fundamental surgical principle of adequate exposure and the recognition that identification of the facial nerve is (again apart from the highly malignant tumours) an essential part of such exposure. Having achieved adequate exposure, the surgeon is in the position to apply the fundamental surgico-pathological principle of treatment of tumours—removal with such margin as the pathology demands. He is also in the best position to preserve, whenever the pathology allows (which is in most cases), important structures such as the facial nerve. All the rest is merely a matter of detailed technique.

May I comment on one sentence in your leading article (4 November, p. 252) in which you refer to the advocacy of radiotherapy "for very frail patients [and] those with a recurrence after surgery. . . ." Operations for parotid tumour, though often technically tedious and therefore time-consuming, are not upsetting to the patient—certainly much less so than intensive radiotherapy. Moreover, though radiotherapy may be a valuable ancillary to surgery in the treatment of muco-epidermoid and adenoid-cystic carcinomas (contrary to what you say, the latter are radiosensitive), the cure by radiotherapy alone of any tumour of the salivary glands, primary or recurrent, is an exceptional rarity.—I am, etc.,

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Transient Cranial Mononeuropathy

SIR,—The interesting paper by Dr. J. N. Blau and Mr. R. Kapadia (4 November, p. 259) on isolated benign recurrent laryngeal nerve palsy brings to mind a similar problem I encountered earlier this year.

The patient, a 61-year-old man, had previously been quite well apart from mild chronic bronchitis. Two days before admission he developed a cough which was particularly troublesome at meal times. The next day he noticed difficulty in swallowing solids and over the course of a few hours the dysphagia became complete. Food seemed to stick in the back of his throat and was regurgitated with difficulty and occasionally nasally. Coughing was unimpaired but his voice had become slightly hoarse over this same period.

On examination there was impaired movement of the left palate. Laryngoscopy showed pooling of saliva in the left pyriform fossa and total immobility of the left vocal cord and hemilarynx. The gag reflex was intact. There were no other obvious neurological abnormalities and general examination was normal. The possibility of mediastinal malignancy was intensively investigated with negative results. In particular, chest x-ray, hilar tomography, bronchoscopy, oesophagoscopy, cerebrospinal fluid examination, and sputum cytology and culture were all normal.

During the first few days of the patient's illness aspiration pneumonia complicated the picture despite feeding via a nasogastric tube and frequent pharyngeal suction. This was effectively dealt with, and 10 days after admission the dysphagia began to improve and his voice became less hoarse. Two weeks later he was able to eat solids and speak normally and indirect laryngoscopy showed return of movement to the paralysed vocal cord. For the past nine months he has been seen frequently in the outpatient clinic. There has been no recurrence of symptoms and his general health seems good.

This man would appear to have suffered a benign cranial mononeuropathy involving the left vagus nerve. However, in this instance involvement was not confined to the

recurrent laryngeal branch and dysphagia was the most obtrusive symptom, with dysphonia a significant but less important accompaniment. One of the patients reported by Blau and Kapadia also had transient dysphagia, but its severity was not mentioned. The present patient showed another difference in that the onset of his symptoms was more acute and resolution more rapid. Unfortunately virus studies were not carried out and no valid comment about a possible viral cause can be made, though clinically there was nothing to suggest such an association.—I am, etc.,

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Haemolytic Anaemia with Nalidixic Acid

SIR,—The case of haemolytic anaemia due to nalidixic acid reported by Drs. L. Alessio and G. Marselli (14 October, p. 110) and the two other published cases cited by them^{1,2} seem to show a variety of haematological mechanisms. We wish to report a case with fatal outcome in which nalidixic acid would appear to have been the responsible agent.

A 65-year-old woman had complained of abdominal pain, vomiting, and dysuria for a few days, and on admission to hospital was profoundly anaemic and jaundiced. She had received nalidixic acid and prochlorperazine from the outset and after 48 hours had become icteric. There was a history of similar symptoms, ascribed to urinary tract infection, for the previous 18 months and on a number of occasions she had received courses of nalidixic acid. Her own doctor recollected that she had developed transient jaundice after the penultimate administration of the drug, three months before admission.

