aetiology of skin conditions does not, as Dr. Lydon suggests, provide an "easy way out." On the contrary it is an arduous and exacting form of therapy, for one is called upon not only to relieve the condition but to counteract the by now fixed idea in the patient's mind that he is suffering from physical disease or is the victim of external noxious agents.

It is difficult, and perhaps unnecessary, to fit these patients into psychiatric classifications. In fact one of the outstanding features of psychosomatic disorders is the total failure of workers in this field to assign a particular disorder to a particular personality group. What is urgently required is relief of the patient's condition, and in order to achieve this the physician should be willing to listen to the patient's, and often immediate relations', stories, however irrelevant they may seem, for therein lies the guide to his treatment. The human spirit cannot be imprisoned in the confines of "the only truly scientific method of study—the objective approach." This way ignores the sufferer in the interests of impure knowledge.—I am, etc.,

London, S.W.1. KATHRYN H. COHEN.

REFERENCES

[1] *Year Book of Dermatology and Syphilology*, p. 37, 1951, Chicago.
[2] Excerpta Medica, 1952, 6, Sect. 13 (Dermat. and Venerol.), 361. Synopsis of paper.

*** This correspondence is now closed.—ED., *B.M.J.*

The Neurology of John Hunter's Last Illness

SIR,—I have read with interest the paper by Sir Russell Brain (December 27, 1952, p. 1371) and find a correlation of the symptom-pathology with that often seen in cases of mental disease. The episodic character of the vertigo, epilepsy, loss of memory, sensory hyperaesthesiae with irradiation, distortion of visual images, auditory pulsation, and diarrhoea, contrasts with the complete absence of gross lesions in the central nervous system and the constant presence of cardiovascular lesions. Similar absence of lesions of the neurone and presence of cardiac pathology has been noted by A. Batty Shaw[1] and other workers, who "suggest that the neurological disturbance may have been due to ischaemia and anoxia from fall in blood pressure, associated with narrowing of the cerebral arteries."[2] In my book[3] I illustrate a localized area of hyperaemia typifying reactive hyperaemia to a temporary occlusion of a cerebral arterial branch (Plate II, a). Such cortical hyperaemia is known to lower the resistances of the synapses, and would thus account for the hyperaesthesiae with irradiation, the epilepsy, and other symptoms depending upon the locality, of the ischaemia predetermined by the vascular pathology.—I am, etc.,

Birmingham. F. A. PICKWORTH.

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[1] *Lancet*, 1952, **1**, 763.
[2] Ibid., 1952, **1**, 652.
[3] *New Outlook on Mental Diseases*, 1952. Bristol.

Christmas Disease

SIR,—We read with enjoyment the letters by Drs. D. H. Collins and P. R. Kemp (January 10, p. 97) and are glad that our name "Christmas disease" gave them a seasonal opportunity to enjoy themselves. In fact, since medical terminology abounds with names far more peculiar than Christmas, an extension of this variety of humour (with due care to avoid impropriety) could enliven your correspondence for weeks.

In compliance with the suggestion of Dr. Kemp, may we submit to you, Sir, for consideration the names we discussed before choosing Christmas disease, with brief reasons for their rejection? Aetiological terms are satisfactory only if the aetiology is clear, and attempts to devise one for Christmas disease led us into deep water. The factor missing in this condition is probably, but not certainly, a co-factor for a precursor of the precursor of thromboplastin and might thus be called "coprothromboplastinogen." Following established practice, the disease could then be termed "hereditary hypocoprothromboplastinogenaemia," but this would be subject to alteration should the factor be found later (as is very possible) to be, say, a co-factor for a co-factor of thromboplastin. What would Dr. Kemp feel about "hypococothromboplastinaemia"?

Terms like these could be avoided by continuing the practice of numbering the clotting factors. But these factors probably react in a sequence at present undetermined, and the numbering must be related to this sequence to be intelligible. We might call the factor under discussion "Factor 8" (or 9, or 10), but if it were later found that another is interposed between those already numbered 8 and 9 we should have to renumber or call the new factor "8A," or even "8½." In any case, would such a name as "Hereditary Blood Clotting Factor No. 8 Deficiency" be acceptable? The unassuming term "Christmas factor" has only been used temporarily, being admittedly unsatisfactory, though there is precedence for calling factors after the conditions produced by their deficiency (for example, "P.A. factor"). Following a suggestion by Dr. Robb-Smith, the name will probably be abbreviated to "X" or "χ," which should forestall any suggestion by your correspondents that its possible precursor should be known as Christmas Eve factor.

Failing the aetiological approach, another is to devise some name suggesting the symptomatology. Christmas disease resembles clinically the inaptly named condition haemophilia, and variants of this word were considered. But pseudohaemophilia, parahaemophilia, and haemophilia 1 and 2 have already been used. "Metahaemophilia" and "haemophilia B" were suggested, but it is actually desired to emphasize the distinction from, rather than the similarity to, haemophilia. Moreover, if Christmas disease becomes "haemophilia B," then haemophilia must be renamed "haemophilia A" and antihaemophilic globulin "antihaemophilic A globulin," with possible repercussions from the International Haemophilia (A ?) Society. By a cautious approach to a classical scholar we might be able to produce some appropriate Graeco-Latin concoction, but angiostaxis, hemogenia, thromboasthenia, and constitutional thrombopathy are in use, and the number of clinically similar diseases awaiting differentiation will probably outrun classical invention.

Finally, coming to eponyms, modesty prevented us from referring to the condition as the "Biggs–Dacie–Douglas–Macfarlane–Merskey–O'Brien–Pitney syndrome," and, faced by the difficulties described, we felt that "Christmas disease" was a legitimate, unassuming, and pleasantly provocative term. After all, Dr. Collins asserts that thousands of your readers turned to our article because it was headed by this name. How many would have given it a glance had it been called, for example, "Hereditary Orthothrombophobia"?—We are, etc.,

ROSEMARY BIGGS.
A. S. DOUGLAS.
Oxford. R. G. MACFARLANE.

Acute Laryngotracheobronchitis

SIR,—I would like to apologize for the delay in replying to Dr. J. S. Taylor's letter (December 20, 1952, p. 1361), but both Dr. Heseltine and I are, at the moment, in Canada. It is true that a combination of penicillin and aureomycin was used in three cases. The reason why penicillin was given in early treatment has been described in the paper. In these cases only one dose of crystallized penicillin was given, and aureomycin was commenced as soon as the child could swallow.

Dr. Taylor is, of course, right when he assumes that penicillin and aureomycin could be antagonistic. A considerable body of work has now accumulated to prove this point, and Jawetz arranges the antibiotics in two groups: (1) Penicillin, streptomycin, bacitracin, neomycin; and (2) aureomycin, chloramphenicol, terramycin. He points out that members of group 1 are frequently synergistic, but never antagonistic. Members of group 2 are neither synergistic nor antagonistic, but when group 1 and group 2 are combined, depending on the susceptibility of the microorganism, antagonism may result. I have listed below some