If Steinmetz's empirical law ($W = \eta(B_{max.})^k$ ($k \simeq 1 \cdot 6$)) is applicable to our experimental data, then there must exist a linear relation between log W and log $B_{max.}$; we find such a linear variation, with the approximate error of 1 per cent, between the flux-densities of 1,000 and 12,000 gauss, with the following values for the constants η and k :

	Sheet 1	Sheet 2	Sheet 3
η	0·00076	0·0010	0·0011
k	1·66	1·65	1·59

It was found that Steinmetz's law is not applicable at high and low flux-densities. We have observed, like Brailsford[1], that for sheets 1 and 3, and very high values of $B_{max.}$, the hysteresis loss increases with $B_{max.}$ according to a linear law.

Moreover, it has been shown experimentally that, in accordance with theory, the influence of a superimposed steady unidirectional flux on the hysteresis loss is nil. The measurements were carried out with steel sheet 1 and for three different values of steady flux : 1,500, 4,100 and 11,300 gauss. 4,100 gauss is the value of the flux-density for which magnetic permeability of the material is a maximum. As an example, the experimental data for two loops of very different shape are: loop with steady flux = nil : $B_{max.} = 13,360$ gauss; $W = 6 \cdot 47 \times 10^3$ erg.cm.$^{-3}$; loop with steady flux = 11,300 gauss; $B_{max.} = 13,320$ gauss; $W = 6 \cdot 31 \times 10^3$ erg.cm.$^{-3}$.

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[1] Brailsford, F., *J. Inst. Elect. Eng.*, **90**, 313 (1943).

Biological Action of Radiations

IT has been shown previously[1] that when radiation (for example, X-rays, α-particles, neutrons) is absorbed in aqueous solutions, an indirect chemical effect is produced due to the formation of free radicals and atoms :

$$H_2O + \text{radiation} = H + OH \qquad . \qquad . \qquad (1)$$

In general, this is followed by the recombination :

$$H + OH = H_2O \qquad . \qquad . \qquad . \qquad . \qquad (2)$$

and by interactions of these radicals with other acceptor substances ($S, S_1, S_2, \ldots$) present, for example :

$$S + OH = E_1 \qquad . \qquad . \qquad . \qquad . \qquad (3)$$

$$S_1 + OH = E_2 \qquad . \qquad . \qquad . \qquad (3 \cdot 1)$$

$$\cdot \quad \cdot \quad \cdot \quad \cdot \quad \cdot$$

$$S_{n-1} + OH = E_n \qquad . \qquad . \qquad . \qquad (3 \cdot n)$$

This scheme of reactions must exist also in the aqueous systems of biological subjects. From the point of view of the older 'hit' theory[2], reaction (3) corresponds to a single 'hit', while if, for example,

$$E_1 = S_1 \ , \ E_2 = S_2 \ , \ \ldots \ E_{n-1} = S_{n-1} \quad . \quad . \quad (4)$$

one gets a successive change of the (starting) substrate (S) by $1, 2 \ldots n$ interactions with, for example, OH radicals, which is equivalent to $1, 2 \ldots n$ 'hits'. It can be shown that—under certain conditions—there is a complete formal analogy between the 'hit' theory, which is described by Poisson's formula, and the mathematical expression for the above system of equations (3, 3·1, . . . 3·n).

If the biologically active substance is represented by the molecule S, the *essential* interaction corresponds to equation 3 and the theory suggests two important cases :

Case A. The recombination process (2) can be neglected (if the interaction with the acceptor molecules is relatively fast). From the above scheme of reactions, it follows that in this case the action of the radiation (for example, the half 'lethal' dose $D_{1/2}$) depends only on the *total* number of ions (radicals) produced, independent of the nature and wave-length of the radiation. This case is represented, for example, by the mutations induced by X-rays. Under the assumption—which is almost certainly fulfilled—that apart from the S molecules there are also some other (biologically non-active) molecules present which react with the OH-radicals (thereby exerting a 'protection effect') the chemical theory leads to the equation :

$$S = S_0 \exp. (-\varkappa D) \qquad . \qquad . \qquad . \qquad (5)$$

where S and S_0 represent the numbers of these molecules at times t and zero respectively ; D, the total dose of radiation and :

$$\varkappa = \ln 2/D_{1/2} \sim q_s(q_{s_m}[S_m])^{-1} \qquad . \qquad . \qquad (6)$$

where q_s represents the (kinetic) cross-section of the S molecules and the term in brackets can be interpreted as the mean linear region available per one molecule of the (protecting) substrate molecules (S_m).

Case B. Recombination reaction (2) taken into account (if the radicals are present in relatively high concentrations or, for example, the acceptor molecules are relatively non-reactive). It follows that in this case the *local* concentration and distribution of the radicals is of importance. While for different radiations the number of ions (radicals) produced is approximately the same for equal doses, their local distribution may differ widely. In this case, therefore, the action of the

radiation must depend on the nature of the radiation, as is known to be the case, for example, of the lethal effect on unicellular organisms.

