but into a Ringer's solution, then it will bulge between the two points A and B on which it is hung, the apparatus being held horizontally. It is easy to understand that the gravitation brings the two points A and B closer to each other, but can never make them diverge. Consequently the gravitation will act against the dilatation. Notwithstanding this the muscle lengthens itself again. Owing to this observation we are able to record curves without the stretching weights, which always deform the curve, while we need not use the poisonous chloroform-benzene mixture.

When it was once stated that a muscle is able to lengthen itself in opposition to the gravitation, I tried if it would even be able to raise its own weight. I succeeded actually in seeing a muscle, placed vertically, fastened at the lower end, lengthen itself after contraction. This fact had been observed some days before, but without my knowledge, by Dr. BAKELS and Mr. PRAKKEN, in this Laboratory.

It being proved that cross-striated muscle tissue dilates actively, two new points of view have been opened:

1. We are able to record curves, excluding all the forces which could deform it.

2. With the aid of this technique it will probably be possible to find a solution to the problem, raised by the result of my experiments. The question is: what intramuscular forces cause this dilatation and through which are they influenced? The dilatation may be caused by the perimysium externum, the sarcolemma, the sarcoplasma or by the fibrils.

I can state at all events, that the perimysium externum is not able to cause the dilatation only by itself (by its elasticity), for even a fragment of muscle tissue, cut out by me, actually lengthened itself again. The sarcolemma, which is a homogeneous elastine-membrane, has a tendency to diminish its surface, in other words: to take the globular shape.

Consequently it will never be able to lengthen the muscle.

December 19th 1919.

Physiology. — "*Identity of the blood-digestive and gelatine-liquefying bacterial actions.*" By Prof. J. J. VAN LOGHEM. (Communicated by Prof. C. EYKMAN.)

(Communicated at the meeting of March 27, 1920).

In investigations on the determination of the so-called specific El-Torvibrios with regard to choleravibrios, I obtained as a result of more general importance a sharper definition of the idea "haemolysis" [1]). By admitting that the changes in the blood, caused by bacteria, may be of different nature, I suggested to understand by *haemolysis* only the causing of *oxyhaemoglobin to come out* from the red blood corpuscles; I opposed to this the *digestion* of blood elements by bacteria, which I indicated as *haemo-digestion*.

Investigations by others (GRIEG [2]) LÖWY [3]) FLU [4]), KRAUS [5]), SOPHIE WOLLMANN [6]) have taught us the practical significance of these for the distinction of choleravibrios. BAERTHLEIN [7]) pointed out the necessity of a right distinction of these ideas, also in the case of other bacteria, whereas SNAPPER [8]) — in connection with his investigations on the decomposition of oxyhaemoglobin in the alimentary canal — has occupied himself with the nature of the digestion.

The following illustrates the latter problem.

Some time ago already, I put the question whether the haemodigestive quality of the choleravibrio is identical with its collolytic capacity and I mentioned several facts which pointed to this possibility.

1. Both the qualities are transient and their decline runs parallel in a certain strain.

2. The processes of the haemodigestion and of the gelatine-lique-

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[1]) Centralbl. f. Bakt. Ie Abt. Orig., vol. 57, 1911; vol. 67, 1913 and vol. 70, 1913; Ned. Tijdschr. v. Geneeskunde, 1915, II, p. 22.

[2]) Indian Journal of medical research, vol. 2, 1914.

[3]) Centralblatt f. Bakt., I, Orig., vol. 75, 1915.

[4]) Geneeskundig Tijdschr. v. Nederlandsch Indië, vol. 53, 1913.

[5]) Die Cholera asiatica und die Cholera nostras, 1914 (with BUSSON).

[6]) Wiener klinische Wochenschrift 1917.

[7]) Centralbl. f. Bakt., I, Orig., vol. 74, 1914.

[8]) Ned. Tijdschr. v. Geneeskunde 1918, II, 1911.

faction are both checked by the appearance of the acid in the nutrient medium.

3. The virtual gelatine-liquefaction-halo, which one can construct by means of EIJKMAN's gelatine-stripe method [1]), is, it is true, not congruent with the haemodigestion-halo in the blood-agar plate; but on the oxyhaemoglobin plate the halos come very nearly together.

4. The strains that are strongly haemodigestive consume casein also intensely; the identity of the casein-digestive and the gelatine-liquefying ferment has been made very plausible by EIJKMAN, by means of the gelatine-stripe method.

SNAPPER's discovery that the digestion of blood has a much quicker process in blood-bile-agar than in blood-agar, incited me to put my hypothesis, stated before, to the test and to enlarge my investigations on the decomposition of oxyhaemoglobin by other bacteria as well.

