30 months of the trial. Among the 13,200 tuberculin-negative participants who were not vaccinated, there were 64 cases of tuberculosis, a rate of 4.85 per 1,000. Among the 14,100 tuberculin-negative participants who were given B.C.G. vaccine, there were 13 cases of tuberculosis, a rate of 0.92 per 1,000. If a ruler is placed across the nomogram from 27,300 on the left-hand scale to 5.77 on the right-hand scale, it cuts the centre scale at the value 1.3 on the 5% side, and 1.7 on the 1% side. Since even the latter is exceeded by the difference in rates of 3.93, the difference is significant at the 1% level.

This nomogram is most accurate when the two samples are of the same size—that is, $m=n$. It is reasonably satisfactory up to a difference in size of 3 to 1.

III. Comparison Between Two Rates in the Same Population

Whereas the second nomogram dealt with two populations, each yielding a sample rate, the present nomogram is concerned with only one population in which, say, two independent conditions need to be compared. It is necessary, in the present context, to assume that no person is subject to both.

The left-hand scale m shows the size of sample; the right-hand scale shows the two rates added together, $P=p_1+p_2$.

If these points are joined by a straight line, the central scale is cut at a point which indicates whether the difference p_1-p_2 is significant or not. That is to say, if p_1-p_2 is greater than the value indicated on the 5% side of the central scale, it is significant at the 5% level, and similarly on the 1% side.

Example 1.—An investigation by McConnell, Pyke, and Fraser Roberts (1956) into blood groups in diabetes mellitus used, in the Oxford area, a control sample numbering 6,492, of whom 2,888 or 445 per 1,000, were group O, and 2,839, or 437 per 1,000, were group A. If a ruler is placed across the nomogram from 6,492 on the left-hand scale to 882 on the right-hand scale, it cuts the centre scale at the value 23 on the 5% side. Since the difference in rates is only 8, it does not reach significance. If it happens that the two rates add up to 1,000 per 1,000—that is, there are two alternative conditions and no remainder—it is possible, and indeed preferable, to use the first nomogram, above. The test is then whether the confidence limits to either rate overlap the value 500 per 1,000. If they do, the rates are not significantly different.

Example 2.—From the same source as Example 1 we find that 3,146 out of a control sample of 6,510 in the S.W. Lancs area were group O—that is, 483 per 1,000. Therefore 517 per 1,000 belonged to other groups. As in Example 1, we can compare these rates and find them just significantly different at the 1% level. Alternatively, using the first nomogram, the 99% confidence limits for group O are 467–499, which just exclude the value 500. (Of course, for the group not –O, the limits are 501–533).

In all three nomograms it is sufficiently accurate to use linear interpolation when a value is not marked on a scale.

Summary

Three nomograms are presented as an aid in assessing the significance of rates per 1,000 in the following situations: (1) providing confidence limits to

a rate; (2) comparing two rates based on different samples; and (3) comparing two rates based on distinct elements in the same sample.

In each nomogram, one outer scale shows the size of sample or combined samples, the other outer scale shows the rate or combined rates, and a straight line joining the two cuts the central scale at a point indicating the required limits or significance.

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[EDITOR'S NOTE.—It has been found impracticable to publish the nomograms here in a size that would make them really useful. They have therefore been separately printed full scale on art paper for those who are interested. Copies may be obtained from the Publishing Manager on application.]

Medical Memoranda

Plunging Parotid Tumour

The term "plunging goitre" designates a goitre which normally lies in the anterior mediastinum but which appears in the neck on deglutition. As is well known, the term comes from the French *plonger*, to dive or submerge, and the metaphor would be more apt if the phenomenon occurred in the reverse direction and a normally placed goitre temporarily disappeared. I have recently met with a similar phenomenon in a mixed tumour of the parotid gland, the temporary complete disappearance of which on two occasions gave rise to diagnostic difficulty amounting to bewilderment. Operation revealed a literal plunging explanation. I am not aware of any previous reports of this phenomenon in the parotid gland.

