16% in those with rheumatoid arthritis alone. The more widespread recognition of this syndrome may distinguish a group of patients in whom extra therapeutic caution is required.

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Idiopathic Cardiomyopathy in Infants

The term "cardiomyopathy" was introduced by Brigden in 1957[1] to describe isolated non-coronary myocardial disease of obscure aetiology. Over the succeeding years the term has been employed to include almost any cardiac disease but restriction is clearly necessary.[2] It has also been suggested that terms "primary" and "idiopathic" should be dropped,[3] and that the term "cardiomyopathy" should be reserved for disorders of heart muscle of unknown cause or association.[4]

The cardiomyopathies can be subdivided clinically into two major types: congestive and hypertrophic (with or without obstruction), and two rarer types, constrictive and obliterative.[5] By definition the aetiology is unknown, and much research has been concentrated on elucidating it. Viral infection has been suggested as a possible cause, particularly of the congestive type, but proof has often been lacking.[6] M. B. Gardner and colleagues[7] successfully demonstrated virus-like particles in a patient suffering from cardiomyopathy. Many reported cases of cardiomyopathy have been described in adults, but to gain insight into the disease process and to find a cause much can be learned from focusing attention on newborn babies and infants because of their relatively short history.

Under the title "idiopathic cardiomyopathy" R. Doshi and K. V. Lodge[8] recently described the cases of nine infants. Their ages varied between 14 days and 9 months; there were four girls and five boys. Two boys were siblings. The common mode of death was circulatory failure (five patients), or sudden collapse (three patients), and in four instances was accompanied or precipitated by bronchopneumonia. At necropsy the hearts showed no abnormality other than dilatation and hypertrophy of all chambers. In three cases mainly the left ventricle was hypertrophied, in two cases mainly the right, and in the remaining four patients equal hypertrophy of right and left ventricle was found.

Histological examination of the myocardium showed irregular swelling and distortion of fibres often widely separated from one another, with occasionally amorphous pink-staining material between them. Some fibres were vacuolated, others necrotic, and still others showed a perinuclear halo which contained periodic-acid-Schiff-positive material. Fibrosis or inflammatory cell infiltrates were absent.

Immunofluorescent tests for bound gammaglobulin showed none in any of the cases. Degenerative cellular changes in the large ganglia were noted, but similar changes were observed in 10 out of the 11 control patients who died from causes other than cardiomyopathy. These changes were interpreted as not being due to virus disease[8] but possibly due to terminal anoxia. This interpretation was strengthened by the fact that these changes in the ganglia were more recent than the myocardial changes, and also they were absent in a further group of six infants of the control series who did not die in heart failure. Viral cultures of necropsy material were undertaken in six of the nine infants, but all were negative. In three of four mothers there were significent antibody titres in the serum against viruses which included mumps viruses, measles, influenza A, adenovirus, influenza, and rubella. No antiheart or antinuclear antibodies were detected in the mothers' sera.

As the disease appeared so early in life, an intrauterine viral cause was a possibility.[8] It was also postulated that the myocardial damage might be caused by a complex immune mechanism resulting from virus or virus antibodies crossing the placenta. In older infants viral infection may have occurred after birth, the maternal antibodies being sufficient to suppress clinical symptoms but not sufficient to prevent damage to the infant's myocardium. On the whole the evidence suggested a viral cause for non-familial infant cardiomyopathy.[8]

It could just be possible that these cases represent a heterogeneous group of cardiomyopathies. The mode of death of some infants, the occurrence of similar cardiac conditions in two siblings, and the histological description of vacuolation and perinuclear haloes containing P.A.S.-positive material[9 10] are reminiscent of the hypertrophic type of cardiomyopathy. One may speculate (and it has been established in a few instances) that the changes which characterize this condition in older children and adults may take time to develop.

This recent contribution,[8] like many previous ones, does not show absolute proof of a viral aetiology of cardiomyopathy, but strong suggestive evidence does exist. Viruses can cause myocarditis—for example, Coxsackie virus,[11 12] measles,[13] mumps,[14] and influenza.[15] The transition from incompletely resolved myocarditis—not necessarily viral in origin—to cardiomyopathy has only rarely been substantiated.[16] Even the presence of raised viral antibody titres is difficult to interpret. G. F. Fletcher and colleagues[17] investigated serum levels of viral antibody (to Coxsackie virus B1-6, echovirus 6 and 9, influenza A and B, mumps, herpes simplex, and psittacosis) in adult patients with primary myocardial disease. These were compared with control subjects. No difference in the titres between test patients and controls was shown. But previous viral infection may be no longer reflected by raised antibody titres and could therefore not be ruled out as the cause of primary myocardial disease.[17]

Thus there is often persuasive evidence that a viral cause seems likely in some cases of cardiomyopathy. The results are frequently difficult to interpret, and further study is necessary to furnish unequivocal proof that viruses are causally related to certain types of cardiomyopathy.

