Inspection. Face thin, anxious, pallid; lips florid. Slight apex impulse; very faint in epigastrio.

Palpation. Apex *in situ*; impulse moderate; pulse normal.

Percussion. No increase of heart's dulness.

Auscultation. A triple sound is very distinct across the lower sternal region, right and left two inches beyond sternum on each side, and at xiphoid cartilage. The second sound is slightly doubled at both aortic and pulmonary cartilages, but greatly less than at points indicated. This reduplication of the second sound becomes very marked during full inspiration. No bruit whatever accompanies or replaces the heart's sounds. Slight *râle sonore* audible over the greater extent of the lungs.

REMARKS. Reduplication of the first or more particularly of the second sound of the heart, either as a persistent or fugitive phenomenon, is, according to my experience, of no very rare occurrence.

Professor Bouilland was, I believe, among the very first to direct special attention to this abnormal action, and, according to his observations, it was ordinarily associated with probable or demonstrative change of the organ. In the cases reported by him, a triple, occasionally a quadruple sound, was heard. In some of these "the pulse was double, as well as the ventricular systole", as if successive rapid ventricular contractions were required to overcome some obstacle at the arterial ostium. He thus clearly supposes the double sound to be developed in a single chamber. On the other hand, very distinguished observers (Dr. Williams, etc.) have imagined that when systolic reduplication existed, it was the natural result of a want of synchronism between the right and left chambers of the heart. But if this explanation were unconditionally true, doubling of the second sound ought surely to be constantly audible also; this sequence, however, most certainly does not always occur. The first sound may be, as it were, divided, *entrecoupé*, and yet the second sound exist in all its clearness and unity.

There are other possible sources of this divided beat extrinsic to the left ventricle: for example, it is possible that in cases of malformation of the chest, the heart, displaced and pressed upon, might, in its systolic tilting, strike some portion of its surface, otherwise than the apex, against the chest-wall, and thus excite a double first sound. The auricle might also, conceivably, engender a sound immediately preceding the ventricular systole, and thus apparently divide the normal sound. This change, however, in the condition of the ordinary aphonous contraction of the auricle might be recognised by such sound immediately preceding the radial pulse.

With regard to reduplication of the second sound at the base, aortic, and pulmonic cartilages, not only is the phenomenon much more easy to recognise from the sharp clear clack of the semilunar valves, but its actual mechanism is also more intelligible. Any retardation in the delivery of a given quantity of blood from either ventricular cavity might, without necessarily doubling the first sound, sensibly affect the quick recoil of the arterial walls. If this explanation be not accepted, we must concur with Skoda that a want of synchrony in the closure of the semilunar valves of a single outlet must be the cause, although there are physical difficulties even in this mode of interpreting the phenomenon. Greatly more difficult is the explanation of the reduplicate second sound, heard loudest at the apex, as in Case IV; and this arises from the acknowledged difficulty of explaining the second normal ventricular sound. Skoda has asserted—and I think most persons familiar with cardiac auscultation will confirm his statement—that a second sound is occasionally audible at the apex of the heart loud and accented, while the corresponding sound at the base is faint and low. Now, it would be unphilosophical to argue that this second sound at the apex could be that of the semilunar valves, transmitted from the base, without some demonstrable source of reinforcement were admitted, such an assumption being opposed by the fundamental laws of acoustics.

But, while we accept the statement of Professor Skoda as

to the actual existence of a second ventricular sound, his explanation of the rationale of such sound must be considered fairly debateable. He imagines it may be produced by the blood passing from the auricles encountering a free ventricular wall; but this view could only hold good on the theory of Beau, that the diastole of the ventricle is one of vacuity and repose—a free wall then existing; but if, according to the current view, the diastole is one not of repose but of gradual repletion, terminated by a rapid contraction of the auricle, then the conditions present are rather such as, according to Skoda's own teaching, should generate murmur instead of sound; viz., one current in motion encountering another comparatively at rest.

In conclusion, it may be remarked that although the so termed reduplication may not subserve any purpose of exact diagnosis, it may, notwithstanding, when permanent, possess a value, even in the absence of all other physical signs, when the rational symptoms are those of cardiac suffering.

MANCHESTER EYE HOSPITAL.

PARALYSIS OF THE THIRD PAIR OF CEREBRAL NERVES: WITH ILLUSTRATIVE CASES.

By JOHN WINDSOR, Esq., F.R.C.S., Senior Surgeon to the Hospital.

[Read before the Manchester Medical Society, March 19th, 1886.]

[Concluded from page 403.]

PARALYSIS of the third pair occurs occasionally in infancy and childhood. Of this I have seen several instances, although it is more rare than at a later period of life. The following is an example of it.

