THE EFFECT OF INTRAVENOUS LIGNOCaine ON CARDiac
ARRHYTHMIAS DURING ELECTROCONVulsive THERAPY

BY
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SUMMARY
The effects of intravenous lignocaine and atropine in preventing cardiac arrhythmias were
evaluated in forty-five psychiatric patients undergoing electroconvulsive therapy. During
the electrical shock a more prolonged asystole was observed in the lignocaine than in
the atropine group. During the tonic-clonic seizure ventricular extrasystoles appeared
in six atropinized patients. During the postconvulsive period fourteen patients premedi-
cated with atropine exhibited ventricular extrasystole and tachycardia, whereas in five
pretreated with lignocaine auriculoventricular block was noted. Lignocaine pretreat-
ment significantly reduced the duration of the somatic convulsion. It is concluded that
lignocaine is a valuable premedicant for electroconvulsive therapy in patients with
ventricular arrhythmias or when atropine administration is contraindicated.

Changes in cardiac rate and rhythm are common
in anaesthetized patients undergoing electro-
convulsive therapy. The most severe ventricular
arrhythmias occur among elderly patients (Kline
and Fetterman, 1942; Richardson et al., 1957).
These effects are of considerable practical
importance because cardiovascular accidents re-
main the leading cause of death during this
therapy (Impastato, 1957). A large number of
drugs have been used in attempts to prevent the
cardiac arrhythmias. Of these atropine sulphate
appears to be the most effective and has been
adopted for the routine use in premedication.
Unfortunately, however, arrhythmias frequently
develop in poor-risk patients despite atropinization
(Lewis, Richardson and Gahagan, 1955; Richard-
son et al., 1957).

As early as 1940 Burstein and associates ob-
erved that a local anaesthetic, procaine, is able to
prevent cardiac arrhythmias induced by adminis-
tration of cyclopropane. Since then local anaes-
thetics have been used extensively in the manage-
ment of disturbances in heart rhythm during
surgical anaesthesia. One local anaesthetic, ligno-
caine hydrochloride, is particularly useful because
in therapeutic doses (1 to 2 mg/kg body weight),
it was shown to produce minimal or no depression
of arterial blood pressure and myocardial con-
tractile force of patients under nitrous oxide,
oxygen, halothane anaesthesia (Harrison, Sprouse

It seemed worthwhile, therefore, to investigate
the effect of intravenous lignocaine in preventing
cardiac arrhythmias induced by electroconvulsive
therapy and to compare its efficacy with atro-
pine in the same group of patients. Since a given
individual tends to have the same type of arrhyth-
mia after each shock treatment, each patient
served as his own control.

MATERIAL AND METHOD
Two hundred and thirty-five modified electro-
convulsive therapy treatments were given to forty-
five psychiatric patients of both sexes ranging in
age from 15 to 52 years. The patients were
screened for any possible medical contraindication
to therapy by the Department of Medicine. Since
all the patients were capable of understanding,
the procedure was thoroughly explained to them
during several interviews and their consent ob-
tained. Electroconvulsive therapy was always ad-
ministered in the morning with the patient fasting.

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They received the same intravenous anaesthetic medication on each occasion, namely, sodium methohexitone (Brevital) 0.5–1 mg/kg, 1 per cent solution; this was followed by suxamethonium 0.4–0.7 mg/kg. On alternate days, however, atropine sulphate 1 mg or lignocaine hydrochloride (Xylocaine) 1 mg/kg body weight, 2 per cent solution, was injected intravenously prior to methohexitone. After the fasciculations induced by suxamethonium had subsided, electroshock was induced by an alternating electrical current of 60 c.p.s. for up to 2 seconds duration. Electrodes were applied either bilaterally or unilaterally but the same technique was always used in each patient. Before, during and after treatment, the lungs were inflated with 100 per cent oxygen until spontaneous respiration reappeared.

The duration of the convulsion was measured in the arm not used for injection. For this purpose a rubber tourniquet was applied to the elbow immediately before the injection of suxamethonium. Thereby, the muscles of the hand were not paralyzed. The duration of apnoea, until the reappearance of the first diaphragmatic contraction, was also measured. Lead II of the electrocardiogram was followed continuously with the aid of needle electrodes and a Sanborn Visocardiette electrocardiograph. The recording of the electrocardiogram was started before the injection of drugs and continued up to 5 minutes after shock treatment. There was no interruption in the electrocardiographic tracing following the electrical discharge because the electrocardiograph is not damaged by the current flow. In this manner early changes in cardiac rate following shock treatment could be detected. Pulse rate was counted in the tracing and arterial blood pressure was recorded with a sphygmomanometer (39 patients) or monitored via an intra-arterial needle connected to a Statham PC3 transducer and a Grass 5 Polygraph recorder (6 patients).

