cases, and aspiration biopsy proved helpful in the diagnosis in cases 2 and 3. Necropsy confirmed the presence of a primary bronchial carcinoma in case 1. In cases 2 and 3 it seems probable that the primary lesion was a carcinoma of the bronchi, in view of the evidence of lung involvement and of the histology of the biopsy material in case 2.

In cases 2 and 3 there was a delay of six and of eight weeks between the onset of symptoms caused by the bony metastases and the appearance of signs of pulmonary involvement. Such a delay is by no means uncommon; Hirsch and Ryerson (1928) described two cases in which the delay was six and sixteen months.

We wish to thank Dr. G. E. S. Ward, Dr. D. McAlpine, and Dr. A. G. C. Taylor for permission to record these cases; Prof. James McIntosh, Prof. R. W. Scarff, and Dr. A. C. Thackray for reports on the morbid anatomy and histology; Dr. H. K. Graham-Hodgson for the radiological investigations; and Mr. E. Bridger and Mr. J. Kilshaw for the photographic work.

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VITAMIN E IN ANGINA PECTORIS

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It has been suggested (Vogelsang and Shute 1946) that vitamin E relieves the pain and improves the exercise-tolerance of patients with angina pectoris. Shute (1949) observed that this drug dilated the local capillaries in senile vulvitis, and he suggested that in angina pectoris it acted as a coronary vasodilator. An alternative hypothesis, supported by the independent experimental work of Govier et al. (1946), was that vitamin E had a beneficial influence on the metabolism of cardiac muscle.

Sudden death has been noted in cattle with a deficiency of vitamin E (Gullickson and Calverley 1946). This was attributed to cardiac damage, and in one animal electrocardiography showed changes in the r—r interval, low-voltage prolonged qrs complexes, and changes in axis-deviation. Atrophy and scarring of the cardiac muscle, with increase in the cellular elements, were found at necropsy. Supporting this hypothesis is the observation that animals with muscular dystrophy, artificially induced by diets deficient in vitamin E, die of cardiac failure. The published work on this subject, however, is not critical, nor are the observations adequately controlled, and the questions asked in the British Medical Journal (1946) regarding the efficacy of vitamin E in angina pectoris led us to carry out a clinical trial.

Our series consisted of twenty-two patients (17 men and 5 women), aged 44-66, and our technique was based on that of Evans and Hoyle (1933) that none of these drugs is of much value in the routine treatment of angina pectoris. Table II suggests that phenobarbitone is the most useful and diminishes the liability to anginal attacks, as suggested by Bramwell and King (1942). Among the patients who found vitamin E the most beneficial no common factor was noted in the electrocardiogram, blood-pressure, age, blood-cholesterol, length of history of the disease, size of heart as measured in the telediagram, or family history.

As regards possible toxic manifestations of vitamin E, one patient complained of a dry feeling in the throat, two had increased headaches, and one noted pronounced vasodilator effects in the form of flushing of the face, throbbing headaches, and giddiness.

SUMMARY

Twenty-two patients with typical angina of effort were treated with vitamin E, phenobarbitone, aminophylline, and calcium lactate, each drug being given for 3 weeks, after which the patients were asked to compare the effects of the drugs.

From this small but clinically significant series it is concluded that vitamin E is not of any therapeutic value in the routine treatment of angina pectoris.

We wish to thank Prof. Crighton Bramwell for his interest in the investigation and for allowing us to use his cases, and Messrs. Roche Products Ltd. for the supply of ephynal.

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