with busy junior medical staff than with nurses, who have formal training and operate a double checking system. Administration of intravenous drugs by nurses not only reduces the workload of senior house officers but should also improve the quality of care.

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Protection of the individual in biomedical research in Africa

EDITOR,—Progress in biomedical science is often due to more and more complex experimentation on humans. The limits of this research need to be defined in terms of science and ethics. Biomedical research in African countries, as in many developing countries, is often carried out without a legal basis: in many African countries there is no legislation on scientific research. Western countries have done, or are doing, their best to adopt legislation about scientific research practice that respects the individual^{1,2}; can Africa stay away from these fundamental changes?

Each African country should have a national ethics committee, and at the top there should be a federal bureau of inter-African ethics and of protection of the individual along the lines of the Organisation for African Unity; this federal bureau should work in close collaboration with the regional bureau of the World Health Organisation. The advantages for the African countries would be considerable: common aims, exchanges of materials and research workers, interchanges of ideas, and stimulation of research in general. Foreign partners wanting to make biomedical assays in, or to conduct experiments on, humans in Africa would have to seek authorisation from the inter-African bureau before submitting these projects to national African ethics committees. These African countries would be the copromoters of the biomedical research carried out there. For big projects the WHO, with the agreement of the inter-African bureau, should stand as co-promoter and assume its obligations in these often impoverished African countries. Foreign investigators should obtain in their home countries the authorisation necessary to carry out such biomedical research in foreign countries.

Although many patients in some African countries may not have the educational background needed to be really informed, informed consent remains necessary. Investigators should adapt to cultural and educational realities in these countries. For biomedical research to be better organised in African countries we need to foster openness and improve ethical standards to better protect the individual.

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Effect of albumin on diuretic treatment in the nephrotic syndrome

EDITOR,—Fehmi Akcicek and colleagues have addressed the question whether albumin has an additive effect on the diuretic treatment of patients with the nephrotic syndrome.¹ We have published similar results, but besides plasma volume expansion we also studied the effect of different plasma and urinary albumin concentrations.² The same plasma volume expansion of about 20% was achieved by infusing either 40 g of albumin or 36 g of dextran 70 (Macrodex, Pharmacia AB, Sweden) in five nephrotic patients on separate study days. The plasma albumin concentration increased from 28 g/l to 33 g/l after albumin and decreased to 23 g/l after dextran. Urinary albumin excretion was unchanged after dextran but increased by 70% from 3.9 to 6.6 g/24 h after albumin. Dextran caused a considerable increase and albumin a small increase in renal blood flow (para-aminohippuric acid clearance). The glomerular filtration rate (inulin clearance) did not change after albumin or dextran infusions.

Both modes of volume expansion caused a moderate increase in the rate of urinary flow but not in the rate of excretion of sodium. The renal sensitivity to frusemide was the same after dextran and albumin. Thus, even within the pathophysiological range of urinary albumin excretion, frusemide binding to urinary albumin does not seem to impair its diuretic effect.

After 24 hours the plasma volume and plasma albumin concentration had almost returned to initial values with all protocols. Moreover, rebound effects of frusemide were seen four to 20 hours after the dose, irrespective of plasma volume expansion. Thus the additive (24 hour) diuretic and natriuretic effects of plasma volume expansion and increased plasma albumin concentration to an intravenous injection of 40 mg of frusemide in nephrotic patients were small and short lasting and thus of negligible clinical importance.

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Suppression of medical conditions by Cushing's syndrome

EDITOR,—Alan Steuer and colleagues describe the unusual occurrence of a medical condition (sarcoidosis) that was suppressible by steroids and presented shortly after surgical treatment of Cushing's syndrome.¹ The corollary of this—namely, that a pre-existing medical condition may be suppressed by the onset of Cushing's syndrome,

reflecting the increased output of corticosteroids—is also true and may help diagnosis.

The case of a 66 year old man who presented with diarrhoea and had been found to have advanced medullary carcinoma of the thyroid gland 11 years earlier illustrates this point. At recent review he complained of polydipsia and polyuria. Several weeks before he had noted that his persistent psoriasis had entirely resolved. (It had been troublesome for over 50 years and had been only partially controlled by dithranol and beta-methasone preparations. Since 1976 it had been associated with arthropathy, for which he required intermittent non-steroidal anti-inflammatory drugs.) Further questioning and examination elicited fatigue with proximal myopathy and a plethoric face but no excess pigmentation. Blood pressure was 120/70 mmHg. Investigations confirmed diabetes mellitus associated with Cushing's syndrome (potassium concentration 2.7 mmol/l, bicarbonate >40 mmol/l, serum cortisol persistently >1660 nmol/l, urinary free cortisol 3120 nmol/24 h, and adrenocorticotrophic hormone 267-316 ng/l (normal 7-51 ng/l)). Carcino-embryonic antigen and calcitonin concentrations were appreciably increased. His Cushing's syndrome responded to metyrapone. He became increasingly pigmented and continued chemotherapy with cisplatin and fluorouracil for his metastatic medullary carcinoma of the thyroid, which was the presumed source of his adrenocorticotrophic hormone.

The onset of this patient's Cushing's syndrome was clearly rapid; he had not developed some of the more typical signs of the condition, such as thin skin and easy bruising, and had not developed excess pigmentation. Despite his metabolic upset and symptoms he considered the clearing of his psoriasis to have been an early and prominent feature. We suggest that it may be worth actively seeking such information from patients with suspected Cushing's syndrome, particularly when the diagnosis does not seem clinically obvious, in the hope of identifying the condition at an early stage. The suppression of pre-existing medical conditions may help to identify the time of onset of hypersecretion of glucocorticoid.

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Lord Moran's prescriptions for Churchill

EDITOR,—In 1992 when my biography of Lord Moran was published, I divulged no information from medical records that had not already been publicly mentioned.¹ In describing Moran's management of Churchill's illnesses the constituents of pills that were prescribed—which various writers, including Churchill himself, referred to by nicknames such as "Morans" and "minors"—remained unidentified.

Some of the drugs have become of interest to medical historians, especially in light of recent advances in knowledge of the treatment of the atherosclerotic disease that would have been the basis for Churchill's repeated cerebrovascular episodes. I therefore sought permission from the present Lord Moran, who owns his father's records, and from Sir Winston Churchill's surviving child, Lady Soames, to identify the various medications. Both have kindly consented.

From 1940 onwards, Moran prescribed "reds" to promote sleep. "Reds" were quinalbarbitone