group which acts as an oxygen-activating unit in cytochrome oxidase. Unfortunately this theory too falls foul of the facts, which have an unpleasant habit of invalidating all the theories about this disease as fast as they are put forward. As S. B. Osborn and J. M. Walshe[11] pointed out earlier this year, it is possible for a patient with Wilson's disease, treated with pencillamine, to live a perfectly normal life with no symptoms of disease and with no detectable copper or caeruloplasmin in his serum—always provided that the drug is continued and the patient maintained in copper balance. So this would suggest an excretory function for caeruloplasmin. But this theory comes up against the work of the Albert Einstein group of workers,[12] who have shown, at least for rabbits, that the copper of catabolized caeruloplasmin "is not directly or preferentially excreted by hepatic, pancreatic, intestinal, or renal routes." Blue blood may be all very well in preventing Wilson's disease, but the way in which it mediates this function is as obscure as its role in ennobling the nobility.

Obstructive Airways Disease

The clinical features of the bronchial and emphysematous types of obstructive airways disease have been defined by A. C. Dornhorst[1] and T. Simpson[2] and their physiological characteristics by C. Ogilvie[3] and by W. A. Briscoe and A. Cournand.[4] The careful and detailed studies of C. M. Fletcher, B. Burrows, and others[5 6] firmly established the criteria whereby these two types can be distinguished. These workers have also tackled the semantic problem posed by the indiscriminate use of the terms "chronic bronchitis" and "emphysema," the first being favoured in Britain and the second in America. Using standardized clinical, radiographic, and physiological techniques, they compared 50 patients from a "bronchitis" clinic in London with 50 patients attending an "emphysema" clinic in Chicago.[7] Apart from a rather greater incidence of disabling chest illness in London, there was no real difference in the pattern of obstructive airways disease for the two cities. In particular, the frequency of the bronchial and emphysematous forms was the same.

N. L. Jones, Burrows, and Fletcher[8] have now extended this study by carrying out an annual review of the original 100 patients over a period of three years. During this time progress and mortality were remarkably similar in the two cities, but the incidence of disabling chest illness was higher among the London patients. However, a detailed questionary showed that bronchitic exacerbations were not in fact more frequent in London than in Chicago but only more disabling in terms of the time spent in bed or off work. The authors attribute this to differences in working conditions, sickness benefits, and cost of treatment between the two cities rather than to differences in the behaviour of the disease process itself.

This recent survey also gives valuable information on the relative prognosis of the two types of obstructive airways disease. The mortality was higher in the bronchial (36%) than in the emphysematous group (15%), and this was related to the greater incidence of hypercapnia among the bronchitic patients. It might have been thought that the complications of bronchitis (infection, hypercapnia, and right heart failure) would prove more amenable to treatment than the irreversible lung changes of emphysema. The actual finding of a much higher death rate for bronchitis surely points to the need for a reappraisal of therapeutic methods used in this formidable disease. The thorough techniques of investigation evolved by Fletcher and his co-workers could well provide the basis for a study of this kind.

[1] Dornhorst, A. C., *Lancet*, 1955, **1**, 1185.
[2] Simpson, T., *Tubercle (Lond.)*, 1958, **39**, 307.
[3] Ogilvie, C., *Thorax*, 1959, **14**, 113.
[4] Briscoe, W. A., and Cournand, A., *Ciba Foundation Symposium on Pulmonary Structure and Function*, ed. A. V. S. de Reuck and M. O'Connor, 1962, p. 304. Boston.
[5] Fletcher, C. M. Hugh-Jones, P., McNicol, M. W., and Pride, N. B., *Quart. J. Med.*, 1963, **32**, 33.
[6] Burrows, B., Fletcher, C. M., Heard, B. E., Jones, N. L., and Wootliff, J. S., *Lancet*, 1966, **1**, 830.
[7] Fletcher, C. M., Jones N. L., Burrows, B., and Niden, A. H., *Amer. Rev. resp. Dis.*, 1964, **90**, 1.
[8] Jones, N. L., Burrows, B., and Fletcher, C. M., *Thorax*, 1967, **22**, 327.

