adenoids removed recently. On examination a sessile tumour was seen on the posterior wall and roof of the pharynx, passing into the nose on the right side. It did not appear to be suitable for removal by operation. Microscopic section showed a fibrosarcoma. Up to six months ago, the tumour was treated by diathermy on several occasions.

Condition on Examination.—The nose was broadened out, and extruding from the right nasal orifice, and completely occluding it, was a fleshy, bluish-red mass. The septum was pushed over to the left. The left nasal cavity was patent. A probe could be insinuated between the tumour and the septum on the right side. The right eye was definitely proptosed, no diplopia, vision and eye movements were normal. There was slight epiphora and the right cheek was bulged; there were no glands palpable. The palate was not bulged and the posterior part of the tumour could be clearly seen with a postnasal mirror. The condition was not painful, and there was no anæsthesia of face.

A skiagram showed absence of normal ethmoidal structures on the right side. The septum was pushed over to the left.

On 31st May 1927, one tube containing 100 mgm. of radium sulphate, with platinum screen 0.5 mm. thick, was embedded in the centre of the tumour; two tubes each containing 50 mgm. of radium sulphate in platinum case 0.5 mm. embedded in the periphery of the polypus. The total exposure was for nineteen hours, *i.e.*, 3800 mgm. hours of the sulphate, equal to 26,677 mgm. hours of the radium element. The application of radium was followed by erythema of the skin over the nose. This cleared up in fourteen days.

Condition on 22nd November 1927.—The tumour had shrunk considerably and was now only seen on rhinoscopy. The swelling of the cheek was less and the proptosis not so marked. A further application of two tubes of 50 mgm. of the radium sulphate, screen 0.5 mm. platinum was given for twenty-four hours, *i.e.*, 2400 mgm. hours of sulphate equal to 1684 mgm. hours of the element.

THE SCOTTISH SOCIETY OF OTOLOGY AND LARYNGOLOGY.

TWENTY-SIXTH MEETING, EAR, NOSE AND THROAT HOSPITAL, GLASGOW.

November 26, 1927.

President—JAMES ADAM, M.D.

Tumour of Cerebello-Pontile Angle—JAMES ADAM, M.D.—Male, aged 44, had commencing deafness in right ear in 1910. In June 1926, had sudden paralysis of whole of right side of face. In September 1926, inflammation of the right eye was present, and by

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December it had become sightless and shrivelled. In May 1927, sudden hemiparesis and hemianæsthesia of the left side of the body occurred.

Examination on admission showed involvement of right fifth to eighth cranial nerves. Deafness was absolute with noise box in left ear; no response to caloric tests; loss of taste on right side of tongue. Left fundus oculi was normal. Steady increase of occipital pain after admission.

On 14th October 1927, Mr Paterson removed the occiput from mastoid to mastoid and from lateral sinus to foramen magnum. It was intended to do a one-stage operation, but owing to considerable escape of cerebrospinal fluid and fall of blood pressure, it was decided to do it in two stages, but the patient died on 15th October from pressure of medulla against the foramen magnum.

Autopsy revealed an unusually large tumour which had eroded the petrous and eaten through the floor of the middle fossa. The internal auditory meatus was large enough to admit the little finger.

Two Fatal Cases of Acute Septic Œdema of the Larynx —W. S. Syme, M.D.—(1) Male, aged 68, was admitted with pain in throat and difficulty in swallowing and breathing for one day; pulse 80, temperature 97° F. The difficulty in breathing was not severe. There was severe inflammatory œdema of the epiglottis and aryepiglottic folds. Parts of the cords could be seen and there was a fair airway. It was not considered necessary to perform tracheotomy. The ice bag was applied to the neck and stimulants prescribed. The breathing did not become more laboured, but he died suddenly next morning. There was at no time a rise of temperature. No autopsy was obtained.

(2) Female, aged 53, while in a medical ward complained of sore throat and evident distress in breathing; she was transferred to the Throat Department. She stated that the sore throat had started two days previously and had become much worse. When seen at mid-day, she looked very ill. The pharynx was coated with a sticky, purulent discharge; the tongue and pharyngeal mucosa were glazed. The temperature was 97.6° F., and the pulse 82. There was severe œdema of the epiglottis and the laryngeal folds, associated with much purulent discharge. (The bacteriological examination showed streptococcus.) Tracheotomy postponed; ice bag was applied and stimulants prescribed. Five hours later she was seen again, and as the dyspnœa had become worse, tracheotomy was performed under local anæsthesia.

For three or four hours she showed decided improvement, but died suddenly at midnight.

