are kept. Thus a 0.66 per cent. solution of serum No. 1 had a strong haemolytic action on human red blood corpuscles, whereas a 1.3 per cent. solution of the same serum had but a slight haemolytic action on the red blood corpuscles of guinea-pigs.

Serum No. 2 is still more specific, for whereas a 1 per cent. solution dissolved human blood corpuscles, a 3.3 per cent. solution had no such action on the red blood corpuscles of guinea pigs.

Similarly, the 3.3 per cent. solution of serum No. 1, kept for eighteen days, no longer dissolved the red corpuscles of guinea-pigs, though a 0.66 per cent. solution of the same serum still dissolved those of man. Serum No. 2, kept for twenty days, even when not diluted, and when fresh guinea-pig's serum had been added, no longer dissolved the red blood corpuscles of guinea-pigs, though a 1.3 per cent. solution still dissolved human red blood corpuscles, provided fresh serum from a normal animal were added.

The fact that such serum is not entirely specific is not astonishing, when one reflects that one is dealing with a liquid as complex as urine, for among the "haemolysogenous" substances contained in urine there must be several substances common to many kinds of animals.

Schattenfroh (*Centralblatt f. allgemeine Pathologie*, vol. xiii, No. 14, August, 1902) also injected animals with urine and found that the serum had strong agglutinating and haemolytic but no precipitating properties.

BOTTLE-FINISHERS' CATARACT.

By WILLIAM ROBINSON, M.D., M.S., F.R.C.S.,

Surgeon to the Sunderland and Durham County Eye Infirmary; Physician to the Sunderland Infirmary.

HARD cataract is very common in bottle-finishers. My experience as surgeon to the Sunderland and Durham County Eye Infirmary during the last seven and a-half years has impressed upon me this fact.

How much commoner it is in them than amongst the rest of the community is shown by the fact that 18 out of 75 (1 in 4) hard cataracts operated upon last year in our Eye Infirmary were in bottle-finishers, there being only 200 or 300 bottle finishers in a population of nearly a million and a quarter in this county.

Amongst bottle-makers the "bottle-finisher" is the man who is almost always the victim of the disease, but occasionally the "founder" suffers.

The Nature of a Bottle-finisher's Work.

The following particulars were learnt from visiting some bottle works in this neighbourhood, where 3-gill black wine bottles were being made. The mixed materials (sand, lime, clay, marl, etc.) are thrown by the "founder" into the large furnace ("tank"), which is kept at a white heat by the burning of currents of coal gas and air directed on to the surface of the metal. The "founder" has to push with a long iron rake the materials away from the mouth of the furnace into which he has thrown them, and consequently he has to look occasionally into the intense light and heat. He is provided with a piece of framed dark-blue glass, through which he can look into the furnace, but this he rarely uses, and consequently some founders get cataract. The "founder" may work six or seven shifts a week of twelve hours each. At the opposite end of the furnace are several openings, at which the different bottle-makers work in squads of five men each.

A lad generally begins as a bottle-maker as a "taker in," whose duty it is to carry the finished bottle from the bottle-finisher to the annealing chamber. He is afterwards promoted to be a "putter-up" or "wetter-off," whose work is to put up to be heated the iron pipe, 6 ft. in length, on which the bottles are blown, into the furnace, and knock off the remains of the bottle that has just been made.

He is next promoted while still an apprentice to be a "gatherer," whose duty it is to dip the iron pipe into the sea of molten metal and take up just sufficient liquid glass to make a bottle. During this part of the work he necessarily looks into the furnace; but he is young and his eyes do not suffer, though the skin of his face is often inflamed and blistered by the heat, especially the left cheek. With his back to the furnace he next rolls the glass on a stone and roughly forms the neck and shoulder of the bottle. He then hands the pipe to the bottle-blower, who shapes the body of the bottle on another stone, then places it in a mould and blows down the pipe into it to form a bottle. During this

process the cheeks of some (not all) of the bottle-blowers balloon out enormously. About two out of every three apprentices become expert enough to be promoted from being "gatherers" to be "bottle-blowers," and they are about the age of 21 at the time of promotion. A "gatherer" sometimes becomes a "founder." The glass-blower hands the blown bottle, still on the iron pipe, to the bottle-finisher, who takes hold of it with a "cage or sticker punty": the bottle is then cut from the pipe. The glass-blower does not look into the glass furnace and his eyes do not become cataractous.