Realism and Addicts

The Ministry of Health is facing increasing criticism because of its alleged dilatoriness in responding effectively to the threat of epidemic heroin addiction in Britain. A brief inquiry published by the *Guardian*[1] last week certainly gives a picture more of disarray than preparedness so far as hospital services are concerned, while a speaker at a recent meeting of the General Medical Services Committee[2] denounced "a typical Ministry paper scheme," which he alleged was simply putting forward proposals which could not be implemented owing to lack of facilities. To what extent are such strictures justified?

The latest "paper scheme" is a memorandum dealing with the rehabilitation and aftercare of heroin addicts.[3] The G.M.S. Committee has already drawn attention to the inappropriateness of the use of the allocation procedure to place an addict on a general practitioner's list, but otherwise the ideas put forward in this memorandum deserve nothing but praise. It presents a humane and intelligent approach to the problem of drug addiction, stressing that detoxification by itself is not enough, and picturing narcotic addiction as requiring comprehensive integrated services—clinics, hospitals, hostels, work-training—rather than mere drug-handout centres. The memorandum supplements the model for hospital treatment which was put forward earlier this year.[4]

The trouble comes when turning from the Ministry's memoranda to the realities. Those in Alexander Fleming House seem to have some of the characteristics of the kind of student who writes alpha papers but muffs the practicals. To take an example: one of the most important proposals in the new circular concerns the role of hostels in the aftercare of addicts, and the benefits that can result from grouping patients are discussed with a degree of sympathy and insight which is admirable: "Although the establishment of a disciplined atmosphere is essential this alone is insufficient to prevent drug taking. It is necessary to build up an atmosphere of co-operation between staff and residents and in this task the psychiatrist can give valuable assistance. . . . The expectation of the hostel must not be set too high and staff must accept the probability that relapse will take place and be ready to contend with its effects on other residents and themselves." A splendid answer in a written paper, but then take the example further and look at the performance in the practicals.

[1] *The Guardian*, 16 November 1967.
[2] *Brit. med. J. Suppl.*, 1967, **4**, 47.
[3] *Ministry of Health Memorandum*, H.M. (67) 83.
[4] Ibid., H.M. (67) 16.
[5] *Brit. med. J.*, 1967, **3**, 692.
[6] Ibid., 1967, **4**, 366.

How many hostels for addicts of the sort which the Ministry envisages are now functioning in London? Not one. Who is going to pay for these no doubt very expensive facilities? The Ministry makes hopeful murmurs about local health authorities footing the bill, but anyone with experience of that method of financing will know that ratepayers are always eager for new ventures to be sited in any borough but their own. Where are the staff of the hostels to be found? These and every other question which bears on reality meet with no answer.

The earlier suggestions on hospital care were of course similarly worthy and invited a volley of equally unanswered questions. Where are the consultant sessions to be found for the new treatment centres? Are new psychiatrists to be manufactured, or will they be withdrawn from other services? What training courses have been arranged? What laboratory facilities are being provided? Will extra psychiatric social workers be hired? How much money will be spent?[5]

It would not be fair to place all the blame on the Ministry for failing to find an immediate solution to this extremely difficult problem. To a large extent the solution (and responsibility) lies in the hands of local health authorities and hospitals, general practitioners and voluntary bodies: imagination, energy, and good organization are needed in the many local communities where drug addiction is established or beginning to be seen. All the blame may not be the Ministry's, but it is reasonable to ask for more practical action and more evidence of a sense of urgency. In 1954 there were 57 known narcotic addicts, while in contrast the present official count is 1,349.[6] With a situation as threatening as this today's minor blunders, procrastinations, or meanness are the stuff out of which tomorrow's disasters are likely to be made.

Surfer's Nodules

All that some people ask is not a tall ship and a star to steer her by but a board that measures ten feet by two, has a core of polyethylene foam with springers of spruce or redwood, is jacketed in fibreglass and resin, and coated in paraffin wax to provide adhesion for its rider. J. G. Erickson and G. R. von Gemmingen[1] have described some of the hazards of surfboarding as it is practised in Southern California. It sounds much less comfortable than riding on a dolphin's back. The devotee "paddles in the kneeling position with his feet extending back and his body weight balanced on the tibial prominences, the middle of the top of the feet, and the dorsum of one or more metatarso-phalangeal joints . . . over months of surfing, at four to six hours a day." In this attitude he travels seaward in search of the perfect wave upon whose back he can ride shoreward in triumph and delight.