I want to refer to the fact that the origin of the greenish and clear halo round the colonies of a haemodigestive choleravibrio on the blood-agar plate is actually based on transformation of the oxyhaemoglobin (SNAPPER entered into the details of this to confirm my previous spectroscopic research): first haematine-like bodies originate, which are decomposed in the course of the experiment.

This is also obvious in the decrease of the greenish colour near the stripe-culture, while the pyridin-chromogen reaction takes place slower at that point than at a greater distance from the culture.

On oxyhaemoglobin plates on which, as I pointed out before, the process of the digestion of the oxyhaemoglobin is to be seen clearly with the naked eye by the zones of different colour, it is possible too to indicate the further decomposition of haematin by means of pyridin and sulphurammonium. On the blood-bile-agar plate the cholera vibrio is sustained in the digestion of the oxyhaemoglobin.

By the action of the bile on the blood, the haemoglobin has not only come out (as is the case in the oxyhaemoglobin plate), but has been transformed into haematin-like substances besides. The process of decomposition is progressing already considerably when the cholera vibrio begins to influence it, which is revealed in the quick formation of a broad clearly transparent and colourless halo round the

[1]) The gelatine stripe method is executed by bringing, by means of a platinum-loop, stripes of liquefied gelatine close to the culture on the agar plate. The gelatine becomes solid at an ordinary temperature; so it is possible to trace how far the gelatine stripe (after some time at 22° C. e.g.) disappears from the culture by the action of a ferment.

The figures in this text show how one may construct the gelatine liquefying halo in this way. (In Fig. 1 e.g. the white dotted line).

stripe-culture as an expression of its haemodigestive power. Even choleravibrios that influence the blood-agar-plate exceedingly slowly, are able to form a halo on the blood-bile plate. I tried this halo-formation of the choleravibrio on the blood-bile-agar plate by means of the gelatine-stripe method and compared this one with the halos on blood plates and casein plates.

The result of these experiments which I made with several new and old cholera strains of a very divergent haemodigestive character, is shown half schematically in the following figures.

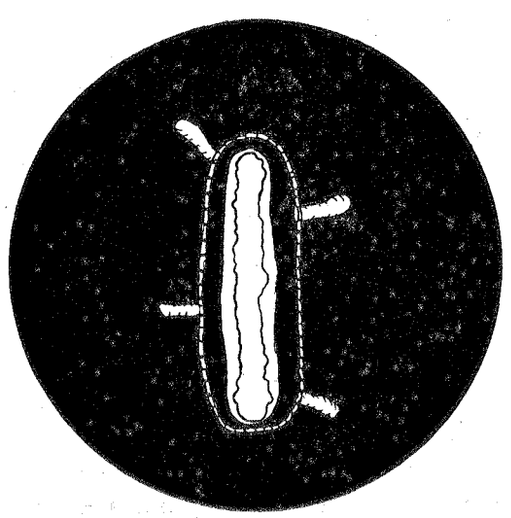

Fig. 1.

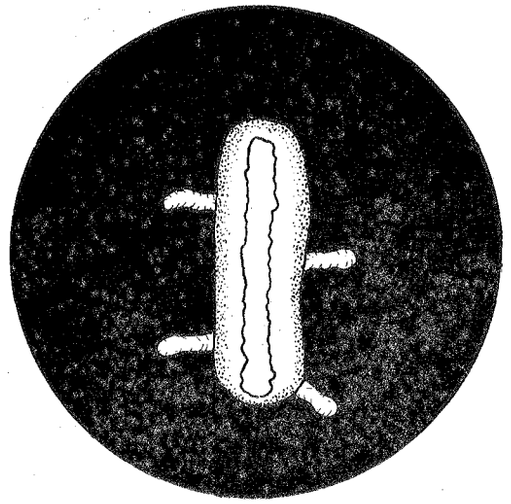

Fig. 2.

Fig. 1. The virtual (white dotted) gelatine-liquefying halo lies considerably beyond the halo of the haemodigestion on the blood-agar plate.

Fig. 2. The zones approach each other very clearly on the oxyhaemoglobin plate; in some cases (as is shown on the figure) there is already an indication of transformation of the oxyhaemoglobin, whose line of demarcation is congruent with the halo of the gelatine-liquefaction.

Fig. 3. *The halos of further oxyhaemoglobin transformation and gelatine-liquefaction are quite congruent on the blood-bile-agar plate,* a condition which agrees with that on the casein plate. (Fig. 4).

By this fact the identity of the oxyhaemoglobin-digestive, casein-digestive and gelatine-liquefying ferment of the cholera vibrio is confirmed.