The bottle-finisher, holding the bottle on the "punty" in his left hand, with an iron rod in his right looks directly with both eyes into the sea of molten glass, and takes just sufficient to form the rim on the neck of the bottle. Having done this he turns away from the furnace and sits under the shelter of a brick shade, in order to put the rim on the bottle. His left cheek is now towards the hot wall of the furnace, and it is therefore generally more inflamed than the right cheek by the heat. The extent to which the eyes of the bottle-finisher are exposed to the most brilliant light and intensest heat from the furnace may be estimated by the fact that he generally works five shifts each week, a shift lasting eleven hours, and at each shift he finishes 110 to 120 dozen bottles at the rate of $2\frac{1}{2}$ bottles per minute. The time in which he is obliged to look into the furnace whilst he gets the metal for the rim of each bottle is about three seconds, so that at each shift his eyes are exposed to the glare of the furnace for at least sixty-six minutes, or about five and a-half hours a week. This is the man, therefore, whose lenses become opaque. With the old furnaces, the left eye was more exposed to the furnace, but in the present furnace both eyes are equally exposed to the light and heat; if there is any difference the right is the more exposed.

Only about one in two bottle-blowers is promoted to be a bottle-finisher, and when he is so, he is generally between the ages of 25 to 40. The bottle-finisher is a much older man at his trade than either the "taker-in," the "gatherer," or the "putter-up," who are usually under the age of 21 and he has passed through all these grades of work as well as that of bottle-blower.

If the cataracts are due to the bright light and heat of the furnace, the question naturally arises: Do men similarly exposed to light and heat in other trades suffer from the same affection?

I have been informed that in other branches of the glass trade such as the making of tumblers, glass jars and plate glass it is only the "founder" who looks into the furnace (the "tank") and he only does so occasionally, and I have met with only one case in men from these works, in which the metal is gathered from "pots" and not from the huge "tank."

In large steel and iron works, such as are at Middlesbrough where the Siemens-Martin process is used, the intense light and heat of the furnace are so great (2,000° F. to 3,100° F.) that looking into it for a short time with the naked eye the observer becomes quite blind for a while—but there the furnace men use dark blue glasses and require only occasionally to look into the furnaces and so far as I know do not suffer from cataract.

In smaller steel and iron works no glasses are used, but as the men only at infrequent intervals look into the furnace and the glare is not so great they do not suffer. The light and heat to which puddlers are exposed are much less and they are not affected by cataract.

Both Eyes are Practically always Affected.

The disease begins about the same time in both lenses, so that they usually present the same appearances when the worker applies for treatment.

The Disease begins comparatively Early in Life.

It commences as often as not before the age of 50 years, and not uncommonly before the age of 40 even, whereas ordinary senile cataract is not often seen before the age of 50.

The Disease progresses Slowly.

The bottle-finisher is often able to work for some years after the opacities begin, especially if the pupil be kept dilated by atropine; the great improvement of the sight by the use of atropine is due to the fact that at first the periphery of the lens is clear.

Mode of Development.

The opacity first appears at the posterior pole of the lens, immediately under the posterior capsule, and is shaped like

a disc or coin, which by oblique illumination is distinctly brass-coloured. The outline of this disc is well defined, but the opacity is less dense and shades off towards the equator. As the haziness spreads it at first clings closely to the posterior capsule, so that it is saucer-shaped in the earlier stages of the disease, the concavity of the posterior capsule of the lens being easily seen by oblique illumination. By direct examination with a +10 lens the central disc is not completely opaque, the opacity presenting often a shape like an irregular Maltese cross within a circle; and the surrounding less-dense opacity is often like a cobweb. From the posterior pole the cloudiness and opacity gradually spread to the rest of cortex, and when the cataract is ripe the lens has a markedly pearly hue. That the posterior capsule of the lens is itself diseased I infer from the following two facts: (1) During the extraction of a ripe cataract, even with the greatest care, vitreous is much more apt to escape than is the case with ordinary senile cataracts; and (2), after a successful operation, occasionally remnants of membrane which, owing to their depth appear to be pieces of the posterior capsule, can be seen to be thick and densely white.

Bottle-finishers' cataract thus differs from the way in which ordinary senile cataract generally develops, namely, (1) as lines or sectors (radii) from the equator of the lens; or (2) as opaque layers surrounding the sclerosed (but clear) nucleus; or (3) as small milk-white opacities scattered throughout the cortex; or (4) in two of these ways at the same time (see Fig. 1). I do not deny that an ordinary senile cataract may begin as a posterior polar cortical cataract; if it does so it is a rare event, whereas practically all cases in bottle-finishers begin in this way.

It differs from ordinary posterior cortical cataract, which is a secondary cataract following on disease of the vitreous, pigmentary degeneration of the retina (retinitis pigmentosa), or disseminated choroiditis, and which is therefore often found in the young, and if removed the vision is still bad owing to the posterior mischief.

