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Tests of Hearing in School

Hearing acuity is usually tested by subjective audiometry, using pure tones or speech. Objective testing has until recently required techniques inapplicable in routine clinical practice. Recently, however, a test based on determination of acoustic impedance by the electro-acoustic impedance bridge (loosely termed "impedance audiometry") has been developed into a useful clinical tool. What is measured is the resistance or impedance to transmission of sound in the middle-ear transformer mechanism. Normally this mechanism is very efficient, and most of the sound energy is transmitted into the cochlear fluids, very little being reflected by the ear drum. The impedance audiometer simply measures the amount of sound energy reflected when a standard low-frequency tone is presented to the ear. In the normal ear this is small: the middle-ear has a low impedance to sound transmission. Acoustic impedance measurements give valuable information on the function of the middle ear, while considerably more information may be obtained about the cochlea, the acoustic nerve, and the higher auditory pathways by studying the stapedial reflex response.

One of the most common abnormalities of the middle ear is serous otitis media, in which the middle-ear cleft becomes filled with fluid. The resulting deafness may lead in children to backwardness at school, while otologists are becoming increasingly concerned with the possible long-term effects of this condition.

In Britain all children over 9 months old are (in theory) screened for congenital sensorineural deafness. A further screen is performed on school entry using sweep audiometry at a level of 25dB. This latter test has come under increasing criticism.[1 2] Its accuracy is impaired by problems with equipment and test conditions and by technical difficulties in performing the test; and, most important, it fails to detect those children with serous otitis media whose hearing level for pure tones is better than 25dB. These children can often be diagnosed by specialist examination and full pure tone audiometry, but they do not always reach a specialist as the parents may be unaware that the child's poor scholastic achievement is the result of a mild hearing loss. These children can be detected and so given early treatment and cure if the school-entry screening test is done with an impedance audiometer.

Many audiologists have advocated the wider use of impedance audiometry,[3] and draw attention to its advantages. Its sensitivity presents some problems, as the test will detect the abnormal middle-ear function that may persist for up to three months after an attack of acute suppurative otitis media, and the results of impedance audiometry are best assessed by specialist consultation. Further problems encountered in this form of screening are the time and technical skill needed to perform the test. Until recently the school screening programme was under the care of the local education authorities, and the staff employed had a variable degree of training in audiometry. Under the reorganization of the Health Services these programmes have come under the care of the area health authorities, and discussions are at present being held on the training of the staff. It is important that adequately trained staff should be made available to operate these programmes.

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Liver Tumours and the Pill

Despite the recent all clear on safety given to the pill there is no doubt that a variety of metabolic abnormalities and adverse side effects may follow its use, albeit rarely. One possibility is that oral contraceptives may cause hepatic neoplasia, though a definite cause-and-effect relationship has yet to be established. In 1973 Baum *et al.*[1] described seven women with benign hepatic adenomata, all of whom were taking oral contraceptives, and four other similar cases have since been described in isolated reports.[2-5] At p. 7 of this issue Dr. J. P. O'Sullivan and Mr. R. P. Wilding report the first three cases from Britain. Thirteen of these women had been taking oral contraceptives for at least two years and usually longer, but one developed symptoms after only six months. Such information as is available suggests that benign hepatic adenomata are extremely rare,[1 6] so that these 14 cases collected over a period of five years represent a striking increase in incidence.

Histologically the lesions are benign and well demarcated from surrounding liver tissue, being composed of nodules of liver cells separated by fibrous septa. Bile duct reduplication was prominent in the cases described by O'Sullivan and Wilding, in contrast to some other reported cases.[1] Most of the tumours have been markedly vascular with small dilated blood vessels. A similar but not identical picture, with phlebectasia and blood cysts, is found in peliosis hepatis, a rare condition associated with a variety of systemic diseases and also with administration of gonadal steroids.[7 8]

Because of their vascularity these tumours are especially apt to bleed, and nine of the reported cases presented with acute abdominal pain and shock from this cause, with four fatalities. Out of the remaining five, four presented with an abdominal mass discovered either by the patient or her doctor, and in one the tumour was an accidental finding at cholecystectomy. O'Sullivan and Wilding recommend elective resection because of the high incidence of life-threatening haemorrhage.