In these days of increased leisure it is becoming harder to draw any sharp distinction between work and sport as an occupation on the one hand and as a relaxation on the other. Indeed, some people would say that the point has been reached at which work has become a relaxation from the obsessive pursuit of a hobby. The surfers of Southern California seem a case in point. They pay for their devotion with nodules, ulcers, ganglia, and bony changes—attributable mainly to the effects of long-continued pressure—and, more hazardously, by taking the chance of being run down by their colleagues in the open sea. Though the pressure effects are avoidable by temporary abstinence or by spreading the load, there is little enthusiasm for avoiding what is acknowledged to be a badge of distinction, since "the prestige of the surfer is proportional to the size of his nodule." "Abstinence," said Blake, if his shade will forgive this use of his words, "sows sand all over the ruddy limbs and flaming hair, but desire gratified plants fruits of life and beauty there." It seems unlikely that modern Britons will try to rule the waves so obsessively, if only because Pacific rollers offer better sport, but even so we could perhaps find examples of surfer's nodules upon our own shores, and students of occupational marks would no doubt like to add this new piece to their collections.

[1] Erickson, J. G., and von Gemmingen, G. R., *J. Amer. med. Ass.*, 1967, **201**, 134.

Platelets in Uraemia

The tendency of patients with advanced uraemia to bleed is associated with a defect in their platelets.[1-3] The more severe this abnormality in the platelets, the more marked are the clinical manifestations of bleeding.[4 5] Thrombocytopenia may also occur, particularly in patients with acute renal failure, but the platelet count is normal in most patients with chronic uraemia. The impaired function of the platelets is manifested by an increase in the bleeding time, defective platelet adhesiveness, reduced platelet aggregation in the presence of adenosine diphosphate, impaired clot retraction, reduced prothrombin consumption, and poor availability of platelet factor 3. The earlier report of A. J. Hellem and his colleagues[6] that intravenous infusion of urea prolonged the bleeding time and reduced platelet adhesiveness has not been confirmed by P. A. Castaldi, M. C. Rozenberg, and J. H. Stewart.[7 5] Substances other than urea are now presumed to cause the functional platelet defect in uraemia.

Recently, Stewart and Castaldi[7] have reported that platelet function in uraemia was considerably improved by dialysis (either peritoneal or haemodialysis), though complete correction—particularly of the bleeding time and platelet aggregation—was not achieved. The clinical improvement in haemostasis paralleled that in platelet function. Dialysis usually improved clot retraction, prothrombin consumption, and platelet adhesiveness. In most patients there was a rise in the platelet count after dialysis. Improvement in platelet function was found as early as one day after dialysis, and occurred in two cases with little fall in the level of urea in the blood. Successful renal transplantation was more effective than dialysis in restoring completely normal haemostasis.

Stewart and Castaldi used measurements of platelet aggregation, availability of factor 3, and clot retraction to estimate platelet function in vitro. No adverse effects were observed after the addition to plasma rich in platelets of urea, dextrose, mannitol, creatinine, urate, phosphate, potassium, or magnesium; or after changes of *p*H or osmolarity within the range which might be found in severely uraemic patients.

[1] Lewis, J. H., Zucker, M. B., and Ferguson, J. H., *Blood*, 1956, **11**, 1073.
[2] Cahalane, S. F., Johnson, S. A., Monto, R. W., and Caldwell, M. J., *Amer. J. clin. Path.*, 1958, **30**, 507.
[3] Cheney, K., and Bonnin, J. A., *Brit. J. Haemat.*, 1962, **8**, 215.
[4] Willoughby, M. L. N., and Crouch, S. J., ibid., 1961, **7**, 315.
[5] Castaldi, P. A., Rozenberg, M. C., and Stewart, J. H., *Lancet*, 1966, **2**, 66.
[6] Hellem, A. J., Ödegaard, A. E., and Skälhegg, B. A., in *Proceedings of the Xth Congress of the European Society of Haematology*, in Basle, 1966.
[7] Stewart, J. H., and Castaldi, P. A., *Quart. J. Med.*, 1967, **36**, 409.
[8] Horowitz, H. L., Cohen, B. D., Martinez, P., and Papayoanou, M. F., *Blood*, 1967, **30**, 331.