Bottle-finishers' cataract is a primary cataract, being purely a local disease of the lens, and after extraction the vision is good, and there is not, as a rule, associated with it any disease of the vitreous, choroid, or retina, though the fundus is generally "tessellated" or "tiger striped," that is, the pigment between the choroidal vessels shows through the retina, owing to disappearance of the pigment in the tessellated layer of the retina, or to increase of pigment in the choroidal intervascular spaces, an effect produced, I think, by the cause of the cataract, namely, the intense light and heat of the furnace.

Etiology.

The disease is undoubtedly due to the great light and heat of the furnace at which the bottle-finisher works, but why does the mischief always begin at the posterior pole of the lens? The explanation I advance is as follows:

1. The nodal point (which practically corresponds to the optical centre) is situated at the exact spot where the cataract begins, and here all the principal rays, of the various pencils of rays, falling on the lens cross and pass without refraction, so that this point of the lens receives the brunt of all the direct rays, harmful by their intensity and heat from the furnace, and is therefore the point first to suffer injury. (See Fig. 2.)

2. All the rays from the furnace, except the direct and principal rays mentioned above, which have fallen on the cornea when they reach the anterior surface of the lens, are again refracted, so that when they reach the posterior surface of the lens they are much more crowded together, and are consequently more injurious there than at any other part of the lens. (See Fig. 3.)

3. The bright light of the furnace contracts the pupil so that the iris shields the periphery of the lens at first from damage.

The rays from the furnace consist of light and heat rays, both of which are radiated, reflected and refracted in like fashion, the difference between them in these respects being one of degree or quality, not of kind. Probably so far as the light rays are concerned it is most likely their actinic rays ("the invisible and more refrangible rays of light at and beyond the violet end of the spectrum, on which the chemical action of light chiefly depends") which help in the production of the cataract. I think, however, that it must be the heat rays that are most to blame by probably causing a drying and shrinking of the normal lens fibres so that spaces are formed between the fibres themselves or between them and the posterior capsule, and that in these spaces fluid (liquor Morgagni) collects, and thus the opacity is produced. I do not believe that the

constant sweating of the men whilst at work produces the cataract, as there are many other classes of workmen who perspire as freely, and who do not suffer in this respect.

Fig. 1. To illustrate the various ways in which a cataract begins in the lens.

Fig. 2

Fig. 3.

Prevention.

The disease can be prevented by workmen wearing dark pure blue spectacles—of this I feel sure; for glass has the property of allowing only 30 per cent. of the heat rays to pass through it; and, if the glass is dark coloured, many of the light rays would also be cut off. I have heard of one man continuing his work who has had a cataract removed from one eye, over which he wears a strong lens, and, the vision of the other eye being good, he uses only a plain glass in front of it. The Siemens-Martin men wear glasses. Although when advised to do so bottle-makers offer various excuses, I am certain that there is no valid objection to the use of the spectacles whilst they are at work, and that it would be very greatly to their advantage to wear them.

I append brief notes of 6 cases of cataract, all in bottle-finishers, who happened to come to my out-patient department on Saturday, November 8th last, whilst I was busy with this paper. They are not, therefore, picked cases, and may be taken as fair

Examples of the Disease in Bottle-finishers.

CASE 1.—J. U., aged 59. Both eyes began about eighteen months ago; he can read newspapers and $\frac{1}{8}$ with each eye. The fundi are normal but "tiger-striped," and both lenses present the early signs of posterior cortical cataract described above. In the left lens there is a single sector or streak of opacity (senile) extending from the equator, half-way to the centre of the lens.

CASE II.—T. G., aged 59. Both eyes affected: the right began at the age of 46, and the left at 49. The right lens is quite ripe and pearly white in colour. I removed the right lens on December 18th; there were some adhesions of the iris to the anterior capsule, which was very dense, so I delivered the lens in its capsule complete, after performing an iridectomy. A small bead of vitreous escaped as the lids were being closed. The anterior capsule of the lens was dense and pearly white and about the thickness of tissue paper; the posterior capsule was much thinner and more translucent but whitish. The result of the operation is excellent. The left lens was removed by me on October 4th, 1902, and with + 10 D he sees $\frac{1}{2}$. There is no posterior mischief.

CASE III.—J. B., aged 59. Both eyes affected; the right began at the age of 55, in January, 1898, and the left shortly after. I removed the left lens on June 18th, 1901; he had had an injury to this eye many years before which had set up iritis and resulted in adhesions of the iris to the lens, necessitating the removal of the lens by the vectis. Some vitreous escaped, and his vision now in this eye is $\frac{2}{3}$, and there is some displacement of the pupil. The right lens I removed on September 26th, 1902; six weeks afterwards with + 11 D he read $\frac{1}{2}$. He has no fundus mischief.