The salient points of the post-mortem examination are these. The body is that of a somewhat obese, elderly female subject. The lungs show purulent bronchitis. The heart muscle is soft and pale. The liver shows very marked cloudy swelling and much fatty change. The

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gall-bladder contains one large and about a dozen small stones. The mucous membrane of the gall-bladder is swollen and the normal honeycomb markings are obscured. The kidneys show very well-marked cloudy swelling. The throat is the seat of intense inflammatory change. The epiglottis, glosso-epiglottic and aryepiglottic folds are œdematous and coated with exudate. There are also numerous ulcers on the folds.

Dr LOGAN TURNER said he wished to draw attention to the great rapidity with which œdema spread along the aryepiglottic folds, which might in a very brief space of time cause occlusion of the upper laryngeal aperture and death from asphyxia. He did not wish to criticise adversely the fact that Dr Syme had not performed tracheotomy, as so much depended upon the condition of the patient and all the circumstances of the case. He (the speaker) thought, however, that tracheotomy might be unduly delayed.

Dr W. T. GARDINER remarked that in these cases he would have intubated and would have felt happier regarding the possibility of death from asphyxia.

Dr J. S. FRASER thought that the absence of fever in these cases was probably a bad sign. One knew that acute streptococcal infections were usually associated with fever, and probably the absence of fever meant an absence of reaction to infection.

Dr HARPER said these two cases were of interest in that both died suddenly. The cause of death in such cases was often obscure. In the past six months he had had two similar cases, both dying suddenly. In neither of them was death due to obstruction to the breathing and there was certainly no dyspnœa. In both, the organism present was a streptococcus.

As regards the question of performing tracheotomy, he (the speaker) was rather against this unless there were definite indications for it. In the Royal Infirmary they had a number of cases of acute œdema of the larynx especially on Sunday mornings—men who had been intoxicated, rolling in the gutter, shouting and probably fighting. They were brought with acute œdema of the larynx involving the arytenoids, aryepiglottic folds, and in some cases the epiglottis. On examination one would have said that tracheotomy was absolutely necessary. But they had not found it necessary to operate on these cases and they had had no deaths from obstruction. The interesting point in the two cases under discussion was the cause of death. What was the cause of death? He (the speaker) was convinced that in most cases it was not due to obstruction to the breathing, but it was of cardiac origin, due to the toxins from the infective micro-organisms. These cases did not as a rule have fever.

Dr BROWN KELLY suggested that this affection would be a suitable subject for discussion. Of the cases of septic sore throat with which he had had to deal, he briefly referred to seven which stood out specially in his memory. Four of these died from heart failure, suddenly and unexpectedly, and a fifth, after bronchopneumonia and suppuration in one knee joint, from pneumococcal pyæmia three weeks from the onset

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of the illness. Two patients recovered; in one the local affection was slight and the general condition excellent. The other was critically ill and unfit to leave the house for three months: a large amount of streptococcus vaccine was given, which produced a violent reaction. Although the larynx was affected in all seven cases, there was no question of tracheotomy excepting in one patient whose general condition contra-indicated operation and who died, but not owing to respiratory obstruction, six hours after coming under observation. The temperature as a rule was not much raised, but in one patient it reached 104° F. The tragic termination in a large proportion of these cases should lead one to give a very guarded prognosis.

Dr JAMES ADAM (the President) said he was interested in Dr Brown Kelly's statement because the cause of death from heart failure was indicated in the post-mortem report on the second case. The absence of pyrexia was a dangerous symptom and meant a bad prognosis.

Dr SYME (in reply) said he felt that these cases did not require to be operated upon and yet he felt guilty afterwards that he had not operated. Now he was much reassured by what Dr Harper and others had said. Even if he had operated earlier, these patients would have died. In neither case was there that distress in breathing that pushed one to operate. He had had another case some time ago, to which he was called at night. On arrival he found the house-surgeon had done tracheotomy and the patient recovered. Still he did not know what he should do if he encountered similar cases again. Dr Gardiner's suggestion that one should introduce a pharyngeal spatula and put a tube through it into the larynx suggested itself as good a way as any and yet he (the speaker) did not know. These patients died from heart failure.

Right Temporal Lobe Abscess.—W. S. SYME, M.D.—Female, aged 9, was admitted on 9.5.27, with a history that six months previously she had a fall and since that time had complained of pain in the right ear. Six weeks later the ear commenced to discharge. This has continued. Since the fall she has had giddy turns, sickness and vomiting, and during the past month she had had shivering attacks. She sweats at night and is losing weight.

