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Deutsche Medizinische Wochenschrift 63(5):182-184, January 20, 1937.

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## **Vitamin C in the Treatment of Croupous Pneumonia**

**A. Hochwald**

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### **VITAMIN C IN THE TREATMENT OF CROUPOUS PNEUMONIA**

The investigations of the pathogenesis of croupous pneumonia (c. p.) were predominantly carried out in two directions in the last few years. Some authors see in c. p. an infectious disease sui generis. Accordingly the therapy is bactericidal-antitoxic. Another investigative direction—Lauche summarized the relevant observations at the time —interprets c. p. (see this number, p. 165) as an expression of a hyperergic reaction of the diseased lung tissue. The most substantial support of this view appears to us to be the observation made by numerous authors that it is only possible to produce c. p. with some degree of regularity in the specifically most sensitive animal. The most essential anatomical criterion is considered not the size of the disease focus, but rather the abundance of the fibrin in the exudate. The question as to the pathogenic significance of a fibrinous exudate was answered by Roessle to the effect that the fibrinous inflammation was the histological expression of an intensified irritation defense.

Our interest was directed at the metabolic processes which introduce the sudden precipitation of large quantities of fibrin, and we also hope to change the hyperergic development by influencing the metabolic pathway. Therefore, we turned our attention to the blood proteins and were inclined to see a pathogenically important symptom in the increase of the readily precipitable proteins found in all allergic diseases.

We were able to expand the finding made by Böger and Schroeder that vitamin C (ascorbic acid) returns the left shift\* of the blood proteins to the norm when administered for a long period of time, in the sense that it is also possible in the short-term experiment to attain a lowering of the globulins or the fibrinogen in the blood with high ascorbic acid doses. Thus, it appeared justified to us to test ascorbic acid for its antiallergic effect.

After we discovered the inhibition of the anaphylactic shock with ascorbic acid in animal experiments and were convinced that only the toxic phenomena of the hyperergic reaction, not its immunizing effect, are eliminated, we began to use vitamin C with c. p. We can illustrate the

\* [Translator's note: Considering the year, this probably refers to paper or starch block electrophoresis.]

therapy carried out in the beginning with a case history.

J. S., 17 years old, at the clinic since April 26, 1935, because of a subacute polyarthritis. May 11, tonsillectomy; afterwards, transient moderate temperature increase and microscopic hematuria. May 17, early temperature, 38.2°. Lungs: right, axillary bronchial respiration. X-ray: Pneumonic infiltration in the right lower lobe. Urine: urobilinogen ++ blood protein, see table. Therapy: 300 mg Redoxon and 10 cc calcium intravenously. The temperature fell to 37.3° by midday; general health somewhat better. In the afternoon, 200 mg Redoxon per os and 10 cc calcium intravenously; cardiac stimulants in addition; evening temperature, 36.5°. May 18, normal temperature. 500 mg Redoxon per os; 2 x 10 cc calcium intravenously in addition. Regarding the lungs, still somewhat bronchial respiration. May 19, temperature continued to be normal and feeling completely well. 2 x 200 mg Redoxon intramuscularly; 2 x 10 cc calcium intravenously. Strong polyuria. Urine: albumen + (trace); urobilinogen, negative. Sediment: isolated leukocytes and erythrocytes. May 20, lungs: by percussion and auscultation, negative. X-ray: negative; urine, negative.

Patient J. S.	Total proteins	Fibrinogen	Globulins	Albumins
Previously	8.37	0.56	3.64	4.2
1 h after 300 mg ascorbic acid	7.76	0.2	2.6	5.0
2 h after 300 mg ascorbic acid	7.8	0.6	4.1	3.1
5 h after 300 mg ascorbic acid	8.2	0.57	4.63	3.0

The case cited here gives the picture of the so-called one-day pneumonia—and because of the flowing transitions and commonalities with a number of other similar cases, as we would like to say, the picture of an artificially produced one-day pneumonia; that is a case in which the inflammation of the lungs was caught while still in the stage of the congestion of the serous inflammation, in which a fully developed croupous pneumonia had not occurred—therefore in which the fibrin precipitation and the necessarily related protracted resolution phenomena did not occur.

