that Deutchmann's hypothesis of an intrauterine inflammation becomes applicable in many cases. The glaucoma in this eye, Mr. Lang said, was difficult to explain; the theory that iridectomy is a safeguard against glaucoma being apparently negated by it. A possible explanation, he thought, might be that the trabecular tissue constituting the spaces of Fontana may have been absent in this case at the coloboma, and the available space for filtration of lymph would therefore have been smaller here than in a normal eye, and hence the liability to glaucoma greater.

This eye subsequently became painful, and Mr. Lang was obliged to excise it, and he gave it to Mr. Treacher Collins for pathological examination. Mr. Collins found the iris absent on the outer side, but the ciliary body was present in that position. On the inner side the root of the iris was in contact with the posterior surface of the cornea, blocking the filtration angle. The case, Mr. Collins said, presented some points for consideration. First, the unusual position—namely, outwards—of the iris coloboma. It is frequently, and Mr. Collins thinks wrongly, assumed that a congenital deficiency on the iris must necessarily bear some relation to the ocular cleft. But, in another remarkable paper on iridemia in this same volume (vol. xiii, p. 128), Mr. Collins showed that the development, not merely of a part of the iris but of the whole of that organ, may be arrested by abnormal adhesions or late separation of the cornea and lens, preventing the ingrowth of the iris; and if this abnormal adhesion or late separation happened to be confined to a portion of the circumference in one or even in two directions, a coloboma, or even two colobomata, might be produced in any direction.

The occurrence of glaucoma in an eye with congenital iris coloboma is remarkable. But here at the coloboma there was a rudimentary iris, as also in Mr. Collins's case of aniridia, which was intimately adherent to the ligamentum pectinatum, and Mr. Collins is of opinion that this adherence was congenital. It then only remained for the root of the iris in the rest of its circumference to become pushed forward in order to bring on high tension. Mr. Collins states he does not agree with Kiefleisch in ascribing intrauterine inflammation beginning in the choroid and extending forwards, and causing perforation near the corneo-scleral margin and escape of aqueous, as the primary cause of the abnormal contact. In this specimen no sign of perforation was present. Again, the corneo-scleral margin is an unusual seat for a perforation. Furthermore, it is unlikely that a bilateral condition, such as aniridia usually is, would be caused in this way. And, finally, it is unnecessary to resort to this explanation, for at the period when the iris is developing, there is no anterior chamber, and hence the lens capsule and the cornea are in contact, the anterior fibro-vascular sheath alone intervening.

In these papers we have examples of Mr. Treacher Collins's admirable pathological work, and it affords me much pleasure to take this opportunity of saying that, highly as that work is valued by us in this country, and especially in this Society, it is esteemed, as I very well know, equally highly in another country, where pathological anatomy is perhaps more at home than with us. It was, therefore, not without a pang of regret that I, for one, heard of the translation of Mr. Collins from the post where he did such good and useful work, to another, which we hope will lead him on to fame in a second direction. And now, in conclusion, I desire to direct your attention to a remarkable case of Mr. Henry Power's in vol. xiv, p. 212, under the title of—

A CASE OF MICROCEPHALUS AND PROPTOSIS.

The case was that of a female child born at full term, after an easy delivery, and you will remember the remarkable picture given of the infant's face and head. It died when about a month old. The head was misshapen, and the eyes were both projecting to so great an extent that the margins of the lids were far behind the equator of the globes. The globes could not be pushed back, but the eyelids could be pulled forwards, and made to meet in front of the eyes. The head was very short in the antero-posterior direction, but of considerable height, especially in the frontal region, where it became cone-shaped. After hardening in spirit, a sagittal section of the head was made