On admission, pulse 84, temperature 97.4° F. Right chronic middle-ear suppuration; tympanic membrane destroyed. Rinné negative; no spontaneous nystagmus. 10.5.27.—Right radical mastoid operation was performed. Nothing was found to suggest the necessity of proceeding further. The child progressed normally for a week, but after that she became irritable and difficult to deal with. The temperature rose slightly and the pulse became slower. 24.5.27.—Lumbar puncture. Fluid turbid, but pressure not appreciably increased. The mastoid cavity was opened up again and the tegmen removed. Thin pus was seen to be coming from the anterior part of the roof, and here an opening was found in the dura mater. This was enlarged and a large abscess in the anterior part of the

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temporal lobe was opened and much thick purulent discharge evacuated. The abscess cavity was swabbed with silver nitrate solution 10 per cent. A gauze drain was put in. This was removed in three days and thereafter no drainage was employed. The cerebrospinal fluid and the discharge from the abscess showed lymphocytes and polymorphs, but from neither was any culture obtained. The child made an uneventful recovery.

Unsuspected Right Temporal Lobe Abscess.—W. S. SYME, M.D.—Male, aged 53, had discharge from the right ear for many years. A year or more ago had severe pain in right ear at times; was very deaf in both ears. Admitted on 10.10.27 with copious discharge from right ear: watch not heard; Rinné negative; no spontaneous nystagmus. Left ear had chronic adhesive changes. Watch not heard; Rinné negative. There were no disconcerting symptoms. The operation was undertaken solely because the suppuration did not yield to simpler measures.

11.11.27.—Right radical mastoid operation performed. The antrum was filled with grey material which was at first thought to be tumour growth, but which proved on histological examination to be brain tissue. On removal of this, the tegmen was seen to be missing and there was a large opening through the dura mater. Dressing forceps passed through this opening entered a large cavity and some thick purulent material escaped. This was followed by a gush of clear cerebrospinal fluid which ceased abruptly. As the opening into the cavity was large, no other method of drainage was employed. The discharge from the brain abscess showed streptococcus. Following the operation, the patient went on well for ten days. Then symptoms of meningitis developed and he died on the 24th October. The post-mortem showed a very large abscess cavity extending into the lateral ventricle, and purulent lepto-meningitis.

Dr SYME said he wished to draw attention in these cases to the absence of drainage, or rather the cessation of drainage after three days. The question of drainage of brain abscess was always a difficult one.

Dr J. S. FRASER asked whether Dr Syme regarded the history in the first case as correct. According to the history, it was really a case of subacute middle-ear suppuration said to be due to a blow or a fall six months previously. The parents of children often stated that a blow by a school-teacher or something of that sort was the cause of the middle-ear suppuration, and yet, examination of the child's other ear might show evidence in the tympanic membrane of former chronic suppuration, *i.e.*, chalk patches, dry perforation, or a scar. The history of middle-ear suppuration from a blow or fall must be received with caution. The change in the child's disposition was of great interest in this case. It was one of the signs upon which we had to depend because abscess of the right temporal lobe was very difficult to diagnose. Another point was the question of turbid fluid on lumbar puncture. Was that to be regarded as evidence of meningitis? Turbid cerebrospinal fluid was sometimes found in cases of brain abscess and yet, when the abscess was opened,

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the case recovered and one heard no more of the meningitis. It seemed likely that leucocytes were poured out from all the neighbouring vessels and that the cerebrospinal fluid was probably cloudy on that account. These cases were not to be regarded in the same light as those of labyrinthine meningitis. Like himself, Dr Syme had used a gauze drain. He (the speaker) agreed that the question of drainage of brain abscess was one which should be discussed. Why was the abscess in this case swabbed out with silver nitrate? It was a useful antiseptic in cases of infection with the *Bacillus pyocyaneus*, but had Dr Syme any special reason for using silver nitrate?

It appeared in the second case as if there had been spontaneous rupture of the brain abscess into the tympanic antrum. He had never met with such a case, but he had seen a suppurating lateral sinus rupture into the antrum and the patient had recovered without any necessity for ligaturing the internal jugular vein.

Dr HARPER said he did not think he had anything to say regarding these two cases beyond the fact that he was interested in the use of the gauze drainage. He was glad to hear from Dr Fraser that there was going to be a discussion on the question of the drainage of brain abscesses. He (the speaker) knew that some surgeons considered that the drainage was an unimportant part of the operation. They held that the opening of the abscess was the chief point and that if the abscess was going to get better, it would get better notwithstanding whether it was drained by gauze or not.