Electrocardiographic tracings in the atropine and lignocaine groups were compared during six periods: the control period, following the premedicant drug (atropine or lignocaine), immediately before electroshock, during the tonic and the clonic phases, and in the postconvulsive period. The matched paired t test was used for comparison within each group and the sample t test was used for comparison between groups.

RESULTS

The control period and following medication.

Heart rate was surprisingly high during the control period, both in the atropine (98.6 beats/min; SE 1.6) and in the lignocaine group (100.8 beats/min; SE 1.8; 0.4>P>0.3). A marked tachycardia (mean increase 27.1 beats/min; SE 1.4; P<0.001) was observed following atropine administration. Lignocaine did not produce consistent effects on the heart rate. Individual changes were always of small magnitude, and the average increase observed of 6.9 beats/min (SE 1.2) was significantly lower than that of atropine (P<0.001). In two patients, ventricular extrasystoles present during the pre-drug period disappeared following lignocaine but remained unchanged following atropine administration. Arterial blood pressure was not significantly altered by atropine or lignocaine.

Immediately before electroconvulsive therapy.

Of the other drugs used, methohexitone and suxamethonium, only the latter produced some effect; slight tachycardia (mean increase 4.9 beats/min; SE 0.9; P<0.001) was seen in the atropinized patients. This could, however, be coincidental with the full development of the vagolytic effect of atropine.

The tonic and clonic convulsion phases.

The effects of electroshock on heart rate and rhythm are shown in tables I, II, and III. In most of the patients, no peripheral pulse was found during the passage of the electrical current. This coincided with a sharp but brief fall in blood pressure appearing in the intra-arterial recordings. During the early part of the tonic phase of the seizure the heart rate decreased significantly as compared with the pre-seizure period (P<0.001). Cardiac slowing was more marked in the patients premedicated with lignocaine (−23.0 beats/min; SE 3.1) (fig. 1) than in those treated with atropine (−14.0 beats/min; SE 1.7) (fig. 2). Asystole lasting longer than 2 seconds occurred in twelve patients of the lignocaine group and in only one of the atropine group. During both the tonic and the clonic phases, a moderate increase in blood pressure was present, the rise being somewhat higher and long-lasting following atropine premedication (fig. 3).
**TABLE I**

*Mean cardiac rates and standard error in 235 shock treatments following atropine or lignocaine medication.*

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Atropine</th>
<th>Lignocaine</th>
<th>Difference</th>
<th>SE</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>98.6 ± 1.6</td>
<td>125.7 ± 1.6</td>
<td>100.8 ± 1.8</td>
<td>2.2</td>
<td>2.4</td>
<td>0.4 &gt; P &gt; 0.3</td>
</tr>
<tr>
<td></td>
<td>(112)</td>
<td>(122)</td>
<td>(113)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Atropine</td>
<td>125.7 ± 1.6</td>
<td>130.6 ± 1.6</td>
<td>107.7 ± 1.7</td>
<td>-18.0</td>
<td>2.3</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(122)</td>
<td>(121)</td>
<td>(113)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lignocaine</td>
<td>130.6 ± 1.6</td>
<td>116.6 ± 2.1</td>
<td>107.9 ± 1.8</td>
<td>-22.9</td>
<td>2.4</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(121)</td>
<td>(91)</td>
<td>(110)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tonic seizure</td>
<td>116.6 ± 2.1</td>
<td>152.2 ± 2.1</td>
<td>85.1 ± 2.9</td>
<td>-31.5</td>
<td>3.5</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(91)</td>
<td>(110)</td>
<td>(91)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clonic</td>
<td>152.2 ± 2.1</td>
<td>128.6 ± 2.0</td>
<td>128.6 ± 2.0</td>
<td>-23.6</td>
<td>2.9</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(110)</td>
<td>(91)</td>
<td>(46)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-convulsion</td>
<td>128.6 ± 2.0</td>
<td>92.5 ± 2.5</td>
<td>92.5 ± 2.5</td>
<td>-21.1</td>
<td>3.2</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td></td>
<td>(91)</td>
<td>(46)</td>
<td>(46)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate the number of observations. Differences between number of observations are due to incomplete e.g. recordings in some patients.

