Inflation of the chest with high intrathoracic pressures inevitably results in gastric distension with an unacceptable increase in the chance of vomiting. This problem may be overcome with the use of a cuffed endotracheal tube or perhaps an obturator airway in the oesophagus[7]—provided that an adequate seal is made with the mask at the face.

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Bronchial adenomas

Bronchial adenomas form a heterogeneous group accounting for somewhere between 1% and 6% of all primary lung tumours.[1-4] They are a continuing source of error and confusion in diagnosis. Though traditionally regarded as benign, more realistically they should be viewed as tumours of varying degrees of low-grade malignancy. Adenomas are derived from the duct epithelium of bronchial mucous glands and are usually either carcinoids or tumours of salivary-gland type.

Carcinoids account for about 90% of most series[4-6] and generally arise centrally in the trachea or main bronchi. Often they present with features of an endobronchial polyp, but extensive infiltration of surrounding lung is common. The characteristic features seen with gut carcinoids—cyanotic flushing, abdominal cramps, diarrhoea, oedema of the face and arms, and wheezing and dyspnoea—are rare with primary lung tumours, occurring in only about 2% of cases.[5] When the heart valves are affected by primary lung carcinoids the lesions occur in the left heart[7] rather than in the right heart, as with gut carcinoids and secondary deposits.

About two-thirds of the tumours of the salivary-gland type are cylindromas. Typically these, too, are central tumours, often encircling the airway and infiltrating extensively into surrounding lung. They are second only to primary carcinomas in incidence as tumours of the trachea. Most of the remainder of the salivary-gland tumours are mucoepidermoid adenomas. Pleomorphic adenomas, resembling mixed parotid tumours, are very rare.

Bronchial adenomas usually present for diagnosis at a much earlier age than carcinomas, commonly before the age of 50,[4 8] and the sex incidence is more nearly equal. Cough and haemoptysis are the first symptoms in around half the patients; often the chest radiograph is normal at that time. Another common pattern of presentation is with infection distal to obstruction by the tumour, causing lobar or segmental collapse and occasionally bronchiectasis, lung abscess, or empyema. Obstructive emphysema with overinflation of a segmental lobe may also occur distal to an adenoma. Peripheral adenomas tend to declare themselves much later and may be discovered as an incidental finding on chest radiography.

Early diagnosis of this group of tumours is essential if local destructive damage and distant metastases are to be avoided. Suspicion should be aroused in any patient with haemoptysis or recurrent chest infections, especially with a normal chest radiograph. Since almost all these tumours are visible at bronchoscopy this is the investigation of choice, but caution is necessary: many have a highly vascular stroma and bleed profusely on biopsy. If fibreoptic bronchoscopy is used an endotracheal tube or rigid bronchoscope must immediately be available to deal with bleeding.

Whenever feasible the treatment of bronchial adenoma is surgical.[4 8] Sleeve resection of the tumour may sometimes be possible, but lobectomy or even pneumonectomy may be necessary because of extensive spread. Favourable responses to radiotherapy have been reported with cylindromas regarded as unresectable.[9 10] Where the primary tumour can be resected the prognosis is good: prolonged survival is possible even with distant metastases, because these tumours grow so slowly.[11]

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Improving the surgical management of biliary atresia

For the infant with biliary atresia the only hope of prolonged survival lies in early diagnosis and effective surgery. The diagnosis must be considered in any infant in the first weeks of life who becomes jaundiced with dark, bile-containing (as opposed to normal, colourless) urine. Such infants always have some hepatobiliary disease; they have a high risk of intracranial bleeding from lack of vitamin K, and require urgent investigation to exclude treatable conditions such as galactosaemia or septicaemia. When these and other common genetic causes, such as alpha-1-antitrypsin deficiency, have been excluded and the stools lack bile pigment biliary atresia becomes a distinct possibility.

To what extent is biliary atresia treatable surgically? A review by Psacharopoulos and co-workers[1] of 47 consecutive infants operated on by one surgeon at King's College Hospital, London, from July 1973 to December 1978, reported 17 survivors aged between 3 and 65 months who were symptom-free with normal growth and normal serum concentrations

of bilirubin, though eight had had cirrhosis at the time of surgery. In three infants no procedure was undertaken because of advanced cirrhosis with portal hypertension and the absence of visible bile-duct remnants. In 15 cases patent bile ducts were identified at the portahepatis and anastomosed directly to the jejunum, but in only four such infants was bile drainage effective—and these were the only four in whom bile was found in the bile-duct remnants. The King's College Hospital group concluded that if no bile was found in the bile-duct remnants the alternative procedure, hepatic portoenterostomy, should be performed; in their hands this was effective in 13 of 29 infants. In this radical operation, first reported by Kasai in Japan in 1959,[2] the bile-duct remnants and surrounding fibrous tissue are transected at their junction with the liver capsule, and bile drains directly into a loop of jejunum anastomosed to the area. Though the procedure flouts the surgical principle of having mucosa-to-mucosa anastomosis, these infants develop bile-stained stools in one to 15 weeks with the serum concentration of bilirubin returning to normal values one to 26 weeks after surgery.[1]

