CARBON DIOXIDE DEPRESSION OF RESPIRATION

BY

E. FALKNER HILL

Professor Pask (1955) says in his review of cases where postoperative care proved inadequate to meet the circumstances that arose: "It has recently been stressed that a disorder due to the accumulation of toxic amounts of CO₂ in the body, may be associated with the use of drugs which impair the respiratory mechanism—drugs such as depressant anaesthetics or relaxants."

Some twenty-five years ago in a research on the action of procaine injected into the theca (spinal anaesthesia) the accompanying record was made.

It shows after the injection of 25 mg of procaine in 0.5 ml saline into the theca in the lumbar region of a cat:

1. Fall of B.P. to 60 mm Hg.
2. Slowing of the drum twenty minutes later.
3. Soon after, gradual decline of the amplitude of respiration; at the first arrow distinct slowing of respiration, then normal rate; six minutes later respiration again slowed, second arrow.
4. Accompanying the decrease in the amplitude of respiration there was a slight fall of B.P., but at the first slow period a
definite but slight rise in B.P. took place.

(5) When respiration slowed again the heart failed, as is evident from the fall of B.P.

(6) Two minutes artificial respiration restored the heart and put the B.P. up to 110 mm Hg.

The explanation of the tracing given at the time was as follows: "A slowing of the respiration produced an asphyxia which, stimulating the vasomotor apparatus, initiated a rise in B.P. which precipitated the cardiac failure. It remains to be seen why the respiratory centre failed. The cat had been given a larger dose of a barbiturate than usual and so the respiratory centre had been rendered less sensitive than it is normally. The increasing CO₂ content of the blood had crept on it unawares, had in fact taken it asleep and poisoned rather than stimulated it. The result of artificial respiration supplies the proof of the accuracy of the foregoing diagnosis. Here again, then, the heart failure is secondary to respiratory failure, but the mechanism of the respiratory failure is somewhat different from those previously seen. It is not due to the procaine but to the depressed state of the respiratory centre due to the overdose of the barbiturate."

It would seem that Professor Pask's opinion as to the cause of some of the deaths of which he writes confirms the accuracy of the explanation of the above tracing given twenty-five years ago, and now appreciated as a cause of death in man.

REFERENCE