CASE REPORT

The patient, a man of 60, had a swelling in the region of the left parotid which had been slowly growing for five to six years. On examination there was a swelling in the lower pole of the left parotid, made more obvious by a thin face and neck. It was approximately 1½ in. (3.8 cm.) in its long diameter, moderately firm in consistency, smooth in outline, mobile, and apparently superficial. It thus had the typical clinical features of an expanding space-occupying lesion of the parotid, of which mixed parotid tumour is the commonest example, and this was the diagnosis made.

The only unusual feature was that a year previously another surgeon had made the same diagnosis and had operated with a view to removal of the tumour. At operation the tumour had completely disappeared and all that he found was the normal lower pole of the parotid, biopsy of which revealed normal parotid tissue with minor inflammatory changes. When the patient recovered from the anaesthetic, to the surgeon's natural chagrin the lump was noted to be back again and unchanged. After discussion with the surgeon, the only explanation I could suggest was that the lesion was a cyst which had ruptured during the operation and refilled immediately afterwards, though this seemed unsatisfactory. In any case, further operation with a view to removal of the lump was decided upon.

On July 17, 1962, under general anaesthesia the left parotid was exposed by the usual cosmetic cervico-facial incision, the scar of the original incision being excised. Shortly after the incision had been made and before any

deeper dissection had been carried out, the lump once more mysteriously disappeared without trace. Nevertheless in view of the previous experience it was decided to proceed with the original plan of removal of the lower half of the parotid. The trunk of the facial nerve was identified sufficiently in front of the stylo-mastoid foramen to preserve its blood supply (Patey and Moffat, 1961), and it was then noted that the nerve was unduly superficial and pursued a longer course than normal before dividing into its two main branches. The superficial parotid—that is, that part of the parotid superficial to the facial nerve—was normal except for fibrosis of the lower pole, presumably the result of the previous biopsy. The lower and posterior half of the superficial parotid was turned downwards from off the facial nerve and its branches. On pulling on the lower attachment of the superficial to the subfacial parotid a tumour of the latter with the typical macroscopical appearances of a mixed tumour reappeared rather in the manner of extracting a cork. After mobilizing the facial nerve, the subfacial parotid, including the tumour, was removed in continuity with the normal lower half of the superficial parotid. On recovery from the anaesthetic the patient showed no facial paralysis, and his post-operative course was uneventful. The pathological examination confirmed the diagnosis of mixed tumour.

DISCUSSION

In light of the operative findings the explanation of the unusual features of this case becomes obvious. This was an example of a mixed tumour developing in the deepest portion of the parotid, a tumour which ordinarily becomes imprisoned in the lateral pharyngeal space to give rise to a bulging of the palate and constitutes the dumb-bell tumour of Patey and Thackray (1957). Presumably in this case the tumour stretched the margins of the space to escape and present superficially. Under the influence of the muscular relaxation of general anaesthesia the tumour twice retreated to its site of origin and apparently disappeared. The case also emphasizes once again that an expanding tumour of the parotid, depending on its site of origin, may push the facial nerve in any direction, and that a tumour which clinically appears superficial may be deep to the facial nerve. The nerve as a result is much nearer to the surface than normal and is thus, unless the possibility is always borne in mind, more vulnerable than normal.

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"Food logistics was critical to the Greek armies during the Peloponnesian war (431 to 404 B.C.). The Athenians lost many decisive battles to the Spartans because of food habits. The battles during this war were conducted mainly on the sea. At mealtimes, the vessels would be beached and the soldiers would journey to the nearest town to buy food for a leisurely repast. On many occasions the Spartans took advantage of this routine. In one instance, the Spartans arranged to have meals for their soldiers at the beach ready for consumption. When the opposing fleets withdrew for mealtime, the Spartans ate quickly and returned to battle. The surprised, confused, and hungry Athenians hastily put out to meet their adversary and suffered major defeat. Much later and less than 200 years ago, the canning process was born out of the Napoleonic Wars when France offered a prize for a method of food preservation which would allow her navy to stay at sea for months and still be well fed. . . . The foods for to-morrow's combat soldier, sailor, and airman will look better, taste better, smell better, and be better preserved." (*New Report*, National Academy of Sciences, National Research Council, September–October, 1962.)

