EFFECTS OF ANAESTHETICS AND THEIR INTERACTIONS WITH NEUROMUSCULAR BLOCKING AGENTS IN CATS

BY

R. HUGHES

SUMMARY

In cats lightly anaesthetized with chloralose, ventilation with 75 per cent nitrous oxide did not affect arterial blood pressure, heart rate or the responses of gastrocnemius muscles to indirect stimulation; blockade by tubocurarine and gallamine was significantly enhanced but that by suxamethonium was almost unchanged. Ventilation with approximately 0.5 per cent halothane caused hypotension and bradycardia and slightly depressed the responses of the muscles to indirect tetanic stimulation leaving the single twitches unimpaired. The intensity and duration of paralysis by tubocurarine were significantly increased but a similar tendency for gallamine did not achieve significance; the action of suxamethonium was virtually unaltered. Infusion of thiopentone, 8–10 mg/kg i.v., lowered arterial blood pressure and heart rate and slightly increased the tetanic contractions of the muscles without altering the twitch responses; blockade by tubocurarine and gallamine was significantly enhanced, whereas that by suxamethonium was unchanged. Halothane and thiopentone potentiated the hypotensive action of tubocurarine.

It has been reported that inhalation of 50–80 per cent nitrous oxide had no paralyzing action in rabbits, either alone or in conjunction with tubocurarine (Naess, 1950), but slightly increased the twitch responses of the tibialis muscle in midcollicular decerebrate cats (Ngai, Hanks and Farhie, 1965).

Halothane did not significantly alter neuromuscular transmission in rabbits but intensified paralysis by tubocurarine (Watland et al., 1957). Burn and associates (1957) obtained similar results in cats and observed that 1.35 per cent halothane antagonized the action of suxamethonium.

Small amounts of thiopentone may increase the responses of isolated rat diaphragms to indirect and direct stimulation but high concentrations cause inhibition (Secher, 1951; Sirnes, 1954). Analogous effects have been produced by subnarcotic and anaesthetic doses in intact dogs, cats, rabbits and rats (Kraatz, Gluckman and Shields, 1953; Sirnes, 1954; Quilliam, 1955; Borgman et al., 1960). The stimulant action of thiopentone may cause a transient antagonism to tubocurarine and decamethonium but the main effect is a depression of muscular activity which potentiates paralysis by these drugs (Kraatz, Gluckman and Shields, 1953; Sirnes, 1954; Borgman et al., 1960).

The following experiments compare, under controlled conditions, the effects of nitrous oxide, halothane and thiopentone on neuromuscular transmission and on blockade by tubocurarine, gallamine and suxamethonium in groups of cats lightly anaesthetized with chloralose. Cardiovascular effects were also investigated.

METHODS

Cats of either sex weighing between 2.4 and 3.4 kg were used; anaesthesia was induced with 4–8 per cent halothane and lightly maintained with chloralose (40–50 mg/kg i.v.). The animals were prepared as described previously (Hughes, 1970a, b) and ventilated with 100 per cent oxygen using a Starling pump. The contractions of both gastrocnemius muscles were recorded in response to supramaximal stimulation of the

sciatic nerves every 10 sec, one with single shocks, the other with tetanic bursts of 30 shocks/sec. Blood pressure was recorded from a carotid artery. The left brachial artery was cannulated for sampling of blood for pH, Pco₂, and Pao₂ measurements; details of the technique have been given elsewhere (Hughes, 1970b). Effects of 75 per cent nitrous oxide, approximately 0.5 per cent halothane and an intravenous infusion of 1.25 mg/min thiopentone sodium (total 8–10 mg/kg) were each studied in combination with light chloralose anaesthesia required for the surgical preparation. The inspired concentration of halothane and the dose of thiopentone were just less than those found to cause severe hypotension.

Experimental procedure.