There is this profitable difference between the blood-bile-agar plate and the blood-agar plate, that the process of haemolysis does not take place in the former.

In this way I was able to compare haemodigestion and gelatine-liquefaction within the group of Proteus-bacteria.

All the Proteusstrains which I have at my disposal (e.g. some indol-producing representatives of *Bacterium vulgare Hauseri, Proteus* X_{19} of WEIL and FELIX producing indol as well and several representatives of the *Bacterium anindologenes* distinguished by me as a separate species) are *haemolytic*, that is to say, they form a halo of blood-agar and cause the oxyhaemoglobin to come out from the blood-broth.

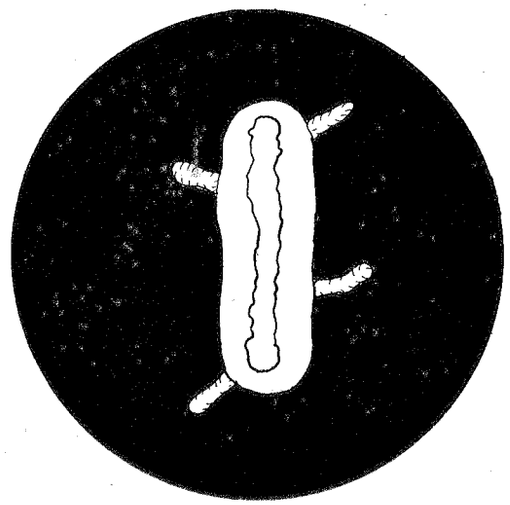

Fig. 3.

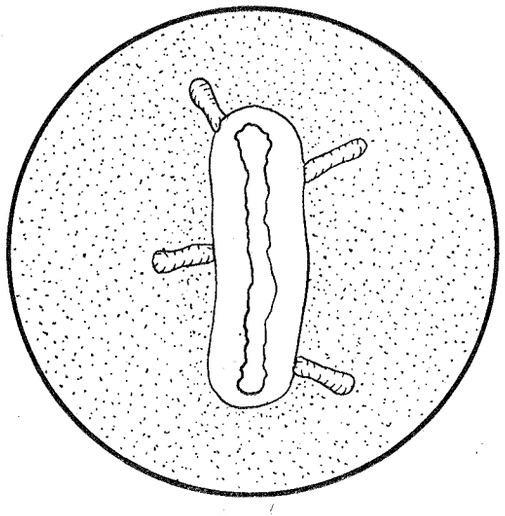

Fig. 4.

They do not all liquefy gelatine. The an-indologenic strain Pneumaturia, which liquefied gelatine strongly 16 years ago, lost this power long ago. *This strain is the only one forming no halo on the blood-bile agar plate.* Other facts may be added to this argument for the conception that also within the Proteus group, oxyhaemoglobin-digestion and gelatine-liquefacting are caused by the same ferment: only the non-haemodigestive Proteus-strain does not digest the casein, as the others do and the liquefaction halos, constructed by means of the gelatine-stripe method are congruent with the halos of haemodigestion on the blood-bile plate.

Moreover I mention the experiments on *B. prodigiosus*, a gelatine-liquefying *coccus* from the air, *B. anthracis*, *Vibrio dunbar*, all of them haemodigestive and liquefying the gelatine, opposed to *B. typhi, coli, B. paratyphi A.* and *B., B. pseudo-tuberculosis rodentium, B. dysenteriae* SHIGA and FLEXNER, which do not form a halo on the blood-bile plate and do not cause the gelatine to liquefy. Both these results are in favour of the identity of the actions in question.

I want to make one more remark; as I pointed out before, sometimes one sees in an organism, of which the casein halo and the gelatine halo cover each other entirely on a nutrient medium, that their congruence has disappeared on another nutrient medium.

When glycerine has been added to the casein plate, on which the cholera vibrio is inoculated, the virtual halo of liquefaction will remain at some distance within the halo of casein-digestion.

So I observed also that the halo of gelatine-liquefaction in a strongly haemodigestive coccus, isolated from the air, is a little larger than the halo of the haemodigestion (on the blood-bile plate). The above-mentioned experimental experience teaches us that this does not contain an argument against the identity of the haemodigestive and collolytic bacterial action.

I conclude by remarking that these experiments teach us that the blood-bile plate as well as the casein plate may serve for the substitution of the broth-gelatine in determining bacteria. This is an advantage while working in tropical littorals, where the use of the nutrient media is subject to difficulties owing to the high temperature of the air.

Amsterdam,
March 1920.

Institute of tropical hygiene, department of the Colonial Institute.