

of parents play some considerable part. In reality the chorio-capillaris of the choroid seems to be first attacked, the retinal degeneration being secondary. As you are aware the outer layers of the retina are nourished by the chorio-capillaris and in retinitis pigmentosa the inner layers show comparatively little change. These facts help us to a reasonable conjecture as to the exact causation of the night blindness.

You will remember that in birds which seek their prey at night, such as the owl, the retina contains only rods; there are no cones in the neuro-epithelial layer. You will also remember that the visual purple is associated with the rods only, so that in animals possessing a fovea, where the rods are absent, there the visual purple is also absent. I may further remind you that with low illumination in man there is much more rapid depreciation of central than of peripheral vision, so that we may reasonably conclude that vision in these circumstances is chiefly carried out through the agency of the rods. Form sense, dependent upon the cones, quickly diminishes in passing from the point of fixation towards the periphery, but at night acute appreciation of variations of light and shade is much more important than accurate delineation of objects. These considerations may afford some explanation of the night blindness in retinitis pigmentosa, as well as of the partial nature of the annular scotoma and the comparative perfection of central vision. Of course, it is not suggested that the cones escape destruction in the affected zonular area, but their loss is discounted by their relatively unimportant functions in this situation.

There is another chronic form of night blindness which we occasionally meet with, differing from retinitis pigmentosa in the fact that it is stationary. This form is always hereditary and shows no gross changes in the fundus. It is a rare disease except in the families afflicted with it, when as has been recently shown in a very striking manner a large proportion of the members are attacked. Thus Mr. E. Nettleship has continued the work of Cunier on a certain family in the south of France and has discovered no less than 135 subjects of congenital night blindness amongst 2121 members of 10 generations, the first member of which, a male, himself night blind, was born in 1637. Unfortunately, no case of this disease has been examined anatomically; it may possibly be found that the retina is deficient in rods or visual purple.

A group of cases of chronic night blindness in some respects intermediate between idiopathic retinitis pigmentosa and congenital night blindness is that of syphilitic pigmentary retinitis. It does not show the same uniformity either of symptoms or of objective signs that are characteristic of idiopathic retinitis pigmentosa. The night blindness is progressive during the active stage of the disease but may then remain stationary for an indefinite period. Fundamentally, however, the night blindness must be regarded as due to the same pathological processes. Besides these chronic forms of disease manifesting this symptom there are also acute forms. Though these show a great variety of clinical types there is nearly always one feature common to all—namely, malnutrition. They are probably much less frequently seen in England now than formerly owing to the improvement in the conditions of the poor. We generally meet with them in badly nourished children and a large proportion of them have xerosis of the conjunctiva. The combination of these symptoms is not so invariable as is sometimes thought. Many cases of xerosis without night blindness and *vice versa* occur. In some cases the cornea becomes ulcerated and in the worst there is keratomalacia; in many of these the age of the patient or the severity of the attendant symptoms prevents the demonstration of night blindness. One fact which may be definitely deduced is that there is no inherent relationship between the xerosis and the night blindness other than a *con com* cause.

Acute night blindness was at one time common among sailors, soldiers, and the inmates of prisons and workhouses. In Russia it was, and probably still is, common during the Lenten fast. Uhthoff, amongst 500 cases of severe alcoholism, found 5 per cent. suffering from xerosis, night blindness, or both together. Less frequently night blindness has been found associated with scurvy, malaria, nephritis, the puerperium, vegetarianism, and so on. Most of these patients have reflex blepharospasm ("photophobia") in bright sunlight. That in many, most likely all, the lesion is peripheral is shown by Mr. Nettleship's interesting observation that if a sailor afflicted with the disease covers up one eye during the daytime that eye has sufficiently good vision at night for the man to carry out the duties of the

watch. It would appear, therefore, that malnutrition acts by lowering the vitality of the retina in such a manner that the process of repair is delayed. Probably the visual purple is restored more slowly than normal—that is, the anabolic processes are defective. To call the condition *torpor retinae* and to regard this as an explanation is futile; it is simply describing the condition by another name.

Another group of cases of night blindness, allied to those last mentioned but deserving separate treatment, are those associated with jaundice. The symptom is not very uncommon in severe cases of jaundice. In some pigmentary changes of slight degree have been found in the retina and the condition has been dignified with the name *ophthalmia hepatica*: in most the ophthalmoscopic signs are negative. It is noteworthy in this connexion to recall the fact that bile salts are a solvent of the visual purple, as was shown by Kühne. Night blindness is common in India among badly-nourished natives, especially during the periodic famines. It has been found that the symptom disappears when the patients are fed on liver, a fact difficult to correlate with the cases occurring with jaundice. The mode of treatment is of extreme antiquity, being advocated in the Ebers papyrus (B.C. 1500).