**TABLE II**

*Changes in heart rate and differences between drugs effect in 235 treatments following atropine or lignocaine medication.*

<table>
<thead>
<tr>
<th></th>
<th>After drug vs. control</th>
<th>After suxa-methionium vs. after drug</th>
<th>Tonic vs. after suxa-methionium</th>
<th>Clonic vs. tonic</th>
<th>Post-convulsion vs. clonic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine Mean</td>
<td>27.1</td>
<td>4.9</td>
<td>-14.0</td>
<td>35.6</td>
<td>-28.5</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>14.3</td>
<td>10.4</td>
<td>16.0</td>
<td>20.2</td>
<td>26.6</td>
</tr>
<tr>
<td>Number of observations</td>
<td>112</td>
<td>121</td>
<td>91</td>
<td>91</td>
<td>52</td>
</tr>
<tr>
<td>Standard error</td>
<td>1.4</td>
<td>0.9</td>
<td>1.7</td>
<td>2.3</td>
<td>3.8</td>
</tr>
<tr>
<td>Probability</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lignocaine Mean</td>
<td>6.9</td>
<td>0.2</td>
<td>-23.0</td>
<td>43.5</td>
<td>-35.6</td>
</tr>
<tr>
<td>Standard deviation</td>
<td>13.1</td>
<td>16.7</td>
<td>26.8</td>
<td>29.0</td>
<td>24.5</td>
</tr>
<tr>
<td>Number of observations</td>
<td>113</td>
<td>110</td>
<td>91</td>
<td>91</td>
<td>46</td>
</tr>
<tr>
<td>Standard error</td>
<td>1.2</td>
<td>1.6</td>
<td>3.1</td>
<td>3.7</td>
<td>3.8</td>
</tr>
<tr>
<td>Probability</td>
<td>&lt;0.001</td>
<td>0.9 &gt; P &gt; 0.2</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Differences between drugs effect</td>
<td>20.2</td>
<td>4.7</td>
<td>9.5</td>
<td>-7.9</td>
<td>7.1</td>
</tr>
<tr>
<td>Heterogeneity of variance</td>
<td>0.5 &gt; P &gt; 0.2</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.01 &gt; P &gt; 0.001</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Standard error</td>
<td>1.8</td>
<td>1.9</td>
<td>3.5</td>
<td>4.5</td>
<td>5.4</td>
</tr>
<tr>
<td>Significance of difference</td>
<td>0.001</td>
<td>0.01 &gt; P &gt; 0.001</td>
<td>0.1 &gt; P &gt; 0.05</td>
<td>P &gt; 0.5</td>
<td>0.05 &gt; P &gt; 0.01</td>
</tr>
</tbody>
</table>

**TABLE III**

*Cardiac arrhythmias in 235 treatments following atropine or lignocaine premedication.*

<table>
<thead>
<tr>
<th></th>
<th>Before drug</th>
<th>After drug</th>
<th>During convulsion</th>
<th>Postconvulsion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>V.A. (122)</td>
<td>V.A. (2)</td>
<td>Asystole 2 sec or longer (1)</td>
<td>V.A. (6)</td>
</tr>
<tr>
<td></td>
<td>V.A. (2)</td>
<td>Asystole 2 sec or longer (1)</td>
<td>V.A. (6)</td>
<td>Respiratory arrhythmia (2)</td>
</tr>
<tr>
<td>Lignocaine</td>
<td>V.A. (113)</td>
<td>(0)</td>
<td>Asystole 2 sec or longer (12)</td>
<td>(0)</td>
</tr>
<tr>
<td></td>
<td>V.A. (2)</td>
<td>(0)</td>
<td>Asystole 2 sec or longer (12)</td>
<td>(0)</td>
</tr>
</tbody>
</table>

Numbers in parentheses indicate number of observations. V.A. = ventricular initiated arrhythmia.
Electrocardiographic changes in a patient undergoing e.c.t. following lignocaine-methohexitone-suxamethonium medication. Control heart rate (110 beats/min) is not significantly changed by drug administration. Following the shock, there is a marked sinus bradycardia. Sinus tachycardia (150 beats/min) develops during the clonic phase. At the end of the seizure (bottom tracing) heart rate diminishes (110 beats/min).

