GEORGE BUDD (1808–1882) AND NUTRITIONAL DEFICIENCY DISEASES

by

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THE EMERGENCE of theories concerning the existence of accessory food factors and the deficiency diseases that result from their dietary absence is usually located at the beginning of the twentieth century. Drummond and Wilbraham have summarized the views of most historians of nutrition:

> As the century drew to a close the orthodox teaching on man's need for food could be expressed in the following manner. There were five *proximate principles* of foods. Three, proteins, fats and carbohydrates, were organic, and two, salts and water, were inorganic. . . . It will be observed that this scheme makes no mention of any such food substances as the curative antiscorbutic principle of fresh fruits, in spite of the hundreds of years of experience of the disease. The nutrition experts were so busy at the end of the century amassing quantitative data about the need for protein and calories they seem to have had little or no time for any wider problems.[1]

Singer and Underwood in their *Short History of Medicine* made a very similar statement: 'During the period when Voit and his colleagues were laying the foundations of the science of nutrition it was thought that it would always be possible to construct an artificial diet, which would satisfy the needs of the body, and that, provided that the constituents were present in their proper proportions, the problem was purely quantitative'.[2]

Casimir Funk, himself a pioneer in the field of vitamin studies, wrote in 1912: 'Diseases . . . such as beri-beri, scurvy, pellagra . . . present certain general characters which justify their inclusion in one group called deficiency diseases. They were considered for years either as intoxications by food or as infectious diseases, and twenty years of experimental work were necessary to show that diseases occur which are caused by a deficiency of some essential substances in the food. Although this view is not yet generally accepted, there is now sufficient evidence to convince everybody of its truth. . . .'[3] The 'sufficient evidence' referred to by Funk, stemmed mainly from the experimental studies of Lunin, Eijkmann, Pekelharing and Hopkins. These early investigations, done during the period 1881–1912, demonstrated quite unequivocably that it was impossible to keep experimental animals alive on diets consisting solely of protein, carbohydrate, fat, salts and water. Additional dietary principles ('accessory factors' was the description used by Hopkins) were necessary. These were later renamed vitamins, and it was shown that the absence of a particular vitamin from the diet resulted in the appearance of a specific 'deficiency disease'.[4] These new concepts were in striking contrast with established nutritional thought at the end of the nineteenth century. There can be very little doubt, however, that very similar ideas were proposed (but never experimentally tested) in 1842 by George Budd, the London physician.

GEORGE BUDD

George Budd was born in North Tawton, Devon, in February 1808. He was

educated at Cambridge (St. John's and Gonville and Caius Colleges), Paris, and the Middlesex Hospital. In 1837 he was appointed physician to the Dreadnought Seaman's Hospital at Greenwich, where, in collaboration with a Mr. Bush, he made extensive researches on cholera and scurvy. He was elected a Fellow of the Royal Society in 1836 and he obtained his M.D. (Cambridge) in 1840—the year in which he was appointed Professor of Medicine at King's College, London. He published a considerable number of papers in the medical press—particularly in the *London Medical Gazette*—but he was best known because of his two books *On Diseases of the Liver* (1845) and *On the Organic Diseases and Functional Disorders of the Stomach* (1855). His Gulstonian and Croonian Lectures (also on 'Diseases of the stomach') were published in the *London Medical Gazette* in 1843 and 1847 respectively. He retired from his chair at King's College in 1863 and moved to Barnstaple (Somerset) where he died in 1882.[5,6]

In 1842 he published in the *London Medical Gazette* a series of five articles based on his lectures at King's College and entitled 'Disorders resulting from defective nutriment' (**2**, 632–36, 712–15, 743–49, 825–31 and 906–15). In these articles Budd quite clearly put forward the suggestion that accessory food factors were obligatory components of normal diets and that clearly definable deficiency diseases resulted from their absence from the diet. His ideas thus preceded by some fifty years the emergence of the currently accepted theory of vitamins and deficiency diseases.