A CASE OF ANEURYSM OF THE FEMORAL ARTERY IN A MAN, AGED 78 YEARS,

IN WHICH SUPPURATION TOOK PLACE
FROM PNEUMOCOCCAL INFECTION; RECOVERY.

By H. H. CLUTTON, M.A., M.B., M.C. CANTAB.,
F.R.C.S. ENG.,

SURGEON TO ST. THOMAS'S HOSPITAL;
AND

LEONARD S. DUDGEON, M.R.C.P. LOND.,

BACTERIOLOGIST TO ST. THOMAS'S HOSPITAL; JOINT LECTURER ON
PATHOLOGY IN THE MEDICAL SCHOOL.

FOR the history and many details of this case we are indebted to Dr. A. Bevan who attended the patient throughout his illness.

The patient was an old man, aged 78 years, living in a hotel who was suddenly taken ill with a rigor and a temperature of 104° F. on Jan. 18th, 1907. He had a cough and rapid respiration but there were no signs in his chest until two days after the onset of his illness. Consolidation of the upper lobe of the right lung was then found. This was followed by a patch of dulness at the angle of the left scapula and at the right base. The temperature touched normal on the third day of the illness and then fluctuated between 100° and 103·4° until the eighth day, when there was a pseudo-crisis with a rise next day to 101·2°. It then fell again on the tenth day to 96·4°. The temperature fell to 96·4° three times with intervening slight rises to 97·2°, remaining subnormal for the following ten days. There was a very dangerous collapse during this time. The pulse-rate fell from 98 to 60, and sometimes could not be counted, as many beats failed to reach the wrist. The heart sounds were very faint from the combined effect of emphysema and feeble action but no cardiac murmur was heard at any time during the illness. The expectoration was of the "prune-juice" type rather than rusty. Dr. H. G. Turney, who saw the patient in consultation with Dr. Bevan, reports that "the pneumonia was clinically on the whole of the influenza type, though the termination was by crisis. The physical signs varied a good deal from day to day; there never was really massive consolidation and he sweated a good deal all through the attack." The patient remained in a low and feeble condition with a subnormal temperature until Feb. 10th. He then improved and the temperature became normal, but from the 20th onwards evening pyrexia (99°) was constant. There was also at this time a little swelling of the left leg and foot from oedema, and a local swelling was noticed about the middle of March in the course of the femoral vessels which was thought to be due to thrombosis of the left femoral vein. In April Dr. Bevan found that this swelling pulsated and regarded it as an aneurysm. Towards the end of April it began to increase in size and the pulsation appeared to be nearer the surface. On examination it was found to be a large swelling situated in Hunter's canal on the left side, with inflammatory exudation into the tissues around, or the aneurysm may even then

have begun to leak. In the latter case its size would be in part due to extravasated blood. It is also of some interest to notice that the femoral artery could be traced over the swelling for an inch or more both above and below its centre. It therefore seemed probable that we had to deal with a sacculated aneurysm situated on the outer side of the femoral artery in Hunter's canal. In this position it would be able to cause compression of the vein against the bone and œdema of the leg and foot. This œdema of the left leg was the first indication of anything abnormal. As the swelling in the thigh increased in size and became more obvious it was again carefully examined and found to pulsate. The central part of the pulsating swelling at the end of April was quite soft and appeared to be approaching the surface. There was evening pyrexia rising to 100°. His arteries were thick and tortuous and the heart action was rather feeble. An operation therefore at the age of 78 years caused us some anxiety. On the other hand, the pulse could be felt quite plainly at the ankle, and it was hoped that ligation of the femoral artery just above the aneurysm at the apex of Scarpa's triangle would be sufficient to arrest its progress. The aneurysm with its surrounding swelling now occupied the middle of the left thigh just reaching to the apex of Scarpa's triangle.