CASE IV.—J. S., aged 53; both eyes affected: left began at age of 48, and right at 51. I removed the left lens in March, 1901. Immediately after the section some vitreous presented and I removed the lens with the vectis in its capsule. He has a beautifully clear pupil, and with a + glass he saw $\frac{1}{2}$ until May, 1902, when the vision began to fail, and now with + 6 he sees only $\frac{1}{3}$. There has developed recently a separation of the retina at the lower half of the fundus and a patch of choroidal atrophy. On September 23rd last I removed his right lens, and on November 8th he read $\frac{1}{2}$ with + 6 D. He has a good pupil and no posterior mischief.

CASE V.—J. W., aged 40, presents in both eyes the appearance of early posterior cortical cataract described above. The defect in his sight he first noticed at the age of 38. He is still at work. The fundi are normal.

CASE VI.—D. McL., aged 55. Early appearances of posterior polar cataract in both lenses; left began at 50, and right at 51 (?); in each lens is a central well-defined dot, a central congenital cataract, which shows no tendency to increase, and by looking well from the side with light reflected from the mirror, clear lens tissue can be readily seen between the central and posterior cortical cataracts in each eye. He is about to give up work owing to increasing blindness. His fundi are normal and "tiger-striped." He has astigmatism.

Cases I, II, and IV were shown at the meeting of the North-umbrian and Durham Medical Society on November 13th last. I have gone into this matter in some detail because, so far as I know, there is no literature at all on the subject, except a short paragraph in Berry's *Diseases of the Eye* (page 177 of second edition), in which it is stated that glass-blowers are subject to cataract, which generally affects the left eye first or altogether, owing to the left eye being nearer the glowing glass.

In this investigation I have been much helped by our house-surgeon, Dr. C. A. S. Dalglish, especially in the observation of cases.

PERFORATION OF THE OESOPHAGUS BY TUBERCULOUS GLANDS.

By CLIVE RIVIERE, M.D. LOND., M.R.C.P.,

Casualty Physician, St. Bartholomew's Hospital; Assistant Physician, East London Children's Hospital, Shadwell.

INSTANCES of tuberculous glands opening into the oesophagus are not often met with, and the following three examples seem, therefore, worthy of record. They occurred at the East London Children's Hospital, Shadwell, and I am indebted to Dr. Eustace Smith and Dr. Coutts for permission to publish the notes. The necropsy in each case was performed by myself.

CASE I.—W. C., a male, aged 2 years, was admitted on November 26th, 1900, with a history of three weeks' wasting and loss of appetite. There had been no diarrhoea or vomiting but cough had been troublesome. There was no family history of tuberculosis.

Condition on Admission.—The child was delicate-looking and showed well-marked rickets. The temperature was 102°; there were scattered rhonchi in the lungs; the liver was found to be enlarged and the spleen reached to the umbilicus.

After-History.—On December 7th it was noted that the abdomen was much distended and the temperature still remained high. On February 1st dullness over the upper sternum and on either side of it was recorded. The temperature kept high throughout the three months' illness, with fluctuations of 2° or 3°. Diarrhoea, which had begun on December 21st, continued throughout with three or four stools daily, and there was occasional vomiting, seven times in all. The child died on February 17th, 1901.

Necropsy.—At the necropsy a mass of caseous glands the size of a tangerine orange was found behind the manubrium sterni. The trachea lay behind it, and an offshoot of the mass surrounded and narrowed the left bronchus. The oesophagus was deflected to the left, and in its front wall, just below the bifurcation of the trachea, was a large opening where a caseous gland had become adherent and had ulcerated through. This was the bifurcation gland, and the opening led into a cavity in its interior containing foul pus and debris. In the stomach were three small ulcers in the mucous coat formed by the softening of caseous tubercles. The intestine contained only two small tubercles in the mucous membrane of the ileum. Some of the mesenteric glands were caseous, and the retroperitoneal glands formed a large caseous mass. There were besides tubercles in most of the other organs of the body.

CASE II.—L. K., a male, aged 10 months, was admitted on April 1st, 1902, with a history of three months' cough and wasting. There had been much vomiting, but the bowels had been regular. The parents were healthy and there was no tuberculous family history.

Condition on Admission.—A pale, wasted child, with yellow-tinted skin. In the lungs were found dullness and feeble breath sounds over the whole left side, and the heart was best heard to the right of the sternum. The abdomen was somewhat distended; the spleen was palpable and firm.

