unipolar stimulation during periods of from 5 or 10 seconds, at intervals of 1.5 to 2 minutes. After showing that several of these liminal stimulations yielded constant responses, 1 per cent. novocaine solution colored by toluidine blue was applied round the point, and the stimulation of the motor focus was continued at regular intervals of from 1.5 to 2 minutes. The area around the motor point, surrounded by the novocainized cortex, usually had a diameter of about 8 mm.

When the general condition of the animal remains constant, one finds in the cat, dog and monkey (*Macacus rhesus*) that after 8 to 15 minutes, usually at about 13 minutes, the excitability of the motor point is *augmented, i.e.,* the threshold for the point in question is diminished, or that when the strength of stimulus is kept unchanged throughout the whole experiment, the responses are distinctly stronger and even wider spread; a point which before the novocainization, at 13 cm coil-distance, gave rise to a slight flexion of the fingers of the contralateral hand, may now yield, at the same coil-distance, not only a much stronger flexion of the fingers, but also flexion of the wrist and often flexion of the elbow and retraction of the shoulder. In the cat and the dog we occasionally observed spread of the response to the hind limb of the same side, or, if the primary stimulation took place on the hind leg, a spread of response towards the front leg. Occasionally, also, a reversal together with augmentation of the response could be observed, *e.g.,* the primary liminal flexion changed after the novocainization into a much stronger extension of the same joint and of other joints of the same limb. Very often a marked clonic, partial epileptoid after-discharge entered into the picture. We have obtained in the monkey this augmentation of responses from the face, arm and leg areas of the cortex. This augmentation, which only sets in after a long latent period of from 8 to 15 minutes, subsides after 20 to 45 minutes. Renewed application of novocaine often gives rise once more to the appearance of the phenomenon.

Cortical facilitation is a well-known phenomenon since Exner discovered it in 1882; and especially so since through the investigations of Graham Brown and Sherrington it is known that cortical motor points do not yield fixed reactions, but are more or less "instable," because upon repeated stimulation of a motor point or after stimulation of another cortical antagonistic point, the response may be augmented or may change in pattern, *e.g.,* from extension into flexion (primary and secondary facilitation). These phenomena occur, at least so far as is now known, only when the two liminal stimuli succeed each other within a few seconds, intervals of one minute being sufficient to do away with any ordinary facilitation known at present. In introducing intervals of from 1.5 to 2 minutes we actually did not observe any facilitation, before the local anesthesia with novocaine.

The long latency of 8 to 15 minutes also points in the direction, that in this curious phenomenon of augmentation of response we have not to do with a primary phase of hyperexcitability of the local anesthesia. So far as we know, such a phase, if present at all, in local novocaine-anesthesia is much less marked than in local narcosis by cocaine and stovaine, and even here this primary phase of hyperexcitability through which the nerve goes (for which most of the investigations on changes in excitability during local anesthesia are carried out) occurs within the first minute or minutes after the application and soon passes away. Furthermore, novocainization of a motor point itself gives rise to a marked depression or even a temporary extinction of its excitability. We may safely assume, therefore, in our experiments, that this explanation does not account for the augmentation of response.

The explanation of our phenomenon is difficult to give in the present state of our knowledge of cortical functions. Perhaps the most probable hypothesis is to look upon it as a phenomenon of "release" of function in the sense of Hughlings Jackson and Head, the excitability of a small area of the cortex cerebri becoming (temporarily) augmented when it is "released" from the influence of the surrounding cortical areas.

We have not succeeded in obtaining this phenomenon after circumcision of a cortical point; the circumcision gives rise, as might be expected, to a long lasting depression or loss of excitability of the motor point. Apparently we succeeded in establishing our phenomenon with novocaine because this drug blocks functionally, but without producing cortical shock.

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THE ETIOLOGY OF SWINE INFLUENZA

SWINE influenza ("hog flu") was first recognized as a clinical entity in 1918 and since then has reappeared in epizootic form each autumn and winter in the swine raising states of the middle west. It bears a striking resemblance to influenza in man. Experimentally, the disease can be readily transmitted by contact and also by the introduction of tracheal exudate from infected animals into the nasal passages of normal swine. Eight strains of the disease have been established among our experimental swine during the three years it has been under investigation in this laboratory.