Laboratory investigation showed a haemoglobin level of 49% and W.B.C. of 27,300/mm³ with 94% polymorphs. Reticulocyte count was 4%. Red cells were spherocytic with marked polychromasia, and nucleated red cells accounted for 6% of the "nucleated cell" count. Direct Coombs test was strongly positive. Fibrinogen level was normal and the blood clotting ratio was 1.8 (normal 1.0). Schumm's test was positive and the serum glucose-6-phosphate dehydrogenase level normal. Serum total bilirubin was 12.6 mg/100 ml, with a direct reading of 1.4 mg/100 ml.

In spite of large doses of hydrocortisone, ampicillin, and kanamycin and blood transfusion the condition deteriorated, with peripheral circulatory failure and a rising blood urea, and she died 72 hours after admission. Permission for necropsy was not granted.

We feel that the autoimmune haemolytic anaemia in this case could be directly attributed to therapy with nalidixic acid. Certainly the rapidity of onset of this illness, shortly after commencing treatment with the drug, and the additional evidence of her general practitioner, who noticed jaundice following a previous course of therapy, would support this view. By analogy with "methyldopa haemolytic anaemia" duration of therapy seems to be an important factor, and in this case intermittent courses of the drug appeared to be responsible for the development of an autoimmune state. The previously reported cases have had relatively short exposure resulting in a "chemical" haemolytic anaemia, and in two of the three there was a deficiency of glucose-6-phosphate dehydrogenase. Experience with the present patient suggests that care should be taken in the long-term use of this drug, and

inquiry about jaundice connected with previous administration would seem to be a necessary precaution.—We are, etc.,

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¹ Bolton, E. M. and Vaughan Jones, R., *Lancet*, 1965, 2, 691.

² Mandal, B. K., and Stevenson, J., *Lancet*, 1970, 1, 614.

Acute Myeloblastic Leukaemia during Pregnancy

SIR,—Pawlinger and others¹ and Maurer and others² have published contradictory views on the effect of cytosine arabinoside and thioguanine on the fetus. The latter reported trisomy for group C autosomes when large doses were given in the second trimester of pregnancy. We would like to report a further case.

A 20-year-old woman was admitted to hospital in March 1972 complaining of tiredness, breathlessness, and increasing pallor. Her Hb was 3.6 g/100 ml, W.B.C. 33,600/mm³, with 86% myeloblasts and 1% myelocytes, and a platelet count of 25,000/mm³. Sternal bone marrow confirmed the diagnosis of acute myeloblastic leukaemia. Fetal movements had been felt and pregnancy was estimated to be 32 weeks. A modified but somewhat similar treatment regimen to that used by Guyer and others,³ was chosen because of difficulties in management and because of the gentler approach to therapy. Bearing in mind the possibility of chromosomal abnormality and the patient's religious objection to termination, she was given the following course of therapy as well as prednisolone 30 mg daily: days 1 to 5, cytosine arabinoside 80 mg/m² intravenously; days 6 to 11, rest period; days 12 to 17, thioguanine 70 mg/m² orally. Only two cycles of treatment were given between the 33rd and 37th week of pregnancy, at the end of which period the peripheral blood picture showed Hb 10.2 g/100 ml and W.B.C. 4000/mm³, with 2% myeloblasts and 2% myelocytes. The platelet count had risen to 105,000/mm³.

Labour was induced on 9 May 1972 and the patient had a normal delivery of a normal male infant weighing 2,948 g, whose appearance was consistent with a gestational age of 38 weeks. Venous blood on the first day showed Hb 14.1 g/100 ml, W.B.C. 23,000/mm³, with 73% polymorphs, 23% lymphocytes, 4% monocytes, and a platelet count of 140,000/mm³. Repeated estimations gave results within the normal range. A chest radiograph showed no thymus shadow. Blood urea and electrolytes were normal. Chromosome analysis showed a normal male karyotype with no breaks observed. After 48 hours the baby developed a typical physiological jaundice (maximum total bilirubin 13.5 mg/100 ml), which lasted eight days.

Nicholson⁴ reported that the risk of congenital abnormality in the fetus resulting from treatment after the first trimester was no greater than normal. Our cytosine-thioguanine regimen was chosen after discussion with Professor E. K. Blackburn because only small doses of the two drugs were required. We were unable to induce marrow remission in the mother but she remained well throughout the pregnancy. In the puerperium she developed an *E. coli* urinary tract infection with septicaemia which was resistant to intensive antibiotic therapy, and the patient succumbed. The baby, now 5 months old, is developing normally.

We are indebted to Dr. C. P. Alexander and Mr. J. A. Riedel, of Leicester Royal Infirmary,