ELECTRIC SHOCK

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(Received July 11, 1955. Presented by V. N. Chernigovskii, Member of the Acad. Med. Sci. USSR)

Electric shocks may cause severe injury or death. Development of the symptom complex of shock, of various degrees of intensity up to so-called "apparent death", is relatively frequently encountered [6]. Electric shock is often followed by a state of general depression, sometimes merging into prolonged sleep, with fall in arterial pressure and a weak, rapid pulse.

The phenomena of depression with lowered arterial blood pressure and shallow respiration have been reproduced experimentally in animals subjected to electric shock. It was not, however, possible to reproduce the full counterpart of human shock, due to the action of an electric current in animals, in which the dominating feature was either cardiac arrest due to fibrillation or asphyxia due to arrest of respiration. For these reasons the problem of electric shock cannot be considered to have been properly elucidated experimentally, and requires further study.

F. M. Danovich [3] has shown that anesthetization of the tissues at the point of application of the electrodes reduces the gravity of the resulting condition. It follows that the action of the current has a reflex element, as well as a direct effect on the heart and other organs involved in the circuit.

The establishment of a reflex component in the pathogenesis of electric shock is supported by data on the existence of an electric form of shock.

The chief obstacle to the study of electric shock is the development of cardiac fibrillation when current is passed through the body of an animal. It has, however, been shown by F. A. Andreev [2] and others that animals can be subjected to prolonged electrical stimulation under conditions which exclude the possibility of fibrillation. The essential condition for this is that the current should not pass through the thorax.

In this connection special interest attaches to a circuit such that the current passes through the head of the animal only. Under such conditions, reflex stimulation should be superadded to direct action on nerve centers, and this renders the development of electric shock very probable, in the absence of fibrillation. An objection to this procedure might be the development of epileptiform convulsions, as a result of stimulation by the current of higher motor centers. It appeared to us, however, that this might be avoided by appropriate placing of the electrodes, so that the curcuit would be closed through the region of the brain stem. The present paper is devoted to a study of this problem.

EXPERIMENTAL METHODS

The experiments were performed on dogs, which were given the usual preliminary injection of morphine (0.5 ml of 1% solution per kg body weight) 30-60 minutes before commencing the experiment. The animals were immobilized and prepared for registration of arterial pressure and respiration on a kymograph. The electrodes (Pean clips) were attached to the ears, which had previously been shaved and moistened, so that the current passed through the head of the animal. Line voltage was reduced from 220 to 90-100 volts by means of a potentiometer.

EXPERIMENTAL RESULTS

The strength of the current was measured during the experiments by means of an astatic milliammeter (precision 0.5).

In all the ten experiments performed passage of the current caused sharp, reproducible changes in arterial pressure and in respiration. The pressure at first rose sharply, to 180-250 mm or more. This stage lasted for a few minutes, and was succeeded by a gradual fall in pressure, to 40-60 mm after 4 to 10 minutes, at which level it stayed for 1-2 minutes. By lowering the voltage, and hence the strength of the current, it was possible at this stage to prolong the condition of low blood pressure (protracted shock). It was possible, by breaking the circuit, to achieve restoration of pressure to normal levels (Fig. 1).

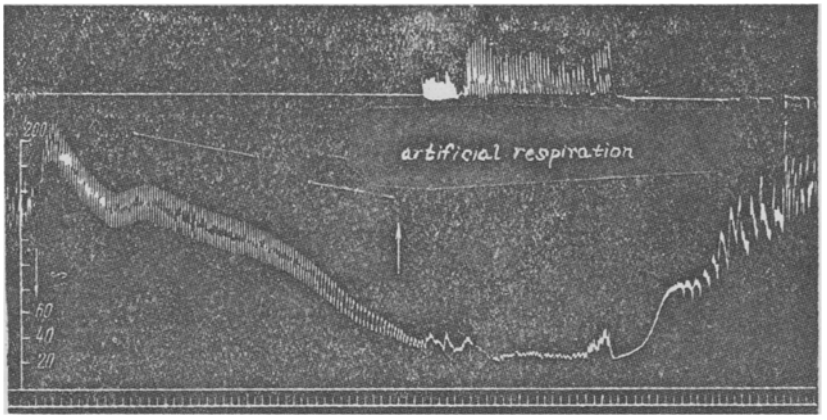

Fig. 1. Changes in arterial pressure and respiration during electric shock. Explanation of tracings (from top): respiration, blood pressure, zero pressure line, time base (5 seconds). (↓) current switched on, (↑)current switched off.

Return to normal levels of arterial pressure often occurred only 1-2 or more minutes after breaking the curcuit; by no means signifying real return of the animal to normality. The dogs had lowered reactivity, in particular to painful stimuli, showing signs of inhibition.

The results of this series of experiments are given in the table.