BUDD'S DEFICIENCY DISEASES

In the opening paragraphs of his articles Budd wrote:

> Large numbers of men at sea and in our prisons and asylums, have, at various times, been kept on a diet insufficient in quantity and variety for the support of the body—diseases of strange kind have appeared among them. . .
> There are three different forms of disease which are already traced to defective nutriment. The first and best known of these is scurvy, properly so called; of the second the most distinctive character is a peculiar ulceration of the cornea; the third is chiefly marked by softness of imperfect development of the bones. . . . These diseases are not only different in appearance, but they arise from different causes—the defect or error of diet on which they depend, is different for each—and any one of them may occur quite independently of the rest.[7]

There can be little doubt that the three diseases described by Budd were in fact avitaminoses C, A and D respectively. It is a measure of Budd's perceptive powers that he not only recognized them as diseases of nutritional deficiencies but that he also introduced (albeit abortively) into nutritional thought the concept that a specific disease could result from the absence of a single dietary component.

SCURVY

Of the three deficiency diseases that he described, Budd's treatment of scurvy (avitaminosis C) is by far the most comprehensive and best balanced. This should not surprise us. Budd had considerable first-hand experience of scurvy during his period as physician to the Dreadnought Seaman's Hospital Ship and by 1840 he had already contributed a comprehensive account of the disease to Tweedie's *Library of Medicine*.[8] The beneficial effects of fresh fruit and vegetables both in preventing and curing scurvy were of course well known by Budd's time. But there is no evidence

that writers previous to Budd regarded a dietary lack of these commodities as the sole—or even as a necessary—cause of the disease. James Lind, whose *Treatise of the Scurvy* (1753) is regarded as a milestone in the history of the disease, was quite specific on this point: 'And I am certain it will be allowed, by all who have had an opportunity of making observations on this disease at sea, or will attentively consider the situation of seamen there, that the *principal and main predisposing cause* [Lind's italics] to it is a manifest and obvious quality of the air viz its moisture'.[9] Later in his treatise Lind referred to: 'an additional and extremely powerful cause . . . the want of fresh vegetables and greens . . .'.[10] However, he nowhere referred to the presence of any specific antiscorbutic dietary principle in the vegetables and greens; instead, he accounted for their antiscorbutic activity in terms of a supposed ability 'either to counteract the bad effects of their before mentioned situation (i.e. moist sea-air); or rather, and more truly, to correct the quality of such hard and dry food as they are obliged to make use of'.[10] Indeed, it appears that he did not regard vegetables and fruit as obligatory dietary principles: 'It would be tedious to give many instances, they being notorious, of ships' crews continuing several months at sea, upon their ordinary diet without any approach of the scurvy'.[11] and' . . . many people for years abstain from vegetables without any inconvenience'.[12]

It is therefore unlikely that Lind regarded scurvy as a disease caused by the lack of a specific principle in the diet. Most commentators of the post-Lind period accepted his teaching virtually unchanged and there was no significant attempt to re-define the etiology of scurvy in nutritional terms.[13] Some writers even reverted to pre-Lind concepts when discussing the causes of the disease.[14]

Budd, on the other hand, was quite unambiguous and uncompromising in his description of scurvy as a nutritional disorder:

> If now we inquire into the circumstances under which scurvy arises, with the view of ascertaining its cause, we find that the only condition which is never wanting, and which seems absolutely essential to its production, is complete and prolonged abstinence from succulent vegetables or fruits, or their preserved juices as articles of food. . . . As agriculture and gardening improved, scurvy gradually became less frequent . . . and (now) the disease hardly ever occurs on land except during sieges or in persons long shut up in prisons and asylums.[15]
> . . . Men had not yet perceived that the disease had its real origin, not in the cold of our rigorous climate but in the abstinence from fresh vegetables and fruits.[16]

This clear delineation of scurvy as a disease of defective nutriment alone was a clear advance on current ideas in the field. Budd made a number of other interestingly new observations on the nature of scurvy. He referred to the comparatively long depletion period necessary before the scorbutic condition developed: 'It is only after this abstinence has been protracted from two to five months that the . . . health suffers so grieviously.'[17] He commented on the occurrence of scurvy: 'Scurvy has not, I believe, been observed in any animal but man. Dogs and other carnivorous animals are kept in perfect health for an indefinite time on meat only; while man, though equally capable of digesting and assimilating animal food, becomes scorbutic at the end of a few months, if not allowed in addition, some succulent vegetables or fruits or some article prepared from them.'[18] and drew attention to the thermolabile nature of the antiscorbutic principle: '. . . The principle, whatever it be, is common

to the juices of a great variety of vegetables and unripe fruits. . . . The antiscorbutic property seems to be impaired by the action of strong heat.'[19] In support of this latter statement he referred to the absence of antiscorbutic activity from Lind's preparation of inspissated juice of orange or lemon: 'Dr. Lind's "rob" . . . was extensively tried but found very inferior in efficiency to the fresh fruit. . . .'[20]