CASE V. Martha Lathward, aged 8 months, from Bow Street, Adelphi, was brought to the Eye Hospital, March 3rd, 1843. The child is pale looking; it has cut the two central incisors of the lower jaw about four months since, but the corresponding ones of the upper jaw are not yet through, nor will the mother permit the gums to be lanced at present.

The left eye is quite closed by a ptosis of the upper lid, and the movements of the eye itself are very limited, except by the abductor. The right eye is quite open. The ptosis was first noticed about two or three days since.

Two grains of hydrargyrum cum creta were prescribed to be taken every eight hours.

March 10th. The infant can now partially, although but slightly, expose the eye. The medicine was continued.

March 24th. She can now raise the upper lid almost as well as in the other eye. The powders were omitted, and infusion of senna was ordered to be taken occasionally.

May 21st. The mother called to say that the child was dead.

The left superior palpebra had recovered its power, but that of the right eye fell for a day or two before it died.

Some convulsive fits had also attacked it for a few days before its death.

CASE VI. John M'Dermott, aged 8 months, became an out-patient of the Manchester Eye Hospital, May 20th, 1852, affected with ptosis of the left superior palpebra. The child sucks well; its bowels regular, and its general health little affected, although the ptosis came on a few days before, simultaneously with a little catarrhal affection, which still continues.

Applicetur hirudo i temporis sinistro.

Sumatur calomelanos gr. i bis die.

Sumatur infusum sennæ pro re natâ.

The affection soon yielded to this treatment.

In cases of paralysis of the third pair, the pupil of the affected eye is generally dilated and irresponsive; but in some cases it is not larger than in the sound eye, and rarely it is more or less responsive to light.

CASE VII. Mary Ball, aged 44 years, from Radcliffe, became an out-patient of the Manchester Eye Hospital,

September 7th, 1848. She is affected with ptosis of the right superior palpebra. The motions of the globe, excepting outwards, are also very limited; the pupil is nearly twice as large as in the left eye: the vision of the right eye, when exposed, is tolerable, but somewhat misty. For the last twelve months the sight of the other eye has been imperfect, and very impressible by cold. She has sometimes been affected with diplopia, but less, of course, since the ptosis of the right eye came on. The left lens looks rather dull, and with this eye she sees worse than with the right, which she ascribes, probably erroneously, to a partial paralytic attack of the left side some time ago.

Sumat pilulæ hydrarg. gr. v ter die.
Habeat pulverem purgantem pro re natâ.
Applicetur vesicatorium pone aurem dextram.

Sept. 15th. Her mouth is very sore; she has less headache; she has more power over the levator palpebræ and the ocular muscles.

Omitantur pilulæ hydrargyri; et sumatur infusum sennæ pro re natâ.
Suntat pulv. ipecac. comp. gr. vij 8vis horis.

Sept. 28th. The power of the muscles is almost fully regained, but the pupil still remains preternaturally dilated. Continuentur infusum sennæ et pulvis ipecacuanhæ compositus.

October 28th. The pupil is still dilated, but the vision is pretty good, and the ptosis is quite gone.

Dec. 10th. She continues in nearly the same state, but the mydriasis is not quite gone.

In the following case the mydriasis was absent.

CASE VIII. James Warren, aged 42 years, from Stockport, was admitted an out-patient of the Manchester Eye Hospital, February 20th, 1849, affected with ptosis on the right side. The pupils of both eyes are about of equal size; and, when the palpebra is raised, he sees pretty well with either eye. The affection was first noticed when he awoke one morning about twelve days since, but he has felt headache both before and since that time. Fomentations externally, and some aperients internally, have been used.

Sumantur pilulæ hydrarg. gr. v ter die, et pulvis purgans pro re natâ.
Applicetur vesicatorium tempore.

He gradually improved by these means, his mouth becoming sore, and in about three weeks he was quite well. I have notes of other cases where the pupil was equally undilated.

I have seen a few cases where the patients have stated that objects have appeared brighter with the affected than with the sound eye, as if the sun was shining on them, and also appearing double, one nearer than the other, and on a different level (vertical diplopia); at other times, perhaps on the same level (horizontal diplopia). This I noticed first particularly in a Mr. Jackson, then of Green Street, Ardwick. He quite recovered, and is still living. This gentleman was occasionally attacked with vertigo so severely as to fall down.

In another person, named Atkinson, whom I attended about the same time (1836-7), there was semi-ptosis of both sides, the two lower thirds of both pupils being visible of the natural size, but little if at all respondent to light. Objects appeared to him magnified further off than they really were (in another case, objects appeared nearer than they were), and often double. The action of the ocular muscles moderate, but not complete. The complaint was of about six months' duration. He gradually recovered by the usual means.

It was remarked in this case, as in some others, that, by a strong effort, he could act somewhat in raising the palpebra; but it was soon followed by a sense of weariness and headache. I have known one or two cases, at least, where the paralysis of the third nerve has gone off, but has been succeeded by permanent paralysis of the second nerve (incurable amaurosis), as in a man named Knapman, etc.