Operation: After-history.—On April 8th an incision was made into the left pleural cavity and 8 oz. of pus evacuated. Thereafter the child steadily wasted, vomiting occasionally (seven times during the two weeks' illness), but having no diarrhoea, and died on April 24th.

Necropsy.—At the necropsy a mass of caseous right tracheal glands, caseous bronchial and pulmonary glands and a limited lung tuberculosis were found. In the oesophagus, below the bifurcation of the trachea, opposite the bifurcation gland, was an opening through the anterior wall large enough to admit the little finger, and leading into a cavity the size of the end of a thumb. This was formed by softening of the bifurcation gland and was lined by a mucous coat over a fibrous wall. It extended upwards in front of the right bronchus to some caseous right tracheal glands. No caseous contents were left. There were no tubercles or ulcers in the stomach, but the intestine was lined with tuberculous ulcers from end to end and the mesenteric glands were caseous.

CASE III.—G., a male, aged 1 year 11 months, was admitted on April 18th, 1902. There was a history of five months' diarrhoea and vomiting, accompanied by wasting. He had had a discharge from the left ear for the past four weeks, and a left-sided fit three weeks before admission. There was no tuberculous family history.

Condition on Admission.—A pale, wasted child, with a profuse purulent discharge from the left ear, and a complete left facial paralysis. In the lungs a few crepitations were heard at the bases. The abdomen was somewhat distended, and the liver and spleen slightly enlarged.

After-history.—The child had a high, fluctuating temperature and much diarrhoea, the bowels acting three to seven times daily. He progressively wasted, and died on April 24th.

Necropsy.—At the necropsy performed next day was found an enormous mass of caseous glands surrounding the trachea. They were largest on the right side. There were racemose tubercles and "caseous blocks" in the lungs, and scattered tubercles in the abdominal organs. The bifurcation gland had completely emptied its contents into the oesophagus, leaving a smooth-walled sac opening through the anterior oesophageal wall by an aperture $\frac{1}{2}$ in. in diameter. There were no tubercles in the stomach. The intestine contained small ulcers and tuberculous nodules throughout its extent. The mesenteric glands formed a caseous mass the size of a turkey's egg, one individual gland being as large as a pigeon's egg. The retroperitoneal glands were caseous, but not very large.

In these 3 cases it was the gland (or glands) situated below the bifurcation of the trachea which caused the trouble, and this seems to be generally the case. I can find but few instances recorded in the literature. In 1890 Dr. Voelcker exhibited a specimen to the Pathological Society from a boy aged 9 years, in whom the bifurcation gland had opened into the oesophagus, and discharged part of its contents, leaving some caseous material still present in the cavity. There was no history of oesophageal trouble, nor is the presence of any tubercle elsewhere in the body recorded. The boy died of quite other disease.

The absence of abdominal tuberculosis in such a case, if the omission of its mention really means its absence, seems to me very extraordinary. In the 3 cases described above it will be noticed that the amount of abdominal tuberculosis is, all through, as we should expect, a striking feature. In one there were tuberculous ulcers in the stomach, a very rare condition; in all 3 there was intestinal tuberculous ulceration and caseous mesenteric glands, and in 2 the retroperitoneal glands were also caseous, a condition which in my experience is by no means common. Dr. Voelcker finds only 3 other cases recorded among 2,504 necropsies made during thirty years at Great Ormond Street Hospital, and in 2 of these 3 the air passages were also involved. This we should expect to occur in many cases, the gland becoming adherent to the oesophagus on one side, and to a bronchus (generally the right bronchus) on the other side, ulcerating into both, and thus forming a passage between the two which finally may become lined by mucous membrane.

In two of the cases mentioned above the gland had completely emptied its caseous contents, its cavity was smooth-walled and appeared to possess a mucous lining.

In March, 1901, Dr. Norman Moore showed at the Pathological Society a case of an oesophageal diverticulum the size of a pea occurring in the anterior wall just below the end of the trachea. The patient was a man, aged 49, who died of bronchitis, and the diverticulum was regarded by Dr. Norman Moore as a congenital abnormality. May not such a diverticulum have originated in the same manner as in the cases described above? The position is at the seat of the bifurcation gland in the anterior wall, and, moreover, the absence of any mention of tubercle in the body does not militate against this suggestion.

In children the bifurcation gland is often, or even generally, the first visible seat of tubercle in the body, and one may suppose that in some cases the disease goes no further. I am inclined to think that many cases of oesophageal diverticulum occurring in this situation are due to this cause. This would not include those instances of "pulsion diverticulum" which have caused so much surgical interest of late years.