is supplied in the report of the Committee of Collective Investigation of the Anatomical Society published in the *Journal of Anatomy and Physiology* in 1894.

Out of eight subjects which I examined a complete bony pterygo-spinous foramen was present in one. In this case it was present on both sides. I have also met with it once in operating. Thus I have met with this variation in two out of nine cases.

The question of the possible existence of the pterygo-spinous foramen deserves consideration in consequence of the impediment it is liable to prove to the successful performance of certain operations. In the operation of excision of the inferior maxillary nerve at the foramen ovale, if a pterygo-spinous foramen is present, that portion of the nerve which passes through it will be protected and probably preserved from division, and thus the object for which the operation was undertaken, namely, the complete removal of the nerve below the foramen ovale, will be defeated. In the case of partial ossification of the pterygo-spinous ligament, the efficiency of the protection afforded to the above-mentioned portion of the inferior maxillary nerve, and the chance of its escaping division in this operation, will of course vary with the amount of ossification present.

In Rose's operation for the removal of the Gasserian ganglion, should there be a pterygo-spinous foramen, this will be the foramen first arrived at in exposing the edge of the foramen ovale by scraping off the pterygoid muscle from its attachment to the sphenoid. When operating it is extremely difficult to distinguish the pterygo-spinous foramen from the foramen ovale. The operator will probably conclude that he has reached the foramen ovale, and will apply his trephine to the edge of the pterygo-spinous foramen. This mistake will much increase the difficulties of the operation, and may give rise to complications which will prevent its successful completion.

The following case illustrates the points to which attention has been called above:—

Mr. P. A. first came to me at the beginning of 1890, at which date he was 54 years of age. He had been a sufferer from trigeminal neuralgia for ten years. The case was a typically severe one. It was confined to the right side.

Two diagnostic points localising the disease to the third branch of the fifth nerve deserve notice.

The patient stated that when his complaint first began the pain was always brought on by movements of the jaw, and was felt just behind the articulation of the lower jaw, from whence it spread over the side of the head and face. It would appear from this that the auriculo-temporal branch was first affected, and that pain was set up by traction on the fibrous sheath derived from the dura mater, in which the inferior maxillary nerve is enveloped, and whose tubular prolongations over the auriculo-temporal and masseteric branches are blended with the capsule of the temporo-maxillary articulation, and are exposed to a certain degree of traction during the movements of the lower jaw. Secondly, I noticed that during very severe and prolonged attacks of pain the right side of the tongue became thickly furred, while the left side appeared natural, or only slightly affected in this way. This appearance seemed to suggest trophic changes resulting from nervous disturbance.

As the patient stated that he had derived benefit from an operation on the auriculo-temporal nerve performed by Dr. Greathead, of Grahamstown, in 1886, I decided to remove the terminal ends of the inferior dental and lingual nerves. This was done in March, 1890. I divided the inferior dental nerve in the dental canal immediately in front of the masseter muscle after exposing the canal by means of a small trephine and dragged out the terminal end of the nerve through the mental foramen. About two inches of the lingual nerve was removed by dissecting it out at the base and side of the tongue. This operation gave perfect rest for one year. During the next six months the patient had occasional twinges. During the following six months the pain increased and began to resume its old character.

In June, 1892, I excised the inferior maxillary nerve at the foramen ovale, reaching the nerve by deepening the sigmoid notch. I removed all that left of the inferior dental nerve by dragging up the part that was left in the inferior dental canal through the dental foramen. This operation gave rest

for about two years and a half, after which time the pain began to return.

I then tried the effect of the *percuteur*. During the first six months of 1895 I gave the patient 100 sittings, each lasting half an hour. During this period the attacks of pain gradually increased in severity. I was not able to satisfy myself that this treatment affected the course of the disease.

In November, 1895, I operated on the Gasserian ganglion by Rose's method. At the time of the operation the possible existence of a pterygo-spinous foramen was present to my mind in consequence of my having met with this variation only a few days previously in the course of a dissection. Nevertheless I decided (wrongly, as it turned out) against this condition. I mention this in order to show how difficult it is to recognise the pterygo-spinous foramen when operating. It was not until the plate of bone cut out by the trephine had been removed that I became aware that what I had taken to be the foramen ovale was really a pterygo-spinous foramen. The trephine hole was therefore wrongly situated, the trephine having been placed against the edge of the pterygo-spinous foramen instead of against that of the foramen ovale. The distance between the outer edge of the foramen ovale and the nearest point of the trephine hole was about $\frac{1}{4}$ inch. There was, therefore, a considerable thickness of bone to be removed before the ganglion could be reached, and in doing this with the chisel the cavernous sinus was unfortunately opened. This accident was, of course, a most formidable obstacle to the successful completion of the operation. The end of the nerve which had been divided at the foramen ovale at the previous operation was fixed to the bone and neighbouring tissues by firm adhesions, while that part of the nerve issuing from the pterygo-spinous foramen appeared to have escaped division altogether. The adhesions were broken down and the superior maxillary nerve divided behind the foramen rotundum, but at this stage I found I must abandon my original intention of dissecting out the ganglion in consequence of the profuseness of the hæmorrhage, and was forced to content myself with cutting through the ganglion and removing the remains of the inferior maxillary nerve together with that part of the ganglion in immediate connection with it. When I last heard from my patient, eighteen months after this operation, he had been absolutely free from pain since its performance.