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Scotland's Drink Problem

Addiction to alcohol is one of the largest and most neglected health problems of our time. It is particularly severe in Scotland, so that any attempt to tackle it there deserves a sympathetic hearing.

Last year the Departmental Committee on Liquor Licensing (chairman, Lord Erroll of Hale) reported on licensing questions in England and Wales,[1] and its main proposals were criticized in these columns[2] as untimely and hazardous. Now the Departmental Committee on Scottish Licensing Law (chairman, Dr. C. Clayson) has issued its report for Scotland.[3] Like the Erroll Committee's report, it has many recommendations that lack medical implications, but some of those that might affect the public health deserve critical scrutiny and are indeed disappointing. For this report also recommends some relaxation of the present restrictions on access of the public to places where alcoholic drinks are sold.

As the report points out, hospital admissions for alcoholism in Scotland according to recent figures were seven times as high for men and five times as high for women as south of the Border, while recorded death rates for alcoholism were six times as high for men and twice as high for women. Convictions for public drunkenness are over 2·5 times as high in Scotland, and death rates for hepatic cirrhosis about 1·5 times as high. No one of these indicators is free from scientific objection, but taken together the evidence overwhelmingly indicates that alcoholism constitutes a considerably more serious problem north of the Border than in the South.

It is in this context that the report's proposals must be judged, for it recommends several relaxations in the licensing law to the end that they might "help to promote civilized drinking and break down the attitude that regards the consumption of liquor as an end in itself." The introduction of a separate type of certificate is recommended for suitable premises which sell light refreshments such as soft drinks, tea, coffee, and snacks so that they could also sell alcoholic drinks. There would be no restriction on the access of children to premises of this kind, though under a minimum age limit they would not be allowed to buy alcoholic drinks. That age the report recommends should be retained at 18. A further recommendation is that so-called "children's certificates" should be issued. "*Where the conditions are right* (report's italics) we see no reason why a child should not be present in a bar in a hotel, a restaurant or even a public house in the company of his family," it says. And in pursuance of this aim holders of such certificates would be allowed to admit children to any part of their premises thought to be suitable, "including parts used mainly or exclusively for the sale and consumption of exciseable liquor, namely, bars." It should be left entirely to local judgement to decide which premises are suitable for the admission of children under 14, states the report.

Such proposals must raise the question whether corner cafes are to become places where drinks can be on sale throughout the day. Would the number of "refreshment house" certificates be limited in any given area? Further, to allow young children into places where alcohol is being drunk may be an encouragement to "civilised drinking," but to many people there would seem to be risks that ought not to be run. Though statistics in this field are notoriously difficult to interpret, it is worth noting that the annual convictions for drunkenness in Scotland of persons aged 14 to 18 rose between 1966 and 1971 from 273 to 445. Consequently, if the report's recommendations are put into effect, there is no doubt that another of its proposals should be taken up seriously, and that is the introduction of a system of monitoring to measure the incidence of the misuse of alcohol.

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Prognosis in Cystic Fibrosis

Cystis fibrosis of the pancreas is one of the commonest lethal genetically determined diseases in north-western and central Europe or among people coming from there. Estimates of its frequency vary from 1 in 2,300 to 1 in 3,000 live births.[1-4] It appears to be rare in African and Far Eastern races.

It was first clearly separated from coeliac disease by G. Fanconi and his colleagues[5] in Zurich in 1936 and by Dorothy Andersen[6] in New York in 1938. In both studies the identification of the disease was based on an examination at necropsy of the gross and histological changes in the lungs and pancreas. The mortality in the early years after its recognition was high and the likelihood of survival beyond childhood was small. But the prognosis has steadily improved, mainly owing to the establishment of special clinics, the introduction of more effective antibiotics, the use of the mist-tent and inhalation therapy, and an appreciation of the importance of physiotherapy. Yet it remains one of the most distressing and disabling conditions affecting children and adolescents and imposes the most exacting demands on patient and parents.

To determine an accurate prognosis in individual cases presents problems. Statistical probabilities are of little help to the worried parent. Comparison between the results in older series and present-day ones are useless, for treatment has improved and ascertainment is more complete, so that many mild or even symptomless cases with diagnostic levels of sweat sodium are now included which would have been missed previously. Another factor impeding comparisons between old and recent series is that mass screening of the newborn is now done in some areas. This means that improved results are likely to be due to the early start of treatment rather than any alteration in the treatment itself.

Consequently, attempts have been made to construct a prognostic scoring system that can be applied to each individual patient and also be used to assess progress. A standard scheme of evaluation of patients was introduced by H. Shwachman and L. I. Kulczycki[7] and modified by C. M. Doershuk and colleagues[8] for use at all ages. A simplified scheme was suggested by E. M. Cooperman and colleagues.[9] Recently Lynn Taussig and colleagues[10] have produced a scoring system based on pulmonary function tests (FEV_1 and vital capacity), chest radiographs, chest symptoms such as acute infective episodes, pneumothorax, haemoptysis, sputum production, and cough, with the addition of scores for physical examination of the lungs and the presence of cor