The experimental design was similar to that already reported (Hughes, 1970b). After preliminary tests in each cat to determine paralyzing dosages, control doses of the neuromuscular blocking agents were given before and after the test in combination with the anaesthetic under investigation. The following intervals were allowed between dosing to reduce cumulative effects and to give sufficient time for administration of the anaesthetics: 60–75 min for tubocurarine; 45–60 min for gallamine; 30–45 min for suxamethonium. Inhalation of nitrous oxide and halothane was commenced about 30 min before giving the paralyzing drug and terminated when some recovery from blockade had occurred. Infusion of thiopentone (5 mg/ml) into the right subclavian vein was begun 10–15 min prior to the test and continued for 5–15 min after administration of drug.

Tables of results show those experiments in which some time after giving the anaesthetic the neuromuscular paralyzing effects of the drugs returned approximately to the earlier control levels. A few animals in which the neuromuscular paralysis became greater with each successive dose of the drug, irrespective of anaesthetic administration, are mentioned only in the text.

Statistical analyses.

The results were assessed by t-test applied to differences in experimental variables during treatments. Significances are indicated in the tables.

RESULTS

Nitrous oxide (table I).

Mean arterial blood pressure and also (not tabulated) the heart rate and the tetanic and twitch responses of the gastrocnemius muscles of cats lightly anaesthetized with chloralose were usually unaltered during ventilation with 75 per cent nitrous oxide in oxygen. Mean Pao₂ was lowered significantly by 221–336 mm Hg while mean pH and Pco₂ remained almost constant.

Paralysis of the twitch responses by tubocurarine and by gallamine (fig. 1) was significantly enhanced in each of four cats during ventilation with nitrous oxide. Blockade of the tetanic responses was often complete in the control situation but in the presence of nitrous oxide recovery times tended to increase. Paralysis by suxamethonium and the time taken for recovery were almost unchanged by nitrous oxide in four cats; a fifth cat became more sensitive to successive doses of the drug irrespective of nitrous oxide administration.

Halothane (table II).

Ventilation with approximately 0.5 per cent halothane in oxygen significantly lowered mean arterial blood pressure by 49–65 mm Hg and reduced heart rate (c. 25 per cent); the tetanic contractions of the gastrocnemius muscles evoked by indirect stimulation were often slightly depressed (mean reduction 8 per cent, SE 4 per cent) but the responses to single shocks were usually unimpaired. Mean Pao₂ was slightly reduced by 16–45 mm Hg, changes in mean pH and Pco₂ being minimal.

Halothane significantly deepened blockade by tubocurarine and delayed recovery in each of four cats (as fig. 2). A fifth experiment was abandoned because paralysis increased with each dose irrespective of the anaesthetic. The hypotensive effect of tubocurarine was often potentiated. Paralysis of the twitch responses by gallamine was moderately intensified and prolonged by halothane in four cats but not significantly. Blockade became deeper with each successive dose of the drug in two other cats. Though paralysis of the tetanic responses by gallamine was often complete in the control tests, recovery times tended to increase in the presence of halothane. The paralyzing action of suxamethonium during ventilation of four cats
TABLE I
Effects in cats, lightly anaesthetized with chloralose, of inhalation of 75 per cent nitrous oxide in oxygen on the pH, $P_{CO_2}$, $P_{O_2}$ and pressure of arterial blood, and on neuromuscular paralysis by tubocurarine, gallamine and suxamethonium. Paralysis was determined on both gastrocnemius muscles stimulated indirectly every 10 sec, one with single shocks and the other with 30 shocks in 1 sec. Mean values and ranges for groups of four cats are shown for before (pre-control), during, and after (post-control) the treatment period. Asterisks denote differences between control and treatment values significant at the 5 per cent (*) and 1 per cent (**) levels respectively.

