted venous current—in all cases, sooner or later, the pressure in the tissue chamber rose till it became higher than that in this distal portion of the membranous tube, and then this portion of the tube collapsed and all flow ceased. The conclusion which Körner drew from his experiment—the result of which was published in 1873—was that there must be some exit for the fluid from the tissues of the body independent of the venous circulation—namely, the lymph vessels—or the rise of pressure in the tissues would ultimately stop the flow in the veins completely. Birnbacher and Czermak, then, in attempting to produce an arrangement which would permit a constant flow to go on, hit on the expedient of placing within the membranous part of the tube a spiral wire which prevented the membrane from collapsing. When this was

set going the proximal (or arterial) end of the tube was seen to be raised up from the wire by the pressure of the contained water, while at the distal (or venous) end of it the membrane was pressed down upon the spiral by the higher pressure in the tissue chamber; and they concluded that a filtration took place from the inside of the membranous tube into the glass cylinder at the proximal end, and that a filtration back again from the chamber into the tube took place at the distal end. The pressure in the tissue chamber became higher when they restricted the "venous" outflow, when they increased the "arterial" pressure, and when they used a more permeable filter at the arterial end, or a less permeable one at the venous end of the membranous tube. There were certain points in the anatomy of the eye which went to make out a strong analogy between the circulation of the blood in that organ and the conditions of this experiment. The blood abstracted from the retinal circulation passed into the eye through fifteen or sixteen ciliary arteries, and nearly all passed out again through the venæ vorticosæ. The venæ vorticosæ, of which there were from four to seven, ran into large sinuses or ampullæ before they made their exit through the sclerotic emissaries. The veins from these ampullæ through the emissaries were much narrower than the ampullæ, and their walls were exceedingly thin. The emissary in the sclerotic had a funnel-shaped opening. The walls of the vein there were not unsupported, but were adherent to the wall of the emissary either directly or by means of fibrous tissue, and were thus protected against compression. The coat of the vein became ten times thicker as soon as it passed through the sclerotic. The result of that anatomical arrangement was that there was a very high pressure on the ampullæ and a sudden and extensive decrease of pressure in the emissary vein. Another reason why the blood pressure on the ampullæ should be high was the unusually large size of the chorioidal capillaries. The pressure in these ampullæ must be nearly as great as the intra-ocular pressure, as there was nothing to prevent them from collapsing. The pressure on the emissary vein must be considerably lower; it could not be very much greater than that in the facial vein (a pressure of 5·2 mm. of mercury has been found in the facial vein of the sheep, while the intra-ocular pressure averages from 30 to 35 mm.), consequently there was every facility for the filtration of the fluid from the eye back into the vein as it left the ampullæ. On the anatomical structure of Schlemms' canal he need not dilate—suffice it to say that the veins there were guarded against compression, and the fluid of the anterior chamber filtered through into those veins. In fact, the veins must be maintained in a permanent condition of dilatation, or else they would not be able—as they were, with dead eyes—to inject those veins from the anterior chamber. Birnbacher and Czermak went further, and injected coloured fluid into the supra-chorioidal space, through which the emissary vein

passed, as it went into the sclerotic; and they found that the coloured fluid came out on the exterior of the globe through the lumen of the vein, and not alongside of the vessel by means of any perivascular space. Their conclusion was that there was a filtering arrangement in the venæ vorticosæ similar to the filtering arrangement in the second apparatus before the Section, in which there was a wire spiral inside the membranous tube to prevent it from collapsing. The best experiments on the effect of the circulation on intra-ocular pressure in the living eye were those of Schultèn, who had found that the intra-ocular pressure varied with the blood-pressure; that it was raised by either an increase of arterial inflow or a diminution of venous outflow; and also that lessened arterial tension raised the pressure. He found that the pressure on the eyeball was lowered if the arterial flow was lessened; and that it was also lowered if the walls of the arteries were put into a state of spasm. The analogy of those observations of Schultèn to those of Birnbacher and Czermak was very perfect. It could be seen in the apparatus that the increased flow at the arterial end raised the pressure not alone in what corresponded to the artery but in the manometer attached to the tissue chamber—both went up together. If the venous outflow were increased the pressure in the tissue chamber diminished; if they diminished the venous outflow the pressure rose, not only in the artery but also in the tissue chamber. There was a very close analogy between the circulation in the eyeball in those respects and the circulation in the brain. The cerebral venous sinuses were defended from collapse by being placed in rigid fibrous channels, which guarded them from the effects of increased intracranial pressure. There were no important lymph paths conducting lymph out of the brain. The cerebro-spinal fluid made its way through such processes as the Pacchionian bodies into the venous circulation. On the special pathological bearings of this experiment he would now touch. It was brought forward by Birnbucher and Czermak at the close of their paper, which stated that they had made a microscopical examination of glaucomatous eyes, and had found that, in addition to the ordinary well-known blocking up of the angle of the anterior chamber by adhesions of the root of the iris to the cornea, the venæ vorticosæ were affected, and that in the emissary veins coming out from the ampullæ there were signs of inflammation, which had thickened the walls of the veins and destroyed the permeability of the space surrounding them; and that there was also an internal inflammation inside the veins, so much so that some of them were completely blocked up by proliferation of the endothelium. The result of the experiment showed that in glaucomatous eyes there was a *vera causa* capable of increasing the intra-ocular pressure, and that that intra-ocular pressure was governed by the physical laws exemplified in the apparatus before them. Whether those arrangements which Birnbacher and Czermak

