DISTURBANCE AND RESTORATION OF THE FUNCTION OF THE INTERNAL ORGANS DURING CLINICAL DEATH AND ENSUING RECOVERY

COMMUNICATION II. DISTURBANCE AND RESTORATION OF CERTAIN FUNCTIONS OF THE STOMACH AFTER DEATH FOR 30 MINUTES DURING HYPOTHERMIA

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In a previous communication [1] changes were described in the secretory and motor functions of the stomach in an animal recovering at first from clinical death for 2½ minutes from loss of blood, and then 7 months later, after clinical death for 7 minutes. The prolonged period of clinical death in the latter case is explained by the onset of fibrillation in the initial period of resuscitation. In spite of this severe complication the period of recover of the vital functions passed relatively quickly, and after 4 months it was found possible to subject the animal to a third experiment, along the lines of the previous ones, but with a slight modification.

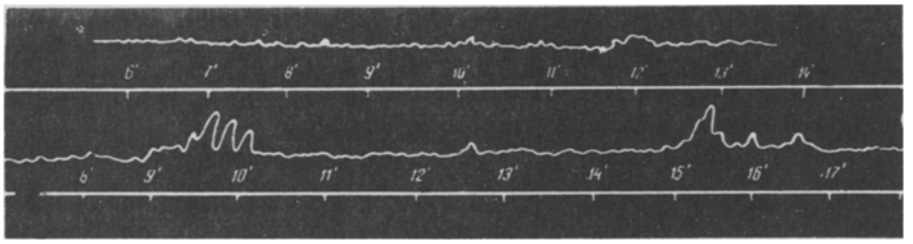
On April 10, 1957 the animal was cooled under pentothal anesthesia.* When the body temperature had fallen to 26°C, bleeding from the femoral artery was started. It took 7 minutes 37 seconds to produce a state of clinical death, which lasted for 30 minutes; restoration of the vital functions was accomplished by means of a combination of resuscitation measures.

The cardiac activity was reestablished 8 seconds after the beginning of resuscitation, and after the intravenous injection of 0.2 ml of ephedrine, the vascular tone was restored on the 27th minute. Spontaneous respiration reappeared later – 109 minutes 52 seconds after the beginning of resuscitation. An attempt to hasten the appearance of respiration by injection of lobeline (on the 80th, 92nd and 95th minutes of resuscitation) led only to the development of a series of rapid respiratory movements (of the inspiratory dyspnea type), which died away again after 28-40 seconds. The spinal reflexes returned 2 hours after the start of resuscitation, and after 10 hours the hearing and vision of the dog was restored. After 16 hours 36 minutes the stance of the animal was fully restored and it once more began to show the aggressive reaction to strangers which was usual for it in normal conditions. After 17 hours the dog walked by itself out of the operating room on the second floor of the building into the vivarium, and after 28 hours it was included in a sham feeding experiment.

EXPERIMENTAL METHOD

For 4 months before the lethal exsanguination experiment under hypothermia, various aspects of the gastric activity of the dog were studied systematically. In this particular series of experiments, besides investigating the magnitude and character of the secretion of the gastric glands in response to sham feeding with raw meat for 3 minutes, and observing the periodic activity of the stomach, we studied the character and rate of evacuation from the cavity of the stomach of a 2% solution of starch or a 0.5% solution of hydrochloric acid introduced separately into it, and also the time of appearance and rate of excretion in the gastric juice of a 2% solution of the dye neutral red, injected subcutaneously into the animal during sham feeding, usually 2 minutes after this had started.

* The experiment was carried out with the assistance of Senior Scientific Assistant V. I. Soboleva. My sincere thanks are due to her.



Character of the periodic activity of the stomach of a dog after undergoing clinical death for 30 minutes under hypothermia. Significance of the curves (from above down): periodic secretion in the first days after clinical death, time marker, (1 minute); periodic secretion 2 months after resuscitation, time marker (1 minute).

After the dog had undergone experimental clinical death for 30 minutes under hypothermia, all the investigations given above were repeated on several occasions in the course of the next 2 months and 10 days.

