

LOGIC AND CLINICAL DEVELOPMENT

William F. Koch, Ph.D., M.D.

(Written in 1963)

In 1912 and 1913, I reported the discovery of fatally toxic amounts of guanidin and methyl guanidin in the urines of parathyroidectomized dogs. They all died in convulsions in about a week after removing the parathyroid glands. The autopsies showed hemorrhagic degeneration of the liver, kidneys and brain and excessive amounts of lactic acid were eliminated in the urines. The large veins showed striated, ante mortem clots, not of the fibrinous normal type but rather a jellation of the blood colloids.

From these findings and the fact that guanidin presents an amine group of high activation due to its conjugation with an imide group, I concluded that the amine group of guanidin liberated its hydrogen atoms to a highly negative group that had to do with the initiating of the tissue oxidations, for lactic acid was formed and hence fermentation took the place of oxidation of fuels in the tissues. One way in which it could accomplish tissue oxidation suppression in this way was to condense with a carbonyl group, which indeed would be agree to make such a condensation because of its high electronegativity. Then I postulated that tissue oxidations were initiated by the dehydrogenation action of a carbonyl group normally, and when this action was blocked, fermentation would take the place of oxidation, and the amount of energy production would be limited and of too low a potential to supply the electric energy to change the tissue and blood colloids, therefore they would not be dispersed and would settle out as a jell and block the fine capillaries causing hemorrhagic degeneration of the vital organs that was observed. Distribution of oxygen to the tissue is thus blocked also.

The facts then led to the conclusion that the tissue oxidations were initiated by dehydrogenating Functional Carbonyl Groups which I named FCG. Now in order for these carbonyl groups to have the preference as dehydrogenators, they must be activated by receiving quotas of electrons from other groups joined to them, and so I postulated that each carbonyl group was conjugated with an ethylenic linkage which passed its electrons on to the carbonyl group. This combined system of carbonyl and ethylene I named the Functional Carbonyl Group system (FCG SYSTEM).

It was evident then if this postulate were correct that oxidation could be blocked by an azomethine condensation with the FCG, or by an addition to one pole of the double bond that activated the FCG. And it was easy to see how the both means of blocking oxidation could be brought about by the very conditions encountered after parathyroidectomy, particularly in general anoxia of the tissues brought about by the failure of the jellated blood to circulate and carry oxygen to the tissues. This led to a basis for a general type of pathogenesis, since if the FCG started oxidation progressions through dehydrogenations, the free radicals would be formed at the point of removal of hydrogen from the fuel substrate and such a free radical, not having its oxygen to combine to form a peroxide free radical that continued the oxidation process,

would have to add to something else and the closest thing to which it could add is the double bond that activated the FCG. In this way the fuel would become locked into or integrated chemically with the cell's energy producing mechanism, and block its action.

Then it was logical to conclude that if the dehydrogenated substance, the free radical that was formed belonged to some pathogen that entered the field (that would ordinarily be burned as fuel as a procedure of the natural immunity as we assumed) in the absence of adequate oxygen it would add to the double bond of the FCG system and become integrated with the cell energy producing mechanism, and be able to exert its pathogenic properties as an integrated part of the cell, and no amount of serological effort could remove it. Indeed such is the case practically for once a virus has intergrated with the cell it can not be removed by serological procedure. The same applied to pathogens intergrated with the FCG by azomethine condensation. Then according to whether the intergrated substance was one that blocked energy production or withdrew energy or contributed energy to the cell