Dr RITCHIE RODGER said, in reference to spontaneous rupture of brain abscess into the tympanic antrum, he had a case about a year ago in which the diagnosis of temporal lobe abscess had been made in a man about fifty years of age. Examination of the ear on admission revealed the usual granulations with a small amount of purulent discharge. When he was wheeled into the theatre from the anæsthetic room, the dressings were drenched with pus, and when they were removed, pus was pouring out of the meatus in large quantity; there seemed to be ounces of it. The mastoid was rapidly exposed and the whole of the roof of the antrum was found eaten away, with a large erosion of the dura mater still discharging pus.

Dr ROSS SOUPER said there was one small point he wished to mention, namely, the alteration in the child's disposition. Dr Fraser referred to that as a dependable sign, but he (the speaker) was disposed to think that in a child it was not so. He had an experience which fortunately ended happily, but very nearly did not owing to laying stress on that very fact. A girl, aged 10, had a mastoid operation performed and the lateral sinus was opened and the clot removed. She did well for a few days and then developed optic neuritis on the same side. She also developed 6th nerve paralysis without fresh local signs or fever, and he had to consider whether the child had developed a temporal lobe abscess or some other localised abscess. The ophthalmic surgeon, who had examined the case for him, had great experience and advised him to delay. The child's disposition was remarked upon by the Sister in the ward; she got drowsy, took no notice of any one, was extremely irritable and altogether looked as if she had definitely developed some intracranial complication. Before a decision was reached the father visited the child. He (the speaker) inquired as to

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the child's disposition and was told that she was a bright child. The father intimated that he had found nothing the matter with the child, who afterwards told the sister that the reason of her indisposition was that she was "fed-up with the whole show," weary with the dressing of the wound, did not like hospital, and wished to go home. He (Dr Souper) was glad that he had not explored for intracranial abscess. The ophthalmic surgeon's explanation of the optic neuritis, which fortunately cleared up, was that he had seen similar cases after a thrombosed sinus and that it was due to some disturbance in the intracranial circulation after obliteration of the sinus.

Dr SYME (in reply) did not think there had been any alteration in the child's mentality since the operation. As regards Dr Souper's statement, he (the speaker) suggested that a brain abscess might be latent for a long time, and that something must have caused the optic neuritis. He had used silver nitrate solution because he regularly did so in septic conditions; in ethmoidal conditions he swabbed the cavity with silver nitrate. He had left the gauze in for three days. The question of turbidity of the cerebrospinal fluid was most interesting. Perhaps one reason why the child recovered was that there was no growth on culture. The man's case had surprised him. He had decided that the radical mastoid operation should be done and his name was put on the list. He was brought in and no one was more surprised than himself when he found the antrum full of greenish material which he thought at first was new growth. Exploration led into a large cavity from which broken-down tissue and purulent material escaped. This was followed by a sudden flow of clear cerebrospinal fluid which he thought was a cystic collection in connection with the lateral ventricle. It was quite possible that the purulent meningitis had developed as a result of infection by this channel.

Left Chronic Middle-ear Suppuration with Fistula Symptom and pronounced Rotatory Nystagmus—W. S. SYME, M.D.—The patient was a female, aged 52 years.

Dr FRASER asked for the report on the Wassermann reaction of the blood and cerebrospinal fluid, because a fistula symptom sometimes occurred in congenital syphilitic cases (Hennebert's sign). This case had some cloudiness of the cornea, but this resembled that due to former ulceration rather than to interstitial keratitis. It was very interesting that the nystagmus was not of the usual horizontal but was undoubtedly of the rotatory type. Hennebert's sign had been attributed to undue mobility of the stapes, and in this case possibly some such explanation might hold good.

Dr SYME (in reply) said he had shown the case because of the rotatory nystagmus. He was quite conversant with the horizontal nystagmus obtained in a fistula symptom, but he had not seen the rotatory nystagmus before. It had not occurred to him that it was possibly specific. The deafness was not what one looked on as a specific type of deafness. He thought she had an old ulcer of the cornea. The Wassermann test would be made.

Patients showing the Result of Correction of Depressed Nose by Rib Cartilage—W. S. SYME, M.D.

Dr DOUGLAS GUTHRIE said that everyone with experience of nasal reconstruction knew how difficult it was to get good results in every case

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and how the technique should be adapted in each case. Dr Syme did not show a photograph or drawing of the condition before the operation. It was of importance in checking the result.

