CHANGES IN PULSE RATE AND BLOOD PRESSURE AFTER EXTUBATION

BY

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SUMMARY

Three groups of fifty adult patients had pulse and blood pressure readings taken after extubation during light anaesthesia. In the first series, patients receiving volatile or gaseous anaesthetics showed mostly a rise in pulse rate and blood pressure. In the second series a nitrous oxide, relaxant, atropine, neostigmine sequence was given, and fewer showed a rise in blood pressure and heart rate. In the third series conditions were similar to those in the second series, but extubation was performed before neostigmine administration, and in this group more showed a decrease in pulse rate than a rise, but most showed a rise in blood pressure. The significance of these results is discussed with regard to the safety of extubation during light anaesthesia.

While the advantages of extubation during light anaesthesia have long been recognized (Kaye et al., 1946; Gillespie, 1950), it has been postulated that vago-vagal reflexes may cause cardiac arrest at the time of extubation (Keating, 1961; Stephenson et al., 1953; Shumacker and Hampton, 1951). This study was undertaken to find what pulse and blood pressure changes normally occur on extubation during light anaesthesia, whether these changes are altered by atropine or neostigmine administration, and if any of the changes found contraindicate extubation under light anaesthesia.

STUDY

One hundred and fifty fit adult patients were studied. Pulse rate and systolic blood pressure recordings were taken 1 minute prior to extubation, then at 1-minute, 3-minute and 5-minute intervals after extubation. All patients were at a very light stage of anaesthesia, some actually being awake, when extubation was performed. Many bucked or coughed on removal of their tubes. Cuffed endotracheal tubes smeared with 5 per cent lignocaine ointment had been used in all cases. Pharyngeal suction was performed immediately prior to extubation, but no tracheal suction was employed. All efforts were made to avoid hypoxia and hypercarbia, throughout.

A Burdick Telecor pulse-monitor with a digital-pulse pick-up was used on all patients, and a standard mercury sphygmomanometer cuff was placed on the upper arm. Pulse rate was counted from the monitor, and systolic blood pressure was measured by inflating the cuff until the signal from the pulse-monitor was obliterated. In the author’s experience, these methods have proved more reliable than palpation of the pulse. Arrhythmias are more readily detected with the monitor than by mere palpation, though not as adequately as by electrocardiography. Unfortunately, an electrocardiograph was not available for this study.

The patients have been divided into three groups:

Series A. These were 50 patients who were intubated after thiopentone and suxamethonium and thereafter were breathing spontaneously. They received light anaesthesia with nitrous oxide, oxygen and either halothane (33 patients), ether (8 patients), or trichloroethylene (4 patients). The remaining 5 patients were given cyclopropane and oxygen. All anaesthetic agents were discontinued some time before extubation, oxygen alone being administered until after extubation.

Series B. Fifty patients were intubated with a thiopentone, nitrous oxide, oxygen, tubocurarine technique with pulmonary hyperventilation by manual intermittent positive pressure; they were given intravenous atropine and neostigmine at
the end of operation to reverse residual curarization. Ten minutes after atropine injection, neostigmine was administered and manual inflation continued with oxygen alone until spontaneous respiration became adequate. Extubation was then performed, this occurring 3 to 5 minutes after the injection of neostigmine.

**Series C.** These were 50 patients having the same anaesthetic sequence as those in series B, but extubation was performed 5 minutes after atropine administration, and neostigmine was given 5 minutes later (10 minutes after atropine). Most of these patients were not breathing spontaneously on extubation, so that artificial ventilation was continued with oxygen given through a facepiece and oropharyngeal airway until respiration was adequate. This was not always an easy procedure.

In series B and C, atropine doses ranged from 0.9 mg to 1.2 mg, while neostigmine doses were 1.25 mg to 2.5 mg. Pulse rate changes of less than 6 per minute and systolic blood pressure changes of less than 5 mm Hg were classified as "no change".

