The local organization is to be based on hospital meetings, and these will elect the regional committees. The larger regions will have bigger regional committees who, in turn, will appoint a larger number of representatives on the Group Council.

These new arrangements give ample scope for democracy to work. The Hospital Junior Staffs Group Council should be able in future fairly to claim that it represents all hospital junior doctors. Its executive committee is to appoint 12 members of the Central Committee for Hospital Medical Services, and this committee is in future to be responsible, through a Negotiating Subcommittee, for remuneration and all terms of service for all hospital doctors.[2] Three of the Central Committee's nine representatives on the Negotiating Subcommittee are to be hospital junior staff members.

Hospital junior doctors will not only have more opportunity to make their voice heard centrally and round the negotiating table but also more power, and therefore more responsibility, in shaping the future of the hospital services as a whole. It would surely be better for them first to try out their new power alongside their senior colleagues rather than independently of them and, sometimes needlessly, in opposition. The medical profession has enough to contend with without also having contention in its own ranks.

Segregation of Tuberculous Patients

Removal of patients with infectious pulmonary tuberculosis from their homes in an attempt to protect the family has been generally accepted as a sound preventive measure. This policy now needs reappraisal.

How effective and necessary is segregation of patients receiving modern chemotherapy, which rapidly reduces infectivity? This question has been investigated in Madras by the Tuberculosis Chemotherapy Centre. It has reported a five-year controlled study of the attack rate of tuberculosis among close family contacts of patients with newly diagnosed infectious pulmonary tuberculosis.[1] The contacts lived in poor, overcrowded conditions and all had been living, feeding, and cooking with the patients for at least three months before the diagnosis was made. None of the contacts were given B.C.G. vaccination or chemoprophylaxis. Attack rates were studied in family contacts whose only infectious member was initially the index case. A comparison was made between 272 "sanatorium" family contacts, whose index case was isolated in a sanatorium for one year, and 256 "home" contacts, who remained exposed to the index case during treatment at home. The study covered five years. Both home and sanatorium patients received isoniazid and sodium P.A.S. (sodium para-aminosalicylic acid) for one year. By the end of it in 86% of the home and 92% of the sanatorium patients the disease was bacteriologically quiescent.

During the five years' follow-up tuberculosis developed in 9% of the home and 14% of the sanatorium contacts, including 5% and 7% respectively in the first year. About one-third of the contacts were initially tuberculin-negative. Among these the attack rate of tuberculosis over the five years was 10% in the home series and 11% in the sanatorium series, including 8.1% and 8.0% respectively in the first year. The corresponding figures for the initially tuberculin-positive contacts were 9% and 16% over the five years and 3% and 7% in the first year. Thus the risk of developing tuberculosis was no greater for the home contacts than it was for the sanatorium contacts either over the whole five-year period or even in the first year.

The sanatorium contacts were exposed to the risk of infection from two sources—firstly, from the patient before the diagnosis of tuberculosis had been made, and, secondly, from other sources in the urban community in which the family lived. The home contacts were exposed to both these risks and in addition to that of continued contact with the patient during home treatment. Exposure to the patient during treatment at home was not an important risk, since the attack rate was similar in the tuberculin-negative contacts in the two groups, while in the tuberculin-positive contacts it was actually higher in the sanatorium group—apparently by chance. The main risk to the contacts seemed to be from exposure to the patient before diagnosis. The continued contact with the patient at home during treatment was little extra hazard.

This demonstration that well-organized domiciliary treatment is practicable, effective, and safe for family contacts is of great importance in developing countries with shortage of sanatorium beds.[2,3] In Britain facilities for domiciliary treatment are good, yet many physicians admit almost all the sputum-positive patients to hospital for several months. This policy can hardly be justified on the grounds of risk to family contacts, for it proved to be unnecessary in the overcrowded conditions of Madras. The risk is likely to be even less in Britain, where patients can often be isolated in their own home. Moreover, B.C.G. vaccination and chemoprophylaxis are freely available for suitable contacts and are known to be effective in preventing tuberculosis.[4,5] It is thus reasonable to conclude that, although there may be several good reasons for treating pulmonary tuberculosis in hospital, admission cannot be regarded as essential to prevent infection of the family contacts of co-operative patients from good homes who can be given well-supervised domiciliary chemotherapy.

Oedema in Pregnancy

Many clinical interpretations have been put on oedema in pregnancy. The relationship of this physical sign to gain in weight and to pre-eclamptic toxaemia is not fully understood. Nor is it clear to what extent oedema means an increase in the total amount of water in the body or a change in the distribution of water between plasma, tissue fluid, and intracellular fluid.

Since these matters have probably been oversimplified, a recent study by A. M. Thomson, F. E. Hytten, and W. Z. Billewicz[1] is of special interest. They examined the records of 24,079 women living in Aberdeen who had given birth to single, legitimate babies. From the records they were able to recognize women who had no oedema, those with oedema of the legs only, and those with generalized oedema—that is, elsewhere than in the legs and ankles. They also classified the women as normotensive or hypertensive (diastolic pressure

[1] Kamat, S. R., *et al.*, *Bull. Wld Hlth Org.*, 1966, **34**, 517.
[2] Fox, W., *Lancet*, 1962, **2**, 413 and 473.
[3] —— *Brit. med. J.*, 1964, **1**, 135.
[4] Medical Research Council, ibid., 1963, **1**, 973.
[5] Ferebee, S. H., and Mount, F. W., *Amer. Rev. resp. Dis.*, 1962, **85**, 490.

of 90 mm. Hg or more), and as being pre-eclamptic when there was proteinuria in addition to hypertension.

