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Skin Impedance Findings in Mental Disease.

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It has been shown¹ that the electrical impedance per unit area of the skin may deviate from normal values not only in thyroid and other endocrine conditions but also in disturbances involving the autonomic and central nervous systems. Gross changes in skin capacity have been reported in certain types of mental disease.² It appeared to be of interest, therefore, to study a group of mental patients with a view to determining whether or not changes in the impedance properties of the skin could be found.

Impedance determinations were made by the 3-electrode method at 11,160 c.p.s., using a concentric and a disc electrode applied over the region of the biceps and triceps as described elsewhere.^{1, 3}

The patients were not selected in any way and included all of the females (54) in the adult wards at the N. Y. State Psychiatric Institute. This group was composed predominantly of schizophrenics and psychoneurotics. The average age was 29 years.

A histogram of the results obtained is shown in Fig. 1 (dotted lines) and represents the distribution of the mean impedances of two 6 cm² skin areas measured on the anterior and posterior sides of the upper right arm. A corresponding histogram of the results obtained on 102 normal females in a previous investigation¹ (solid lines) is given for purposes of comparison.

As will be seen from Fig. 1, there is a considerable shift of the mentally diseased group toward low values, their mean impedance *m* falling well to the left of the normal mean *M*. The group of patients having impedances lying entirely outside the normal histogram on the low side are of particular interest since it is in this low impedance region that the values for thyrotoxic fall.¹ None of the patients in this low impedance group showed signs of thyrotoxicosis.

Since the tendency towards low impedance values in these patients might be due to a diminution in their skin phase angles, measurements were also made of the phase angle of the skin at 15,300 c.p.s. using the same electrodes. The phase angles, in every case, fell within

¹ Barnett, A., *West. J. Surg.*, 1937, **45**, 540.

² Roggenbau, C., and Lueg, W., *Monat. f. Psychiat. u. Neur.*, 1929, **73**, 301.

³ Barnett, A., *J. Physiol.*, 1938, **93**, 349.

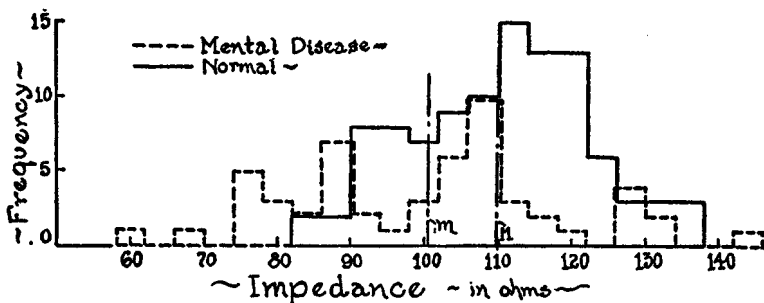


FIG. 1.

Histograms of skin impedance findings in normals (solid lines) and in the mentally diseased (dotted lines). Abscissæ in ohms, ordinates in frequency. The impedances represent values for a 6 cm² skin area.

normal limits and were well distributed over the normal range.³ The skin phase angle of thyrotoxic has also been found to fall within normal limits.¹

The fact that the skin phase angles of normal, thyrotoxic and mentally diseased subjects fall within the same limits while the impedances of thyrotoxic and mental patients show a group shift towards low values is of considerable interest. It may be interpreted to mean that although the quality of the dielectric (dielectric loss, power factor) remains the same, its thickness changes. This would be true for sheets of glass differing only in thickness. Their phase angles would be the same. The impedances would vary with the sheet thickness. It has been pointed out elsewhere³ that impedances measured by the 3-electrode technic represent the properties of only the poorly conducting portions of the skin, *i. e.*, the epidermal layers. We appear to be dealing, then, with differences or changes in the thickness of the epidermis. This structure is known to be in a continuous process of growth. The thinning of the skin in thyrotoxicosis and its thickening in myxedema has long been known to clinicians. The effect of thyroid substance on tadpoles has been shown to be one of inhibiting growth and hastening differentiation,⁴ *i. e.*, rapid evolution from tadpole to frog with symmetrical dwarfing. The thinning of the skin in thyrotoxicosis may, therefore, be due to a similar hastening of differentiation in the basal proliferating layers of the epidermis with "dwarfing" of the fully developed structure. In the case of mental patients, no such mechanism can be invoked for thinning since thyroid function appears to be, generally, normal. Evans⁵ has shown that the proportion of growth hormone in the pituitary glands of adult cattle is as great as in those of young calves.

⁴ Gudernatsch, F., *Cold Spring Harbor Symp.*, 1934, 2, 94.

⁵ Evans, H. M., *J. A. M. A.*, 1935, 104, 464.

No satisfactory explanation for this interesting finding has, as yet, been given. The tendency towards low impedance skin values in mental disease may be a symptom of deficiency in growth hormone affecting directly or indirectly both the epidermis and the central nervous system. It is proposed, therefore, to study the therapeutic effect of growth hormone and other growth stimulating substances⁶ in mental patients particularly where low skin impedance readings are obtained.

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Preparation of Follicle-Stimulating Extracts by the Use of Trypsin.*

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In a previous publication we have reported that the luteinizing activity in sheep pituitary gonadotropic extracts is destroyed by trypsin.¹ This has been confirmed by Chen and Van Dyke.² Our procedure for destroying the luteinizing activity has been utilized, therefore, in developing a convenient method for obtaining follicle-stimulating preparations.

The method of preparation is as follows: Acetone-dried sheep pituitary powder (100 g) was shaken with 1 liter of water and 0.5 cc of toluene for 12 hours and centrifuged. The extraction was repeated twice. The activity was recovered from the supernatant liquids by precipitation with acetone and centrifugation, after which the precipitate was suspended in 400 cc water, shaken and supercentrifuged.

The supercentrifuged supernatant liquid was treated at 37°C for 3.5 hours at pH 8 with 40 mg of trypsin† per gram of original pituitary powder and centrifuged. The precipitate was discarded. The clear supernatant liquid was placed in 50 cc centrifuge tubes and

⁶ Various authors in the Symposium on Growth, *Cold Spring Harbor Symp.*, 1934, 2.

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¹ McShan, W. H., and Meyer, R. K., *J. Biol. Chem.*, 1938, 126, 361.

² Chen, G., and Van Dyke, H. B., *Proc. Soc. Exp. Biol. and Med.*, 1939, 40, 172.

† The trypsin used was samples No. 360427 and No. 390120 prepared by Fairchild Bros. and Foster, New York.