Short-term electronarcosis: a new method of general anaesthesia for defibrillation

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Summary
Electronarcosis has been induced 138 times in 72 patients between the ages of 17 and 68 years, after premedication with droperidol, phentanil, and atropine, before electrical defibrillation. Electronarcosis was induced by passage of a current of 170–180 mA at a frequency of 500 Hz through two pairs of electrodes applied to the mastoid region. Electronarcosis was given because it avoided possible respiratory and circulatory complications of anaesthesia. Sinus rhythm was restored after electronarcosis and defibrillation in 69 patients; of these 68 improved clinically, and were subsequently discharged from hospital; one died 11 h later. The patients regained consciousness 2–3 s after treatment, and were completely orientated in time and space. The case history of a 66 year old female patient with mitral disease, pulmonary infarction and circulatory insufficiency is described.

Introduction
Electrical defibrillation of the heart under general anaesthesia in cases of severe circulatory and respiratory disturbances is difficult because of possible interactions with the anaesthetic. This sometimes makes one reject surgical intervention and resort to less effective conservative measures.

For this reason electrical defibrillation cannot be recommended as emergency aid at the patient’s home, except in some cases of sudden death, caused by ventricular fibrillation. Attempts to apply pulsation outside hospital have only emphasized the complexity of the problem (Tabak, 1965; Ageev, 1971).

Our own experience with the treatment of cardiac arrhythmias has impelled us to search for less dangerous techniques of general anaesthesia. With this aim in view electronarcosis has been tested and employed in surgery with inductive currents (Kuzin, Liventsev, Zhukovskiy & Sachkov, 1966; Zhukovsky, 1971). The technique of electronarcosis has been modified according to the specific needs of cardioversion.

Methods
Before electroanaesthesia, patients were given intravenous injections of droperidol (5 ml), phentanil (1–2 ml) and atropine sulphate (0.5 ml of 0.1% solution in 20 ml of 5% glucose solution). In cases of severe hypotension due to cardiogenic shock, arrhythmia,
lung oedema, or asthma, premedication was restricted to atropine or a combination of droperidol and atropine.

Electronarcosis was started when everything was ready for electrical defibrillation. A current of 170–180 mA at a frequency of approximately 500 Hz was passed through two pairs of electrodes attached to the mastoid process by means of an elastic band in the occipital region. The defibrillator (ID-66 or DKI-01) with a power of 4–4.5 kW was used 1.5 s after the beginning of electronarcosis, and the latter was stopped immediately. If the first discharge of the defibrillator was ineffective or the arrhythmia recurred, the defibrillation with short-term electronarcosis was repeated once or several times, with gradual increase of the voltage of the condenser.

Short-term electroanaesthesia was applied 138 times to 72 patients between the ages of 17 and 68 years. Electrotherpay was normally indicated for paroxysmal tachycardia or paroxysmal auricular fibrillation. Tachycardia or tachyarrhythmia, tachypnoea and low arterial pressure dominated the clinical picture of most patients. Arrhythmia was often accompanied by angina pectoris. During prolonged attacks signs of developing circulatory insufficiency such as peripheral oedema, liver enlargement, cardiac asthma or pulmonary oedema were seen, which prompted urgent and necessary application of cardiac stimulation.

Results
The sinus rhythm was restored after one or several defibrillator discharges in 69 patients (Table 1). In 68 patients the return to normal of the cardiac rhythm resulted in improvement of their general state, and all the patients were subsequently discharged. One patient died 11 h after a defibrillation session because of unsuccessful measures to treat the patient’s shock. The sinus rhythm was retained until cardiac arrest. Autopsy confirmed the presence of extensive transmural myocardial infarct of the anterior and lateral walls of the left ventricle and of the anterior part of the interventricular septum.

In three patients the initial rapid auricular fibrillation was not reversed. Shortly afterwards the patients succumbed to progressive cardiovascular insufficiency. Observations

<table>
<thead>
<tr>
<th>Disease</th>
<th>No. of patients</th>
<th>Fibrillation</th>
<th>Paroxysmal tachycardia</th>
<th>No. of cardioversion sessions</th>
<th>Restoration of sinus rhythm</th>
<th>No effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction</td>
<td>23</td>
<td>4</td>
<td>19</td>
<td>29</td>
<td>23</td>
<td>0</td>
</tr>
<tr>
<td>Cardiac aneurysm</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Hypertension</td>
<td>22</td>
<td>9</td>
<td>13</td>
<td>40</td>
<td>21</td>
<td>1</td>
</tr>
<tr>
<td>Arteriosclerotic myocardiopathy</td>
<td>20</td>
<td>13</td>
<td>7</td>
<td>57</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>7</td>
<td>2</td>
<td>5</td>
<td>12</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of patients</td>
<td>72</td>
<td>28</td>
<td>44</td>
<td>138</td>
<td>69</td>
<td>3</td>
</tr>
</tbody>
</table>
have shown that patients lose consciousness as soon as the inductive currents begin to affect the brain, causing anaesthesia of the chest when the condenser discharge passes through. This was gathered from a detailed questioning of patients subjected to short-term electroanaesthesia, none of whom reported any painful sensations in connection with reversion to normal rhythm. The objective measures of the depth of narcosis may be given only by recording the electroencephalogram during short-term electroanaesthesia. This requires further investigation.

