this work was virtually ignored for 40 years, I think at least he deserves the courtesy of a reference.

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¹ Clarke, J K. *British Journal of Experimental Pathology*, 1924, 5, 141.

Oculocutaneous reactions to beta-blocking drugs

SIR,—The "alternative explanation" produced by Drs P M Gaylarde and I Sarkany (16 August, p 435), while plausible, does not in fact stand up to critical evaluation. In the first place there have been very few reports of the oculocutaneous reaction in patients on any of the beta-blocking agents apart from practolol, and most of those in whom oxprenolol or propranolol has been incriminated had also received practolol in the past. Ten of the 21 patients reported by Felix *et al*¹ subsequently received oxprenolol and four were given propranolol and suffered no adverse effect, although 12 of the group were challenged with practolol and developed reactions.

The clinical features of the oculocutaneous syndrome, the development of a positive antinuclear factor test, and the irregularity of the occurrence of this condition are very much in favour of an immunological rather than a pharmacological mechanism being involved. Not that they are mutually exclusive. However, the evidence adduced in favour of a possible pharmacological mechanism is faulty.

It is unfortunate that the term "psoriasiform" has stuck to the cutaneous component of this condition. It is also lichenoid, erythematous, and eczematous at times. Histologically, the lesion shows evidence of intraepidermal cell death and there is not the typical hypertrophy of psoriasis. One certainly cannot assume that increased epidermopoiesis is playing an important role in the pathogenesis of the epidermal disorder. While the importance of the cyclic nucleotides as regulators of cell division in some systems cannot be denied, their role in the control of epidermopoiesis has not as yet been firmly established. Our own experience with phosphodiesterase inhibitors and that of others with other compounds active in the adenylyl cyclase—cyclic nucleotide system as depressants of epidermopoiesis and as clinically effective agents in psoriasis has been disappointing.

There would appear to be good evidence for the involvement of immune mechanisms in the pathogenesis of the oculocutaneous syndrome and very little in favour of a pharmacological mechanism.

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¹ Felix, R H, Ive, F A, and Dahl, M G C, *British Medical Journal*, 1974, 4, 321.

SIR,—We read with great interest the case report by Mr R B Cubey and Dr S H Taylor (8 November, p 327) of an ocular reaction to propranolol and its subsequent resolution on continued treatment with a different beta-blocker. We have held for four years a special clinic for patients with coronary arterial disease and have seen no oculocutaneous reactions to propranolol, nor

indeed to timolol maleate (Blocadren). Routine ophthalmic examinations have been carried out in the clinic for the four years, and in the event of any suspicion of an adverse reaction the patient is then screened by the ophthalmologists in the normal manner.¹

There was no reference in the case report to the autoimmune status of the patient at the time that the ocular reaction was found or subsequently. We believe that determination of antinuclear factor may detect early adverse reactions, and other workers have found the antinuclear factor (ANF) titre to be elevated.^{1,2} There may well be a place for screening patients before starting treatment with the newer beta-blockers and indeed, since Mr Cubey and Dr Taylor's report, also those on long-term propranolol therapy.

We have found no significant ANF titre in 12 anginal patients who have been on timolol continuously for four years. Similarly, they have had no oculocutaneous reactions.

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¹ Wright, P, *British Medical Journal*, 1975, 1, 595.
² Raftery, E B, and Denman, A M, *British Medical Journal*, 1973, 2, 452.

Enteric-coated aspirin overdose and gastric perforation

SIR,—Dr R J Farrand (15 November, p 409) suggests that I am simplistic and jump to immediate conclusions. To the contrary, I still do not have sufficient information even to understand the "facts" in their apparently clearcut case. May I put some unanswered doubts on paper?

(1) The patient clearly ingested 67 Safapryn tablets. To assess whether an 83-year-old patient with a 16-g overdose of paracetamol would behave like some of the rather younger patients in the paper he quotes one needs to know that there was no biochemical evidence of previous hepatic or renal disease or that these organs were histologically normal at post-mortem and showed no evidence of paracetamol toxicity.

(2) Besides the six different drugs mentioned in the paper, the post-mortem report specifies an additional four: propoxyphene (in Distalgesc), digoxin, frusemide, and effervescent potassium as well as the bile-stained silver threepenny bit found in the stomach as mentioned by Dr F J A Bateman (15 November, p 409). If the patient, while in hospital, was able to accumulate 67 Safapryn tablets, recognised by their residues, how did Dr Farrand and his colleagues exclude the others, all soluble in the stomach? He gives evidence only for phenylbutazone in his letter.

(3) From Dr Bateman's letter I realise that the point of the report was that sufficient aspirin may be leached out to cause local irritation despite an apparently intact coat. Did Dr Farrand check the aspirin content of the original capsules or look for salicylate after his elegant experiment with Safapryn tablets, simulated gastric juice, and a 1913 silver threepenny bit? Such a simple test would have proved or disproved his theory.

