SCIENTIFIC PROCEEDINGS

ABSTRACTS OF COMMUNICATIONS.

One hundred second meeting.

New York Post Graduate Medical School, New York City, November 19, 1919. President Calkins in the chair.

17 (1477)

Blood sugar curves with glucose, lactose, maltose, mannite, and cane sugar.

By CYRUS W. FIELD.

These curves were obtained by feeding normal males colored, with 100 grams of the pure sugar, on a fasting stomach. The dose was as a rule given after the first sample of blood had been taken; this was as a rule at 8 A.M. The second, third and fourth samples were taken one, two and three hours after the ingestion of the sugar, which had been dissolved in a large glass of water.

The urines were tested for glucose up to three hours after the last sample of blood had been taken, and in none of the cases did a specimen ever show the slightest trace of a reducing substance with Benedict's qualitative solution.

The glucose curve was that with which all are familiar, that is rising to its highest point one hour after the ingestion, and then dropping to the normal at the end of the next hour or two.

Maltose gave the same curve as the glucose. Mannite gave the same time curve as the glucose, and maltose, but did not rise to the same height as the other two; the average of five cases gave an increase of only 10 milligrams per 100 c.c. of blood, while that for glucose was 40 milligrams, and for maltose 34 milligrams.

Cane sugar showed a curve that reached its height at the end of the second hour after its ingestion, and had dropped to normal at the third hour. Its average rise for 10 cases was 20 milligrams

per 100 c.c. of blood. Lactose shows only a very slight rise, 4 milligrams, and that at the end of the second hour, as was the case with the other disachrade, cane sugar. I have been unable to consult the literature and so will offer the figures for what they are worth.

18 (1478)

The bacteriology of infectious gaseous gangrene.

By MARSHALL C. PEASE.

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Infectious gaseous gangrene can no longer be conceived of as being necessarily a monomicrobic disease. On the contrary it is frequently the result of an association of bacteria, not all of which are by themselves pathogenic or even under the most favorable condition of animal inoculation capable of causing a pathological lesion. The causative agents of infectious gaseous gangrene are found in a certain group of anaërobes, all of which are capable of elaborating a powerful toxin which has not only a local but also a systemic action. Death in gaseous gangrene is not the direct result of the local lesion but of the absorption of toxin into the general circulation with a consequent general toxemia.

The spread of the local lesion is dependent upon local tissue necrosis. The tissue necrosis in turn is dependent upon the elaboration of bacterial toxins, which are distributed along the line of the muscle sheaths and facia, and through the lymph spaces. There is no evidence that the toxin producing the local tissue necrosis differs from the toxin which is the cause of the general toxemia. If for any reason toxins are not elaborated within the wound or are not absorbed from the wound a gaseous gangrene does not develop despite the fact that there may be within the wound a large number of potentially pathogenic anaërobes.

All the aërobes can be dismissed as a cause of infectious gaseous gangrene. Any effects which they produce are in the nature of a complication. At the most their rôle in this disease process is confined to the absorption of oxygen, the turning upon