How oral contraceptives might produce hepatic neoplasms is a matter for speculation. Their long-term use in healthy asymptomatic women has been shown to lead to ultrastructural changes in the hepatocytes with hypertrophy of the smooth endoplasmic reticulum and mitochondrial abnormalities, in the absence of changes on light microscopy.[9] These changes may be important, for hepatic enzymes which metabolize

foreign compounds in the body are situated on the smooth endoplasmic reticulum, and stimulation of their activity—the process of enzyme induction—is associated with hypertrophy of this organelle. Enzyme indction may potentiate the carcinogenicity of certain compounds, probably by increasing their conversion to toxic metabolites,[10] and it has been suggested that oral contraceptives might act in this way.[11] Progestogens are known to have enzyme inducing properties,[12] and, though oral contraceptives contain only a small amount, the subclinical cholestasis which occurs in a high proportion of women who take the pill[13] could result in an accumulation of these gonadal steroids, and perhaps also of carcinogenic compounds, within the liver. This would explain the long latent period between starting oral contraceptives and the onset of symptoms. Closely structurally related to the synthetic progestogens are the anabolic steroids, methyltestosterone and oxymetholone, and recently cases have been described of hepatocellular carcinoma, sometimes with peliosis hepatis, complicating their long-term administration.[14 15] In one case there was regression of the tumour when the steroid was withdrawn. Alternatively, the oestrogen component of the pill could be to blame, for the carcinogenic potential of oestrogens in other organs is well recognized,[16 17] and small doses of oestrogens have been shown to promote liver regeneration in rats.[12] Conceivably they could lead to the hamartomatous malformations described if given over a long period of time.

The link between benign liver tumours and oral contraceptives cannot be regarded as proved, however, until their actual incidence in women of childbearing age has been determined, and it would be unwise to jump hastily to conclusions based on anecdotal evidence, however persuasive. Furthermore, the prevalence must be very low in relation to the millions of women in the world who are taking the pill, though now that more attention is being drawn to the existence of a possible association it is likely that more cases will be recognized.

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Diet and Coronary Heart Disease

Some of the confusion about which dietary measures—if any—will help to prevent coronary heart disease (C.H.D.) arises from the complexity of the disease: diet is by no means its only cause, and we still lack conclusive direct proof that any dietary change will reduce its incidence in man. Yet doctors and the public need some clear provisional guidelines, which should be based on the judgement of experts after sifting the large volume of circumstantial evidence.

For this reason the last six years have seen recommendations on measures to prevent C.H.D. published by five bodies[1–5] in the U.S.A., two [6 7] in New Zealand, a combined Scandinavian[8] board, committees in Australia[9 10] and in Holland, and by the International Society of Cardiology.[11] Some of these concluded that persons at increased risk of C.H.D. should be identified and treated; others made recommendations for the whole population. Some considered risk factors other than diet, including smoking, hypertension, and lack of exercise, while other bodies were set up specifically to advise on diet. To this collection of official recommendations the Department of Health and Social Security has now added the report of an Advisory Panel of the Committee on Medical Aspects of Food Policy, published last month.[12] The panel had been asked to evaluate the evidence on the relation between diet and cardiovascular disease and its recommendations are for the population as a whole.

The report makes five recommendations. Firstly, obesity should be avoided or treated, in both children and adults. Secondly, most members of the panel thought the amount of fat in the British diet, especially saturated fat, should be reduced. But they unanimously agreed that they could not recommend an increase in the intake of polyunsaturated fat as a measure to reduce the risk of C.H.D. They further recommended that the consumption of sucrose should be reduced "if only to diminish the risk of obesity." Lastly they advised caution in any proposals to soften the water supply in any part of the country. The chairman, Sir Frank Young, is to be thanked for guiding the panel to almost complete agreement in this first official advice on diet and C.H.D. in Britain. There is one note of reservation in the report: Professor J. Yudkin considered that dietary sucrose has a specific role in the pathogenesis of C.H.D.

The report stated that the order in which the recommendations were listed did not denote any ranking of their importance. Readers of the report should not miss this point, because the epidemiological evidence[13–16] shows that obesity is rather a weak risk factor for C.H.D., and there has been no primary prevention trial of weight reduction. The panel did not suggest what dietary components people should eat in place of the reduced saturated fat intake, nor did they explain why they took such a firm line against its partial replacement by polyunsaturated fat, which is recommended in other countries[1 3–5 7 9 10] and has been shown to be a practicable way to reduce plasma cholesterol in Britain.[17 18] Since the report is intended for food manufacturers and the Ministry of Agriculture, Fisheries, and Food as well as for doctors, it may have the effect of further increasing the price of polyunsaturated margarines (which are not subsidized) relative to butter (which is). It is also likely to discourage introduction here of the new ingenious methods of producing polyunsaturated beef and dairy products.[19]

Sadly, the panel did not include a recommendation that salt intake should be moderated. This could hardly do any harm in healthy people, and the F.A.O./W.H.O. Expert Committee has advised that the amount of salt in baby foods should be limited.[20] Some of the newer evidence on the relation between dietary sodium and blood pressure has probably appeared since the main meetings of the panel.[21] So too has a careful review by the National Food Survey, which shows that people in the upper income group in Britain now obtain more of their dietary energy (calories) from fat than from total carbohydrate.[22] Since 1955 the proportion of fat in the diet