**RESULTS**

**Series A.** In this group, before extubation, the average pulse rate was 92 beats/min, and the average systolic blood pressure was 134 mm Hg. Within 3 minutes of extubation, the majority of patients showed an increase in pulse rate and blood pressure, as shown in tables I, II and figure 1. The highest pulse rate after extubation was 144 beats/min, while the slowest was 72 beats/min. Whatever the anaesthetic, the predominant response was a rise in pulse rate and blood pressure.

**TABLE I**

*Pulse changes on extubation (Series A).*

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>37</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Per cent</td>
<td>74</td>
<td>20</td>
<td>6</td>
</tr>
</tbody>
</table>

**TABLE II**

*Blood pressure changes on extubation (Series A).*

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>31</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Per cent</td>
<td>62</td>
<td>20</td>
<td>18</td>
</tr>
</tbody>
</table>

**TABLE III**

*Pulse changes on extubation (Series B).*

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>23</td>
<td>17</td>
<td>10</td>
</tr>
<tr>
<td>Per cent</td>
<td>46</td>
<td>34</td>
<td>20</td>
</tr>
</tbody>
</table>

**Series B.** In this series, where atropine and neostigmine were given prior to extubation, the average pre-extubation figures were a pulse rate of 92 beats/min and a blood pressure of 144 mm Hg. Within 3 minutes of extubation, more patients showed a rise in pulse rate and blood pressure than a fall (tables III, IV and fig. 1), but changes were not as marked as in series A. The maximum pulse rate was 144 beats/min, while the slowest was 60 beats/min.

**Series C.** This group, where atropine was given before extubation (and neostigmine after), showed
average pre-extubation levels of 116 beats/min for pulse rate, and 150 mm Hg for blood pressure. Within 3 minutes of extubation, more patients showed a pulse rate decrease than an increase, while a large number showed no change (table V and fig. 1). Most cases showed a rise in blood pressure (table VI and fig. 1). Pulse changes were less in this group than in the other series, but the maximum pulse rate was 156 beats/min, while the slowest pulse rate recorded was 72 beats/min. The range of pulse changes for each group of patients is shown in figure 2, while blood pressure changes are presented in figure 3. In all series, there was a tendency for both pulse and blood pressure levels to return to pre-extubation values at the 5-minute period.

**Arrhythmias.**

One patient in series A, receiving halothane, showed bigeminal rhythm which reverted to

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**TABLE IV**

**Blood pressure changes on extubation (Series B).**

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>24</td>
<td>15</td>
<td>11</td>
</tr>
<tr>
<td>Per cent</td>
<td>48</td>
<td>30</td>
<td>22</td>
</tr>
</tbody>
</table>

**TABLE V**

**Pulse changes on extubation (Series C).**

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>13</td>
<td>19</td>
<td>18</td>
</tr>
<tr>
<td>Per cent</td>
<td>26</td>
<td>38</td>
<td>36</td>
</tr>
</tbody>
</table>

**TABLE VI**

**Blood pressure changes on extubation (Series C).**

<table>
<thead>
<tr>
<th></th>
<th>Increase</th>
<th>Decrease</th>
<th>No change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>35</td>
<td>13</td>
<td>2</td>
</tr>
<tr>
<td>Per cent</td>
<td>70</td>
<td>26</td>
<td>4</td>
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</table>
normal on extubation. In series C, one case showed extrasystoles after extubation, but a normal rhythm returned spontaneously within a few minutes. No arrhythmias were noted in series B.

**DISCUSSION**

While Keele and Neil (1961) state that stimulation of sensory nerves produces various effects on the heart, it can be seen that in this study the predominant features after extubation were a transient rise in pulse rate and systolic blood pressure. Increased heart rate is probably due to afferent impulses from the larynx, reflexly causing either an increase in sympathetic impulses or a decrease in vagal (parasympathetic) impulses to the heart. When bradycardia occurs it is probably due to a reflexly increased vagal tone, or decreased sympathetic impulses.

A rise in systolic blood pressure would be due to increased sympathetic impulses to the peripheral vessels causing vasoconstriction while a fall would be due to decreased sympathetic impulses causing vasodilatation. Autonomic effects on heart rate and force of contraction will also influence blood pressure changes.