<table>
<thead>
<tr>
<th>Drug, standard dose i.v. (mg/kg)</th>
<th>Procedure</th>
<th>pH</th>
<th>$P_{CO_2}$ (mm Hg)</th>
<th>$P_{O_2}$ (mm Hg)</th>
<th>Blood pressure (mm Hg)</th>
<th>Paralysis (per cent)</th>
<th>Recovery time (min)</th>
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<tr>
<td>Tubocurarine</td>
<td>Pre-control</td>
<td>7.31</td>
<td>39.4</td>
<td>494</td>
<td>130</td>
<td>28*</td>
<td>18**</td>
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<td></td>
<td>Post-control</td>
<td>7.33</td>
<td>39.5</td>
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<td>110</td>
<td>(15-40)</td>
<td>(13-24)</td>
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<tr>
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<td>Pre-control</td>
<td>7.34</td>
<td>34.5</td>
<td>431</td>
<td>130</td>
<td>20</td>
<td>10</td>
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<tr>
<td></td>
<td>Post-control</td>
<td>7.31</td>
<td>32.5</td>
<td>401</td>
<td>110</td>
<td>(13-30)</td>
<td>(6-16)</td>
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<tr>
<td>Suxamethonium</td>
<td>Pre-control</td>
<td>7.34</td>
<td>32.8</td>
<td>454</td>
<td>121</td>
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<td>Post-control</td>
<td>7.33</td>
<td>30.5</td>
<td>451</td>
<td>111</td>
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<td>(5-11)</td>
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TABLE II
Effects in cats, lightly anaesthetized with chloralose, of single and sustained response to ph, $P_{CO_2}$, $P_{O_2}$ and pressure of arterial blood, and on neuromuscular paralysis by tubocurarine, gallamine and suxamethonium. Other details as table I.

<table>
<thead>
<tr>
<th>Drug, standard dose i.v. (mg/kg)</th>
<th>Procedure</th>
<th>pH</th>
<th>$P_{CO_2}$ (mm Hg)</th>
<th>$P_{O_2}$ (mm Hg)</th>
<th>Blood pressure (mm Hg)</th>
<th>Paralysis (per cent)</th>
<th>Recovery time (min)</th>
</tr>
</thead>
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<tr>
<td>Tubocurarine</td>
<td>Pre-control</td>
<td>7.37</td>
<td>27.9</td>
<td>468</td>
<td>120</td>
<td>24*</td>
<td>21**</td>
</tr>
<tr>
<td></td>
<td>Post-control</td>
<td>7.39</td>
<td>25.3</td>
<td>468</td>
<td>110</td>
<td>(15-30)</td>
<td>(12-31)</td>
</tr>
<tr>
<td>Gallamine</td>
<td>Pre-control</td>
<td>7.34</td>
<td>35.9</td>
<td>482</td>
<td>151</td>
<td>16</td>
<td>9</td>
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<tr>
<td></td>
<td>Post-control</td>
<td>7.37</td>
<td>29.9</td>
<td>478</td>
<td>92</td>
<td>(21-60)</td>
<td>(12-31)</td>
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<td>123</td>
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<td>Post-control</td>
<td>7.32</td>
<td>32.3</td>
<td>471</td>
<td>108</td>
<td>(17-95)</td>
<td>(4.5-16)</td>
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</tbody>
</table>

TABLE III
Effects in cats, lightly anaesthetized with chloralose, of intravenous infusion of 1.25 mg/min thiopentone (total 8-10 mg/kg) on the pH, $P_{CO_2}$, $P_{O_2}$ and pressure of arterial blood, and on neuromuscular paralysis by tubocurarine, gallamine and suxamethonium. Other details as table I.

<table>
<thead>
<tr>
<th>Drug, standard dose i.v. (mg/kg)</th>
<th>Procedure</th>
<th>pH</th>
<th>$P_{CO_2}$ (mm Hg)</th>
<th>$P_{O_2}$ (mm Hg)</th>
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<th>Paralysis (per cent)</th>
<th>Recovery time (min)</th>
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<td>(17-95)</td>
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</tr>
</tbody>
</table>
ANAESTHETICS AND INTERACTIONS WITH BLOCKING AGENTS

Fig. 1
Records from a cat 3.4 kg, lightly anaesthetized with chloralose.
(a) Control dose of gallamine, 0.5 mg/kg i.v., depressed the tetanic contractions of the gastrocnemius muscle stimulated with 30 shocks/sec every 10 sec and slightly reduced the single twitches of the contralateral muscle.
(b) Inhalation of 75 per cent nitrous oxide in oxygen.
(c) Paralysis by gallamine, 0.5 mg/kg i.v., was slightly intensified during treatment with nitrous oxide; the PaO2 was lowered.
(d) Further control doses of gallamine, 0.5 mg/kg i.v.
Intervals of 45 min were allowed between doses.

with halothane was virtually unaltered. A fifth cat became more sensitive to successive doses of the drug independently of the anaesthetic.