In the normotensive women oedema, mainly of the legs, increased with age, being present in 14% of those under 20 years of age and in 29% of those over 30. Preliminary evidence suggested that women who had been oedematous in one pregnancy were twice as likely to develop oedema in a subsequent pregnancy as those who did not have this sign. Women too heavy for their height at 20 weeks of pregnancy were more likely to have oedema, usually of the generalized type, than those of average weight or less. If overweight women gained weight at the rate of 1.2 lb. (545 g.) or more per week from 30 to 36 weeks of pregnancy, there was a 42% chance that they would have generalized oedema and a 24% chance that they would develop oedema of the legs. When the rate of weight gain was less, so was the chance of developing oedema. It should be emphasized that these were the changes found in normotensive women.

When the blood pressure rose above 90 mm. Hg the incidence of oedema, particularly of the generalized form, rose too, and such generalized oedema was even more likely when there was proteinuria as well. Oedema of some kind was present in 35% of normotensive women, 65% of hypertensive, and 85% of those with pre-eclampsia as defined. It is perhaps worth noting the considerable numbers of women who did not have clinically recognizable oedema even when they had hypertension with or without proteinuria.

In general, women with oedema tended to have larger babies than those who did not have oedema, though the difference was only 0.17 lb. (77 g.). The prematurity rate was, on the average, less when there was oedema, especially of the legs, while the perinatal mortality rate was the same whether there was oedema or not. The conclusion the authors reach is that "Oedema, in the absence of other evidence of pre-eclampsia, is associated with a slight advantage in terms of birthweight and incidence of low birthweight, and is not associated with any disadvantage in terms of perinatal mortality. To that extent, it is possible to affirm that oedema in pregnancy is not necessarily harmful and unphysiological." This careful investigation therefore begins to put oedema in perspective.

Hytten, Thomson, and N. Taggart[2] investigated total body water in pregnancy. In 93 patients at least three readings of total body water were made in pregnancy and one 6 to 8 weeks after delivery, all the patients being clinically normal. The mean body water gained in pregnancy amounted to 8.5 kg. Between 10 and 38 weeks of pregnancy the gain in body water was 6.84 kg. in women with no oedema, 7.19 kg. in those with oedema of the legs, and 9.80 kg. in those with generalized oedema. It seems probable that those without oedema held between 1 and 2 litres of water in the extracellular spaces. The women with generalized oedema would appear to hold a further 3 litres somewhere which cannot be accounted for in the products of conception, the uterus, or elsewhere. Such women can be essentially normal, being healthy themselves and having normal babies. If the weight of the total body water gained is deducted from the total gain in weight, the "dry weight" gained can be estimated. When this is done it is found that overweight patients put on less "dry weight" than do those who are of average weight for height or underweight. This "dry weight" is probably mainly fat when it cannot be accounted for in other known factors of the total weight gain. The conclusion seems to be that those women who have large fat stores already do not store much extra in pregnancy, while those with small fat stores lay down some more fat.

Hytten and Thomson[3] measured total body water in six patients who developed pre-eclampsia as shown by a rise in blood pressure, proteinuria, and some oedema. The amount gained was within the limits found in normal women without pre-eclampsia, though the rate of gain of water in the last ten weeks of pregnancy was faster than in normal women with generalized oedema.

It is still not easy to assess the significance of oedema in the individual pregnant patient, but this work shows that age, oedema in a previous pregnancy, site of oedema, weight-for-height ratio, blood pressure, proteinuria, and rate of weight gain may all have to be taken into account. Oedema of itself would not seem to be invariably a bad prognostic sign and indeed might signify a slightly better prognosis for the baby when all other factors are considered. This work helps to clarify the relationship between oedema and increase in total body water in pregnancy, but the difference between normal and pre-eclamptic pregnancy in terms of water metabolism is still conjectural.

Investigating Hypertension

In general the significance of a patient's renal lesion to his hypertension is difficult to decide. For instance, renal-artery stenosis may occur with[1] or without[2] hypertension. Thus there is still no single reliable investigation for selecting those hypertensive patients with unilateral renal disease who may be expected to benefit from surgery.

The help of various complex inpatient investigations is sought before resorting to surgical operation. They include aortography, possibly with renal-function studies of each kidney, assay of pressor substances in systemic or renal circulation, angiotensin-infusion tests, studies of renal blood flow, and bilateral renal biopsy. These are formidable tests to put any patient through, and it is not easy to find agreement on which patients should be subjected to them. H. W. Smith[3] estimated the incidence of curable renal hypertension to be at most 2% of all hypertensive patients. This fairly gloomy picture is confirmed by M. J. Chamberlain and J. A. Gleeson,[4] who found that in a series of patients subjected to aortography the number permanently benefited by surgery was unduly small in relation to the morbidity of the investigation and later operation. But a higher proportion of cases of correctable renal hypertension has been reported by M. E. DeBakey and colleagues[5] and E. F. Poutasse.[6] A compromise figure, 5–10%, of renovascular hypertension has been proposed by M. H. Maxwell and G. B. Prozan,[7] who thought that the great majority of such patients could benefit from surgery. In Britain renal-artery stenosis has been reported in 10% of hypertensive patients at aortography,[8] and the surgical treatment of carefully selected patients has certainly been rewarding.[9 10]

Viewed against this background any advance in the accuracy of outpatient screening tests to select patients for more complex inpatient studies would be welcome. If the

[1] Thomson, A. M., Hytten, F. E., and Billewicz, W. Z., *J. Obstet. Gynaec. Brit. Cwlth*, 1967, 74, 1.
[2] Hytten, F. E., Thomson, A M., and Taggart, Nan, ibid., 1966, 73, 553.
[3] —— —— ibid., 1966, 73, 714.