passing through the vertical meridian of the right cornea. The right orbit, which was thereby opened, was found to be extremely shallow, its depth being only 2.5 c.cm. from its inferior border to the optic foramen. The height of the orbit, on the other hand, was increased, measuring 32 mm. at its most anterior part, while 20 mm. is the normal measurement at this period. The corpus callosum was absent, and in certain localities the surface of the brain presented a microgyrous condition. After removal of the brain, a photograph of the skull was taken by Dr. D. J. Cunningham, Professor of Anatomy in the University of Dublin, who, like myself, was much interested in the specimen. The floor of the orbit was horizontal, but the roof was almost vertical in position, being directed upwards and forwards at an angle of 45°, and becoming continuous with the vertical plate of the frontal bone, into which it passed with only a small projection to mark the orbital edge. A perpendicular line from the front of the vertical portion of the frontal bone would fall entirely behind the globe. The optic nerve, from its entrance at the optic foramen, ascended sharply and remained in close relation with the displaced roof of the orbit, till it bent downwards to enter the sclerotic. A complete explanation for these extraordinary conditions is not given in Mr. Power's communication, and as I was particularly desirous to have one for a special purpose, I applied to Dr. Kanthack, late Curator to the Museum of St. Bartholomew's Hospital, and he in the kindest manner lent me the specimen for further examination, and I then submitted it for that purpose to Professor Cunningham. He ascertained that with the single exception of the joint between the occipital and the basi-occipital, every suture and synchondrodial joint in the skull was firmly ossified. The consequence of the solidification of the base, and the ossification of the vault into a continuous bony sheet, was the general uplifting of the front part of the roof of the cranium by the growing brain, and the simultaneous dragging up with it of the orbital plates, to which is due the extreme shallowness of the eye sockets.

It was also found that the convolutions of the brain had pressed so hard against the ossifying cranial vault, that the normal dimples had become deep bony pits, and the floors of these pits had become exceedingly thin and diaphanous. The case affords a valuable instance of the effect of premature synostosis in producing such deformities, and their resulting displacements of the eyeballs.

REFERENCES.

¹ Schapinger, *New York Med. Monats.*, January, 1893. ² *Die angeborenen Beweglichkeitsdefecte der Augen*, p. 45; Deutschmann's *Beit. z. Augenhk.*, xix Heft, 1895.

CASE OF BRADYCARDIA WITH RAPID REMISSION AND FATAL RECURRENCE.

By J. M. RATTRAY, M.A., M.D.,
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MUCH has yet to be learned as to the pathology of bradycardia, and I venture to think that the case I now record is not without special points of interest. My determination to publish these notes was prompted by the perusal of an important and interesting account by Dr. Watson Williams, of Clifton, in the June number of the *Bristol Medico-Chirurgical Journal*, of the whole bearings of paroxysmal tachycardia. Both in this and in its opposite, bradycardia, Dr. Watson Williams draws special attention to the suddenness of onset and decline, and shows that the one condition may suddenly pass into the other. This occurred in my patient's case, and I think it worth noting, so that in any future similar condition under observation a careful continuous record of the pulse-rate may be taken.

In the early part of this year I saw the last of Mrs. C., a farmer's wife, aged 70, whom I had attended intermittently for twelve years. She suffered from paroxysms of pain in the left lumbar region. These came on suddenly and so intensely that nothing but a stiff injection of morphine, administered the moment I entered her bedroom, would quiet her or enable her to resign herself to giving details of the onset of her attacks. No information was obtained by physical examination of the affected area, but the urine was scanty, over-acid, and loaded with lithates. With recoveries that were pretty sudden, and sometimes long-sustained, a diagnosis of neuralgia was given to the patient and her

friends; but it was constantly in my mind that a renal calculus might be the source of the mischief, and that further developments would substantiate this suspicion.