Changes in Arterial Pressure Due to Passage of Current

No. of expt	Weight of dog (kg).	Voltage.	Strength of current (ma).	Duration of passage of current (min.)	Arterial pressure (mm).		
					initial	at height of rise	at lowest point
1	9.0	100	80—100	8.0	180	220	70
2	6.1	100	60— 70	10.0	160	230	44
3	4.6	60	50—120	28.0	170	270	40
4	1.1	90	60	3.5	160	280	90
5	5.0	110	80—150	3.0	180	190	30
6	16.0	100	70— 95	6.0	180	220	25
7	5.5	90	100	8.0	110	230	76
8	6.0	90	110—155	5.0	108	240	40
9	8.9	100	140—355	10.0	125	215	50
10	8.0	80	110	5.5	120	230	40

Considerable changes in respiration took place during the passage of the current. Switching on usually caused arrest of breathing. In some experiments isolated respiratory movements were observed after the current had been passing for some time, but these were not adequate for restoration of arterial pressure and for improving the general condition of the animal.

These findings permit of the conclusion that it is possible, by passing a current between appropriately placed electrodes, to induce a typical pathological condition, with the usual shock sequence of a stage of stimulation followed by a stage of depression.

The effects observed can in no way be ascribed to cardiac fibrillation; this condition rapidly leads to the death of the animal, which would preclude the possibility of development of prolonged sequential changes in the vascular system.

These changes may, however, be due to asphyxia resulting from arrest of breathing. Although the process continued in spite of the appearance of isolated respiratory movements, these were inadequate, and might not have prevented the development of uncompensated asphyxia.

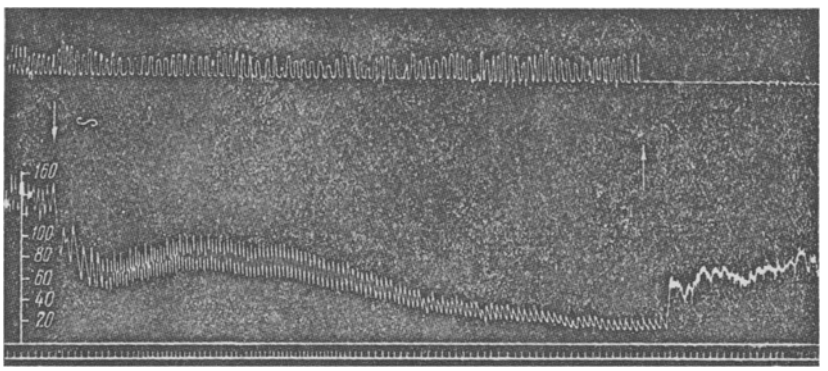

Fig. 2. Changes in arterial pressure in electric shock, under conditions of artificial respiration. Explanation of tracings as in Fig. 1.

This possibility could be investigated by means of experiments in which prolonged passage of current could be effected without the risk of both fibrillation and asphyxia. Ten experiments were performed as before, with the difference that artificial respiration was maintained throughout, by means of a bellows and a tracheal tube.

Under such conditions the passage of the current caused a rise in arterial pressure; this is fully in accord with published data.* It follows that the rise in arterial pressure (first stage) is due not to asphyxia, but to the stimulating effect of the current.

As before, this stage was succeeded by a second stage of lowered arterial pressure. Although the onset of this stage was somewhat delayed under conditions of artificial respiration, it is nevertheless, possible to obtain considerable and rapid fall in pressure if the sensitivity of the animals to shock is increased. It is known that readiness to develop shock can be increased if a certain amount of blood is first removed from the animal [4, 11]. We found the same effect when we repeated our experiments using previously bled animals.

As is evident from Fig. 2, artificial respiration does not under such conditions prevent the onset of the second stage of lowered arterial pressure, and the dogs entered into a state of profound electric shock. It is of interest that with such shock-prone animals the first stage of increased pressure was barely evident, fall in pressure being observed almost immediately. It is also noteworthy that lowered arterial pressure and marked inhibition of respiratory movements persisted for some time after the current was switched off.

It may therefore be consluded that the pathological process observed by us in our experiments on electric shock cannot be ascribed exclusively to the effects of asphyxia, although it is intensified by accompanying asphyxia.

DISCUSSION OF RESULTS

Electrical stimulation causes a sharp fall in arterial pressure, and inhibition of respiration, and leads to a state of general depression of the animal, under conditions which exclude development of fibrillation and of asphyxia. It follows that the morbid process observed by us cannot be considered as being due exclusively to heart failure or to asphyxia.

The state of profound depression observed, and the fall in arterial pressure, preceded by a rise, allow us to consider the condition of the animals as one of shock. Further support for this view is afforded by the observation that switching off the current does not always result in immediate restoration of breathing and of normal arterial pressure; breathing was either not resumed for 1-3 minutes, or was intermittent, and blood pressure remained at a low level. These effects may be ascribed to inhibition of the respiratory and vasomotor centers.

The work of Soviet authors has shown [4, 6, 7] that traumatic shock is due basically to a state of parabiotic inhibition, localized chiefly in the region of the respiratory and vasomotor centers, and arising as a result of their excessive stimulation.

It may be supposed that in our experiments the excessive stimulation was effected by the prolonged passage of an electric current. This is confirmed by the precipitating effect of shock-proneness factors on the response of the organism to electrotrauma. All this points to the existence of electric shock, as a variant of traumatic shock; this should be taken into consideration in the treatment of severe electrotrauma.

It should, however, be remembered that in this paper we are concerned with the existence of electric shock as a natural phenomenon; this is irrespective of whether and to what extent it occurs when the location of the circuit is different. In studying this form of shock we did not overlook the fact that the most common cause of fatal electric shock is auricular fibrillation, as has been shown by V. A. Negovskii [5].

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