Of course there is only relatively seldom, the opportunity to reach a c. p. in its very first hours. The later one begins the treatment, the less influence one will have on the course of the disease, in spite of very large doses of ascorbic acid. From the 3rd day on, pneumonias appear to remain completely uninfluenced. Even on the 2nd day, after the initial chills it is possible to attain only partial successes; a shortening of the disease and alleviation of the symptoms, but no longer an

arrest of the disease. Afterwards, we went on to substantially increase the doses of ascorbic acid; to administer them only intravenously; and to undertake no other therapeutic measures besides that. I would like to summarize the observations we made on the 13 c. p. cases treated in this way up to now.

The most remarkable finding was the improvement of general health (prostration, dyspnea, etc.), which already begins after the first injections and is very noticeable, even if the temperature still remains high. In addition, the rapid defervescence was clear; in our cases it was mostly lytic. In accordance with the lowering of the temperature, often even earlier, there is a leukocyte drop and an improvement of the morphological blood picture. The infiltration of the pulmonary parenchyma, which can be clinically detected, but mainly through X-rays, and which tends to persist for several weeks after the decline of the fever, very quickly vanished in the cases which we observed.

Another symptom of the c. p., the urobilinogenuria, is also often eliminated early. With regard to chloride retention, we were not able to determine a clear effect in the investigation, which is not yet sufficiently precise, however. Within two hours the blood proteins exhibited a lowering of the globulins or of the fibrinogen 3 times; once it remained the same; once a moderate increase in the globulins, with the dosage of  $\frac{1}{2}$  g ascorbic acid (Redoxon, Röche) per dose which we selected, only during this period did we find the ascorbic acid effective with intravenous administration in an acute experiment.

The following gives an overview of our results. The immediate ascorbic acid effect lasts approximately 2 hours. The dose of  $\frac{1}{2}$  g has proven best as the individual dose; an increase in the dose can be undertaken without any danger. From this it follows that this dose is to be constantly repeated at intervals of approximately 2 h until the therapeutic effect is attained. However, one should particularly note that the outlook for favorably influencing the c. p. is all the more favorable the earlier the treatment begins. It is a matter of hours, and only in cases in which the ascorbic acid treatment can be introduced in the first hours does one have the prospect of shortening the treatment. Also, only relatively small doses of ascorbic acid are needed. Here one should however particularly note that from time to time we found failures in which a clear improvement was not noted. Details on this can be found elsewhere.

Our investigations were confirmed by observations made by Gander and Niederberger, who started out with a study of vitamin C retention. The authors were nice enough to allow us to have a look at the manuscript of their research, which is still at press. Gander and Niederberger found out, above all, that simultaneous with the decline in the fever, the vitamin C deficit of the body is saturated and reducing substances once again appear in the urine; also that perorally administered

ascorbic acid is effective. The authors have therefore substantially simplified the vitamin C therapy for the purposes of actual practice, in that they continuously determine the excretion of vitamin C with a very manageable method that they developed themselves and supply ascorbic acid intravenously and perorally until it is eliminated in the urine. Gander and Niederberger also stress the necessity of administering sufficiently large quantities of vitamin C as quickly as possible.

*Briefly summarized*, it is clear from our experiments that ascorbic acid has an inhibitory effect on the anaphylactic shock of experimental animals, and that it can exert certain healing effects on c. p. in humans. With the caution which is recommended for any therapeutic experiment, it is perhaps possible to draw the conclusion that the view of the importance of an allergic mechanism in the pathogenesis of c. p. receives additional support. One has to proceed even more carefully with conclusions for practical-therapeutic application. It is not possible today to say what the optimal therapy of c. p. will look like in the future. Perhaps a combination therapy will be the most successful one, which takes into consideration the bacterial-toxic component (quinine, serum) and the allergic component (ascorbic acid, calcium and requires symptomatic measures (vascular cutoff, circulatory agents) only secondarily. Before that, however, it is absolutely necessary to determine the sole effect of the ascorbic acid and to delimit the indication for its application with a large amount of material in precise investigations.

**References, see:**

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