The practical conclusions to be drawn from the above case may be summed up as follows:—

1. The possible existence of a pterygo-spinous foramen is a point against the performance of excision of the inferior maxillary nerve at the foramen ovale.
2. It is a point in favour of the Hartley-Krause operation on the Gasserian ganglion as compared with Rose's operation.

PERFORATE SEPTUM VENTRICULORUM, WITH INFECTIVE ENDOCARDITIS OF THE PULMONARY VALVES.

By WILLIAM GORDON, M.D., M.R.C.P.,

Physician to the Devon and Exeter Hospital and to the West of England Eye Infirmary.

THE following case presents, perhaps, some points of interest from (a) its bearing on the intracardiac blood-currents of malformed heart; (b) the difficulty of diagnosis caused by the limitation of the endocarditis to the right side; (c) the assistance rendered in this difficulty by alterations in the area of cardiac dulness, (d) a few other features in the course of the case.

The patient, W. F., a small boy aged 5, was admitted to the Devon and Exeter Hospital under my colleague, Mr. Harris, on April 13th, 1893, complaining of pain round the left heel and in the cervical spine, evidently very ill, and with a high temperature. Nothing definite being discoverable at either seat of pain—the pain in the cervical region indeed soon disappearing—and the condition of the heart suggesting infective endocarditis, he was transferred to my care on April 17th.

History.—The history, which was not forthcoming till late in the case, was rather scanty. The boy had been subject, since the age of 10 months, to attacks of palpitation, and a doctor who examined the chest when he was 12 months old said that "he never could be reared." Till three weeks before his admission he had been in his usual health, but then began to complain of pain in his left heel. No cause for this could be assigned. Four days later he said his neck hurt him, and his mother noticed that he was very feverish.

Present State, April 17th.—The fever when I saw him was high and oscillating, with profuse perspiration at each fall of temperature. He was a very pale, thin, pigeon-breasted child, looking extremely ill, but without any trace of cyanosis. Nothing definite could be made out in the cervical region, but about the left heel there was some slight puffiness

and tenderness, especially under the os calcis and between its posterior upper surface and the tendo Achillis. There was, however, no fluctuation. On placing the hand over the heart, one became at once aware of a very marked thrill, systolic and palpable over the whole cardiac area to the right of the apex beat. At the apex beat itself, which was in the sixth space and left mammary line, it was not palpable. The area of cardiac dulness extended from the apex beat upwards to the third left costal cartilage, and rightwards as far as a finger breadth to the right of the right edge of the sternum. A very loud and harsh systolic murmur was heard over the whole front of the chest, loudest at the junctions of the third and fourth left costal cartilages with the sternum. This murmur could not be said to be conducted especially in any one direction. The

second sound was scarcely audible anywhere. The pulse was regular at 118 a minute. The respirations were 32 a minute, and there was some cough. The tongue was perfectly clean, the stools natural, only rather dark; there were no spots or petechiae on the abdomen or elsewhere; the spleen came down below the ribs with a soft edge, and the liver was considerably enlarged. A largish tender gland was found in the left axilla and a few small glands over the left clavicle. The urine was 1020, clear, and contained no albumen.

The character and position of the murmur were strongly suggestive of perforate septum ventriculorum, and, as we had then no history of importance, the diagnosis became a matter of considerable interest. The problem was: Had one to do with an ordinary case of infective endocarditis and an unusual murmur, or with some other infective disease *plus* a malformed heart, or with a malformed heart *plus* infective endocarditis? With regard to the murmur, this was difficult to associate with any ordinary valvular defect, and although the anaemia was very pronounced the murmur was not the least like any hæmic murmur one had heard, and it was, moreover, associated with a pronounced thrill. As to other infective diseases, one's choice practically lay between local suppuration, typhoid, general tuberculosis, and pyæmia other than cardiac. There was no evidence of any local collection of pus. There were no symptoms justifying the diagnosis of typhoid. There was no cerebral disturbance, no choroidal tubercles, no definite signs (then) in the lungs, and no evident old tuberculous lesions anywhere. Every part of the surface was searched for wounds, the bones were gone over with a view of possible osteomyelitis, the ears were examined, the muscles, and the throat with negative results, the only doubtful part being the left heel. In respect of infective endocarditis, however, there was also no certainty; in neither retina, skin, vessels, brain, nor kidney was there any evidence of embolism, and the spleen was not tender as an infarcted spleen would be. Altogether it was felt impossible to make a decided diagnosis at once. Meanwhile large doses of quinine, with abundant food and stimulants, were ordered.