The patients regained consciousness 2–3 s after the procedure was over and were orientated in time and space. They usually reported painless sensations such as flashes of light and some tremors of the head during short-term electroanaesthesia. It is of interest that patients usually estimated the duration of electroanaesthesia approximately correctly as lasting a few seconds.

This kind of general anaesthesia did not affect the arterial pressure or the respiration. This was noted when one or several condenser discharges happened to be ineffective. Recovery of normal circulation and respiration in successfully treated patients coincided with cessation of the high-frequency cardiac contractions.

In surgical practice electronarcoosis was applied in combination with muscle relaxants to prevent convulsions during the inductive current. During short-term electroanaesthesia generalized convulsions did not have time to develop. As a rule, only the facial muscles and rarely the shoulder muscles contracted. The only complication of electroanaesthesia was slight biting of the tongue.

Case history

A 66 year old female patient was admitted for treatment with the diagnosis of inactive rheumatic fever; she had mitral insufficiency and stenosis of the left venous orifice. She had infarction of the lung, and circulatory insufficiency of IIIB degree. Some hours after her admission she had an attack of paroxysmal auricular fibrillation at a rate of 160–185/min accompanied by severe retrosternal pain, increased dyspnoea, a respiratory rate of 38–42/min and the appearance of an acute left ventricular insufficiency. Her blood pressure fell from 139/90 mmHg to 80/60 mmHg.

Conservative medical treatment with short-acting digitalis, potassium–magnesium asparaginate, propranolol (Inderal), glucocorticoids, antihistamines and morphinomimetics proved to be ineffective. It was decided to apply electroimpulse therapy.

After premedication with droperidol (4 ml) and atropine sulphate (0.5 ml of 0.1% solution in 20 ml of 5% glucose solution) intravenously, electronarcoosis was applied and a defibrillator discharge of 4.5 kV was administered 1.5 s later. On electrocardiography, restoration of the sinus rhythm with a frequency of 92/min was noted, with single supraventricular extrasystoles. During the procedure, the patient experienced a sensation of ‘sparkling in the eyes’, but she did not report either pain or a blow in the chest.

Within a few minutes of restoration of normal rhythm, the arterial pressure was up to 140/80 mmHg, the number of moist rales in the lungs decreased sharply, the respiratory rate came down to 22–23/min and retrosternal pains and dyspnoea stopped. There were no relapses into arrhythmia during further follow-up at the hospital. After completion of the treatment the patient was discharged in a satisfactory condition.

Discussion

Requirements for very short periods of anaesthesia by inductive current do not differ from those used to suppress the motor and autonomic reaction to pain and surgical inter-
vention, or, according to Huguenard (1966), of the subjective sensation of pain. However, this can be achieved only by comparatively deep inhibition not only of the cerebral cortex but also of subcortical nuclei, which determine behavioural and vegetative defensive and adaptive reactions. Short-term application of inductive current to the nervous system (180–200 mA) does not seem to inhibit the reactions to pain completely.

Acutely unpleasant sensations were more marked in patients with stable haemodynamics and respiration, that is, with efficient neural reflexes. On the other hand, patients in the terminal state hardly reacted to restoration and normal rhythm, which can be explained by an increased threshold to external stimuli during hypoxia.

This makes it reasonable to include an analgesic in the premedication and a preparation decreasing the neuro-vegetative tension but preserving defensive and adaptive reactions. A combination of phentanil (a morphinomimetic which causes a peculiar indifference to pain) with a neuroleptic (droperidol) activating the sympathetic system (Janssen, Niemeges, Verbruggen & Van Neuren, 1963) met these requirements.

The premedication was carried out according to Henshel's (1966) standard technique except that the drugs droperidol (2.5–5 mg), phentanil (0.1 mg) and atropine sulphate (0.25–0.5 mg) were injected intravenously, which allowed us to proceed within a few minutes.

The use of droperidol and phentanil in the dosages stated is permissible in patients with haemodynamic disturbances, particularly in arrhythmic collapse and cardiogenic shock. Chazov, Smetnyev, Petroso, Tabak & Suvorova (1970) have used such a cocktail successfully to treat the pain of the severest forms of coronary attack. The favourable effect of droperidol on the haemodynamics is believed to be associated with redistribution of the regional vascular resistance and regional blood flow due to its ability to reduce the alpha-receptor sensitivity to catecholamines (Corssen, Domino & Sweet, 1964; Bynatyan, Mestcheriakov & Santo 1972). Phentanil is known sometimes to have an unfavourable effect on respiration during dyspnoea and it should be used with care.

The absence of severe complications with short-term electroanaesthesia, the simplicity of its application, and the portability of the NEIP-I apparatus, give grounds to suppose that this method will be used more extensively in the clinic, particularly by first-aid teams outside hospitals. In our opinion, the clear indication for electroanaesthesia is a severely ill patient, in whom it would be risky to give normal anaesthetics. However, it may be a method of choice in cases of planned electroimpulse therapy.

Unpleasant sensations in patients during electromarcosis must be considered. One patient did not consent to a further treatment for relapse into arrhythmia, because he had experienced an unpleasant "tremor in the head". This is insufficient to question the idea of short-term electroanaesthesia, but does indicate the necessity to refine further the application of this technique.

References

Papers in Russian


Papers in other languages