I saw little of my patient during 1896 until the last week of the year. She was then suffering from dyspepsia, and I could not help noticing that she had become considerably thinner and altered in appearance. On examination in bed I detected a slight musical murmur, which was persistently heard a little to the right of the apex beat. Her pulse-rate was about 48 a minute at the outset of my attendance, and her symptoms were headache, restlessness, and insomnia. Later on fainting attacks supervened, which soon became frequent and very alarming, and I considered a great change for the worse had come over her. I had very little success in the management of the case during the very dull wet days of late January and early February of this year; rest in bed, occasional doses of antipyrin and bromide of ammonium to relieve headaches, and a course of treatment aiming at checking acidity were the means mainly relied on. Later on free heart stimulation by digitalis, alcohol, and subcutaneous injections of strychnine with ether were employed to remedy the faintings, which were incessant. With the warmth of bed and a supervised diet the attacks of lumbar pain completely ceased. One day on exposing her chest for stethoscopic examination I was surprised to find an eruption of herpes zoster extending round the same region where she had suffered previously from the neuralgic attacks.

During the whole course of my patient's last illness her temperature was subnormal and pulse abnormally slow, ranging about 40 per minute. A fortnight before her death on counting the radial pulse and comparing with the beat at the apex the rate did not rise above 35. The character of the pulse was that of moderate tension, the rhythm was regular, and, with the exception of its curious behaviour on one day, its course was that of gradually increasing slowness.

As the condition was obviously serious the case was seen with me by a neighbouring practitioner, a careful observer, to whom I had sent details by letter. We had some distance to go to meet at the house, so I had not seen the patient previously during the day. I was much surprised when on auscultation the consultant said to me in an undertone: "Your bradycardia is gone." On examining for myself I then found that the slow laboured action of the heart had disappeared, being 128 beats per minute. Our conjoint prognosis was a grave one, the consultant agreeing with me that the patient's condition was very serious. How the changes in the pulse-rate took place I am unable to say, but on my subsequent visits the bradycardia was again pronounced. In a day or two the pulse had returned to the 35 per minute rate, and on the tenth day after the consultation the radial pulse and the heart beat, counted together, registered 26. On the evening of that day, while the nurse and daughter were preparing some article of food in the room, the patient suddenly died.

It was a matter of extreme regret both to the consultant and myself that the relatives would on no account permit the patient's body to be subjected to a post-mortem examination.

The clinical facts in this case were:

1. Old age and degenerated blood vessels; uric acid diathesis. The patient had enlarged small joints, and the urine was habitually acid, with deposit of lithates. One brother had suffered from gout severely.

2. Intercostal neuralgia, the climax of the foregoing condition. The cycle of habits previous to her last illness was: Intense pain, doctor, morphine, examination of urine, treatment for acidity, recovery, care for a time, less care, no care at all, recurrence of pain, etc.

3. Cardiac dilatation, with probably diseased semilunar valves. The patient exhibited short breath and had a slightly cyanosed appearance with blue lips. My patient suffered from headache and vertigo towards the last, but had no convulsive seizures.

Bradycardia, as is generally known, is coupled with tachycardia, as the extremes of aberration from the pulse-rate of health; 40 beats per minute and anything under may be considered as morbid, and bradycardia established. It has been held that the cause of the affection is the application of an irritant to the ends of the vagus in the heart, and that the inhibition of the heart is increased by the presence of certain morbid matters circulating in the blood which act directly on the heart muscle. In the case I have recorded one would certainly have found some gross pathological condition associated with the abnormal cardiac rhythm. Probably the changes on which bradycardia depends are degeneration of the heart walls, fatty heart, and depression of the nervous system through the action of poisons, bile, urea, etc., which are present in the blood in certain conditions. In my case the development of the slow pulse was simultaneous with the changes in the heart and system, evidenced by the symptoms and physical signs of progressive cardiac dilatation. Neuritis and acidity were present during many years of intermittent suffering, but not until definite structural changes in the heart appeared was the bradycardia distinctly pronounced.