In one or two, though not in all of Dr Syme's cases, the cartilage did not reach as far as the tip of the nose. It was essential that the graft, introduced from an incision at the base of the nose, should reach as far as the tip so as to give support to it. In certain cases it was also necessary to give support to the columella. In making the pocket from above, one must project the elevator right down to the tip of the nose so as to secure a good bed for the graft at the lower end, otherwise the result would not be satisfactory. Had Dr Syme tried the incision in the columella, because it presented certain advantages? The incision seemed to be very useful, and one could always be certain of getting the graft into good position at its lower end. One of the cases showed a splaying out of the ascending process of the maxilla on each side. In such a case one could never restore the nose merely by a cartilage graft. It was necessary to divide the ascending process of the maxilla so as to restore the contour of the nose. He (the speaker) had had two cases of this nature and was able to divide the ascending process from the inside of the nose, making an intranasal incision and then introducing a fork-tailed chisel and dividing up towards the inner canthus of the eye. Even though only one side was displaced, the operation must be performed on both ascending processes and then the entire external nose was rendered quite mobile and could be moulded into the desired shape.

Dr ADAM (the President) said the question of sepsis was worth keeping in mind. He had done a number of operations, and in one, with healed tuberculosis of the septum starting after trauma, he had used Gillies' incision. It became septic and the implanted cartilage had to be removed. That was the only one in which he had that result. He preferred either the central incision which Dr Syme used in one case, or one inside on the septum.

Dr SYME (in reply) said he had never had sepsis necessitating removal of the cartilage. Once or twice he had delay in the healing of the small incision. He liked the incision in front of the columella best and had only recently used it. All the same he did not think that a Roman or a Grecian nose would suit every face. One of the patients shown had a slightly retroussé nose and he thought that she was nicer looking than if she had been given a straight nose. The case with splaying of the nose was shown purposely to demonstrate the deformity and to elicit opinions. He had had intended to attack the deformity from the canine fossa upwards, and then remove the prominent part of the ascending process of the maxilla, but that would leave a certain hollow in the side of the nose. He thought that Dr Guthrie's suggestion to fracture the part was the better one.

Tuberculosis of the Larynx—H. WHITEHOUSE, M.B.—Male, aged 21 years, had influenza followed by left-sided pleurisy one and a half years ago. Later, hoarseness and tubercle of left side of larynx developed. He has had Sanatorium treatment for one year. He returns now complaining of sore throat. The tonsils are septic. The larynx is quiescent, but he is gradually losing weight and has cough and spit. Should enucleation of tonsils be performed now?

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Dr SOUPER asked what the condition of the larynx was a year ago. He thought it would be very interesting to know what the condition of the larynx was when it was first described as a tuberculous laryngitis. He did not agree with the present description of the larynx, which in his opinion showed active disease.

Dr BROWN KELLY agreed that the disease was not quiescent. The posterior wall was infiltrated and the left cord red.

Dr WHITEHOUSE (in reply) said that as far as he remembered the condition (he only saw the case once) there was infiltration of the left arytenoid and also of the left false cord. The report on the sputum was positive and the patient was removed to the Sanatorium.

Suggested Explanation of Cardiospasm based on Pathological Changes — MUNRO CAMERON, M.B. (introduced by Dr A. Brown Kelly).—The view put forward was based on the histological examination of eight examples of œsophagectasia without distal (pathological or anatomical) stricture; briefly, the dilatation of the œsophagus was not due to any cause extrinsic to that organ, but to a degeneration of the cells in Auerbach's plexus at the lower end of the tube.

Seven of the cases were of long standing and the ages varied from 22 to 67 years at the time of death. Allowing for individual differences in the extent of the fibrous change the sections generally showed: inflammatory changes in the submucous layer with œdema, desquamation of the epithelial layer in parts, and in others, small ulcerated areas of varying depths. The muscularis mucosa, instead of consisting of two or three thin layers as in a normal œsophagus, was invariably definitely hypertrophied and occasionally showed fibrous, hyaline, or other degenerative change. The circular layer was always increased in size and usually out of all proportion to any increase in the longitudinal layer; it almost always showed fibrotic changes, in one example complete muscle bundles being replaced by fibrous tissue. The longitudinal layer often exhibited some degenerative change and occasional inflammatory cells were found in the interstices. Between these two layers, the ganglionic cells were invariably affected and varied from a chronic inflammation with increase of the cells in the ganglionic capsule to complete replacement of the ganglia by fibrous tissue.

In the eighth case, the child concerned was six years old at the time of death and had suffered from attacks of vomiting coming on immediately after food, since six months old, and just after bronchopneumonia. The condition had varied in severity, and an occasional period of six weeks' duration occurred in which no vomiting was evident. At two years of age the child was sent to a Sanatorium, where he was treated apparently very successfully for phthisis. At the post-mortem examination, only a small, healed scar was found at the apex of the left lung. One month before admission the condition was so bad that the child would not, or could not, swallow anything, and gastrostomy was performed.