On May 2nd, four months after the onset of the pneumonia, the femoral artery was ligatured just above the margin of the swelling. Dr. Bevan gave the anæsthetic and had a good deal of anxiety over his patient, who showed signs of cardiac distress during the operation. Mr. C. A. R. Nitch gave much valuable help and assistance. The sheath was unusually adherent to the artery which had very thick walls and was rather tortuous in its course. Two double strands of floss silk, making four ligatures in all, were passed beneath the artery and separately tied without dividing the coats. Pulsation in the aneurysm and at the ankle was arrested by the ligature. The pulsation never returned at the ankle, but in the aneurysm it was occasionally detected on careful examination during the next fortnight. The foot remained warm and of good colour throughout. The temperature began slowly to rise after the sixth day from the date of the operation, when the sutures were removed. From May 10th to 22nd there was pyrexia (102°) every evening, although the wound appeared to be perfectly sound and to have healed by first intention. The aneurysm, on the other hand, whilst the temperature was rising, steadily increased in size, and finally gave one the impression of being an abscess. It roughly extended from the inner side of the knee to the buttock; the most prominent part was situated about the centre of the inner side of the thigh. As it was still doubtful whether the swelling was due to extravasated blood or to suppuration the blood was examined and the result strongly suggested suppuration.

On May 23rd an anæsthetic was given by Dr. Bevan and with Mr. Nitch's assistance an incision was cautiously made through the centre of the swelling. What appeared to be blood escaped in large quantities. Having preserved some of this fluid in sterile tubes the cavity was fully explored and found to be very large, extending far beyond the limits of the original aneurysm. As it did not appear now that there was any direct arterial communication with this cavity and that the fluid was old softened blood-clot, the wound was left open without either a drainage-tube or gauze plug being inserted. The dressing had to be frequently renewed as there was a constant discharge of a large amount of blood-stained fluid. At each dressing this fluid could be squeezed out of the wound and on careful measurement amounted in 24 hours from five to eight ounces. The temperature during this time dropped from 102° to 101°. As no further diminution of the discharge or fall in the temperature took place an anæsthetic was given on June 11th and the wound was freely enlarged. Two counter openings were also made, one towards the knee and the other at the back of the thigh towards the gluteal fold. A drainage-tube was introduced into each opening. The highest temperature in the 24 hours quickly dropped to 100°, and by June 20th it was only 99°. The whole thigh also began to diminish in size, the œdema of the foot disappeared, and the patient improved rapidly in general condition. By the end of June the discharge was of slight amount and the highest temperature was always below 99°. Our anxiety was thus at an end. On July 14th, when the wounds were nearly healed, the temperature rose slightly and he complained of pain in the left groin. The femoral artery, which had been previously

pulsating up to the seat of the ligature, could not be recognised below Poupart's ligament, and œdema of the leg and foot was again apparent. Thrombosis of the artery from the seat of the ligature up to Poupart's ligament had obviously taken place. In about a week's time the pain had gone, the temperature was 99°, and the artery could be felt as a solid cord up to and beneath Poupart's ligament. The œdema of the foot, however, remained. The last drainage-tube was removed on July 21st. Early in August he was able to leave London for Mr. H. C. Crouch's home at Ascot. The wounds were practically healed and the œdema had almost disappeared. There was slight pitting on pressure over the shin but none on the dorsum of the foot. The artery in Scarpa's triangle was a painless solid cord. Once again, at the end of August, he had an attack of inflammation in the left groin. Pain, tenderness, and swelling round the femoral artery with a slight rise of temperature gave the same indications as before, and naturally aroused a suspicion of further extension of the thrombosis. But as there was no œdema of the foot it was hoped that the inflammation was confined to the part previously affected, and that there was no extension of the thrombosis to the external iliac artery. Within a week the attack had disappeared, leaving the artery as it was before.

Pathological report.—On May 21st an examination of the blood was made with a view to determine, if possible, whether suppuration had occurred in the aneurysmal sac. The result was as follows: leucocytes, 22,740 per cubic millimetre. A differential count of 500 cells was made, of which 86 per cent. were polymorphonuclear neutrophils and 1.25 per cent. the Ehrlich type of neutrophilic myelocytes.

A bacteriological examination of the contents of the sac of the aneurysm was obtained on May 24th. Film preparations were made from the coagulated blood which had been received in sterile tubes. The cells were chiefly microphages, the remainder macrophages. Diplococci were numerous; they were Gram-positive and the majority were extracellular. A pure culture of the diplococcus was obtained from the coagulated blood. This organism was found to be the pneumococcus. It gave the reactions which are most commonly obtained in the artificial media employed by Andrewes and Horder for the differentiation of the streptococci. On May 27th a second bacteriological examination was made. The contents of the tubes consisted of blood-stained pus, in which there were large numbers of both extra- and intra-cellular Gram-positive diplococci. A pure culture was obtained but on this occasion the organism was extremely difficult to cultivate and died out very rapidly. On June 4th a third examination was made. Film preparations of the pus showed numerous Gram-positive diplococci. A few bacilli were also present. The cultivation experiments were found to be sterile.