Electrocardiographic changes in a patient undergoing e.c.t. following atropine-methohexitone-suxamethonium medication. Heart rate increases following atropine. Immediately following the shock there is a slowing of the heart rate. During the clonic phase muscle jerks are superimposed on the tracing. In the middle tracing it is appreciated that every QRS complex is preceded or followed by a convulsive fit. This synchrony between heartbeats and convulsive movements could be seen in some patients at the end of the clonic phase. At the end of the convulsion (heavy mark) there is no change in heart rate (180 beats/min). Three minutes later (bottom tracing), during the after-seizure period, heart rate is 160 beats/min.
During the clonic phase, the heart accelerated in both groups. The mean increase was larger in the lignocaine (43.5 beats/min; SE 3.7) than in the atropine group (35.6 beats/min; SE 2.3), but the difference between drug effect was not statistically significant (P>0.5). Ventricular extrasystoles, single or coupled, appeared in six of the atropine patients and in none of those who received lignocaine.

During the postconvulsive period, the tachycardia diminished abruptly in the lignocaine, and progressively in the atropine premedicated patients (fig. 4). During the next minutes heart rate remained higher in the atropine (123.6 beats/min; SE 2.1) than in the lignocaine group (92.5 beats/min; SE 2.5; P<0.001). Most cardiac arrhythmias appeared during the postconvulsive period. Uni- and multifocal ventricular extrasystoles appeared in fourteen of the atropine premedicated patients. In two of them ventricular tachycardia developed, and was terminated by intravenous injection of lignocaine 100 mg (fig. 5). No ventricular arrhythmias appeared in the lignocaine group. However, three instances of second-degree auriculoventricular block and two cases of nodal rhythm were observed. At the termination of the seizure, the size of the P wave increased in twenty subjects. The incidence of this change was equally distributed between the atropine and lignocaine groups. When spontaneous respiration reappeared a cyclic type of sinus arrhythmia was observed coincidental with respiration. Of the nineteen subjects exhibiting respiratory sinus arrhythmia, seventeen had been premedicated with lignocaine.

The duration of tonic-clonic seizure and postconvulsive apnoea is shown in table IV. Mean duration of convulsions was significantly shortened by lignocaine administration (mean decrease 16.5 sec; SE 1.6; P<0.001). Of the two phases of the convulsive episode, intravenous lignocaine shortened mostly the clonic phase. By contrast to convulsions, the mean duration of postconvulsive apnoea was longer in the lignocaine (69.1 sec; SE 5.0) than in the atropine group (55.8 sec; SE 4.1; P<0.001).

### Table IV

<table>
<thead>
<tr>
<th>Groups</th>
<th>Duration of convolution (sec)</th>
<th>Duration of apnoea (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>41.4</td>
<td>55.8</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard deviation</td>
<td>13.4</td>
<td>26.0</td>
</tr>
<tr>
<td>Number of observations</td>
<td>110</td>
<td>40</td>
</tr>
<tr>
<td>Standard error</td>
<td>1.3</td>
<td>4.1</td>
</tr>
<tr>
<td>Lignocaine</td>
<td>24.9</td>
<td>69.1</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Standard deviation</td>
<td>9.6</td>
<td>29.5</td>
</tr>
<tr>
<td>Number of observations</td>
<td>104</td>
<td>35</td>
</tr>
<tr>
<td>Standard error</td>
<td>0.9</td>
<td>5.0</td>
</tr>
<tr>
<td>Mean difference</td>
<td>-16.5</td>
<td>13.3</td>
</tr>
<tr>
<td>Standard error</td>
<td>3.2</td>
<td>6.4</td>
</tr>
<tr>
<td>Probability</td>
<td>&lt;=0.001</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

**Postconvulsion.**

During the postconvulsive period, the tachycardia diminished abruptly in the lignocaine, and progressively in the atropine premedicated patients (fig. 4). During the next minutes heart rate remained higher in the atropine (123.6 beats/min; SE 2.1) than in the lignocaine group (92.5 beats/min; SE 2.5; P<0.001). Most cardiac arrhythmias appeared during the postconvulsive period. Uni- and multifocal ventricular extrasystoles appeared in fourteen of the atropine premedicated patients. In two of them ventricular tachycardia developed, and was terminated by intravenous injection of lignocaine 100 mg (fig. 5). No ventricular arrhythmias appeared in the lignocaine group. However, three instances of second-degree auriculoventricular block and two cases of nodal rhythm were observed. At the termination of the seizure, the size of the P wave increased in twenty subjects. The incidence of this change was equally distributed between the atropine and lignocaine groups. When spontaneous respiration reappeared a cyclic type of sinus arrhythmia was observed coincidental with respiration. Of the nineteen subjects exhibiting respiratory sinus arrhythmia, seventeen had been premedicated with lignocaine.

