ALKALOSIS, SODIUM POISONING, AND TETANY.

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In a paper entitled "The supposed relation between alkalosis and tetany," the author (1) reported the results of a series of experiments upon dogs receiving intravenous injections of sodium carbonate or bicarbonate. It was found that it required large amounts of these substances to produce convulsions and that the concentration of sodium in the plasma at that time was approximately the same as in other dogs in whom convulsions were induced by the injection of sodium chloride or sulfate. A specific connection between an increased alkalinity of the blood and the appearance of convulsions was denied.

In a recent paper, Denis and von Meysenbug (2) have reported the results of a repetition of these experiments upon dogs anesthetized with ether and have confirmed the author's statement as to the concentration of sodium in the plasma of dogs in convulsions after the administration of sodium bicarbonate, chloride, or sulfate. However, they differentiate between two kinds of convulsions, those following injections of sodium bicarbonate being due to "tetany," whereas those following injections of either sodium chloride or sulfate are not. The reason for this distinction lies in the differences they observed in the responses to electrical stimulation. The dogs receiving sodium bicarbonate are said to have become hyperexcitable, the others not.

Denis and von Meysenbug (2) state:

"... it is, we believe, generally conceded by both clinicians and physiologists that it is practically impossible to state with absolute certainty whether convulsions observed in man or in animals are or are not due to tetany without a determination of the electrical reactions."

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1 Denis and von Meysenbug (2), p. 48.
It is true that clinicians do generally diagnose as tetany only those convulsive or spastic conditions in which an increased electrical excitability can be demonstrated. But this is really nothing more or less than a matter of definition. Cases showing electrical hyperexcitability are those of tetany; those that do not, are not so classified. “Tetany” is not a clinical entity. There is as yet no satisfactory evidence that tetania strumipriva (or parathyreopriva), idiopathic tetany, and gastric tetany, to mention only three types, have any relation whatever to one another, except for the resemblance between certain of the symptoms. As a matter of fact, clinicians do recognize cases of tetany without electrical hyperexcitability. Thus Falta writes:

“Kahn and I observed a case of chronic tetany with acute exacerbations, in which all the important symptoms of tetany were present pronouncedly, but Erb’s phenomenon, in spite of the presence of severe spasms, was absent during the first days.”

Holmes states:

“1. The appearance of cathodal opening contractions under 5 ma. (and in the absence of certain conditions already mentioned) in children under 5 years of age is pathognomonic of tetany. Cathodal opening contractions are, however, not infrequently absent in cases of clinical tetany.

“2. The appearance of anodal opening contractions with less current than that causing anodal closing contractions, and under 5 ma. during the first 6 months of life is probably pathognomonic of tetany in all cases; their appearance with less current than that causing anodal closing contractions and under 2 ma. is probably pathognomonic up to the fourth or fifth year; thereafter it is of little significance.”

There seems to be no reason whatever for the inclusion of the words “or in animals” in the statement of Denis and von Meyenburg. Most of those working on animals, as indeed many of those working with men, have been apt to use the word “tetany” to denote a spastic condition of the muscles, which is accompanied or followed by tremor, twitching, and tonic-clonic convulsions. And this use of the word seems to have been countenanced by at least some of those clinicians who have had occasion to refer to the animal experiments. Thus Howland and Marriott (5), refer-

*Holmes (4), p. 29.
ring to the experiments of Binger (6) state that these "show that typical tetany may be produced in dogs when the parathyroids are left entirely undisturbed." Binger did not test the electrical reactions of his animals.

Denis and von Meysenbug neglect to mention the fact that MacCallum and his coworkers (7) had previously tested the electrical reactions of dogs receiving injections of sodium carbonate. They had found that, although the electrical excitability was sometimes increased, this was not always the case, although the other symptoms, such as tremors and convulsions, did not differ from those observed when the electrical excitability was increased.

Just what is to be considered electrical hyperexcitability? Denis and von Meysenbug4 state:

"Reactions which are considered characteristic of tetany or spasmodophilia are those showing either anodal reversal (AOC<ACC and <5 milliamperes) or COC<5 milliamperes or CCTe = 5 milliamperes."

