

pulse-length, 0.6–50  $\mu$ sec., or rate of recurrence, 100–500 per sec., power-density, maximum rise of temperature, 14–2°C., or electrolyte concentration.

These results posed the main physical problem of relating energy in the body of the solution, expressible as total calories per c.c., with the localized effect on the micro-organisms, taking into account that major effects could be produced with a rise of 2 or 3°C. Now it can be shown that the energy expended in the double-layer adjacent to the organism and that in the body of the solution are both proportional to (ionic drift velocity)<sup>2</sup> × concentration. The localized action can be further correlated with that measured in the solution by comparing linear separations between ions in both regions<sup>6</sup>; these prove to be so nearly the same, about 20A., that the two energies could well be of the same order, whereas for selective heating of so small a body as a cell the local energy would have to be enormously the greater. Hence the idea arises of ascribing the primary physical action to sufficient ionic disturbances in the double-layer. This idea is supported by the reproductions of the main pulse effects observed after another kind of ionic disturbance caused by change of the ionic environment when organisms are centrifuged from one solution and re-suspended in another<sup>7</sup>, and more strongly by the fact that when the pulse treatment is preceded by such an ionic change its effect is much modified.

The pulse action may thus be considered as an orderly march of the cations of the double layer over the surface of the organism, superposed on their random motions, and as successive ions pass a point producing disturbances at intervals of the order of 10<sup>-8</sup> sec. which are comparable with the relaxation times of water molecules in the neighbourhood of ions<sup>8</sup>. To such disturbances, unusual in number or kind, the organisms are not apparently adapted, and viability fails; a single 0.6  $\mu$ sec. pulse can so kill some 7 per cent of *E. coli* or 17 per cent of *Cl. butyricum*. Further, because of the very short time intervals involved, it can be supposed that the disturbances take effect biologically before the equipartition of energy in subsequent collisions degenerates their energy into random motions recognizable as heat. That such disturbances are capable of affecting sensitive chemical equilibria is shown by the displacement of the equilibrium between methylene blue and oxygen.

A detailed account of this work has been prepared for publication.

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<sup>1</sup> *Nature*, **157**, 51 (1946).

<sup>2</sup> *Nature*, **183**, 1450 (1959).

<sup>3</sup> *Proc. Roy. Soc.*, B, **98**, 312 (1925).

<sup>4</sup> Fricke, H., and Curtis, H. J., *Nature*, **135**, 436 (1935).

<sup>5</sup> Cole, K. S., *Trans. Faraday Soc.*, **33**, 966 (1937).

<sup>6</sup> McQuillen, K., *Biochem. Biophys. Acta*, **6**, 66 (1946).

<sup>7</sup> Gosling, B. S., *J. App. Bact.* **21** (2), 220 (1958).

<sup>8</sup> Kozlyev, B. M., *Disc. Faraday Soc.*, **19**, 135 and 185 (1955).

### Biological Half-Life of Radioiron in Man

THE recommendations of the International Commission for Radiological Protection, 1955<sup>1</sup>, give in Table C.V. values of the effective half-life for iron-55 and iron-59 of 61 and 27 days respectively. The fact that these values are so similar in spite of the big difference in their physical half-lives (1.06 × 10<sup>3</sup> and 46 days respectively) results from the use of a biological half-life for iron in the blood and blood-forming organs of 65 days (Table C.IV). This result

is probably based on early data obtained with animals (for example, Greenberg and Copp<sup>2</sup>) and would now appear to be considerably too low for the adult man.

An increase in the value of the biological half-life would not affect the calculation of the maximum permissible body burden, but would lead to a decrease in the maximum permissible levels in water and air, these being inversely proportional to the effective half-life in the conditions of final equilibrium with chronic exposure as considered by the Commission.

Under these equilibrium conditions one can assume that the body store in the critical organ is constant and the daily excretion and ingestion are equal. Then the biological half-life  $T_b$  may be calculated (as in their equation C4) from:

$$T_b = \frac{0.693 (\text{mass of element in initial organ})}{\text{daily uptake into (or excretion from) critical organ}}$$

Table C.II gives the mass of blood as 5,400 gm., containing 5 × 10<sup>-4</sup> gm. iron per gm. blood, and the uptake as 80 per cent of a dietary daily intake of 12 mgm. of iron. Using these figures in the above equation leads to a biological half-life of 195 days—already some three times the value used in the calculations.

However, recent absorption studies<sup>3,4</sup> indicate that not more than 10 per cent of the dietary iron is, in fact, absorbed in the normal adult.

Further, although iron is released after red cell destruction, it is diluted in body pools and re-utilized for further red cell production, so that the net excretion loss is very small indeed. Callender<sup>5</sup> states that the adult male has about 2–3 gm. of iron in haemoglobin and 1–1.5 gm. in body stores (ferritin and haemosiderin). This total is somewhat higher than the 2.7 gm. of iron in the critical organ given by the Commission's data. The excretion of iron in the faeces does not exceed 0.5 mgm./day<sup>6</sup>, the loss from all sources being only about 1.5 mgm./day, corresponding to some 10 per cent (that is, the absorbed fraction) of the dietary daily intake of 10–15 mgm. Then, using values of 1.5 mgm. daily loss and a store of 3 gm. leads to a biological half-life of 1,400 days, corresponding to effective half-lives of 600 days for iron-55 and 44.5 days for iron-59, increases of 10 times and 1.6 times, respectively, over the present figures given by the Commission.

It should be appreciated that in childhood the iron stores are in the process of being built up and during this period iron uptake will always exceed the excretion. Thus, equilibrium conditions are not achieved and the effective half-life will approximate to the physical half-life.

That such discrepancies should exist in data, which are widely quoted and accepted as standard, emphasizes the need for continual review of such tables, and for the need for workers to pass on to the respective International Commissions any new experimental results which may have a bearing on their calculations.

*Note added in proof.*

We have been informed by Dr. K. Z. Morgan, chairman of Committee II of I.C.R.P., that corrections of this type have already been incorporated in the revised recommendations which are being prepared.

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<sup>1</sup> *Brit. J. Radiol.*, Supp. 6, 37 (1955).

<sup>2</sup> Greenberg, D. M. T., and Copp, D. H., *J. Biol. Chem.*, **169**, 377 (1946).

<sup>3</sup> Moore, C. V., *J. Clin. Nutr.*, **3**, 3 (1955).

<sup>4</sup> Callender, S. T., Mallett, B. J., and Smith, M. D., *Brit. J. Haemat.*, **3**, 186 (1957).

<sup>5</sup> Callender, S. T., *Brit. Med. Bull.*, **15**, 5 (1959).

<sup>6</sup> Dubach, R., Callender, S. T., and Moore, C. V., *Blood*, **3**, 526 (1948).