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Nothing of special interest was found at autopsy except the condition of the œsophagus which, above a hypertrophied cardiac canal, displayed a fusiform dilatation extending to within two inches of the lower border of the cricoid.

Histological examination of sections made through the wall of the cardiac canal showed pitting and erosion of the mucosa with inflammatory reaction round the glands. The submucous coat was œdematous, the vessels very congested and the muscularis mucosæ thickened. Considerable hypertrophy of the circular and some hypertrophy of the longitudinal layers were noted; all the muscular coats showed hyaline changes of greater or less extent, and odd bundles of lymphocytes were scattered between the bundles and lodged in the interstices. The inflammation in and around the ganglia was acute. Masses of leucocytes were found around the ganglia and the nerve cells when present showed chromatolysis, sometimes a swollen, glassy, "ghost-like" appearance. In some parts, spaces filled with colloid were present where normally ganglia might have been expected. The most careful search revealed no tubercle bacilli, but some Gram-positive diplococci, presumably pneumococci, were found.

Common then to all these cases was an inflammatory lesion of the ganglia, acute or chronic in its manifestation. The condition of the vessels in the first seven suggested that an arteriosclerotic process might be the primary cause; but the direct and indisputable evidence of bacterial invasion in the last case makes a toxic basis the more credible for all, especially as the antecedent development of an acute infectious disease, influenza, or pneumonia, is not uncommonly reported in the literature of this disease. The invasion of the ganglia may be a specific selection of these structures by bacteria or their products, parallel to what is found in post-diphtheritic and meningococcal paralysis, or it may be the result of the outward spread from a primary œsophagitis.

We know from the work of Kronecker and Meltzer that a stimulus applied to any point on the œsophageal mucosa results in an area of contraction above and of relaxation below that point, and Bayliss and Starling have shown that repetition of this stimulus results in contraction alone, relaxation not taking place. Physiologically, therefore, the first result of interference with normal peristalsis is abeyance of the relaxed area below the stimulated spot. The lesion I have described here will interfere with stimuli reaching the ganglia and with transmission from them, and at the area supplied the peristaltic wave will be held up. Sooner or later food will accumulate and the œsophagus will dilate, while the walls hypertrophy in an attempt to expel its unaccustomed contents.

The older authorities, Zenker, von Ziemsen and Morell Mackenzie, had no difficulty in accounting for this condition, as they looked upon it as due to a myasthenia or at least to a diminished contractile power of the œsophageal musculature. To-day we know that the muscle is nearly always hypertrophied and that usually it persists throughout the

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dilated area; in some examples it appears to develop proportionately and *pari passu* with the dilatation.

Dr BROWN KELLY congratulated Dr Munro Cameron on his striking results. He said that the finding of changes in the intermuscular ganglia at or near the hiatal level in all the five cases of cardiospasm examined, supported the view that the underlying cause of the disease was a disturbance in the innervation and functioning of the cardia. It would therefore appear that certain factors, to which etiological importance had been assigned, could henceforth be discarded, viz., pressure external to the œsophagus exercised by the crura of the diaphragm or other conditions. A. F. Hurst maintained that the affection under consideration was not due to spasm, but to absence of the relaxation at the cardia (achalasia) which normally took place during deglutition. The speaker, on the other hand, held that the clinical evidence supported a spasmodic origin because in these cases closure at the cardia was at least firmer than in normal individuals, while sometimes it was strong and difficult to overcome especially when the disease had existed for long; also, because spasmodic closure of the cardiac canal could usually be excited and observed with the œsophagoscope.

Dr J. S. FRASER asked Dr Cameron if he could say what was the cause of dilatation of the stomach and of dilatation of the colon. It was well to look at analogous conditions in other regions of the intestinal tract. Were the changes found in the ganglia presiding over the contraction and relaxation of the colon the same as those found in the œsophagus of cases of so-called cardiospasm or achalasia? What was the beginning of the pathological process? Was it an œsophagitis followed by small-cell infiltration round Auerbach's plexus, or was it a local toxic absorption or a toxic condition of the blood? He wished to ask Dr Cameron for his explanation of a case mentioned at the last meeting of the Society in Edinburgh. When the tube was passed he (the speaker) found the œsophagus, as usual, full of fluid bismuth which had been given previously. The operating table was then turned into the Trendelenberg position and the œsophagus was washed out. In most cases this could be done without difficulty, the water being returned through the tube, the tube itself being gripped by the sphincter at the upper end of the gullet. In this case, however, the fluid had escaped between the œsophageal walls and the tube and came into the patient's mouth. He supposed that there was a want of tone of the muscles at the upper end of the œsophagus, and, if at the upper end, why not through the whole length of the œsophagus.

