The Intolerance of Quinine in Influenza-Pneumonia Patients

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The reckless way in which quinine has been doled out wholesale to the public as a prophylactic, and in the treatment of this virulent epidemic of influenza-pneumonia through which we have recently passed, induced me to conduct a few experiments and observations with a view to ascertaining its value or otherwise.

Theoretically the treatment is not sound, and clinically I have had evidence which made me discard it altogether.

Let us take the therapeutic action of quinine internally. In the stomach any salt of quinine is converted into a chloride, and some of it, as such, is absorbed there. Into the blood it is readily absorbed as a chloride, and although the blood is alkaline, it is not precipitated, being probably held in solution by the gasses of the blood. The excess of quinine not absorbed by the stomach—passes on into the intestines and is there precipitated by the alkaline secretion, and
is often excreted unchanged in the faeces. Let us review the action of quinine on the blood and divide it up into what we should consider would be the “pros” and “cons” in its administration in this malady. We will take the “cons” first, as they appear to be the more numerous.

“Cons.”—(1) The arrest of movements in the white corpuscles and their “diapedesis” through the capillary walls; (2) the ozonising power of the blood is reduced; (3) the stability of oxy-haemoglobin is strengthened, consequently the blood cannot yield or absorb oxygen so readily; (4) in large doses it lowers blood-pressure probably by its action on the blood-vessels; (5) it diminishes the action of metabolism, because of its retardation of oxidation; (6) the excretion of uric acid and of other nitrogenous bodies in the urine is diminished. If frequent doses are continued over a prolonged period, then we get congestion of the labyrinth and middle ear. I have also known it to cause epistaxis. Moreover, it is contra indicated in persons subject to gastro-intestinal irritation; and as the victims of this epidemic frequently commenced their illnesses with vomiting and gastric irritation the drug was often intolerant. So much, then, for the “cons.”

The “pros” are: (1) Its usefulness as a tonic, and even a stomachic in non-gastric cases; (2) its antipyretic action on the temperature of the blood; (3) its antiseptic action.

Whilst we are all familiar with its action as a direct poison to the haematozoa which infest the blood in malaria, there is no evidence to show that it acts similarly in the septicaemic, virulent toxæmic affection of the blood such as we have all met with in this recent epidemic.

In several cases I gave a four-hourly dose of the bi-sulphate of quinine (per os) during the acute and early stages of this disease, and in most of them I noticed a decided increase in the cyanosis of the patient after the fifth or sixth dose. In one case with a temperature of 105 (acute lobar pneumonia) I gave a 3-grain dose of the bi-hydrochloride hypodermically on two occasions, and after each administration he developed symptoms of angina pectoris, with distressing dyspnoea.

I am afraid I cannot lay claim to being a bacteriologist, because we general practitioners as a class usually forget more than we retain of our knowledge on the subject; but I would ask the expert: Is it possible for the opsonic action of normal or diseased blood to be arrested or affected in any way by the chemical action of the drug quinine?

We know that the opsonic action—which is dependent on the presence of complement and a small quantity of amboceptor—is capable of acting on the bacilli in the blood, and that these, although not killed, can be so far damaged that they can be taken up by the leucocytes (vide my remarks on white corpuscles).

We all know that the opsonisation of heated normal serum which has been in contact with staphylococcus pyogenes aureus (after removal by centrifugalisation) acquired the property of greatly inhibiting the opsonic action of fresh normal serum, and it would appear that the administration of quinine might have the effect of further doing so.

In a few cases I injected—in various doses—both antistreptococcus and anti-pneumococcus serums on the theory (according to Neufeld) that there are bacteriotropic substances in both the above serums which are thermostable, and which promote phagocytosis, not by stimulating the leucocytes, but by acting directly on the microbe.

Needless to say that I have given quinine a wide berth in the treatment of my influenza-pneumonia patients, at any rate during the latter part of the epidemic, and in future shall refuse to prescribe it in all cases of pyogenic affections of the blood other than its antipathy, malaria.

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