acid gas, without formation of hydrogen gas. In these conditions there is no ferment; on the contrary, cultivated *in vacuo*, or in presence of pure carbonic acid gas, it multiplies, but not without, this time, giving a true fermentation, with evolution of carbonic acid and hydrogen gases, as its life is accomplished without air. This is a new confirmation of our principle, that *fermentation accompanies life without air* —a principle which I am persuaded will one day dominate our conceptions of the *physiology of the cell.*

In the first hours of the development of our vibrio, the rapidity of which, principally in contact with air, is considerable, is under the form of small rolls, very short, gyrating, making pirouettes, advancing, swelling out, and in a soft, gelatinous, flexuous state, which is very marked, notwithstanding the small size of the individuals. In a short time all movement stops, and then it resembles absolutely the *bacterium termo*, slightly constricted as this is, in its thickness, yet specifically very different from the *bacterium.*

Inoculate, with a few drops of this culture, a guinea pig, or a rabbit, and pus commences to form, and to become visible in a few hours. In the succeeding day an abscess is formed, which contains an abundance of pus. This, it will be said, is nothing surprising, because it is known that any solid object whatever, as particles of carbon, a bit of wool which a bullet drives before it, will cause pus to form. I will add that these last experiments have been realized by us with matters previously water-killed, and not containing microscopic germs. But the activity of our microbio, considered as a generator of pus, even though this property may be due only to the quality of a solid body, will be sensibly augmented by the fact of its possible multiplication in the bodies of animals.

The following experiment may serve for confirmation of this fact; let a culture of this organism be divided into two equal portions; one to be water-killed, at a temperature of 110° C. (230° F.) which kills the microbio without in anyway altering its form or volume; then inoculate separately, on two similar animals, equal portions of the water-killed and the non-water-killed half. It is then easily verified that the latter gives more pus than the former, which, in the mean time, furnishes it as any inert solid body would do. Let us add that if the pus formed in each of the two living animals, be sown separately, we shall see that what has been tried on the animal which received the water-killed organisms is absolutely sterile, whilst the pus of the animal which received the organisms of the non water-killed, readily produces, in abundance, the same organisms.

(*To be continued.*)

FATTY EMBOLISMS IN FRACTURES.

(*Translated from Le Progrès Medical.*)

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In 1862 Zenken, while making a post-mortem examination of a man crushed between two waggons, found the capillaries of the lungs full of fat. He believed that this fat proceeded either from the contents of the stomach, or from the liver which was steatomatous, as both these organs had been affected by the traumatism. Zenker considered the circumstance very interesting from an anatomical point of view, but he did not recognize the relation which existed between fatty embolisms and traumatisms, neither did he accord great practical importance to the case he had the opportunity of noticing. The same year Wagner published several cases of fatty embolisms, but he regarded them as proceeding from a metamorphoses of pus, as one of the causes of pyemia. It was only in 1865 that Wagner and Busch arrived about the same time, at an exact and completely similar description of the nature and causes of fatty embolisms in osseous alterations. From that time, the doctrine of fatty embolisms was placed on an unassailable foundation, and anatomical, pathological and chemical researches rapidly followed. It was established that in every fracture there was fatty embolism having its origin in the medulla of the bones; that this embolism was more or less considerable, and that it was very rarely localized in the lungs; but that it might be found in all the tissues of the organism. Finally, it was shown that in a certain number of cases it could be diagnosed during life, and that it was necessary to regard it as a frequent cause of death and as explanatory of the mechanism of the fatal termination in a large number of cases of death more or less sudden after great traumatic lesions, and previously attributed in a comprehensive manner to that which in surgi-