Fatal Marrow Aplasia Complicating Infectious Hepatitis

Major haematological complications in infectious hepatitis are rare, although leucopenia and thrombocytopenia are often seen. Aplasia of the bone-marrow as a result of virus infections is also rarely reported, and only two examples of this condition occurring in infectious hepatitis can be traced in the literature. In the following case fatal marrow aplasia occurred during the course of a typical attack of virus hepatitis.

CASE REPORT

A woman of 64 was admitted to hospital on August 11, 1960. For 10 days she had noticed anorexia, nausea, and the passage of dark urine. One week before admission the stools became pale, and on August 8 she noticed yellowness of the skin. Three days before admission a tendency to bruise was noticed. There was no history of transfusions, of injections, or of contact with any known hepatotoxic agent. She gave a history of angioneurotic oedema, exacerbated by aspirin and treated continuously since 1956 with an antihistamine, meclozine hydrochloride.

Examination on admission revealed icterus, bruising over the sacrum, and petechial haemorrhages over the knees. The liver was palpable half an inch (1.5 cm.) below the costal margin, but the spleen was not felt. The capillary fragility test (Hess) was negative. The haemoglobin was 13.3 g./100 ml. and leucocytes 5,000/c.mm., with a normal differential count. Platelets were 40,000/c.mm. and the bleeding-time (Duke) was 15 minutes. The one-stage prothrombin time (Quick) was normal. The direct Coombs (antiglobulin) test was negative and no platelet antibodies were detected by the mixed antiglobulin technique (Chalmers *et al.*, 1959). The Paul-Bunnell test was negative and no atypical mononuclear cells were seen. The serum total bilirubin was 5.2 mg./100 ml. and the direct van den Bergh reaction was positive. Alkaline phosphatase was 25 King-Armstrong units, thymol turbidity 4.5 units, zinc sulphate turbidity 4.5 units, and serum glutamic oxaloacetic transaminase 180 units. The serum total protein was 8.4 g./100 ml. and electrophoresis showed a reduction in albumin and increases in beta- and gamma-globulin. The urine showed a trace of protein and some bilirubin but no urobilinogen.

Further changes in the serum bilirubin and the haematological findings are shown in the Chart. The sternal marrow on August 18 showed minor abnormalities in the red-cell precursors, many of which contained Howell-Jolly bodies. The myeloid precursors were normal, but there was a decrease in megakaryocytes. A further examination on September 7 showed moderate numbers of red-cell precursors, but myeloid precursors were extremely scanty and no megakaryocytes were seen.

The patient was treated with a 40-g. protein diet and prednisolone was administered from the fifth day in a dosage of 40 mg. daily. The serum bilirubin fell rapidly after the administration of steroids but the petechiae and bruising extended and haematuria developed. A total of 6 pints (3.4 litres) of platelet blood, collected into plastic containers containing sequestrene, was given, with transient and decreasing effect. On September 26 the patient suddenly collapsed and died.

Necropsy Findings.—Apart from multiple petechial haemorrhages and ecchymoses the main finding was massive gastro-intestinal haemorrhage originating in the oesophagus or stomach. The cut surface of the liver showed slight fatty change but little disorganization of structure. Microscopically the liver showed well-preserved architecture but some infiltration of the portal tracts with chronic inflammatory cells. Appearances were compatible with the healing stage of a mild virus hepatitis. The marrow showed groups of erythroid precursors but almost complete absence of myeloid precursors or megakaryocytes.