'Window Fields' in Muscle

The 'membrane', that is, the electric double layer, surrounding the muscle fibre generates no field of forces inside the system. If, however, part of the membrane becomes depolarized, the depolarized part acts, so to say, as a 'window' in the closed system and an electric field is generated. If the depolarization should occur with a sharp border, the maximal intensity of the field in the axis of the fibre would correspond to a field generated between the two plates of a condenser placed at a distance equal to the diameter of the fibre and having the same potential difference as the two sides of the 'membrane'. If the wave of depolarization travels along the membrane, this will have the same effect as if we had pulled electrodes along the axis charged correspondingly. Experiments, performed along this line by St. Hajdu and one of us, show that such a field will actually bring the contractile matter directly to contraction even if the membrane is inoperative.

It is an interesting property of the 'window field' that its time integral is independent of the sharpness of the borderline of the wave of depolarization, and that the time integral of the component of the field parallel to the axis is identical for any point of the cross-section. It is equally noteworthy that the time integral of the component of the field, vertical to the axis, is zero. The parallel component will act as a direct current, the vertical one as an alternating current.

There seems to be thus no 'transmission' of excitation from the membrane to the contractile matter; depolarization and the elicitation of contraction merge into one single physical event.

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Dec. 15.

A New Blood-Group Antigen

A serum that identifies a new blood-group antigen was obtained from the parturient mother of a baby with erythroblastosis fœtalis. Mrs. Kidd had had five previous pregnancies but no blood transfusions. No previous babies had erythroblastosis fœtalis. She was delivered on April 17, 1950, of a male infant who developed typical erythroblastosis fœtalis, and whose cells gave a positive direct Coombs's test. On routine testing of Mrs. Kidd's serum at that time, it was apparent that it contained a previously unidentified antibody.

Typing tests showed Mrs. Kidd to be *O*, *CDe/cDE* (R_1R_2), *MsMs*, *Pp*, *Le(a−b+)*, *Fy(a−)*, *kk*. Her cells were not agglutinated by her own serum. The baby is *O*, *CDe/cDE* (R_1R_2), *MSMs*, *pp*, *Le(a+b+)*, *Fy(a+)*, *Kk*.

The serum was further tested against the bloods of 210 unrelated persons. Of twenty-one *Kell*-positive bloods, all were agglutinated by the Kidd serum. The presence of anti-*Kell* was later proved. Of the remaining 189, all *Kell*-negative, 146 (77 per cent) were agglutinated. Chi square tests failed to show any serological relation of the new antigen to the antigens of the *ABO*, *MNS*, *P*, *Rh*, *Lewis* or *Duffy* blood-group systems, or to sex. Insufficient evidence has been obtained as to the relation, or lack of relation, to the *Lutheran* and *Kell* blood-group systems. Further selective absorption studies and family studies will be required.

The new antibody gave specific reactions at 37° C. against red cells suspended in saline, in a titre of 1 : 16. The reactions were no stronger in bovine albumin; but a titre of 1 : 64 was obtained by Coombs's method. On storage, the saline-active component has disappeared, leaving a component active by the Coombs's method. A saline-active component of the anti-*Kell* made it impossible to distinguish *Kell*-positive, *Kidd*-negative bloods.

On the basis of these preliminary tests, it is possible to say that a new blood-group antigen has been identified, present in the red cells of about 77 per cent of Americans. If it is inherited as a dominant, the gene frequency in Americans is approximately 52 per cent. It is proposed to name the new antigen Jk^a (after Mrs. Kidd's son); its inheritance is now being investigated.

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Sweating in Man from the Intradermal Injection of *nor*Adrenaline

It has been generally believed that sweating in man is produced in response to sympathetic nervous activity by the liberation at the termination of the post-ganglionic fibre supplying the sweat gland of acetylcholine[1], the transmitter being different from that liberated by sympathetic fibres supplying other organs, which, until recently, was believed to be adrenaline. However, evidence has accumulated that there is an adrenergic component in sweating in man. Sweating is a prominent feature in patients with phæochromocytomata[2], which were believed to secrete adrenaline. Spontaneous sweating in man is abolished by the adrenolytic agent 'Dibenamine'[3]. The intradermal injection of adrenaline has been shown to produce sweating[4]

Recently, evidence has accumulated that the post-ganglionic sympathetic transmitter is probably *nor*adrenaline[5-9]. Also the ergone predominantly present in phæochromocytomata has been shown to be predominantly *nor*adrenaline[2,10].

It was therefore considered of importance to determine whether the local injection of *nor*adrenaline could produce sweating. The method used for detection of sweat-gland activity was that described by Masao Wada[4]. Briefly, the area of skin to be examined is painted with a 3 per cent iodine in absolute alcohol solution and dried completely; the area is painted again with a mixture of 100 gm. fine starch powder and 100 ml. of castor oil. Sweating is evidenced by the appearance of blue dots at the mouths of the sweat glands.

Adrenaline hydrochloride and *nor*adrenaline bitartrate monohydrate were prepared in dilutions of 10^{-3} to 10^{-8}. A small amount of a saline control solution and the different dilutions of the adrenaline and *nor*adrenaline solutions were injected intradermally in the skin areas prepared as described. Sweating was produced with the dilutions 10^{-3} to 10^{-5} with both the adrenaline and *nor*adrenaline solutions in all of five subjects, and in some cases with a dilution of