EXPERIMENTAL RESULTS

The results of our previous investigations [1] showed that after lethal experimental exsanguination, the period of after-effects in the form of abnormalities of certain functions of the animal lasted for about 2 – 3 months. For this reason we accepted the results of observations made not less than 2 months after the second lethal exsanguination experiment on the dog, on December 7, 1956, as controls.

We carried out a small number (3 – 4) of experiments, which in general gave similar results that may be summarized as follows: the volume of gastric juice collected in one hour after sham feeding for 3 minutes varied from 105 to 130 ml, with a total acidity of 0.51% HCl and with a digestive power of 6 mm. After clinical death for 30 minutes under hypothermia, the volume of gastric secretion under the conditions described above showed severe fluctuations: from 19-31-36 to 128-153 ml in one hour throughout the first month after the lethal exsanguination experiment, and from 101 to 196 – 229 ml in one hour throughout the second month of observation. Only during the last 10 days of the experiment was the volume of the gastric secretion established at the level of 139 – 155 ml of gastric juice in one hour of observation after sham feeding for 3 minutes.

Thus during the first period the gastric secretion was sharply depressed: the average value in 10 observations was 73 ml in one hour; but in the second period the average of 8 experiments was 164 ml (hypersecretion).

The comparative data of acidity and digestive power of the gastric juice showed weakening of the functional activity of the gastric glands. The total acidity of the gastric juice, for instance, fell from an average value of 0.51% (results of 16 experiments) to 0.47% (results of 19 experiments). The digestive power also was reduced: from 6.1 mm to 5.4 mm, an average reduction of digestive power of 1615 enzyme units calculated on the basis of one experiment. This reduction of the digestive power and acidity remained stable throughout the period of observation (over 2 months).

In this series of experiments a severe disturbance of the motor function of the stomach could be found by observation of the periodicity of the stomach. In the period preceding the exsanguination experiment, lasting 4 months, we carried out 17 experimental observations of the character and duration of the working periods of the "fasting" stomach. After clinical death for 30 minutes we were able to carry out only 10 experiments on the same lines. The general pattern of the changes in motor function by this method resembles that described by us in the first communication, namely that during exsanguination and subsequent clinical death there is a marked increase in the duration of the periods of "work" (the average duration of one period of "work" rose from 41 minutes before the experiment to 45 minutes) and a decrease in the periods of rest (from an average of 30 minutes to 24 minutes 45 seconds).

The experimental results support our view of the intensification and enhancement of excitation of the nerve apparatus regulating the motor function of the stomach. The significant shortening of the resting periods, which was not observed in the two previous experiments, affords an even greater degree of confirmation of this change in the regulating factors in the direction of an essential weakening of inhibitory influences.

We also determined the character of the motor function of the stomach by experiments on the evacuation of 200 ml of a 2% solution of starch or 200 ml of 0.5% HCl solution from the stomach. These experiments showed

that 200 ml of 2% starch solution, introduced into the stomach, was evacuated within 30 – 50 minutes (the average of the control periods was 38 minutes).

After experimental clinical death for 30 minutes, evacuation of starch from the stomach was prolonged to 46 minutes. In these experiments signs of a slight weakening of motor function were observed, somewhat reminiscent of atony. A completely different picture was seen in response to infusion of 200 ml of 0.5% hydrochloric acid, warmed to body temperature, into the stomach. In the control experiments the hydrochloric acid was kept in the stomach from 2 to 4 hours, and was evacuated extremely slowly and gradually. In this case there took place the "acid" reflex effect on the pyloric sphincter, known since the time of I. P. Pavlov's research, allowing the passage of the strongly acid liquid only so long as it could be neutralized by the pancreatic juice. Experiments in which hydrochloric acid was introduced after clinical death for 30 minutes were invariably unsuccessful throughout the period of over two months. Very soon after the hydrochloric acid was introduced into the stomach, violent antiperistaltic movements began in the duodenum and stomach; a mixture of gastric and intestinal contents was vomited and the experiment had to be abandoned sooner or later. In this way our view of the increased excitation of the motor apparatus of the stomach was confirmed by the experimental findings.

