Many failures result from neglect to remove the enlarged posterior end of the inferior turbinals, which are far more obstructive to respiration than are the faucial tonsils; of course, in many cases they would diminish in size after removal of the pharyngeal tonsil, but as this would occupy from two to three years, during which time the nasal obstruction would continue, why operate at all? A patient who undergoes an operation for nasopharyngeal obstruction deserves a perfectly functional result. It is no use scraping the pharynx as a routine when the entire obstruction is due to nasal stenosis.

The posterior ends may be removed by snipping them away with one of the guillotines invented by Lake, but often it is impossible to get these instruments easily down both sides of the nostril. In this case a strong nasal snare may be employed; the loop should be guided over the posterior ends with a finger in the nasopharynx.

Of special interest, also, is the persistence of symptoms of "throat cough" and laryngeal catarrh after operation. In many cases this will be found to be due to either a septic condition or actual hypertrophy of the lingual tonsil, which may be in contact with the epiglottis. It is very possible that after complete extirpation of the faucial and pharyngeal tonsils the remaining lingual tonsil undergoes some compensatory hypertrophy.

If there is found such a degree of cartilaginous septal deviation that one nostril is completely blocked, it is essential to obtain a free airway or sooner or later an acute catarrh supervening will cause either collapse of the membrana tympani or middle-ear suppuration on that side. The best operation is some form of Watson-Gleason operation which can be performed with a scalpel and a pair of Killian's septal compression forceps. If the deflection is slight it may be rectified by a dental splint in many cases.

After-treatment is quite as important as the operation itself. It consists of (1) breathing exercises daily; (2) politzerisation weekly for six weeks, then omitted for one week, and repeated for six weeks if required; and (3) dental appliances necessary perhaps to restore a narrow palatal arch and septal deviation which is not severe enough to require operative treatment.

Manchester.

A NOTE ON PERFORATION OF GASTRIC ULCERS INTO THE HEART ITSELF,

WITH REPORT AND PHOTOGRAPH OF A CASE.

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THIS condition is one of extreme rarity. After a prolonged search I have been able to find only four cases recorded in the literature, and in only one of these was the diagnosis suggested before death.

The first case to be recorded was that of Oser⁵ in 1880. His clinical record is accompanied by full details of the post-mortem findings of Hans Chiari.⁸ The patient was a woman, aged 71, who lived three and a half days after the first hæmatemesis. The ulcer was a round one measuring 2 cm. across; it was situated on the lesser curvature, at the cardiac end of the stomach, 5 cm. from the cardiac orifice.

In the next year Brenner,¹ also in Vienna, published the second case. In this case both

hæmatemesis and melæna occurred before death, which did not take place for fully four days after the onset of the symptoms. The ulcer was again situated on the lesser curvature, 5 cm. from the cardia, but was of the size of the palm of the hand ("fast handtellergröss"). At the left base there were old pleural adhesions of rheumatic origin.

The third case, the first to be recorded in English, was that of Finny,⁴ in 1886. The ulcer was on the anterior wall of the stomach, 2½ inches from the cardiac orifice and 2 inches from the lesser curvature. The pericardial cavity had been obliterated by old rheumatic pericarditis. The perforation was direct into the left ventricle, about an inch from the apex. It was hidden by a musculus papillaris attached to the posterior surface of the mitral valve. There was a serous exudate in the left pleural cavity. There were adhesions between the pulmonary and diaphragmatic pleura and also between the stomach and the diaphragm. The bronchial glands showed caseation and some calcification. The stomach and the whole length of the intestines were full of fluid blood.

Brunniche² published the fourth case in 1887. His patient was a male, and the ulcer was on the lesser curvature. On the posterior surface of the left ventricle was an ulcer of the size of a shilling. The stomach contained a blood clot of the size of the fist. The ulcer was not covered by any membrane, there was no sign of any pus, and the heart itself did not contain any stomach contents.

In 1894 Ludwig Pick⁶ published a monograph on perforation of the diaphragm by gastric ulcer, and of 28 cases collected by him only 10, or 35·7 per cent., involved the pericardium. Of these 10 only the 4 I have referred to, or 14·3 per cent. of the whole, involved the heart muscle itself. His monograph makes it clear that in these cases a perigastritis is always present, and that it is *via* the most anterior portion of that part of the diaphragm on which the heart rests that the entry is gained. Of 10 of Pick's cases in which the ulcer was on the lesser curvature, 6 perforated the pericardium, 3 the left pleura, and 1 the mediastinum. Where the ulcer was (5 cases) situated in the fundus, the left pleura was always perforated. Of 3 in the cardiac region, 2 perforated the left pleural sac and the third the pericardium. All ulcers on the posterior wall perforated the left pleura, while the one case on the anterior wall involved the heart muscle. All were chronic ulcers with thickened and indurated edges, and their bases adherent to neighbouring organs, so that they were not able to cicatrise, contract, and heal up. As one would expect—taking into account the relationship of the pylorus to the liver—in spite of the frequency of pyloric ulcer, it was only met with 3 times in Pick's 28 cases of perforation of the diaphragm.