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FIG. 4
Differences in heart rate changes at the end of the convulsive seizure. L: patients premedicated with lignocaine. A: patients premedicated with atropine. Bars under tracings indicate the end of the convulsion; thus tracing before the bar is preconvulsion, and after bar is postconvulsion. Numbers refer to cardiac rate. Immediately after the end of the convulsion there is a sharp bradycardia in patients premedicated with lignocaine. No sudden changes in heart rate are observed in patients premedicated with atropine. In the first patient premedicated with atropine (A), clonic jerks are superimposed with cardiac beats. Their disappearance indicates the end of the seizure.

There are other systemic effects which were not quantitated. For instance, there appeared to be less salivation and lacrimation in the atropine group, and a more prompt recovery of consciousness in the lignocaine group. Similar results have been observed by one of the authors elsewhere (Wikinski et al., 1966).

DISCUSSION
The present experiments demonstrate that lignocaine can prevent cardiac arrhythmias induced by electroconvulsive therapy. This protective action differs from that of atropine, since lignocaine blocks premature ventricular contractions otherwise unaffected by atropine, and is ineffective against atrial arrhythmias which are prevented by atropine. These differences may be related to differences in mechanisms of drug action. Atropine binds to specific acetylcholine receptors located mainly at the sinus node and therefore it blocks disturbances of the rhythm associated with slowing of the atrial pacemaker. Lignocaine, on the other hand, has a more widespread action on the heart. By prolonging the refractory period of the conduction system and by increasing myocardial threshold to abnormal stimulation, lignocaine more effectively counteracts the initiation and spread of ectopic ventricular activity (Hoffman and Cranfield, 1964).

The finding that atropine and lignocaine selectively affect certain types of cardiac arrhythmias supports the classic concept that cardiac disturbances induced by electroconvulsive therapy may be due to a complex interplay of sympathetic and parasympathetic influences. Either vagal or sympathetic arrhythmias have been produced by selective stimulation of certain areas of the central nervous system, namely the hypothalamus and reticular formation (Colville et al., 1958; Attar et al., 1962). During generalized stimulation of the brain, as in electroconvulsive therapy, the vagal and sympathetic systems are simultaneously activated. Diaphoresis, pupillary dilatation, increased salivation, are some of the manifestations of this generalized somatic response. For some unknown reason, the cardiovascular response is multiphasic. Immediately after the electrical discharge, there is a vagal predominance manifested by arterial hypotension, extreme bradycardia and even asystole. During the clonic phase, sympathetic activity is predominant with
Effect of intravenous lignocaine on cardiac arrhythmias

**Fig. 5**

Effect of lignocaine on ventricular arrhythmias (e.c.t. = electrical shock).

A: Atropine premedication. Bigeminal rhythm precedes and follows e.c.t. Lignocaine 100 mg i.v. (*) is followed by return to sinus rhythm.

B: Same patient, next treatment. A coupled ventricular arrhythmia is suppressed by intravenous lignocaine pretreatment (*). No arrhythmias develop during or immediately after shock. Ventricular arrhythmia returns 5 minutes following e.c.t., when the protective effect of lignocaine has worn off.

C: Same patient, atropine premedication. Shortly after shock, ventricular arrhythmia develops, rapidly progressing towards a ventricular tachycardia. In the bottom tracing lignocaine 100 mg i.v. (*) suppresses the arrhythmia.

characteristic arterial hypertension and sinus tachycardia. Finally, both vagal and sympathetic influences could manifest during the postconvulsive phase.

This sequence was changed by drug administration. In the lignocaine group, vagal activity could be detected throughout the seizure. Automaticity of the sino-atrial cells was depressed, and occasionally the pacemaker site shifted from the sino-atrial to the auriculoventricular node. When, however, vagal effect was blocked by atropine, neurovegetative discharge enhanced the automatic activity of ventricular pacemakers; thus various forms of ventricular arrhythmias appeared (fig. 6). It is worth noting that the incidence of ventricular arrhythmias of nearly 15 per cent among the patients medicated with atropine is rather low. Lewis, Richardson and Gahagan (1955) recorded ventricular arrhythmias in over 50 per cent of treatments despite full atropinization. The differ-
Cardiac arrhythmias observed during the postconvulsive period. All records were taken within 3 minutes of the end of the convulsion.