This case is of interest, even if it were regarded merely as an example of suppuration in an aneurysm without reference to the intrinsic cause, but owing to the fact that the pneumococcus was obtained in pure culture from the contents of the aneurysmal sac, and that the patient had only recovered from acute pneumonia quite recently, it may be considered as a case of exceptional rarity in surgical pathology. The first question which naturally arises is whether an aneurysm was present previously to the attack of pneumonia. Dr. Bevan tells us that he is unable to answer this question. If we allow that a small aneurysm of the femoral artery was present previously to the acute illness, then probably either a clot in the interior of the sac became infected by the pneumococcus, or an atheromatous patch in the wall of the aneurysm became the seat of an acute infection by this organism, with subsequent thrombosis. It has already been stated that the contents of the aneurysm, when first received in the laboratory, consisted of coagulated blood, which had undergone acute inflammation, while a few days later true suppuration had occurred. If we allow for the sake of argument that there was no aneurysm previously to the acute pneumonia, then we have to decide whether the patient developed an acute embolic aneurysm as a result of infective endocarditis, or whether the atheromatous condition of his arteries led to direct pneumococcal infection of the wall of the femoral artery.

It is well known that even the most severe forms of infective endocarditis may not be diagnosed during life, but even allowing for this fact there was no reason to suspect in the case under discussion that acute endocarditis had occurred. Dr. Turney, who was called in to consultation

on several occasions with Dr. Bevan, saw no reason to suspect infective disease of the endocardium. There were no cardiac murmurs during the whole course of the illness. The pyrexia at one time corresponded to the attack of acute pneumonia and later followed the course of the acute infection of the femoral artery. As soon as the suppuration was relieved the temperature fell to normal and has remained normal ever since, except for slight pyrexia which lasted for a few days during the period of convalescence.

An acute embolic aneurysm which develops during the course of acute infective endocarditis is characterised by its rapidity of formation, and also by a considerable amount of pain at the seat of infection. It cannot be said that either of these phenomena was present in the case under discussion. Perhaps the most important argument against infective endocarditis is that the patient continues in good health and shows no evidence of cardiac disease. The other explanation which we have already referred to is that the patient developed an acute pneumococcal infection of an atheromatous patch in the femoral artery. Careful consideration of the facts of the case strongly suggests either direct infection of the artery leading to the formation of an aneurysm or direct infection of an aneurysm which was present previously to the acute illness. In either case the pathology is somewhat similar. The slow and feeble action of the heart, which was such a noticeable feature in the first few days after the crisis, and the alteration in the coagulability of the blood which is so commonly found in such conditions, would favour the formation of thrombosis either in the sac of an aneurysm or over an atheromatous patch in a degenerated artery. It is impossible to say whether acute infection of the arterial wall took place previously to, or subsequently to, the formation of thrombosis. In either case the pneumococcus, which may probably be regarded as the cause of the acute pneumonia, set up an acute arteritis which finally gave way into the surrounding tissues. It is probable that the infection of the tissues took place through the wall of the artery before actual leakage occurred. In any case, whether the aneurysm was primary or secondary, it appears to have been of the sacculated variety, and direct communication between the sac and the vessel was obliterated before the aneurysm was opened. It is unfortunate that it is impossible to say at what period of the patient's life the aneurysm was formed, but at any rate this does not detract from the great interest which centres round such a remarkable condition.

THREE CASES OF POISONING BY CARBONIC OXIDE; ONE RECOVERY.

BY R. SPENCER PEARSON, M.R.C.S. ENG., L.R.C.P.
LOND., D.P.H.R.C.P.S.I.,

LATE MEDICAL OFFICER OF HEALTH, WIGAN RURAL DISTRICT.

CASES of poisoning by carbonic oxide appear to me of sufficiently rare occurrence to justify a description of those which recently happened in the workhouse infirmary, Leighton Buzzard, for such a tragedy to take place in the wards of an institution the arrangements of which had been passed by expert engineers is fortunately uncommon and has a special interest.