said existed in the eye to prevent the occurrence of abnormal pressure were those which did prevent its occurrence or not, it was too early to say; but there must be some arrangement similar to what they described to prevent the complete stoppage of the circulation of the blood, having regard to the conditions under which it circulated in the eyeball. There must necessarily be some arrangement to prevent the compressing and collapsing of the veins.

The PRESIDENT said the reasoning founded on this experiment was applicable to the circulation of the blood in other organs besides the eye. He would ask Dr. Story did his ophthalmic brethren consider that this experiment went far towards solving the disputed problem as to the ætiology of glaucoma? Also, he wished to ask what difference did the quality of the fluid make? Experiments made with pure or coloured water would hardly represent conditions *intra-vitam* where a fluid impregnated with saline materials had to be dealt with.

DR. BENNETT said if these experiments gave the true reading of the action of the vessels in producing the aqueous humour, the latter should be more akin to serum in its constitution than the name indicated. If it were the result of such constantly changing pressure as had been described, its condition would be not far from that of albumen.

DR. STORY, in reply, said it was not implied by him, and those from whom he took those observations and experiments, that they represented the whole of the actions that took place, in the interchange of fluids, between capillaries and tissues. To some extent that interchange must be governed by mechanical conditions, such as the amount of pressure inside and outside the filtering wall of the capillaries; and so far as that was the case, the laws represented by these experiments held good. Of course it might make a difference if the fluid was not pure water, but was a saline, diffusible solution. Birnbacher and Czermak stated in their paper that the pressure varied according to the quality of the fluid, as well as according to the quality of the filter. But, as far as he (Dr. Story) had been able to make out, they did not make experiments in that direction. Variations in the quality of the fluid practically did not come in. It was extremely probable that the walls of the capillaries had the power in themselves of altering their power of filtration so as to offer more or less resistance to the passage of the fluid. Contraction in the walls of an artery at once diminished the degree of the pressure in the area of the district supplied by that artery. There were a number of interesting experiments in the paper of Birnbacher and Czermak showing that increase in the pressure was neutralised by contraction of the artery. It was raised by restricting the venous outflow; but if the arterial pressure was subsequently diminished sufficiently to make the pressure of the so-called "artery" in the mechanical apparatus the same as it was at the commencement of the experiment, the pressure in the tissue chamber did

not fall down to what it was at the commencement of the experiments, but remained permanently higher. So that the contraction of the vascular walls must not alone compress the artery sufficiently to make the pressure on it what it was before the vein was constricted, but it must lower the arterial pressure even still below that—in fact, there must be a spasmodic contraction of the coats of the arterioles to keep down the pressure on the tissue to the normal degree. In reply to the question as to what his ophthalmic brethren thought of this experiment, he was afraid that none of them had read it or knew anything about it. The experiment of Körner was published in 1873; but in spite of the vast amount of work that had since been done in connection with glaucoma, no one seemed to have noticed its importance till Birnbacher and Czermak had taken up the subject in their paper in the December (1886) number of von Graefe's Archives.

Fracture of the Skull.

Dr. Heuston exhibited the skull and brain of a gentleman, aged sixty, who had fallen from a window into a flagged area on his head, a distance of twelve feet. After the accident he presented no evidence of serious lesion for five days, when symptoms of cerebral irritation began, eventuating in hemiplegia of the left side of the body on the tenth day, when he died from paralysis of the muscles of respiration. The skull presented a fracture occupying the posterior portion of the right parietal bone, which included a somewhat quadrilateral area, the enclosed portion of bone being further broken into three portions, the uppermost of which was depressed, while the two lower were elevated at their junction. From the posterior inferior angle of this fracture a fissure extended through the masto-occipital and petro-occipital sutures to the jugular foramen, from which a fracture extended into the foramen magnum posterior to the occipital condyle. An extensive clot was also found occupying the position of the fracture, and also filling the posterior fossa of the skull between the dura mater and the bone; the dura mater in this situation presented a laceration an inch in length. The right cerebral hemisphere presented a laceration at the junction of the occipital with the temporo-sphenoidal lobe extending obliquely upwards and backwards for an inch and a quarter, and being half an inch in breadth. The inferior aspect of the right lobe of the cerebellum showed a laceration occupying the biventral lobe half an inch in length.