Course.—Naturally attention was mainly directed to the heart and to the tender part of the left heel. The tenderness of the heel persisted, but did not increase, and the swelling remained about the same—very slight; an exploring needle discovered nothing.

On April 16th the right side of the heart was noted to be dilating, the right margin of dulness moving rightwards without any change in the position of the apex beat. At the same time the cough became more troublesome. The spleen was increasing in size, but the liver decreasing.

On April 21st the right side of the heart was greatly dilated, the dulness reaching to the right mammary line. Spleen still larger, liver two finger-breadths below ribs in the right mammary line.

April 21st.

April 24th.

On April 24th the patient's condition had improved. The cardiac impulse was stronger, the second sound audible for the first time. The cardiac dullness had receded, and its right edge was now only a finger-breadth to the right edge of the sternum, as it had been on the 17th. The spleen was, however, still larger, and the liver now reached three finger-breadths below the ribs in the right mammary line; the cough was worse, there were rhonchi at both bases, and harsh breathing and rales could be heard at the apex of the right lung.

On 27th all improvement had ceased, the heart was once more dilating, cough was increasing, the temperature, which had somewhat fallen, was again high and swinging with profuse sweating.

On May 1st, as the quinine seemed useless, it was abandoned, and large doses of steel substituted.

May 5th.

On May 9th the pulmonary second sound was very marked; the second aortic unheard. The pulse felt "almost like that of aortic regurgitation." On May 16th, for the first time, a very faint diastolic murmur could be made out at the lower end of the sternum.

On May 18th oedema of both feet set in, and on May 23rd death took place.

Necropsy.—The accompanying photographs (kindly taken for me by the house-surgeon, Mr. H. Andrew) show the condition of the heart. The pericardium was normal. The opening between the ventricles was congenital and of fair size, easily admitting an ordinary lead pencil. The aortic, mitral, and tricuspid valves, and the walls of the left cavities, were free from disease. The pulmonary valves and neighbouring walls of the right ventricle displayed a number of large grey-green vegetations, most of them rather friable; of these the largest was situated on one of the pulmonary cusps, and a small calcareous nodule at its apex had scored the adjacent arterial wall. They extended below the level of the interventricular opening. There were many recent infarcts in the lungs, especially the right, but no other infarcts anywhere. The os calcis and the bones about the left ankle were carefully examined, but no osteomyelitis was discovered.

REMARKS.

1. *Bearing on the Intracardiac Currents of a Malformed Heart.*—One generally assumes that, in cases of perforate septum ventriculorum, a blood current during systole of the ventricles

sets from left to right, from the stronger ventricle to the weaker. And judging by the thrill, that was what occurred in this heart; but I do not know that we possess any in-

formation with regard to currents set up at other periods of the cardiac cycle. Consequently it adds, I think, to the interest of the case that we seem to have here an opportunity

of deciding whether a current at any period sets in the reverse direction, from right to left. A fair-sized, smooth-walled opening existed between the ventricles, large enough to permit a strong jet to be thrown through it in systole, as proved by the thrill; round this opening and close above it were masses of friable vegetation, actually throwing off emboli and producing pulmonary infarcts. Yet no extension of disease to the left side of the heart occurred, nor did emboli find their way into the systemic circulation. One can scarcely escape the conclusion that no current of any importance, at any period of the cardiac cycle, set from right to left.

2. *The Difficulty of Diagnosis.*—In dealing with infective endocarditis, the textbooks lay stress very rightly on the importance of the evidence afforded by embolism in various parts of the systemic circulation. But though they allude to pulmonary embolism, the distinction is not drawn, I venture to think, quite so clearly as it might be between the symptoms of right-sided and left-sided disease. In those cases where murmurs are absent, or doubtfully recent, the difficulty of diagnosing infective endocarditis is decidedly increased if the left side of the heart is not implicated.

3. *The Assistance rendered in this Case by Changes in the Area of Cardiac Dulness.*—When the patient is old enough to expectorate, hæmoptysis would, no doubt, help to set one on the right track. But failing hæmoptysis I would suggest that help might sometimes be derived from a daily mapping out of the area of cardiac dulness. In the present instance the rapid changes were probably largely due to the jet of blood shot through the congenital opening against the right ventricle wall, and the patient's age made the wall more yielding. But an inflamed heart wall dilates more readily than a sound one, and rapid cardiac dilatation in a doubtful case would be, to my mind, suggestive of intracardiac inflammation. At all events, rightly or wrongly, it was the rapid alteration in the cardiac dulness that led us in the above instance to a definite diagnosis of infective endocarditis.