AVITAMINOSIS A

Budd's observations on his second deficiency disease, are, if anything, more interesting than his account of scurvy. He began by describing Magendie's experiments of 1817 in which dogs given a diet of pure sugar and water became emaciated, showed corneal ulceration and ultimately died. These experiments were widely quoted during the first half of the nineteenth century as proof of the essential nature of nitrogenous compounds (proteins) as dietary components. Budd's interpretation of them was less restrictive: 'It was thence inferred that the ill effects of this diet resulted from deficiency of nitrogen. . . . This inference is, no doubt, to a certain extent true, since Liebig and Dumas have satisfactorily proved that animals obtain their nitrogen exclusively from their food; but it is far too exclusive. It does not recognize the importance of other principles which are wanting in these articles of food but which, although less in amount, yet, like nitrogen, enter constantly into the composition of the body, and are therefore required for its support.'[21] Budd then referred to cases of dietary-induced corneal ulceration in man: 'It is not in the lower animals only and in express experiments, that ulceration of the cornea takes place from defective nutriment. The same phenomenon is not infrequently witnessed in natives of Indostan, who subsist chiefly on rice'[22] and gave examples of cases that he personally had treated: 'While visiting-physician to the Dreadnought, I observed a similar condition, though less in degree, in Lascars on their arrival in this country from India [he then describes four such cases where corneal ulceration had occurred but apparently without any accompanying marked emaciation] . . . All these men came, as I have remarked, from the same ship, and during the voyage had fared alike. Their food consisted of rice, salt-fish, ghee (a sort of butter) curry of fish and sometimes peas. In their own country, in addition to these articles of food, they have fresh vegetables, milk, bread and occasionally meat.'[22]

Budd effected a cure with a diet of milk, eggs, fish etc. The structure of the diet taken by the Lascars during their voyage from India would appear to preclude the possibility of protein deficiency—a point emphasized by Budd in his commentary on the cases: 'The frequent occurrence of this ulceration of the cornea in Lascars, whose diet, whilst at sea has little variety, the fact that it shows itself only towards the end of a voyage, and that fresh sailors are not affected after their arrival in port, when their diet is again more varied, and the circumstances that both eyes are generally affected alike, show that it has its origin in imperfect nourishment. Its not being always attended with great emaciation, further shows that it does not result from simple inanition but from want of some elementary principle in the diet. What this principle is we cannot as yet pronounce with certainty. The circumstances noticed in the account of Mudor (*one of the cases described by Budd*) that he was not emaciated, tends to show that in this case nitrogen is not the principle at fault. . . . Without a

knowledge of the particular element wanting in the food of Lascars we cannot point out a more definite means of prevention than a more varied diet.'[22]

Budd clearly realized that he was dealing with a deficiency disease not attributable to lack of dietary nitrogen. The 'principle at fault' to which he referred was in all probability vitamin A (or its precursor, β-carotene) and there can be little doubt that Budd was in fact recording cases of avitaminosis A, the most characteristic feature of which is xerophthalmia with subsequent corneal ulceration.[23] Xerophthalmia is still widely prevalent in certain areas of the world and particularly in South and East Asia where the dietaries are lacking in vitamin A and β-carotene. Heavily implicated in this respect are rice-based diets unsupplemented by green vegetables; the diet eaten by the Lascars described in Budd's report would fall into this category.