Dr MUNRO CAMERON said he had always the greatest difficulty in understanding why one group, which used the term achalasia of the cardia, in describing this condition, should object so much to the use of cardiospasm, and *vice versâ*. He saw no difference except one of degree.

If an involuntary muscle (in this case the physiological cardia sphincter), which ordinarily should relax, did not relax because of a lesion affecting its controlling ganglia, what was to prevent that muscle swinging over to the other side, so to speak, and becoming spasmodically contracted?

Then, as regards Dr Fraser's question about similar conditions in other parts of the body: the most obvious parallel was megacolon or Hirschsprüng's disease. He (the speaker) had examined two cases of this

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condition, but had not found any lesion quite comparable to what he had described in the œsophagus to-day. In one there appeared some diminution of the cells in Auerbach's plexus, in the other, some atrophic change in Meissner's plexus.

As to Dr Fraser's next point, whether the changes in the ganglia followed a preliminary œsophagitis, he (the speaker) thought sometimes this was probably the sequence of events. At other times there appeared to be a specific selection of ganglia by bacterial toxins similar to what was found in diphtheria and cerebrospinal fever.

He thought Dr Fraser's explanation of the case mentioned, where fluid escaped between the œsophageal wall and the tube, was correct, and that it was due to a diminution in the contracting power of the muscle. Indeed he (the speaker) showed one case on the screen in which the replacement of the circular muscle by fibrous tissue could have had no other result.

Three Fatal Cases of Cavernous Sinus Thrombosis, with Post-mortem Reports on Two—GAVIN YOUNG, M.B.—CASE I.—Male, aged 31, was said to have suffered from "septic throat" commencing about 13.9.27. This was present on admission, as also bronchopneumonia. The left eyeball was proptosed and fixed, the conjunctiva injected, both eyelids red and œdematous; patient was seen by Dr Lewis Macmillan, who diagnosed provisionally "cavernous sinus thrombosis," or "orbital cellulitis" following sinus infection. Patient was too ill to undergo X-ray examination and died 26.9.27.

Both lungs showed septic pneumonia with abscess formation; the nasal accessory sinuses were healthy, the cavernous sinuses thrombosed with pus present.

The suggested route of infection was septic tonsillitis, septic pneumonia, blood-borne infection to cavernous sinus.

CASE II.—Female, aged 21, was diagnosed as "mumps" on 10.2.27, with otitis media, left, on admission.

18.2.27.—Both parotids were incised and pus evacuated. 26.2.27, left cortical mastoid operation was decided on because of slight swelling over mastoid; perisinus abscess present; sinus wall unhealthy and exposed over considerable distance; primary suture, which healed well.

Temperature remained unsettled for several weeks, until 19.3.27, but this was attributed to the presence of several abscesses in the scalp which required to be drained; patient tended to become drowsy.

19.3.27.—Left pupil became dilated, left external rectus showed paresis and left eyeball was slightly proptosed.

20.3.27.—Lateral sinus opened, some clot seen, sinus ablated. Patient improved for a few days and the left eye was less proptosed, but she became worse and died on 31.3.27. No autopsy.

CASE III.—Male, aged 6, was diagnosed as "pneumonia." On admission, neck was retracted, Kernig positive, and a lumbar puncture was carried out at once. The cerebrospinal fluid was turbid, with increase of polymorphs but no organisms. History of slight earache,

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left side, but no discharge; tympanic membrane was intact, but showed signs of past suppuration; no mastoid tenderness.

21.10.27.—Lumbar puncture, cerebrospinal fluid turbid, containing Gram-positive cocci, mainly intra-cellular: left eyelid red and œdematous, with proptosis, and right pupil dilated.

27.10.27.—Patient died.

Post-mortem report stated:—Pus in left middle-ear, mastoid cells apparently healthy; thrombosis of left lateral sinus, petrosal sinuses and left cavernous sinus: meningitis.

Dr ADAM (the President) asked if there was any thrombosis of the lateral sinus in the first case.

Dr GAVIN YOUNG said the lateral sinus was thrombosed.