4. *Other Points.*—The age of the patient was unusual. Of Professor Osler's 209 cases, only 3 or 4 occurred in children.¹ The odd way in which heart dulness and liver dulness varied in opposite directions is difficult to explain. The pulse-rate for a considerable time moderate in proportion to the temperature deserves notice; also the low pulse-tension to which Dr. Sanson has drawn attention.² Pronounced anæmia is common in the disease. Uncommon as is the association of infective endocarditis with congenital heart disease, it would seem to be less so than one might imagine. Similar cases were described by Dr. Turner and Dr. Hector Mackenzie at a meeting of the Pathological Society on January 19th, 1892, in both of which, however, the left ventricle also was affected. At a meeting of the same society in November, 1892, Mr. Jackson Clarke described a case, and alluded to another, where right-sided ulcerative endocarditis co-existed with congenital pulmonary stenosis. If with Dr. Peacock we assume that intrauterine endocarditis is a cause of cardiac malformation, it is reasonable to consider that a valve so damaged should be prone to infective endocarditis, just as valves damaged in after-life by ordinary endocarditis are prone to become infected. At all events, such a case as the above is apparently not such a curiosity that one need hesitate to diagnose it when the indications are reasonably clear.

REFERENCES.

¹ *Outsonian Lectures, BRITISH MEDICAL JOURNAL, 1885, vol. 1, p. 467.*
² *The Diagnosis of Diseases of the Heart and Thoracic Aorta, p. 450.*

DIPHTHERIA TREATED WITH SERUM.

THE FIRST 300 CASES OF DIPHTHERIA TREATED WITH SERUM, COMPARED WITH THE LAST 300 CASES TREATED WITHOUT IT, AT THE DIPHTHERIA BRANCH OF THE SYDNEY CHILDREN'S HOSPITAL.

By CHARLES P. B. CLUBBE, L.R.C.P. LOND., M.R.C.S. ENG.,
Hon. Surgeon to the Sydney Hospital for Sick Children.

As there is still a doubt in the minds of many men (even at this date), both in England and America, as to the efficacy of the serum treatment of diphtheria, it seems to me to be the duty of those who have the opportunity of watching this

form of treatment in a special hospital to publish their results. It has been argued that a comparison of results is often valueless because the cases have not been treated in the same place, by the same man, and are not numerically the same. These arguments will not apply to the cases now under review. All these 600 cases have been treated in the same hospital,¹ and have been under my care. They have been treated exactly in the same way as to food, stimulants, drugs, and local applications. They have all been examined bacteriologically. Cases in which the Klebs-Loeffler bacilli were not found are not even included. The only difference in the treatment is that the last 300 had serum injected.

TABLE I.

Last 300 without Serum. Females, 169. Males, 131.

	Cured.	Died.	Death-rate.
Tracheotomies 199	64	135	67.8
Simple diphtheria... .. 101	78	23	22.7
Total 300	142	158	52.7

First 300 with Serum. Females, 158. Males, 142.

	Cured.	Died.	Death-rate.
Tracheotomies 129	80	49	37.9
Simple diphtheria... .. 171	160	11	6.4
Total 300	240	60	20.0

It will be noticed from this table that the total death-rate is diminished by 32 per cent., so that in the 300 cases 96 lives may be said to have been saved.

There were 70 fewer tracheotomies in the last series, so that the serum treatment lessens the necessity for operation in 23 per cent. of cases. The death-rate in the tracheotomy cases is decreased by 30 per cent.

The following table shows that the ages were very much the same in the two series:—

TABLE II.

Last 300 without Serum.

Cases.	Cured.	Died.	Tracheotomy.	Cured.	Died.
Under 1 year 12	5	7	8	3	5
From 1 to 3 years ... 108	37	71	84	21	63
" 3 " 5 " ... 106	55	51	78	33	45
" 5 " 10 " ... 59	35	24	25	6	19
Over 10 years 15	10	5	4	1	3
Totals 300	142	158	199	64	135

First 300 with Serum.

Cases.	Cured.	Died.	Tracheotomy.	Cured.	Died.
Under 1 year 13	5	8	10	2	8
From 1 to 3 years ... 83	60	23	46	28	18
" 3 " 5 " ... 105	88	17	52	37	15
" 5 " 10 " ... 88	76	12	21	13	8
Over 10 years 11	11	—	—	—	—
Totals 300	240	60	129	80	49

A cottage, containing twelve beds, in connection with the Children's Hospital.