RICKETS

Budd's third deficiency disease was rickets. Rickets was probably the most common nutritional deficiency disease in Britain in the 1840s and it would be surprising had Budd not included it in his discussion. His description of it is brief but to the point. 'A disease of imperfect nutrition frequently seen in children is rickets, which is characterized chiefly by pliability of the bones which yield to the weight of the body and become crooked and much distorted.'[24] Budd subscribed to the, then, novel theory that rickets resulted from an impaired intake of calcium.[25] '[Rickets] . . . may assuredly arise . . . from deficiency of food, or rather from a diet deficient in lime and other elements of which the bones are made up'[26] and he quoted Liebig's statement that children whose food did not contain enough lime to form their bones ate the lime off the walls 'with as much relish as their meals'.[27] 'A wineglassful of lime-water, two or three times a day in milk, is said to be of considerable efficacy', wrote Budd.

It is now known that the main dietary determinant in rickets is vitamin D. Budd's suggestion that butter (a useful source of the vitamin) should be removed from the diet of rachitic persons is probably the least valuable of all his dietary precepts and contrasts with the observational and experimental approach which forms the basis of the bulk of his nutritional thought.

BUDD'S PLACE IN NUTRITIONAL HISTORY

Perhaps the most striking feature of Budd's nutritional thought was the way in which he succeeded in formulating his ideas in an essentially unfavourable—and possibly an alien—conceptual environment. His concept of accessory dietary principles and of associated deficiency diseases, stood in almost complete isolation from the mainstream of mid-nineteenth-century nutritional thought. Many contemporary writers on diet and nutrition still subscribed to the 'humoral' doctrine that man could be characterized in terms of: 'four principal constituents, long familiarly known as the bilious, the lymphatic, the nervous and the sanguine . . .' and that the '. . . necessity of adapting the diet and regimen to the individual constitution and mode of life, becomes so obvious . . . as to excite surprise that it should ever have been neglected.'[28] Supporters of this theory held that dietary adjustments were a necessary method for redressing humoral imbalances within the body. Within this

loose conceptual framework it was possible to think in terms of dietary-engendered diseases and their cure—as in the claim that 'veal is not of a heating nature, and may therefore be allowed to febrile patients in a very weak state.'[29]

But this was a far cry, and qualitatively, involved quite a different approach, from the concept of deficiency diseases within a framework of rational physiology. And in any case, by about 1820 a distinct shift was discernible in nutritional thought in Britain. As the work of the continental physiologists became known the humoral approach gradually gave way to a more functional characterization of foodstuffs coupled with a more quantitative assessment of their nutritional significance. Classification of nutrients on a chemical basis became more popular and a truer appreciation of the physiological significance of the main dietary components began to emerge. Kilgour arranged foods into 'Farinaceous, Mucilaginous, Saccharine, Acidulous, Oleaginous, Caseous, Albuminous, Gelatinous and Fibrous' and made the observation that: ' . . . experiments with these principles has shown a fact to physiologists which they have as yet found a difficulty in explaining, viz. that not any *one* of these principles used for a *continued period* will support life.'[30]

This system was still further simplified as Liebig's views on the relative importance of nitrogenous (protein) and non-nitrogenous (fat and carbohydrate) dietary components became known. Scientific nutritionists at the beginning of the 1840s occupied themselves almost exclusively with questions concerning the physiological significance of the macronutrients and their quantitative relationships. Budd's interest in *qualitative* aspects of *micronutrients* therefore represented a complete break with the contemporary pattern of nutritional thought.

Discontinuities and breaks in processes of conceptualization are characteristic of the broad general advance of scientific knowledge, but such breaks are meaningful only within the context of the situation that they seek to modify or supplant. Their emergence is dependent upon the existence of a pattern that already partakes of a partial degree of validity. Budd's ideas were without doubt a measure of his considerable originality of thought. Nevertheless, his scheme would have been meaningless in 1800—or even in 1820—before the emergence of a rational and quantifiable system of nutrition. Even so, he put forward his scheme some decades before established scientific opinion was ready to assimilate it.

Budd's endeavour may be summarized as follows: (1) he postulated the existence of essential nutritional principles other than the macronutrients; and he pointed out that they could be required in only small amounts; (2) he suggested that the absence from the diet of any single principle would result in the appearance of a specific deficiency disease; (3) he correctly forecast the conditions under which deficiency diseases were likely to emerge (long periods on an unvarying diet of simple structure); (4) he was probably the first to state unequivocally that scurvy resulted from a dietary lack of one such principle and from this alone; he described the antiscorbutic principle correctly as a thermolabile one present in fresh fruit and vegetables; (5) he almost certainly described cases of avitaminosis A, cured them, distinguished them from changes produced by protein deficiency and attributed them to the absence of a specific but unknown dietary principle.

