drugs, namely, bromide, chloral, alcohol, propylene glycol, paraldehyde and avertin with amylene hydrate (tribromoethanol). Of the coal tar antipyretics, acetylsalicylic acid did not change the convulsive threshold, whereas acetophenetidin and acetanilid raised it, but only in very high doses. Morphine, in doses of 10 and 15 mgm. per kilo, did not change the threshold for the convulsive stimulus. The analeptic drugs strychnine, metrazol, coramine and caffeine were irregular in their effects, indicating a lack of specificity for this phase of cerebral function. On the other hand picrotoxin lowered the threshold to a considerable degree and in relatively low doses. Cocaine and mescaline raised the convulsive threshold moderately, indicating that these drugs depressed rather than stimulated cerebral function at this level. It is suggested that the psychic effects or hallucinations after the use of these drugs may be due to release of cortical inhibition. The sympathomimetic amines generally raised the convulsive threshold to a moderate degree. d-Benzedrine was more effective than dl-benzedrine, whereas l-benzedrine was almost completely inactive; d- and l-ephedrine produced practically no change even in very high doses. Propadrine depressed the excitability, although it is not commonly believed to have a pronounced action on the central nervous system. Tyramine, paredrine and epinephrine caused only inconsistent changes, but neosynephrine and cobefrine depressed the excitability in very high doses.

"The changes with the sympathomimetic amines did not agree well with their effects on the central nervous system as indicated by other methods or observations, and, therefore, leave some doubt as to the causal significance of the convulsive threshold changes for central stimulant effects of these amines. However, this conclusion cannot be accepted without reservation until the possible role of the simultaneous circulatory changes is evaluated. Thyroxine lowered the threshold to epileptiform convulsions more than any of the other agents tested, the threshold being reduced from an average of 2[illegible] ma. in the control period to 15.7 ma. after 19 days of thyroxine medication. This reduction suggests a possible physiological basis for the impaired neuromuscular control and poor poise of clinical hyperthyroidism." [illegible] references.

J. C. M. [illegible]

MADAN, K. E.: *Reflex Cardiac Inhibition under General Anaesthesia.* Brit. J. Anaesth. **18**: 129–131 (Jan.) 1943.

"During an operation under general anaesthesia there may arise various conditions which can cause shock, and reflex cardiac inhibition. One of the causes which I have sometimes observed in my long experience and which has not so far been pointed out, is the therapeutic use of ether by the surgeon, by pouring it into the abdominal cavity in tuberculous diseases of the intestines. . . . Ether is very cold, irritating, and lowers the local resistance of the tissues to infection, and in the most sensitive and rapidly absorbing serous sac it sometimes sets up a dangerous reflex sufficient to cause reflex cardiac inhibition. . . . Death is due to fibrillation and reflex cardiac inhibition. This may happen in spite of atropine given as premedication to lessen vagal irritability. . . . Just as irrigation and the injection of antiseptics in the pleural cavity have been given up due to the shock it produces, so also the instillation of ether in the peritoneal cavity should be condemned, as it may prove dangerous as stated above."

J. C. M. [illegible]