(4) Dr Farrand excluded phenylbutazone as a cause of gastric ulceration as there was no history of phenylbutazone intolerance. Those who use aspirin, phenylbutazone, indomethacin, etc, regularly know only too well that peptic ulcers or gastric perforation may occur without any previous intolerance.

Dr Farrand asks "How much room for speculation is there in a 'short report'?" That

of course depends on the editor, but the authors must ensure that sufficient facts are included to allow readers to understand the authors' conclusions.

Even with Dr Farrand's amplification, I feel the original report belongs to the apocrypha of aspirin toxicity, and in my book it is firmly inscribed as the tale of the bile-stained silver threepenny bit.

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Nutrition rehabilitation units

SIR,—Nutrition rehabilitation (NR) is a practical form of health education aimed at the mothers of malnourished children (1 November, p 246). Wherever such children are being treated, particularly in the paediatric wards of teaching, city, and district hospitals, the management is incomplete without a practical educational component to combat the high recurrence rate.¹ An associated NR unit is not a luxury for it can greatly increase the effectiveness of these children's wards.

Several further points should be noted. An important additional benefit of NR is the practical nutrition training for all members of the medical, nursing, and supporting staff. Too often their nutrition instruction has been only in theoretical terms of the major dietary components, megajoules (Calories), international units of vitamins, etc. Many staff members have never seen such figures in actual volumes of local foods. If they have to spend a period of in-service training in NR units they are better equipped not only to manage malnutrition but also to advise mothers about feeding children in a realistic way.

Both the day-care and residential forms of NR have limitations because no country with large numbers of malnourished children can provide enough NR centres to care for all who need help. In this context domiciliary NR as developed by Professor P M Shah is worthy of consideration.²

Evaluation of the effect of NR is complicated because of multiple factors which influence nutrition in an individual family or community. Currently an attempt is being made to test a questionnaire that will help the staff of NR units to assess the effectiveness of their own work.³ The results of follow-up after rehabilitation are much more encouraging than those after standard hospital treatment!⁴ (see table).

Two groups of workers have specifically studied the evaluation of NR.^{5,6} These were both retrospective studies, but Beaudry-Darisme and Latham examined experimental (nutrition-rehabilitated) and control groups of children in Haiti and Guatemala. At follow-up in Haiti the weight-age of the experimental group was significantly higher than that of the control group. This was not the case in Guatemala and this was partly attributed to undue emphasis on the feeding and not enough on maternal education. However, the mortality in the experimental group was much lower than in the control group in both countries—4% and 14% respectively in Haiti and 1% and 9% respectively in Guatemala.

A practical educational component is

Nutrition rehabilitation: numbers of children¹

Nutritional status (weight for age)	Admission	Discharge	Follow-up
Adequately nourished (>80%, Harvard mean)	0	1 (1%)	1 (1%)
I* malnutrition (71-80%, Harvard mean)	4* (3%)	10 (7%)	34 (24%)
II* malnutrition (61-70%, Harvard mean)	20* (14%)	45† (32%)	60 (43%)
III* malnutrition (<61%, Harvard mean)	116 (83%)	84† (60%)	45 (32%)
Total	140	140	140

*With oedema. †Without oedema.

essential in the management of protein energy malnutrition. Some form of nutrition rehabilitation can provide this, but modifications are necessary in different areas and controlled prospective evaluation would clarify the real benefits.

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- 1 Cook, R. *Journal of Tropical Pediatrics and Environmental Child Health*, 1971, 17, 15.
- 2 Shah, P M. *Journal of the Indian Academy of Paediatrics*, 1971, 8, 805.
- 3 Cole-King, S M. personal communication, 1975.
- 4 Cutting, W A M. *The Child in the African Environment*, pp 387-397. East African Literature Bureau, 1975.
- 5 Beaudry-Darisme, M. and Latham, M C. *Journal of Tropical Pediatrics and Environmental Child Health*, 1973, 19, 299.
- 6 Beghin, I D. and Viteri, F E. *Journal of Tropical Pediatrics and Environmental Child Health*, 1973, 19, 404.

SIR,—In the attempt to salvage serious cases of malnutrition it is only the most myopic doctors who can persist with standard clinical methods without being concerned with the wider aspects of the problem. But for those medical and health workers who have tried to look beyond the sphere of pure clinical treatment the extent and complexity of the problem rapidly become apparent.

I am afraid that the immediate appeal of nutrition rehabilitation, referred to in your recent leading article (1 November, p 246), is as a palliative for many frustrated workers. It removes the problem one stage away from the precious paediatric ward, where malnourished children not only take up valuable space but frequently die despite the most intensive measures or take an inordinate time to recover sufficiently to be discharged. Nutrition rehabilitation units also often depend heavily on gift-food handouts, another favourite palliative for the headache of malnutrition. Centres of this kind give some relief to the donors but are of doubtful value in tackling the real problem.

It is easy when considering nutrition rehabilitation to be distracted by the "centre" with all its problems of structure, staffing, etc and miss the *process*, which must follow right back to the homes and communities from which the malnourished children have come and prove to be successful *there*. The problems vary not only from country to country but for the various cultural, social, and family groups within a community. A nutrition rehabilitation programme must carefully analyse each problem and test means of coping with it before including it in any teaching. Blanket nutrition teaching, which is the all too frequent pattern, is as dangerous and irresponsible as the indiscriminate use of antibiotics without a medical diagnosis.