In developing these ideas Budd was some fifty years ahead of his time. There is

evidence that even in 1840—two years before the publication of his *Medical Gazette* articles—he already regarded scurvy as a disease resulting from a dietary lack of an 'essential element'. L. J. Harris has drawn attention to Budd's early ideas on scurvy and has aptly described him as 'The Prophet Budd': 'But in 1840 a medical writer called Budd, more far-sighted than his contemporaries, seems to have foreseen the existence of the anti-scurvy vitamin—for he definitely expressed the belief that scurvy (to quote his own words) was due to the 'lack of an essential element which it is hardly too sanguine to state will be discovered by organic chemistry or the experiments of physiologists in a not too distant future.'[31]

It was not until 1842 however, in his *Medical Gazette* articles that Budd extended his analysis to other conditions and put forward his generalized theory of 'diseases of defective nutriment'.

BUDD'S INFLUENCE ON NUTRITIONAL THOUGHT

Budd's theory was nevertheless of little relevance to the main areas of contemporary nutritional thought with their emphasis on quantitative aspects of the physiological function of macronutrients. This probably accounted, at least in part, for his failure to influence subsequent nutritional developments in the nineteenth century. Certainly, there is no strong evidence that his ideas achieved any element of permanency or influence, although it may be noted that Pereira in the preface to his *Treatise on Food and Diet* (1843) made the following point: 'The author [Pereira] did not venture, without considerable hesitation and doubt as to its propriety, to deviate from Dr. Prout's beautifully simple and generally admitted classification of alimentary principles into the *aqueous*, the *saccharine*, the *albuminous* and the *oleaginous*.[32] After mature consideration, however, he satisfied himself of the impossibility of reducing all nutritive principles to these four heads . . . lemon juice, which constitutes one of our most valuable antiscorbutic foods, does not owe its efficacy to water, sugar, albumen or oil.'[33]

Later Pereira actually referred in a footnote to Budd's articles in the *London Medical Gazette* in 1842;[34] it is more than likely therefore that his implied suggestion that accessory dietary principles existed was at least in part derived from Budd's writings. There is no further reference in Pereira's book to such accessory principles nor is there any discussion of diseases of defective nutriment. The characterization of the macronutrients and the assessment of their nutritional significance remained the main interest of both theoretical and experimental nutritionists almost until the close of the century. F. W. Pavy (1829–1911), a pupil of Claude Bernard and probably Britain's leading metabolic physiologist during the second half of the nineteenth century, like Pereira, made only a passing reference to the possible existence of accessory food factors: 'Experience has shown that, for a proper maintenance of health a certain proportion of the food must be consumed in the fresh state. . . . Without being able to give the precise reason for what occurs, it is evident that there is something absent from dried and salted food which the system requires'.[35] Pavy obviously did not consider this to be a matter worth pursuing further and later in his text he gives the requirements for a normal diet as nitrogenous matter, fat, carbohydrate and mineral matter. The re-emergence of Budd's theory had to await the

work of the experimental nutritionists at the turn of the century. Nevertheless, a comparison of the following four passages will give some measure of his originality of thought.

> (a) But further, no animal can live upon a mixture of pure protein, fat and carbohydrate, and even when the necessary inorganic material is carefully supplied the animal still cannot flourish. . . . The field is almost unexplored, only is it certain that there are many minor factors in all diets, of which the body takes account. (F. Gowland Hopkins, 1906).[36]
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> (b) From this we must infer, that the ill effects of a diet consisting of sugar, starch, oil, fat, do not result from want of protein only but from want of other principles also requisite for the support of the body. Perhaps the deficiency of each principle shows itself in a particular way (George Budd, 1842).[37]
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> (c) . . . the observations now to be described . . . bring out in particular the marked influence of *minute additions* of normal food constituents in promoting the nutritive power of synthetic dieteries (F. Gowland Hopkins, 1912).[38]
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> (d) Numerous facts, both in nurture of animals and in agriculture, point out the importance of various incidental principles, *small in absolute amount*, but apparently not the less essential to the due development of the structures into which they enter as constituents. With all these principles we are perhaps not yet acquainted. You will see, therefore, the prudence of recommending a varied diet; and you might expect, what indeed really happens, that if man, and more especially children, be kept long on the uniform and regulated diet of our prisons, workhouses and asylums, some disorder, the result of defective nutriment, is sure to arise. (George Budd, (1842).[39]