In these experimental infections as well as in diseased animals studied in the field an organism, first isolated by the late Dr. Paul A. Lewis, with whom this investigation was started, has been found constantly

present. This organism is very similar if not identical to non-indol-producing strains of Pfeiffer's bacillus. Oftentimes the organism has been obtained in pure culture from the involved lung and bronchial exudate. It has not been found in the respiratory tract of normal swine. Freshly isolated cultures of the organism, when administered intranasally, may produce a disease which might be confused with the natural infection in swine but which, unlike the natural disease, is not contagious. Cultures on artificial media for two months or longer are non-pathogenic.

Suspensions of tracheal exudate and lung from infected animals passed through Berkefeld N filters, when introduced into the nasal passages of normal swine, cause a variable disease complex, apparently dependent upon the strain of infectious material under study. One strain, obtained in 1928, produced a clinical picture and lesions which closely resembled those following the intranasal injection of unfiltered infectious material. With other strains the disease produced by the filtrate has been very mild and transient and sometimes difficult of certain recognition. The contrast between the mild disease caused by the filtrate and the typical disease induced by unfiltered infectious material has been particularly striking with two strains of the disease obtained in 1930. In all instances bacteriological examination of the lung and tracheal exudate of filtrate infected swine has failed to reveal the influenza-like organism and sometimes these sites have been found bacteriologically sterile. The mild disease induced by the filtrate is contagious.

If a small amount of a culture of the influenza-like bacillus, carried on artificial media for over two years and long since non-pathogenic for swine, is added to a Berkefeld N filtrate and this mixture injected intranasally into normal swine, a typical swine influenza results and this disease is transmissible by contact to other swine. In such experiments control animals inoculated with culture alone remain perfectly normal, animals receiving filtrate alone develop a mild, transient, scarcely recognizable disease, while animals receiving a mixture of the two develop typical swine influenza.

The experimental data obtained in the investigation and briefly outlined in this note indicate that the primary inciting agent in swine influenza is filterable. However, since the influenza-like bacillus is always found in field and experimental cases and is capable experimentally of converting the mild disease caused by the filtrate into clinically and pathologically typical swine influenza, it seems probable that both the filterable agent and the bacillus are etiologically essential to the production of the disease and that, in this rôle, they act synergistically.

It is conceivable that these results may be suggestive in the study of influenza and certain other respiratory infections in man and animals.

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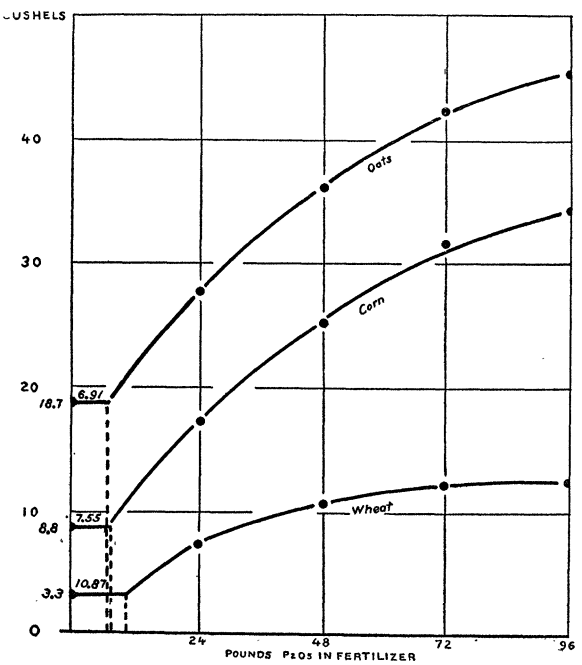
MEASURING ABSORBED PHOSPHATES AND NITROGEN

WHEN phosphate or nitrate fertilizers are applied in certain cases small amounts of the plant food element appear to be absorbed by the soil and held in a condition unavailable to the growing crop. The usual increase in yield with increasing applications appears not to begin until the quantity applied exceeds the amount that can thus be absorbed. Whether potash is similarly absorbed is not yet known.

The purpose of this communication is to point out what seems to be a method of measuring the amount of a plant food element absorbed in the manner above described. The accompanying drawing shows the relation between amount of phosphoric acid applied and corresponding yields of oats, corn and wheat at the Snowshoe Branch Station of the Pennsylvania Experiment Station, as reported in Pennsylvania Bulletin No. 166.



The dots along the curves show actual yields. The curves are calculated from the yields for 24, 48, 72 and 96 pounds of P_2O_5 for each crop by means of the equation

$$Y = M - AR^x \qquad (1)$$