On the night of Jan. 20th three women retired for the night in a small ward and at 9 P.M. were known to be in their usual health. A fire had been lighted in the stove as on this day the ward had been cleaned and it is probable that the women had closed the only ventilator before going to bed. On the following morning at 7 A.M. a workhouse inmate went to call the women and found them, as she thought, all dead. There was no smell in the room and the gas was burning, and the fire in the stove was still just alight. The three women were lying as if asleep. On further examination one of them, aged 76 years, was found to be alive, the workhouse master, Mr. Swaffield, at once with commendable promptitude performing and continuing artificial respiration, thus most probably saving her life. The other two women, aged 52 years and 47 years respectively, were quite dead. The master sent at once for Mr. L. Worts, the medical officer, to whom I am indebted for my subsequent conduct of the case and for permission to publish these notes.

I was sent for at 10 A.M. on the morning of Jan. 21st and found a woman, aged 52 years, and a woman, aged 47 years, lying dead. The body of the first woman had been removed from the bed to the floor. She was quite dead; some white foam was issuing from the mouth. Her attitude was one of repose and her complexion and lips were fresh and of a bright cherry colour. The body was warm and rigor mortis was hardly discernible. The body of the second woman was on the bed; the face was pallid and she had vomited some pultaceous matter. She lay on her back with her arms folded over her chest; rigor mortis was marked and the body was quite cold. The third woman, aged 76 years, was lying on the floor, totally insensible, the conjunctival reflex being absent. The pupils were equal and of moderate size. She was practically pulseless and was breathing stertorously, the cheeks blowing with each expiration. Respiration was slow (about 12 to the minute), the lips and mucous membranes were bright, and the complexion was clear. Both wrists showed abrasions the result of the continued efforts at artificial respiration, and it was noted that the reddened skin around the wrists had a peculiarly bright hue. As there was no information to go upon, and as there was a suspicion that all three had partaken of the contents of a mysterious parcel left by a visitor, after injecting $\frac{1}{10}$ th of a grain of strychnine I decided to wash out the stomach. This was done with warm water and afterwards with weak Condy's fluid till the washing was clear; the washings were first of a coffee-ground colour. I then gave one and a half ounces of brandy through the stomach-tube, and after injection of ether and the application of the interrupted current in the course of the phrenic nerve, after about three hours the pulse improved, the conjunctival reflex returned, and she responded to outside stimuli. Oxygen was sent for but she recovered before it arrived. She was ordered to be fed per rectum with "panopeptone" and by 6 P.M. she was able to take it by the mouth, and she is now out of danger but can remember nothing of what happened.

Realising that the desperate illness of the third woman must be brought about by the same cause which had led to the death of the other two I decided to make a spectroscopic examination of the blood of the living woman and, being fortunate in possessing an excellent stellar spectroscope, used it with most decided results. A drop of blood from the finger was received into a test-tube two-thirds full of normal saline solution and the tube was shaken up; the resulting solution was of a bright cherry colour. On being placed before the slit of the spectroscope there were seen the two absorption bands of CO hæmoglobin most distinctly, and the reaction was unaltered on the addition of ammonium sulphide. Next day I was present at the post-mortem examination made by Mr. Worts. Both bodies presented similar appearances. The blood 30 hours after death was perfectly fluid and cherry-red in colour. The muscles, too, were bright red. There was no blood clot found at all. The mucus membranes were all bright red and the complexion fresh. The stomach of one woman was empty and presented numerous petechial hæmorrhages, and similar hæmorrhagic points appeared in the cerebral white matter on section; the cerebral ventricles were full of fluid. In both cases the lungs were markedly œdematous and the kidneys showed interstitial nephritis; the capsules were adherent. I took specimens of blood from both dead women and examined them as before, with a like result, the spectroscope giving definite evidence of CO hæmoglobin.

The cause of the carbonic oxide in the atmosphere was, on investigation, not hard to find. At first I thought that it must be due to the permeability of red-hot cast iron to gases, but since then I have had an opportunity of examining the stove with the representative of the firm who manufacture them. The stove is of ornamental cast iron with doors in front which can be opened; on the top is a circular aperture covered by an ornamental perforated cap. The stove stands in the room about two feet from the fire-place, which is blocked up. A six inch cast-iron horizontal pipe passes into the brick flue, terminating on its entrance. The flue is a long one leading from the floor below, one flue common to both landings. Inside the stove is a flame baffle, which when rightly set serves to direct the heat into the room and direct the current both from the fire and the chamber at the top of the stove into the flue. Instead of being set thus the baffle was set as shown in the diagram, thus allowing any products of combustion which passed the baffle to come into the room (Fig. 2). The