Such results with hepatic portoenterostomy are similar to those reported by Altman[3] and approach the 55% success rate reported by Kasai.[4] They fall well short of Kasai's highest reported short-term success rate of 90% in patients operated on by 60 days of age[5] but then the average at referral of cases was 10 weeks and extended up to 34 weeks. Though five of the 12 infants operated on at King's College Hospital after 12 weeks are currently well and have clearly benefited from surgery, the best reported five-year survival figure of 36% (again from Japan)[6] is found in those operated on by 60 days of age, falling to 16% in those operated on between 90 and 110 days and to nil with later surgery. The condition of 84 survivors aged more than 5 years reported in a comprehensive review of the Japanese experience of the surgery of biliary atresia[7] is encouraging. All are engaged in normal activities for their age and have no symptoms of hepatic disease, although 31 have enlarged livers and 14 enlarged spleens. With good bile drainage hepatic fibrosis regressed and portal hypertension rarely caused symptoms.

The attitude of Japanese and North American paediatric surgeons to the management of biliary atresia has also been analysed in this recent report.[7] A detailed study of 500 Japanese infants with biliary atresia cared for by 53 surgeons in 22 centres was compared with the results of a questionnaire completed by 60 of 70 paediatric surgeons in North America and Canada with a declared interest in the condition. Japan has developed regional centres for the treatment of biliary atresia in which 95% of the operations would be done by one surgeon —and it was in these centres that the best results were achieved. In North America the overall results were less satisfactory since, with one or two notable exceptions, such concentration of experience had not occurred. The lessons of the Japanese experience must surely be applicable to other parts of the world, including Britain.

In spite of these encouraging observations major unsolved problems remain. Postoperative cholangitis is the most feared complication, not only because of the acute illness it causes but also because each attack adds to the liver damage and makes cirrhosis more likely. The cause of these attacks of cholangitis is not entirely clear. Japanese surgeons have introduced over 20 complex surgical techniques aimed at minimising contact between the bowel flora and the porta hepatis in attempts to reduce its frequency. These operations add to the surgical morbidity and mortality of this procedure, and have not been shown to reduce the incidence of cholangitis below the 45% reported in the King's series, using a relatively simple Roux-en-Y technique. In over half the infants surgery still fails to achieve bile drainage and a jaundice-free state, and over half such infants will die from cirrhosis by 2 years of age.[8]

Earlier consideration of the diagnosis and earlier referral would reduce the toll from biliary atresia, but family doctors and paediatricians should also be aware of the tremendous emotional strain caused by the disease and its complications, highlighted in a report from Denver, Colorado.[9] A study of 24 families with affected infants showed a very high incidence of severe marital discord and divorce, even one abandoned child, as well as unemployment and severe financial pressures. Families in Britain may be spared the more severe financial pressures experienced in the United States, but clearly need much help and support.

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Chronic fluorosis

Roholm's classic monograph[1] on chronic skeletal fluorosis appeared in 1937, and little has been added to the clinical picture since then. The initial diagnosis is usually based on the characteristic radiographic changes: an increase in the density of spongy and other bones. Symptoms are usually confined to back stiffness and vague pains in the joints. Less commonly, tendons, ligaments, and muscles may be infiltrated by mineral deposits, and periosteal outgrowths of exostotic bone may occur in various forms. The fluoride concentration in the blood may be slightly raised; phosphate and, usually, calcium concentrations remain within normal limits. When the source of the excess fluoride is airborne the lungs may be damaged, causing respiratory symptoms. Changes in hormonal and enzyme systems have been described, which may be associated with the non-specific symptoms occasionally reported—headache, reduced appetite, vertigo, and nausea. Teeth may be discoloured if excess fluoride is ingested during the period of development of the enamel.

Fluorosis may be caused either by ingesting or inhaling relatively large amounts of fluoride. Usually fluoride is rapidly absorbed, producing a temporary rise in its concentration in the blood: only a small amount appears in the faeces. The raised concentration in the blood quickly subsides, since half or more of the ingested fluoride is excreted in the urine, while the