Doctors must face the unavoidable reality that the chances of survival and full recovery

for a malnourished child and the future for further children depend largely on the means and ability of the parents to provide adequate care. Although health services may play a part in tackling the problems of malnutrition, the root causes are nearly always social, economic, or political, and as such must be dealt with at that level. It is an unfortunate irony that developments such as the "green revolution," although they have increased overall production, have widened the gap between the rich and the poor and have not resulted in equitable distribution. The mechanisation, irrigation, and fertiliser levels that are necessary can be afforded only by the wealthy and, being capital-intensive, reduce the number of jobs, causing further unemployment and poverty.

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Dietary fibre hypothesis

SIR,—On behalf of my co-editor, Mr Denis Burkitt, at present abroad, and the contributors to our book,¹ now published, we welcome Mr C L Copeland's letter (15 November, p 404) asking the Department of Health Committee on Medical Aspects of Food Policy to assess the dietary fibre hypothesis concerning several diseases of the large bowel and also diabetes mellitus and atherosclerosis. We suggest that large amounts of unrefined plant foods, especially all starchy foods rich in dietary fibre, may offer protection against these diseases.

White bread has many excellent nutrients and no toxic substances but it is harmful, in my opinion, because it lacks enough fibre, intimately mixed with the flour, and cell-wall structures (dietary fibre) in a close physico-chemical relationship with cellular contents, especially starch. In other words, to improve atherosclerosis² or diabetes mellitus³ one must eat very much unrefined starchy food of many different varieties. This is not possible unless one decreases very considerably all fats and sucrose. With such a diet bran is unnecessary.

Independently of my views doctors at the Long Beach Veterans Administration Hospital, California, have been treating severe peripheral vascular disease with this type of diet in a metabolic ward for six months. Thirty-eight patients were randomly divided into two equal groups. One group ate the usual low-fibre, high-fat, Western-type diet and received the approved drug therapy; the other group ate completely unrefined starchy foods (80% of energy content), fats, any variety (10%), and protein (10%) with no added salt or sugar. Their cholesterol intakes were less than 100 mg/day. The dietary-

treated group were encouraged to increase physical exertion markedly several times a day, stopping only when pain became intolerable; they came off all drug therapy as soon as possible. The control group of patients showed negligible improvement. The group treated by diet and exercise showed much improvement, which was statistically significant ($P < 0.001$); most have discontinued all drugs; their maximum walking distance has increased to many thousand feet; three who had angina have all lost it; of 13 diabetics, 11 are now off all hypoglycaemic drugs and/or insulin.⁴ The work has been extended to other studies and Mr N Pritikin has informed me that about 80% of maturity-onset diabetics respond well but that no improvement has occurred in younger diabetics considered to be of the juvenile type.

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- 1 Burkitt, D P, and Trowell, H C, eds. *Refined Carbohydrate Foods and Disease: Some Implications of Dietary Fibre*. London and New York, Academic Press, 1975.
- 2 Trowell, H C. *American Journal of Clinical Nutrition*, 1975, 28, 798.
- 3 Trowell, H C. *Diabetes*, 1975, 24, 762.
- 4 Pritikin, N, et al, paper presented on 19 November 1975 at 52nd Annual Session of the American Congress of Rehabilitation, Atlanta, Georgia.

Irrigation of the vas for immediate sterility after vasectomy

SIR,—Urquhart-Hay¹ described a method of obtaining immediate sterility after vasectomy by irrigating by slow injection the proximal end of each vas with 2.5 ml of sterile euflavine 1:1000. The purpose of this procedure was to render unnecessary the two or three postoperative semen analyses at 8, 12, or 16 weeks and thereby reduce the inconvenience that such tests cause to patient, laboratory, or vasectomy clinic. Albert *et al*² recently reported the use of nitrofurans as the sperm-killing medium and concluded after only 24 patients had been studied that irrigation dispenses with the need to do subsequent semen analyses. The purpose of this report is to illustrate that these methods cannot guarantee immediate sperm death and therefore immediate sterility.

The technique described by Urquhart-Hay was exactly followed, but semen analyses were carried out at 8 and 12 weeks in all patients. In the 84th case in which this method was used examination of uncentrifuged semen at the eighth week showed the presence of motile sperms—2/3 per low-power field. Subsequent examination one and two months later of this patient's centrifuged semen showed the absence of sperms by this time.

This case illustrates that immediate sterility cannot be guaranteed by using vas irrigations, even though in most cases this procedure may hasten the onset of sterility. Therefore I conclude that postvasectomy semen examinations are still essential to confirm the absence of sperms in all patients.

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- 1 Urquhart-Hay, D. *British Medical Journal*, 1973, 3, 378.
- 2 Albert, P S, et al, *British Journal of Urology*, 1975, 47, 459.