I have mentioned, in a preceding part of the paper, a

case where the superior portion of the nerve only, supplying the levator palpebræ and the rectus superior, was affected. I believe I might adduce cases where the inferior portion only was involved; but my notes of these are not hitherto sufficiently precise to be quite satisfactory: besides, in such, the prominent symptom—ptosis—being absent, they might, without careful analysis, be easily mistaken for partial paralysis of the second nerve, or incipient amaurosis.

The preceding cases and remarks have been perhaps already too much prolonged, although I have wished to adduce few or none but what presented some peculiar feature of its own as to degree, extent, or complication, etc.; otherwise I might have extended them almost indefinitely. In regard, however, to the most prominent sign of paralysis of the third pair—viz., ptosis—I beg leave to add a few lines; and to observe that, in addition to its usual origin in a constitutional or cerebral cause, it sometimes is altogether a local affection, the effect of accident, or some morbid state of the palpebra itself. In the former case, constitutional treatment is indicated, and is generally sufficient for the cure. Not so in the latter: having a local origin of a somewhat mechanical nature, a local or operative proceeding is indicated, and generally proves very satisfactory and successful, as the following cases exemplify.

CASE I. *Hypertrophy of Palpebra.* Thomas Walwork, a middle-aged man, came under my care, at the Manchester Eye Hospital, on the 15th of May, 1822, on account of an elongation and thickening of the right superior palpebra, so considerable as to prevent him opening his eye, except in a very imperfect manner. The same affection troubled him five or six years since, but was relieved for some time by an operation performed by my colleague, Mr. Barton.

May 29th. I removed a considerable portion of the external part of the palpebra nearly its whole length, and much of its breadth. Some small arteries bled freely at first, but soon contracted. I brought the integuments together by three sutures.

June 10th. The wound is healed; the deformity in a great measure removed; and he can expose his eye so as to get tolerably well. On account of a little conjunctival inflammation, he was desired to apply a mild lotion and ointment, and to take an aperient.

June 18th. Nearly in the same state. He can open the eye moderately, but not so well as the other, although the distance of the supercillum from the edge of the tarsus is only about half of that on the other side. Being considerably, yet not perfectly relieved, he ceased his attendance.

CASE II. James Wild, aged 19 years, a collier, became a patient of the Manchester Eye Hospital, August 2nd, 1847. About two months previously, a stone fell on the back of his head, and threw him against a coal-waggon. His eyes, he says, were swelled up for a fortnight afterwards. On the right side, there is a cicatrix at the upper part of the eyebrow; on the left, one a little below the eyebrow; and on this side there is complete ptosis; but when the lid is raised for him, he can see pretty well. This state of the palpebra, he says, took place immediately after the accident, and has remained stationary since. There is considerable stiffness about this cicatrix, as if it was firmly bound down.

Sept. 30th. The ptosis continuing in the same state, so that he had no power whatever of opening the eye, and the boy being very desirous of relief, I dissected out a somewhat elliptical portion of the palpebra, embracing the cicatrised part; viz., from just below the eyebrow to about the middle of the palpebra. The wound was united by two stitches and plaster. He was now able to open or close the eye.

Oct. 4th. I removed the stitches, and dressed with plaster.

Oct. 6th. The lid has fallen a little since the stitches were removed, exposing a healthy granulating surface. No eversion.

Oct. 13th. The part has been touched every other day with sulphate of copper, and is now just healed. The gradual contraction of the granulating surface has removed the remaining disposition to ptosis: he can open and close his eye pretty well.

A degree of ectropium afterwards occurring, with a projection of the palpebral conjunctiva, the latter was excised by the curved scissors, and the parts afterwards touched occasionally with nitrate of silver or with sulphate of copper. By these means the cure was soon completed.

CASE II. Joseph Wood, a middle-aged man, was admitted a patient of the Manchester Eye Hospital, June 26th, 1854. The right eye was covered by the superior palpebra, which he could not elevate so as to expose the eye. Six months previously, the lid had been split up vertically in a line from the inner commissure by a gas-pipe. The part divided was united by sutures somewhere in the country, but ptosis has been the result. I took out a portion of the integument of the upper palpebra, and brought the parts together again by two sutures and tissue plaster.

July 13th. The dressing was not removed until to-day. The wound is well united, and he can open his eye much better; but there is some conjunctival eversion.

I afterwards removed a portion of the conjunctiva, soon after which the eye presented and preserved quite a natural appearance.

CASE III. *Congenital (?) Ptosis*. 1854. Michael Burn, aged 15 years, has been for a short time an out-patient of the Manchester Eye Hospital, on account of right ptosis almost covering the eye. He says he has been affected with it as long as he can recollect. He has no headache or other complaint.