The statement of Holmes, which is at least partially contradictory, has already been quoted. But, after all, we are dealing here, not with children, whether 6 months, 2 years, or 5 years old or older, but with dogs; and comparisons must be made with dogs. As far as the author is aware the only previous workers who have tested the electrical excitability of dogs are MacCallum and Paton and their respective associates.

The former (8) wrote:

"Bekanntlich bildet die erhöhte elektrische Erregbarkeit der motorischen Nerven das am leichtesten zu erkennende und konstanteste Symptom der Tetanie. Die Leichtigkeit, mit der eine Reaktion auf die Kathodenöffnung hervorgerufen werden kann, ist besonders charakteristisch, während die Veränderungen in der Erregbarkeit bei Kathoden- und Anodenschlusszuckung und selbst die Anodenschlusszuckung, obgleich sie gewöhnlich der Kathodenöffnungszuckung parallel geht, weder diagnostisch so wichtig noch so umfangreich sind.

"Beim Hunde tritt in gesunden Zustande gewöhnlich keine Kathodenöffnungszuckung bei Strömen unter 5 Milliamperes auf, während die Anodenschlusszuckung bei einem Strom von 1, 5–4 Ma. und die Kathoden- und Anodenschlusszuckung bei Strömen unter 1 Ma. auftreten können."

4Denis and von Meysenbug (2), p. 49.
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MacCallum seems not to have anesthetized his dogs. Certainly his figures for the strength of current normally required to elicit contractions are much lower than those of Denis and von Meysenbug.

Paton and his associates anesthetized their dogs, though only for a short time, before testing the electrical reactions. Their values for the normal excitability are of the same order as those of Denis and von Meysenbug. In view of the importance that Denis and von Meysenbug attach to "anode reversal," it is interesting to read in the paper by Paton, Findlay, and Watson:

"The normal response of nerve and muscle varies greatly. The usual sequence is K.C.C., A.C.C., A.O.C., and K.O.C., but not infrequently A.O.C. is more marked than A.C.C."

And again, under the heading "Tetania parathyreopriva and idiopathic tetany:"

"As a rule no alteration of the normal sequence of the various responses results, but the A.O.C. is more frequently than in health greater [lower in sense of Denis and von Meysenbug—I.G.] than A.C.C., and on occasions may equal or be greater than K.C.C. This is, however, of no diagnostic value."

Since the animals used by Denis and von Meysenbug were anesthetized with ether for periods of from 2½ to 6½ hours, the following quotation from Paton, Findlay, and Watson is not without significance:

"Ether may cause a transient increase which may be followed after about 15 minutes by a slow progressive decrease."

Just as did MacCallum, Paton and his associates regarded the lowering of the strength of current required to elicit the cathodal opening contraction as the most accurate indication of tetany. Of the five experiments with sodium bicarbonate, reported by Denis and von Meysenbug, the cathodal opening contraction was practically, if not entirely, unaffected in four and, in the fifth, the determination was made impossible because of cathodal closing tetanus at 4.5 or 6 milliamperes.

*Paton, Findlay, and Watson (9), p. 312.
*Paton, Findlay, and Watson (9), p. 313.
In view of all these inconsistencies and contradictions, it seems to the author that the work of Denis and von Meysenbug can only be regarded as a confirmation of his conclusion that the convulsions after injections of sodium carbonate or bicarbonate are not due to alkalosis but to "sodium poisoning," a disturbance due to excess of sodium ion of the normal relations between this and other cations. They also indicate that the symptoms observed after the injection of alkalies have nothing to do with tetania parathyreopriva and give additional support to the view previously expressed by the author:7

"Tetany and convulsions are not due to any single cause. Any one of a multitude of disturbances in the equilibrium within certain tissues may be responsible. Convulsions are to be regarded as a sign of approaching or partial disintegration of the neuromuscular apparatus. The defect may occur in any one of several structures and may be due to any one of many causes."

BIBLIOGRAPHY.


7Greenwald (1), p. 298.