ing fine print, until finally even large print became obscure. One week before her admission to hospital she could distinguish the largest objects only as vague shadows. Apart from her defective vision, no abnormalities were detected on physical examination, and her chest symptoms were minimal. Her visual fields were found to be markedly constricted, and on both sides there were dense central scotomas. Her visual acuity was down to 5/400. On fundoscopic examination, the veins were seen to be moderately engorged and tortuous, and there were flame hæmorrhages radiating from the discs, which were slightly elevated.

Her chloramphenicol therapy was stopped at once, and she was treated with intramuscular injections of vitamin B^{12}, and oral doses of ascorbic acid, thiamine and multivitamin capsules (of six different types!) on a presumptive diagnosis of optic neuritis. Her vision slowly improved, although she needed streptomycin and colistin to control her chest symptoms, and after five months her vision had nearly returned to normal. Some residual signs of damage still remained, however.

In the other series, N. N. Huang, R. D. Harley, V. Promadhattavedi and A. Sproul from Philadelphia [2] tell of nine children with severe symptoms of fibrocystic chest involvement, whose ages ranged from six and a half to 14 years, and whose general condition varied from fair to poor (their condition required treatment with various other antibiotics and aerosol therapy, as well as the chloramphenicol), who were given courses that ranged from 81 to 252 days. The total doses given at the time the ocular symptoms became noticeable were from 81 to 283 grammes, and, as in the first case mentioned, frequent blood examinations had failed to demonstrate any abnormality.

The first complaint of the children was of impairment of vision. On being tested for visual ability they were all found to have a marked decrease in acuity, and bilateral central scotomas. Fundoscopic examination revealed disc blurring in six of the nine children, and three had retinal hæmorrhages. In addition, two of the children complained of numbness and cramps in their feet which caused them more discomfort than their visual disturbances. Subsequent developments varied, apparently quite haphazardly, and the confusion is such that no very definite conclusion can be derived from them. Two children died from the effects of their disease shortly after the eye signs were discovered, on having stopped taking chloramphenicol, the other having continued the therapy. No ocular improvement was noted in either of them before their deaths. Two others were given no vitamin or other treatment aimed specifically at the eye symptoms, which cleared up spontaneously in both cases, although one continued to take chloramphenicol. In the one who stopped treatment, vision improved in a matter of hours from the time of stopping the drug. Of the remaining five children, all of whom were given vitamins in varying combinations and quantities, one showed a quick improvement on stopping chloramphenicol, two showed improvement despite continuing with the drug, and two showed only slight improvement after first continuing with chloramphenicol, and then stopping it and taking corticosteroids instead.

The authors consider that the conditions they report represent forms of optic neuritis and retrobulbar neuritis, although they are at a loss to explain the mechanism of their causation by chloramphenicol. They do suggest, and this would seem very sensible, that those patients who for one reason or another require long-term courses of chloramphenicol therapy should, in addition to their usual hæmatological check-ups at regular intervals, have a frequent assessment of visual acuity, and that parents of patients should be advised to test their children's sight regularly with a small visual chart. In some instances, too, the development of numbness and cramps in the feet may be a forerunner of visual disturbance, and should also be watched for.

However, in explanation of some of the conflicting data mentioned above, there is evidence to suggest that some of the toxic effects of chloramphenicol may be related to a deficiency of vitamins of the B group, particularly riboflavine, and in a paper read at the recent annual meeting of the Australian Pacdiatric Association in Canberra on April 3, G. Morgan, G. Wise and D. O'Gorman Hughes, from the University of New South Wales, tell of four patients with chloramphenicol toxicity of differing varieties, one of whom developed amblyopia which was alleviated by the administration of B-group vitamins, despite his continuing with chloramphenicol.

None of which is to suggest that chloramphenicol is not a thoroughly efficient and often life-saving drug. But, as with such things as nuclear fission and motorcars, the greater the potential advantages, the greater the parallel dangers, not all of which can always be forecast.

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[2] *J. Pediat.*, 1966, 68 : 32 (January).