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9. LIND, J., *A Treatise of the Scurvy*, Edinburgh, 1753, p. 106.
10. Ibid., p. 115; later, in a footnote, Lind ascribed a synergistic causative activity to the two factors 'they are both of them (viz. *diet* and *moisture*) predisposing causes to the disease. They are each but half causes, neither of them singly being able to produce it'.
11. Ibid., p. 91.
12. Ibid., p. 94.
13. Francis Home in his *Principia Medicinae* (3rd ed., Edinburgh, 1770, p. 204) referred approvingly to Lind's *Treatise* ('Bene de civibus meret Lynd [*sic*], ob id volumen quod de scorbuto composuit') and his treatment of the disease was for the most part a précis of Lind's ideas.
14. E.g. lack of fresh vegetables and/or fruit was not included in the eight possible causes of scurvy listed in the twelfth edition (1791) of William Buchan's well-known *Domestic Medicine*.
15. BUDD, G., op. cit., p. 633.
16. Ibid., p. 636.
17. Ibid., p. 636; it is of interest to note that in human volunteers on a scorbutogenic diet a period of seventeen weeks elapsed before the emergence of the disease (Bartley, W., Krebs, H. A., and O'Brien, J. R. P., Special Report Series, Medical Research Council, London, H.M.S.O., 1953, No. 280).
18. BUDD, G., op. cit., p. 743.
19. Ibid., p. 745.

20. Ibid., orange juice loses over half its content of vitamin C when converted into 'rob' according to Lind's directions; moreover, over threequarters of the vitamin C content of the 'rob' is lost during storage for two months (R. E. Hughes—unpublished observations).
21. BUDD, G., op. cit., p. 745.
22. Ibid., p. 746.
23. MCLAREN, D., in *The Vitamins*, ed. W. H. Sebrell and Robert S. Harris, New York and London, Academic Press, 1967, vol. 1, p. 267.
24. BUDD, G., op. cit., p. 825.
25. DRUMMOND, J. C. and WILBRAHAM, A., op. cit., p. 381.
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27. The belief that a nutrient deficiency could produce a craving for foods containing the missing principle was a point frequently emphasized by Budd in his writings.
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30. KILGOUR, ALEXANDER, *Lectures on the Ordinary Agents of Life*, Edinburgh, 1834, p. 232. Similar to Kilgour's approach was that of Paris in his widely known *Treatise on Diet*, 2nd ed., London, 1827.
31. HARRIS, L. J., *Vitamins in Theory and Practice*, 4th ed., Cambridge, 1955, p. 6. Budd wrote in his article on 'Scurvy' in 1840: 'We are ignorant of the essential element, common to the juices of antiscorbutic plants, on which the properties in question depend; but shall, probably, not be deemed to sanguine, if we anticipate that the study of organic chemistry, and the experiments of physiologists, will, at no distant period throw some light on this subject' (Budd, G. in Tweedie, A., op. cit., p. 77).
32. I.e. water, carbohydrate, protein and fat respectively.
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CENTENNIAL MEETING TO COMMEMORATE THE FOUNDING OF THE ANDERSON SCHOOL OF NATURAL HISTORY BY LOUIS AGASSIZ

A week-long meeting is being planned to commemorate Louis Agassiz and his Anderson School of Natural History at Penikese Island on the hundredth anniversary of its founding. Meetings at Woods Hole and on Penikese Island, Cape Cod, Mass., 13–18 August 1973 will be devoted to the historical importance of Agassiz and his School (organizer H. L. Burstyn), and to an in-depth study of island biology (organizer E. O. Wilson). A field trip (organizer J. S. Ranklin, Jr.) to Penikese Island will be made to continue the work of previous anniversary trips in 1923 and 1947. Those interested in attending should write to Dr. Donald J. Zinn, General Chairman of the Committee on Arrangements, Department of Zoology, University of Rhode Island, Kingston, R. I. 02881, for further information and an application form.