The above scheme leads to the following expression (with certain simplifying assumptions) :

$$S = S_0 \exp. \left(- \frac{k_3 D}{k_2[H]_l} \right) = S_0 \exp. (- \alpha D) \quad (7)$$

where $[H]_l$ represents the *local* concentration of the radicals in the regions where the actual reaction takes place. As a first approximation $[H]_1$ can be estimated for two important stages of the reaction. (i) *In the initial stage*, that is, before any appreciable diffusion of the radicals (formed in the tracks of the ionizing particles) has set in. Here one may assume that :

$$[H]_{1(i)} \propto \text{number of ions per unit length of track} \quad . \quad (8)$$

This quantity is known experimentally[3,4] for different radiations. More generally, one has approximately[5,6] (if V denotes the energy of the radiation) :

$$[H]_{1(i)} \propto V^{-1} \text{ (for X-rays)} \qquad . \qquad . \qquad (9)$$

$$[H]_{1(i)} \propto V^{-1/2} \text{ (for } \alpha\text{-rays)} \qquad . \qquad . \qquad (9a)$$

(ii) *In the advanced stage* (but before any appreciable intermixing of the individual ionization tracks has occurred) :

$$[H]_{1(ii)} \propto V \qquad . \qquad . \qquad . \qquad (10)$$

Taking into account both these stages for the action of, say, X-rays, one obtains for α of equation 7,

$$\alpha = \alpha_{(i)} + \alpha_{(ii)} \propto \frac{k_3}{k_2} \frac{1}{V} \left(\frac{V^2}{a} + 1 \right) \quad . \quad (11)$$

where a denotes a proportionality factor. Remembering that $V \propto \lambda^{-1}$ and that for the range (R_e) of the (ionizing) fast electrons one has the relation[5] : $R_e \propto V^2$, one obtains finally from equations 7 and 11

$$D_{1/2} = \ln 2/\alpha \propto \frac{1}{\lambda} \frac{a}{(R_e + a)}, \quad . \quad . \quad (12)$$

which is formally identical with Glocker's well-known expression.

It is now possible to predict that if sufficiently hard γ-rays or X-rays are used, the dependence on the wave-length should gradually diminish and finally disappear. This can be illustrated by Wykoff's own figures[8] for *Bacterium coli*, where the mean lethal dose decreases in the series of decreasing wave-length as : 8·4, 6·67, 4·65, 4·20 × 10³r, and in order to achieve the same effect with α-rays, 24 × 10³r are required. On the other hand, while it is known that the doses of radiation for the production of mutations are independent of the wave-length in the case of γ- and X-rays[9] a much higher dose of neutrons is required to produce the same effect[10]. This follows directly from the above theory, as the recoil protons produce a relatively high local ion (radical) density leading to increased recombination.

A full account will be published elsewhere which will include also a discussion of 'multiple hit effects', where the situation is somewhat different[11].

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[1] Weiss, *Nature*, **153**, 748 (1944).
[2] cf. Mayneord, *Proc. Roy. Soc.*, A, **146**, 867 (1934).
[3] cf. Gray and Read, *Brit. J. Radiol.*, **15**, 72 (1942).
[4] Lea. *Proc. Camb. Phil. Soc.*, **30**, 80 (1934) ; *Brit. J. Radiol.*, **16**, 338 (1943).
[5] cf. Bethe and Bacher, *Rev. Mod. Phys.*, **8** (1936).
[6] Bagge, *Ann. Phys.*, **30**, 72 (1937).
[7] Glocker, *Z. Phys.*, **77**, 653 (1932).
[8] Wykoff, *J. Exp. Med.*, **51**, 921 (1930) ; **52**, 435, 769 (1930).
[9] cf. Schrödinger, "What is Life ?" (Cambridge, 1944).
[10] Timoféeff-Ressovsky and Zimmer, *Naturwiss.*, **26**, 362 (1938).
[11] cf. Hevesy, *Rev. Mod. Phys.*, **17**, 102 (1945).

Effect of Container Walls on Packing Density of Particles

IN recent correspondence[1] in *Nature* a general theorem on the ordered packing of equal spheres is cited as implying that "the overall voidage of any one of the four possible ordered arrangements of equal spheres packed into a container is independent of the relative size of the container provided its size and shape are such that it will contain an integral number of 'unit' cells of the particular ordered arrangement and in all other cases an ordered packing cannot be obtained". This is due to a misconception of the 'unit' cell, which in the first three out of the four ordered packings, namely, a sphere having 12, 10, 8 or 6 neighbours, really contains fractions of a sphere totalling to one, and it is only in the fourth case that the unit cell, being a simple cube, contains a whole sphere.

Furthermore, the inescapable higher voidage adjacent to the container walls as compared to the interior of the container has to be taken into account[2], and its contribution to the overall voidage has to be allowed for ; unless it happens to be negligible, as with a container of very large size compared to the particle size of the packed material. Characteristic voidage for a compacted packing of a sized material is then found to depend more on the particle shape[3,4,5] than on the particle size. This increases with increased deviation of the irregular particles from a spherical shape, unless conditions exist to