August 14th. A moderate portion of the integuments of the palpebra was excised between the eyebrow and ciliary margin. The parts were approximated by three sutures and tissue plaster. The result was satisfactory.

In cases similar to the above, the successful result in the future command over the elevation of the lid is probably aided by the action of the occipito-frontalis, as suggested by my colleague, Mr. Hunt.

The distinctions which I have thus endeavoured to point out, of ptosis into two forms, one of cerebral, the other of local origin, is evidently important as to the appropriate treatment of each. This distinction I recognised and acted upon in the case of Thomas Walwork related above, occurring in the year 1822; and subsequently in several others, as mentioned in this paper.

P.S.—Since the above was written, the following case of *accidental or traumatic ptosis* has occurred to me; and although still under treatment, I very briefly adduce it here.

CASE. James Grimshaw, aged 24 years, was admitted an out-patient of the Manchester Eye Hospital, May 5th, 1856. About a month since, he had the superior palpebra of the left eye divided transversely by a gas-pipe. It was sewed up directly by a surgeon at Staleybridge, but considerable inflammation of the eye followed, attended with violent pain, but he is now much easier. A lotion was applied, and he took some aperient medicine. At the present time there is complete ptosis, the superior palpebra quite covering the front of the globe, and there is some eversion of the inner side of the swollen (fungous) conjunctiva palpebræ.

On raising the eyelid, the cornea appears considerably opaque, from the presence of a somewhat sloughy ulcer almost covering it. He was prescribed a mild lotion, a little ointment at night, and some laxative powders.

May 10th. Little change has occurred since his last visit. I removed to-day (my colleagues, Messrs. Hunt and Redfern being present) the everted fungous-looking conjunctiva. As soon as the inflammation of the eye has subsided, I purpose operating on the lid in the usual manner, as in the cases related above, except the state of the cornea and of the vision should render an operation objectionable.

Original Communications.

ON THE CLOSURE OF ARTERIES AT THEIR ORIGIN; AND ON SOME MORBID CHANGES OF THE HEART.

By THOMAS HODGKIN, M.D.

[Read before the Harveian Society, April 3rd, 1856.]

[Concluded from page 381.]

ALTHOUGH I am unable to produce the proofs furnished by inspection, that many of the various changes in the substance of the heart to which I have alluded were, as I have suspected, the results of closure or obstruction of the coronary arteries at their origins in one or other of the modes which I have described, we may safely infer that they are, for the most part, rather gradually brought about. The histories of the cases, the duration of the symptoms, and the concomitant change in the form of the organ, are all but decisive on this point. This view is almost equally sustained by *a priori* reasoning, for we can scarcely conceive the possibility of the persistence of the heart's action, were its muscular structure to become the seat of acute inflammation. It is next to impossible for a muscle in this state to be brought into action. As an instance of this, we notice the far greater difficulty of breathing in severe pleurodyne, or in inflammation of the diaphragm, than in pleuritis, pneumonia, or bronchitis. If we permit these examples to have any weight with us, we can scarcely conceive the possibility of acute inflammation of the muscular substance of the heart being set up, to any considerable extent, without causing immediate death by syncope. The production of such inflammation appears to be providentially guarded against by the want of susceptibility of this structure in the heart, as shown by the fact of both ventricles having been transfixed with a steel graver, which remained infixed in the substance of the heart for many days, during which the man survived, with comparatively little suffering. I do not remember to have examined but one case in which I found unequivocal evidence of the existence of acute inflammation of the muscular structure of the heart, and as a rarity, at least to myself, I may perhaps be allowed to relate it here. It is true that I have not, at least of late, searched amongst the records of cases inspected by others, but I have no doubt that parallel examples might be found. At the same time I must confess that I should require some very strong evidence of acute inflammation before I could fully admit that it had really existed: it is so easy for a chronic alteration of structure, causing more or less of softening and preparing for rupture, to be confounded, in a tissue like that of the heart, with the effects of inflammation.

CASE. J. G., aged about 64 years, clerk to the Excise, had led rather a sedentary life, and was of temperate habits, rather a remarkably small eater, but unusually corpulent. He was occasionally subject to husky cough in the winter, and was habitually rather short breathed. He had had one or more attacks of oppression of the chest and difficulty of breathing some months, or perhaps even years, before his death, and he recognised his last illness as being of the same character. A few days before his death—I believe not more than ten—he had an attack of increased difficulty of breathing, with great oppression of chest, accompanied with cough, and some expectoration. It was considered that his affection consisted in a smart attack of bronchitis. He was bled repeatedly, by which he was considerably relieved. His bowels were gently opened by a mercurial purgative, and some mild salines were given in conjunction with ipecacuanha. The blood drawn was much cupped and buffed. On the 15th of the month, in the morning, he became much worse, and the oppression of chest and dyspnoea were quite alarming; he had already lost about