The last group of investigations was concerned with the excretory function of the stomach. During a sham feeding experiment, 2 minutes after its commencement we injected 1 ml of 2% neutral red in physiological saline subcutaneously into the dog. Six control experiments were carried out. As a rule weak staining (beginning of excretion of dye) was observed at the 7th minute of secretion of gastric juice: staining of marked intensity was usually observed during the first 45 minutes of secretion, and then the staining began to fade rapidly. In 5 experiments carried out in the course of the 2 months immediately after clinical death for 30 minutes, the beginning of staining of the gastric juice was somewhat delayed by comparison with the controls – on the average by 2 minutes 42 seconds (against 7 minutes to 9 minutes 42 seconds), and the intense staining of the gastric juice persisted for 65 minutes, whereas in the controls the intense staining did not last for more than 45 minutes. Hence a perceptible weakening of the excretory function of the mucous membrane of the stomach was also present.

As an illustration of the character of the periodic activity of the stomach we show the kymogram obtained in the experiment on June 9, 1957, i.e. 2 months after the dog had undergone clinical death for 30 minutes under hypothermia (see Figure).

It can be seen from the Figure that only the second period is expressed in the form of clear, periodic large waves of contractions, whereas the form of the motor activity in the first period of "work" is characterized by prolonged and chaotic movements resembling those in our previous communication, consisting of small waves of the most diverse size and shape. This example, it seems to us, very clearly illustrates that the motor function of the stomach is far from completely restored even 2 months after experimental clinical death for 30 minutes. In other words, prolonged clinical death, even under conditions of hypothermia, is followed by a very long period of aftereffects, which are shown particularly clearly on the excretory and evacuatory activities of the stomach.

Observations of the state of the animal as a whole after 3 lethal exsanguination experiments enabled us to detect one further special feature which evidently concerns the character of the metabolism in such an animal. This is an unremitting fall in the weight of the dog during the 13 months of observation. On the day of the first experiment (May 31, 1956) for instance, the weight of the dog was 17.5 kg, on the day of the second experiment (December 7, 1956) it was 16.3 kg, and on the day of the third experiment (April 10, 1957) – 15 kg.

The dog died on July 1, 1957, from an accidental cause, when its body weight was only 11 kg. Such a persistent fall in weight cannot be attributed to a change in the diet. On the contrary, we tried in every way to raise the calorie value of the diet by adding fish oil, milk and egg to the food. Twice a day the dog received hot gruel with vegetables, meal, bread and stewed meat; in addition it was always possible for it to take about 200 g of raw meat. We can make two suggestions on the cause of this persistent fall in the animal's weight: either it was the result of a higher level of basal metabolism, in consequence of the lethal exsanguination experiments, or the dog lost weight because the modification in the function of the gastrointestinal tract after the repeated experiments led to a sharp fall in the assimilation of food ingested by the animal. This question can be answered correctly only after special investigations.

The experimental results described thus demonstrate the considerable and prolonged pathogenic aftereffects on various aspects of the gastric function in animals which are associated with lethal exsanguination and clinical death of varying duration.

SUMMARY

A dog was twice subjected to exsanguination with 2- and 7-minute clinical death. In 2 months after the second experiment it underwent a 30-minute clinical death in condition of hypothermia. There was a favourable process of reestablishment of the vital functions, which made it possible to conduct examination of the stomach function (in the chamber on a stand) in 24 hours after exsanguination. Notwithstanding this, not a single function of the stomach was reestablished for a long time. The excretory and the evacuatory functions were found to be the most to suffer.

Prolonged observation of the restorative processes after 3 experiments with exsanguination of dogs demonstrated the presence of acute metabolic disturbances which resulted in systematic, continuous loss of weight, notwithstanding an increased full-value diet.

LITERATURE CITED

[1] M. A. Usievich, Byull. Eksptl. Biol. i Med. 44, No. 11, 36 – 41 (1957).*

* Original Russian pagination. See C.B. Translation.