Keele and Neil (1961) also state that mild anoxia and hypercarbia produce tachycardia, while severe anoxia causes bradycardia, so while efforts were made to eliminate these states, it is possible that a mild degree of anoxia or hypercarbia could have influenced the results.

For the purposes of this discussion, a rise in pulse rate or systolic blood pressure is described as a sympathetic response, while a fall in pulse rate or systolic blood pressure is described as a parasympathetic response.

The predominant response to extubation in series A is sympathetic, which is similar to the response obtained by King and his co-workers (1951) in their study of intubation. This appears to be the normal response to extubation, but it is possible that a mild degree of anoxia or hypercarbia might result in more showing a sympathetic response than if the presence of hypoxia and hypercarbia could be absolutely precluded. It would seem that the normal balance of autonomic impulses to the heart and vessels is more readily altered to sympathetic preponderance, than parasympathetic, but as some patients showed bradycardia and a fall in blood pressure, the balance of autonomic impulses may vary from person to person.

In series B, where atropine and neostigmine preceded extubation, sympathetic responses were more common than parasympathetic, though not as marked as in series A. The figures for series B are comparable with a similar group studied by Rawstron and Hutchinson (1963), and it appears that the injection of neostigmine, which increases vagal tone on the heart, modified the usual response so that a greater number showed parasympathetic effects.

The routine in series C was employed in order to establish whether the parasympathetic response could be abolished by extubating after atropine (which blocks vagal action) but before neostigmine administration. The results show that there was actually a slight increase in the number of patients showing a fall in pulse rate, a considerable decrease in those showing cardiac acceleration, and a larger number showing no change in pulse rate, compared with series B. Thus atropine did not block the parasympathetic response, but the number of patients showing pulse rate changes and the degree of these changes was less in series C than in the other series, so that atropine did have a stabilizing effect on the heart rate. As the pre-extubation pulse rate in series C was higher than in series B, because of the action of atropine, some heart rates may have already been at a maximum when extubation took place, so that they could not accelerate further. Also, elevation of blood pressure was more common in series C than in series B, which indicates that a sympathetic response may still have been dominant.

Over all, there was a fair range of changes in both pulse rates and systolic blood pressures, but no case caused alarm by either an excessively fast or slow pulse rate (maximum 156 beats/min, minimum 60 beats/min), or by an excessively high or low blood pressure (maximum change 50 mm Hg). Also, changes were transient, as in all series there was a tendency to return to pre-extubation levels within 5 minutes.

Few arrhythmias were noted on extubation, but the limitations of a pulse-monitor must be recognized.
CHANGES IN PULSE RATE AND BLOOD PRESSURE AFTER EXTUBATION

Converse et al. (1952) showed that, with a variety of anaesthetic agents, the response to extubation, if any, was sympathetic. Keating (1961), Stephenson et al. (1953) and Shumacker and Hampton (1951), however, all state that cardiac arrest may follow extubation because of a vago-vagal reflex. They also agree that hypoxia or anoxia is involved in these cases of cardiac arrest, and Glenn (1953) stresses that decreased oxygenation of the myocardium is the important causal factor, considering it rare for vagal stimulation alone to cause arrest.

From this study, it can be seen that when hypoxia and hypercarbia are avoided, the predominant response to extubation is sympathetic, as shown by a rise in pulse rate and blood pressure. Some vagal responses did occur, but none was of sufficient intensity or duration to cause concern. These parasympathetic reflexes were more common after neostigmine, but extubation after atropine and before neostigmine did not abolish this effect although it did stabilize the heart rate. However, any advantage this offers is largely outweighed by the difficulty of maintaining artificial ventilation that this method entails.

CONCLUSIONS

Under normal conditions, the predominant response to extubation is a rise in pulse rate and systolic blood pressure, but whatever the nature of these circulatory changes, none appeared dangerous in either degree or duration, so that it seems safe to extubate during light anaesthesia, as long as efforts are made to avoid hypoxia and hypercarbia.

ACKNOWLEDGMENTS

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REFERENCES


LES MODIFICATIONS DU POULS ET DE LA T.A. APRES L'EXTUBATION

ZUSAMMENFASSUNG