Patients A, B, C and D, premedicated with lignocaine. Patients E, F and G were premedicated with atropine.

A: Respiratory arrhythmia.
   Bradycardia during expiration, tachycardia during inspiration. The mark underlying the tracing corresponds to the inspiratory phase.

B: Sinus bradycardia and atrial-initiated beats respectively.

C: Nodal rhythm.

D: Nodal beats intercalated in a sinus pattern.

E: Isolated ventricular beats.

F: Two examples of ventricular coupled beats.

G: Short runs of ventricular tachycardia developing in the same patient.

ence can be explained by the fact that the series reported by Lewis, Richardson and Gahagan included a large number of patients with pre-existing cardiovascular disease, whereas ours did not. Furthermore, in this series every patient was oxygenated before and throughout the convulsive episode.

The utilization of local anaesthetic-like drugs during electroconvulsive therapy is by no means new. Quinidine (Bankhead, Torrens and Harris, 1950) and procainamide (Green and Woods, 1955) have been used before for the same purpose. Both drugs provided a long-lasting protection against ventricular arrhythmias but were soon abandoned because they also produced circulatory depression. In the course of a study of muscle relaxants during electroconvulsive therapy Usubiaga and associates (1967) and Wikinski and associates (1965) found that four local anaesthetics—lignocaine, procaine, cinchocaine (dibucaine) and amethocaine (tetra-caine)—were effective anti-arrhythmic agents. Lignocaine is a short-lasting drug which compares favourably with the agents mentioned above, because in therapeutic doses it exerts no undesirable effects on the tone of the myocardial muscle and peripheral vessels. Furthermore, there is no advantage in using longer-lasting drugs, since protection against electroshock-induced arrhythmias is required for only a few minutes.

Although lignocaine is an effective anti-arrhythmic drug, there are many reasons why it should not be used routinely as a premedicant. First, it does not protect against all types of electroshock-induced arrhythmias. For example, vagal arrhythmias, although less common than without premedication (Gravenstein et al., 1965), are still present. Second, the antimuscarinic action of lignocaine is too feeble to counteract the increase in salivary and bronchial secretions during treatment. This could impede the management of the airway and impair ventilation. Third, lignocaine slightly prolongs postconvulsive apnoea. This action has been suggested as a non-specific displacement of suxamethonium from circulating proteins (Usubiaga et al., 1967). This effect of lignocaine could be a complicating factor when a patient does not recover spontaneous ventilation shortly after the treatment. Finally, the most serious argument against routine use of lignocaine for this purpose is, perhaps, the fact that it shortens the seizure episode and this could diminish the efficacy of the therapy.
The present results, however, show the potential value of lignocaine as a selective premedicant. Patients with premature ventricular contractions before treatment, or those having narrow-angle glaucoma in whom administration of atropine is questionable, could be pretreated with lignocaine and thus receive the benefits of electroconvulsive therapy otherwise unavailable to them. Lignocaine can therefore be regarded as a useful drug to be added to our armamentarium for electroconvulsive therapy.

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REFERENCES


L’EFFET DE LA LIGNOCAINE INTRA-VEINEUSE SUR LES ARYTHMIES CARDIAQUES PENDANT LE TRAITEMENT ELECTROCONVULSIF

SUMMARY

Les effets de la lignocaine intra-veineuse et de l’atropine dans la prevention des arythmies cardiaques ont été examinés chez 45 malades psychiatriques subissant un traitement anticonvulsif. Pendant l’électrochoc on a observé une arythmie plus prolongée dans le groupe de la lignocaine que dans celui de l’atropine. Pendant la phase tonico-clonique des extrasystoles ventriculaires apparaissent chez 6 malades atropinisés. Pendant la phase post-convulsive 14 malades prémédiqués par l’atropine présentent des extrasystoles ventriculaires et de la tachycardie, alors que chez 5 sujets prémédiqués avec lignocaine on observa un bloc auriculo-ventriculaire. La prémédication par la lignocaine reduisit considérablement la durée des convulsions somatiques. On en conclut que la lignocaine représente une prémédication valable pour l’électrochoc chez des malades atteints d’arythmies ou en cas où l’atropine est contre-indiquée.

DER EINFLUSS VON INTRAVENÖS VERABREICHTEM LIGNOCAIN AUF HERZRYTHMUSTÖRUNGEN WÄHRENDE DER ELEKTROSHOCKTHERAPIE

ZUSAMMENFASSUNG