Dr LOGAN TURNER said that we were much indebted to Dr Gavin Young for bringing forward these cases. They illustrated the fact that infective thrombosis of the cavernous sinus was not so infrequent as we might be inclined to think. Dr Young was able to describe three cases occurring in one hospital in the space of twelve months. At the present moment he (the speaker) had, in the laboratory, material and post-mortem notes of thirty-one cases dying with intracranial complications, and sixteen of these (50 per cent.) had infective thrombosis of that blood sinus. He had made a note of the primary focus of infection in these cases and found that one had a focus in the eyelid, one, suppuration round the lacrymal sac, two, furuncle of the nasal vestibule, one, a septic focus of the upper lip, one, a peritonsillar abscess, one, middle-ear suppuration, and nine, disease in the accessory nasal sinuses.

Dr Young had asked how infection reached the cavernous blood sinus from the tonsil. The tonsillar veins drained into the pharyngeal plexus, a tributary of the pterygoid plexus. The latter communicated through the foramen ovale or by the vein of Vesalius with the cavernous sinus. Hence infective thrombosis of the pterygoid plexus might produce, by retrograde thrombosis, an infection of the cavernous blood sinus. Middle-ear suppuration might bring about thrombosis of the cavernous sinus without involvement of the lateral sinus as an intermediate stage, because veins opened directly from the ear into the superior and inferior petrosal sinuses.

Dr RITCHIE RODGER said that Dr Wells P. Eagleton in the book he published last year stated that infection of the cavernous sinus from the tonsil might occur by one of two routes—by the pterygoid plexus or from the small vein surrounding the tonsil conveying infection to the internal jugular vein, thence to the inferior petrosal sinus. As Dr Turner had said, thrombophlebitis of the cavernous sinus was more common than was supposed. When reporting four cases in 1921, he (the speaker) suggested that there were two distinct types of the condition, and this was borne out by the cases of Dr Young. There was the usual type described in the text-book, with rapid onset and dramatic display of symptoms, extreme proptosis, chemosis and discoloration. This was due to infection carried direct to the cavernous sinus in the direction of the blood flow. A septic clot from the region of the nose or sphenoidal sinus reached the cavernous sinus and in the slow current of the latter, thrombosis quickly

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occurred with rapid onset of the symptoms described. In cases of otitic origin, however, there was first, as a rule, thrombosis of the sigmoid sinus or of one of the petrosal sinuses, and the infection of the cavernous sinus was of slow onset. With stasis in the cavernous sinus due to blocking of the petrosals, the infection spread slowly by continuity, against the direction of flow. A mastoid case might show puffiness about the eyes for many days before distinct proptosis or chemosis could be detected.

Osteoma of Left Orbit, Ethmoid and Antrum. Operation: Recovery—GAVIN YOUNG, M.B.—Male, aged 26, had exophthalmos and lateral displacement of his left eye, with defective vision. The middle turbinal was replaced by a bony growth of extreme density. X-ray examination showed a large area of increased density in region of middle turbinal, anterior ethmoidal cells and orbit.

Operation by Moure's incision; upper part of the antrum was occupied by dense bony growth which was followed up into ethmoid, orbit, and middle turbinal region.

Dr J. S. FRASER suggested that a radiogram should now be taken of this case. It was necessary to account for the displacement of the eyeball. A radiogram would show whether there was still some osteoma at the back of the orbit.

Dr ADAM (the President) said he had shown one case about 1911; there was displacement of the eyeballs in the same way. Both antra were involved. He removed the osteomata with local anæsthesia quite easily. They certainly originated from near the floor of the antrum and not in the ethmoid.

Dr RITCHIE RODGER wished particularly to refer to a case because of the unusual history. At the Edinburgh Meeting of this Society, seven years ago, he showed photographs of a patient before and after the removal of an osteoma from the ethmoid region. The first tumour was as large as a small cherry, situated in the middle line; after making great preparations for the operation, he found that on the first application of the mallet and chisel it came away easily and completely. He could see into both frontal sinuses which were healthy, and the ethmoid cells were practically gone. Healing took place by first intention.

After seven years the patient returned with complaint of severe pain over the left eye. There was protosis of this eye, displacement of the eye downwards and outwards, swelling of the supraorbital region, and a hard swelling at the inner canthus. X-ray examination showed a dense tumour filling the whole of the left frontal sinus, involving the ethmoid region and extending downwards into the antrum, half filling the latter cavity. He operated through Moure's incision which, however, had to be extended very far outwards in the supraorbital region. There was hard ivory bone filling the frontal sinus. The anterior wall of the sinus was as thin almost as an egg shell, and the roof of the sinus was eroded to the extent of a square inch, with exposure of the dura mater. There was pus in the attenuated air space surrounding the tumour. The ethmoid and antral parts of the tumour were equally dense. The patient again made a good recovery with quite a good cosmetic result. It would seem that we have to be prepared for recurrences of osteomata in this region.

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