In my own case the subject was a woman aged 70. She went to bed as usual at 11 o'clock on a Sunday night. At 8 o'clock on the following morning she was found kneeling at the side of the bed, dead and cold. The bed had been slept in. The chamber utensil contained blood. I ascertained that she had not found it necessary to obtain medical advice for nine or ten months previously; the medical man she then consulted has been good enough to inform me that she complained of indefinite pains about the lower part of the chest and in the epigastrium, there being no physical signs of organic disease, either thoracic or abdominal. She was given one or two bottles of medicine by him, and as she did not consult anyone again presumably felt better.

I made a necropsy about 40 hours after death, in a very poor light and in very unsuitable surroundings. The body was covered with vermin. There was some oedema of the legs. The meninges of the brain were thickened and the convolutions atrophied. The pleural cavities were everywhere obliterated from old pleurisy, and the pericardium was also adherent throughout. The myocardium was pale and flabby and the mitral orifice incompetent. At the posterior part of the left ventricle, about half-way between the apex of the heart and the base of the ventricle, there was a small communication with the cavity of the stomach, through which a small glass rod could be passed, as shown in the figure. In this region the heart, pericardium, dia-

From a photograph showing the site of the perforation.

phragm, and stomach wall were firmly adherent together. It was quite impossible to separate any of these structures one from another. The left ventricle itself was quite empty; there was no sign in it of any stomach contents. On opening the stomach its cavity was found to be nearly filled by a large blood cast, but there was also some meat. The blood in the stomach was one large firm clot, but down the whole of the rest of the alimentary canal fluid blood was found. There were some perisplenitis and perihepatitis. The aorta showed well-marked atheroma. Dr. H. Heathcote has kindly made sections of portions of the heart muscle for me, and examined them for fatty changes. With regard to sections through the left ventricular wall he reports that there was an excess of leucocytes in the epicardium, that there was some fatty infiltration of the muscle, and a good deal of yellowish-brown pigment in the cells. In the papillary muscle he found some fragmentation of the myocardium, and (staining with charlach R and hæmalum) a diffuse purplish-pink

staining of the fibrous ends of the papillary muscle at the attachment of the chordæ tendineæ, probably indicating fatty deposit.

The ulcer of the stomach was of the size of a half-crown. Its edge was slightly raised owing to firm adhesions to the subjacent structures which had filled in the gap in the stomach wall by a false wall consisting of the structures which naturally lie between this point and the heart wall. Its floor was therefore deep and crateriform, and soft as compared with the true stomach wall. The exact position of the ulcer was on the anterior wall of the stomach near to the lesser curvature, the edge being an inch from the right edge of the cardiac orifice. The distortion of the stomach owing to the adhesions was very great, and in the photograph it has perforce been accentuated, owing to the difficulty experienced in bringing both the ulcer and the opening into the heart into suitable positions to show in the same view. The greater curvature has been turned upwards and to the left, while the lowest portion of the œsophagus and the cardiac orifice appear in the figure to the right of the ulcer.

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Manchester.

THE HEALTH OF BIRMINGHAM.—The medical officer of health's report for the third quarter of this year records a birth-rate of 26·8 per 1000, a slight increase on the rate in the same quarter of the two preceding years. The death-rate was 14·0 per 1000, a higher rate than in the corresponding quarter of some recent years, though lower than in any year prior to 1907. In 1912 the death-rate was 10·9; in 1911, 16·0; and in 1910, 10·9. Increases occurred in the death-rates from diarrhoea and enteritis, cancer, pneumonia, diseases of the heart, infantile debility, cerebral hæmorrhage, bronchitis, whooping-cough, non-pulmonary tuberculosis, Bright's disease, and premature birth, whilst decreases were recorded for measles, pulmonary tuberculosis, and old age. Eight of the great towns of England had a higher death-rate and seven a lower. The infantile death-rate was 161 per 1000 births as compared with 91 in 1912 and 238 in 1911. The principal cause of the increase was the prevalence of diarrhoeal disorders. Up to three months of age about 82 per cent. of the babies in Birmingham are breast fed, and the mortality among these from diarrhoea and enteritis was 4·7 per 1000, whilst among the bottle-fed babies or partly bottle-fed it was 95·5 per 1000. 2229 cases of scarlet fever were notified during the quarter, as compared with 1008 as the average in the third quarters of the years 1903-12; 259 cases of diphtheria were notified, the same number as the average; and there were 919 notifications of pulmonary tuberculosis. The elementary school teachers reported 89 cases of measles during the quarter, compared with 862 in the same period last year, and 587 cases of whooping-cough, compared with 232. Scarlet fever was more prevalent than in any third quarter for many years past. The type of disease was mild and the case mortality only 1·7 per cent. The incidence of the disease was more severe in the suburban than central districts. Of the 2229 cases, 1647 were removed to hospital. 762 patients who had applied for treatment for tuberculosis were examined, and of these 423 were recommended for admission to a sanatorium, 260 for dispensary treatment, and 47 for domiciliary treatment. The total of notified cases of tuberculosis numbered 1076.