I have related the case mainly on account of the interest attached to its unexpected termination. Its lesson is the grave significance of the onset of the very slow pulse, combined with the symptoms of fainting, sufficiently alarming at first, but not enough to lead one to predict that treatment would be altogether ineffectual, and that the patient's end

was so very near. Again, the sudden interruption in the course of the bradycardia is to be noted. This is difficult to explain. Emotional excitement of a patient submitted to a consultation between two medical men is scarcely a strong enough reason; but nevertheless its occurrence corresponded with that transaction.

PEMPHIGUS CONTAGIOSUS.

By GERALD SICHEL, F.R.C.S.Eng.,
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[Communicated by the DIRECTOR-GENERAL OF THE MEDICAL DEPARTMENT OF THE NAVY.]

UNDER this name, Dr. Patrick Manson (in Davidson's *Hygiene and Diseases of Warm Climates*) describes a skin disease of damp tropical countries. It is unaccompanied by any constitutional symptoms, and the elementary lesion is described as a large, hemispherical, clear, tense vesicle or bulla; these vesicles become flaccid and burst. Two forms are described, according to their distribution: an axillary form, met with in adults; and a diffuse form, mostly seen in infants. As the name implies, Dr. Manson considers it a contagious disease.

This description more or less tallies with a series of cases which occurred on board H.M.S. *Gibraltar* during the first three or four months that the ship was at Zanzibar (arriving there during the "lesser" rains or "vuli"); cases also occurred on board H.M.S. *Sparrow*; she had just recommissioned, so that the men were new to the climate. The disease does not appear to be met with on shore, so that I take it to be more or less special to newcomers.

The disease as I observed it appeared to occur in three forms, two of which were met with as sequelæ to lichen tropicus, and the third in which the eruption appeared on previously healthy skin. By far the most common was a pustular rash, following closely on "prickly heat." The following case is a typical one:

D., aged 29, a chief stoker, who had been suffering from an attack of fever (from which he had entirely recovered), had also had an extensive outbreak of lichen tropicus, especially on his legs, the remains of which still lingered both on these and on the trunk. The following rash then appeared: flat-topped, bright yellow pustules, surrounded by a narrow, well-marked, hyperæmic ring, varying in size from a pin's head to half an inch in diameter, for the most part discrete but closely set, with nothing noticeable as to arrangement. These burst after a few hours or a day or two, the contents and covering collecting in a semilunar or circular scab around the edges of very well-defined circular scars, at first pink and shining, soon becoming pigmented stains which persisted for about three weeks, looking like syphilitic pigmentation. The rash was roughly symmetrical, being limited to the lower extremities and the lower part of the trunk. It itched very much. His urine was free from albumen, and there was no constitutional disturbance.

Many other cases occurred, identical in character, except as to distribution, the rash occurring not only on the legs, but also on arms, axillæ, face, neck, and upper part of trunk. The most troublesome were those which attacked the inner side of the upper part of the thighs.

In one case of a man who was suffering from a very severe attack of fever, to which he ultimately succumbed, the rash was mixed with numerous boils, and was also peculiar, inasmuch as many of the spots were umbilicated almost like small-pox. In no case could contagion from other cases be definitely traced.¹ In some patients the disparity in size of the pustules was well marked, but in others they were much more uniform, generally about the size of a split pea, sometimes two or three small ones ran together to form a large irregular pustule.

Another class of cases may be described as follows: A rash appears—on the face and neck especially, but also on the trunk and limbs—in patients who have suffered for some time from prickly heat; spots come as scattered vesicles, at first clear, but soon opaque and milky-looking, surrounded by a narrow pink areola; the size varies from a pin's head to a small split pea; these soon burst, either by being scratched or naturally, without becoming pustular, leaving dry, pink stains, with a circular or semicircular border of dried secretion and cuticle of the vesicle; the colour of these stains does not entirely disappear on pressure, and, although discrete at first, they rapidly become confluent, and then present large pink patches surrounded by sinuously-curved, well-defined borders. The rash itches slightly; it is not symmetrical, but occurs in irregular patches. If neglected and much scratched it becomes almost impetiginous in