These specimens were examined during 1957 at Cornell University by Dr. Peter Olaffson, who failed to find any indication that the piglet may have had muscular dystrophy lesions.

Thus results to date indicate that vitamin E supplementation of sows and gilts in Fiji, receiving what would be considered a normal farm ration, apparently eliminated the birth of piglets exhibiting clinical symptoms of muscular weakness, and in addition decreased the number of still-births per litter and increased the number of piglets born alive, though these effects were not quite statistically significant. Also the addition of vitamin E to the diet of two baby piglets exhibiting the muscular weakness resulted in their dramatic recovery. Histological examination of tissues from one affected piglet failed to disclose any signs of muscular dystrophy lesions.

It is considered that the balance of evidence is in favour of the theory that there was some form of vitamin E deficiency in the breeding stock. So far as is known, no one has previously reported symptoms of vitamin E deficiency in a herd of pigs fed standard type rations, though Gorham *et al.*[3] and Davis and Gorham[4] have reported a condition known as 'yellow fat' disease in pigs fed on a rather unusual ration of fish scraps, which responded to vitamin E supplementation of the ration, and which is probably caused by excessive amounts of unsaturated fatty acids in the diet. Obel[5] has also reported that death from liver dystrophy is high in pigs in Sweden aged 3–15 weeks, where 6 per cent of cod-liver oil is fed in the ration, and that this disease is prevented by substituting lard for cod-liver oil and supplementing the ration with vitamin E.

The rations fed to the pigs in Fiji contained a high proportion of coconut meal which was usually slightly rancid. As the oxidation of vitamin E is especially rapid in foods containing high levels of unsaturated fatty acids or rancid fats together with minerals that catalyse the oxidative reactions, it is possible to postulate that the available vitamin E in the ration was being oxidized. This theory is supported by a report from Jamaica by McDonnough[6], who states that two calves fed on irrigated grass with a concentrate supplement of coconut meal that may have been rancid died, the post-mortem examination revealing that they were suffering from 'white muscle disease'. It is hoped to investigate this theory further at this establishment.

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W. J. A. Payne

East African Agriculture and
Forestry Research Organization,
P.O. Box 21,
Kikuyu,
Kenya.
Jan. 2.

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Cardiac Output in Cattle

The determination of cardiac output by the dye dilution techniques requires the taking of serial arterial blood samples at short regular intervals. Direct puncture of the brachial artery of cattle[1] enables such samples to be collected. *T*.1824 (Evans blue) was injected into the jugular vein of the subjects at a dose of approximately 0·40 mgm./kgm. body-weight and arterial blood samples collected at 1-sec. intervals for a period of 30 sec. Adopting the procedure described by Hamilton *et al.*[2] the average concentration of the dye in the arterial blood samples was found, together with the time required for one circulation of the dye and the cardiac output calculated from the formula :

$$\text{cardiac output} = \frac{60 \times \text{amount } T.1824 \text{ injected}}{\text{mean concentration } T.1824 \times \text{circulation time}}$$

The subjects were allowed to settle in their surroundings for some time prior to making a determination, and during the manipulative procedures excitement of the subjects was avoided so far as possible. Pulse-rates were taken during injection of the dye and collection of arterial blood samples ; and during the procedure on each animal the pulse-rate remained within the normal range of 65–90 beats per min.

Twenty-six cardiac output determinations were made on 22 female Ayrshire cattle of body-weight 100–605 kgm. The outputs ranged from 12 to 68 l. of blood per min. which, when calculated as output of blood in ml./kgm. body-weight per min., give a mean value of 113 ml. with an *S.D.* ± 11·0 ml.

The result calculated for cattle as cardiac output of blood in ml./kgm./min. is similar to that obtained in sheep by Schambye[3] using a dye-dilution technique and by Cross *et al.*[4] using the Fick method. It also compares with the value obtained by Barcroft *et al.*[5] using the Fick method on goats. Doyle *et al.*[6] used the Fick method on three adult cows to determine the cardiac output and found a mean value of 44 l./min. but made no mention of the breed or weight of the animals.

Edward W. Fisher
Rodger G. Dalton*

University of Glasgow Veterinary School,
Veterinary Hospital, Bearsden Road,
Bearsden, Glasgow.
Jan. 6.

* Agricultural Research Council Research Student.

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Production of Acute Insulin Deficiency by Administration of Insulin Antiserum

The diabetic syndromes observed in experimental animals and in man are due to metabolic abnormalities induced directly or indirectly by insulin deficiency and to lesions which affect tissues other than the β-cells of the islets of Langerhans. It is difficult to