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PULMONARY ANTHRAX AND CONTRIBUTORY FACTORS

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PULMONARY ANTHRAX AND CONTRIBUTORY FACTORS

/Following is the translation of a French-language scientific paper by H. Velu, P. Soulié and B. Bellocq in Bulletin de L'Academie de Medecine (Bulletin of the French Academy of Medicine), Vol. 125, Paris, 1941, pp. 159-161./

Since the work of S. Lodge (1) on the woolsorter's disease and the contributory factor of chalk dust in this disease, many researchers have attempted to specify the mechanism of pulmonary anthrax. The general significance of this problem is very important because we must know if the air inhaled can be a decisive agent of the infection.

The theoretical side of the question, the contamination of laboratory animals with high doses of spores, was immediately solved by Buchner (2). However, in spite of continuous experimental investigation and long and sometimes heated discussions, the practical problem has remained unsolved. Can air which carries only an infinitely small number of anthrax spores be actually a source of contagion or does the ambiant medium play a more important role as assumed by Lodge? This is the question which remains to be solved.

Some authors believe that a lesion is not necessary to permit the spore to enter the alveolae and to germinate; others believe on the contrary that a local lesion is required to bring about infection by the bacillus but, except for Ronzani who appears to have been completely ignored, all others utilized, for the introduction of the bacillus and the irritating factors of the passages (thorax, diaphragm, trachea, intranasal with the syringe) which are never the natural passages, always high dosages and often in the mycelian form which is never that of the usual contamination. They overlooked that there is a sure method to let the virus of anthrax enter the lung in a simple manner at calculable dosages, and without any risk of trauma, through inhalation of an either dry or moist aerosol as originally used by Buchner, Flugge and their school in their work with bacillus anthracis and taken over since then by Trillat in France (3) and W. F. and M. W. Wells in the United States (4).

It must be admitted that neither the remarkable work of Sanarelli (5) which covers the complete bibliography of the question until 1925 but has unfortunately not had sufficient attention on the part of researchers, nor that of numerous authors who attempted to demonstrate or contradict the hypothesis of Besredka on the supposedly extreme receptivity of the skin (6), have brought the solution of this problem.

During his excellent work on the role of industrial gases in pulmonary infections carried out from 1907 to 1909, Ronzani investigated

only chronic irritation, the results of which are quite different from that of single and fleeting action and utilized bacterides, active in themselves, and not spores inherently absolutely inactive at small doses.

We have again taken the problem up with experimentation on mice. Together with Bellocq (7), we initially demonstrated that anthrax could be induced in this species only by inhalation and the deposition in the lung of about not less than 600-1,000 spores.

Our new series of experiments was intended to confirm if a single exposure to one of the industrial gases utilized by Ronzani and more recently by Arloing et al (8), chlorine, insufficient in itself for producing acute symptoms of pulmonary irritation, was able to eliminate the natural defense against infection by the spores of anthrax and to induce the development of pulmonary anthrax identical to the woolsorter's disease.

Our experiments were carried out on 12 groups of five mice each and led us to the following conclusions:

1. A very low dose of chlorine (at a titer of 500 and/or 1,000 derived from the product C x t in which C represents the overall concentration in milligram per cubic meter and t = the time in minutes of the exposure to irritation) has no irritating effect. We should point out that mice are able to control apnea which allows them to tolerate this dosage much more easily than man.

2. With Ct = 45,000, all mice succumbed in 18 hours. This is the minimum fatal dose because not one of them died at a concentration of 30,000. For the rabbit and the guinea pig, Flury admits a dose of 900 milligram per cubic meter for one hour, i.e. a concentration of 900 x 60 or 54,000.

3. Between 1,500 and 30,000, the mice were more or less seriously affected but rapidly recovered.

4. All those subjected to the inhalation of spores through the method developed by Buchner, and followed at the very short interval of hardly more than one hour by exposure to irritation at concentrations of 3,000, 7,500, 15,000 and 30,000, died of typical pulmonary anthrax with terminal septicemia within about 50 hours; the number of spores deposited in the lungs after inhalation varied among the different groups from 75 to 150.

5. The rate of 1,500 also utilized by Arloing et al constitutes a threshhold because, out of 5 mice subjected to such light irritation, 3 died of pulmonary anthrax with terminal septicimia in 48 hours, one died within 72 hours, and the last one survived.

6. Exposure to irritation at Ct = 15,000 effected 24 hours before inhalation of the spores, brought about death under the same conditions as for the preceeding groups (number of spores inhaled: 500).

Obviously all these groups were checked against control groups for

exposure to irritation by chlorine and for inhalation of spores separately and no fatalities occurred in the control groups.

Summary:

a) Exposure to such an irritating gas as chlorine, even at doses insufficient to produce acute symptoms of pulmonary irritation, still resulted in the development of typical pulmonary anthrax with terminal septicemia in mice.

b) Widely used in wool processing, chlorine should therefore be added to the chalk dust indicated by S. Lodge as a contributory factor in pulmonary anthrax among subjects which have inhaled a very small number of spores.

c) For the same reasons for which both the contributory causes and the protective role of the very particular -- we might even say very peculiar -- foci of inflammation of the subcutaneous connective tissue or of the derma have been specified and, in the expression of A. Boquet, "let disappear" (9) the anthrax infection under conditions which have absolutely nothing in common with practice, for the same reasons it appears pertinent to specify, in order to combat or eliminate them, such factors as irritating dust or gas which are likely under customary industrial working conditions and even at scarcely pathogenic doses, to eliminate the natural defense against microbe infections entering through the natural passages. This is a problem of industrial hygiene of the greatest interest.

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