Thiopentone (table III).
Intravenous infusion of 8–10 mg/kg thiopentone in 15–25 min reduced mean arterial blood pressure by 29–59 mm Hg and slightly reduced heart rate (c.15 per cent); the responses of the gastrocnemius muscles to indirect tetanic stimulation were usually slightly increased (6 per cent, SE 3 per cent) whereas the responses to single shocks were not usually affected. Changes in mean pH and Pco2 were small; mean PaO2 was often slightly increased by 17–20 mm Hg.

Paralysis of the twitch and tetanic responses by tubocurarine was potentiated and significantly prolonged during the infusion of thiopentone in each of four cats. The hypotensive effect of the drug was increased. Blockade by gallamine was also significantly enhanced, but the paralyzing action of suxamethonium was not modified by thiopentone.

DISCUSSION
The method used in this study was a compromise in that the action of anaesthetics on neuromuscular paralysis in cats was studied in the presence of light chloralose anaesthesia; according to Secher (1951) chloralose had no peripheral action in the intact animal.
Records from a cat 3.1 kg, lightly anaesthetized with chloralose.

(a) Control dose of tubocurarine, 0.17 mg/kg i.v., depressed the tetanic contractions of the gastrocnemius muscle stimulated with 30 shocks/sec every 10 sec and partially reduced the single twitches of the contralateral muscle.

(b) Inhalation of c.0.5 per cent halothane in oxygen lowered blood pressure.

(c) Paralysis by tubocurarine, 0.17 mg/kg i.v., was increased during treatment with halothane; marked hypotension and bradycardia occurred.

(d) Further control dose of tubocurarine, 0.17 mg/kg i.v.

Intervals of 60 min were allowed between doses.

**Nitrous oxide.**

Ventilation of cats with 75 per cent nitrous oxide had no cardiovascular or neuromuscular effects, in conformity with observations in rabbits and dogs (Naess, 1950; Smith and Corbascio, 1966; Price and Price, 1967).

In the present experiments, but not those in rabbits (Naess, 1950), paralysis by tubocurarine and gallamine was significantly increased; this effect is unexplained.

**Halothane.**

The concentration of halothane in the inspired oxygen stream was set at 0.5 per cent on the calibrated vaporizer for a flow rate of 1 l./min. This concentration was rather low but there may have been accumulation of halothane in the body as the lungs were artificially ventilated at a constant volume. Severe hypotension and bradycardia ensued if the inspired concentration was increased to 1 per cent.

Hypotension with halothane in cats and dogs has been attributed to blockade of sympathetic ganglia (Raventós, 1956) and this effect is synergistic with that of tubocurarine (Burn et al., 1957). Observations in man (Johnstone, 1956; Bryce-Smith and O'Brien, 1956; Burns et al., 1957) led to the generally accepted practice of using tubocurarine at reduced dosage in the presence of 1–3 per cent halothane to avoid a profound fall in arterial blood pressure.

Single twitch responses were unaltered by c.0.5 per cent halothane in the present experiments or in earlier studies in cats, rabbits or man.
using concentrations of 1–4.4 per cent (Burn et al., 1957; Watland et al., 1957; Katz and Gissen, 1967; Baraka, 1968).

The slight depression of the tetanic contractions reported here agrees well with the observation in man that 1–2 per cent halothane reduced the tone and electromyographic activity of the abdominal muscles without depressing the twitch responses of the thumb (Katz and Gissen, 1967). Inhibition of tetanic contractions by halothane may be in part attributable to the associated hypotension and reduced muscle blood flow (Schweitzer, 1945; Lindgren, Westermark and Wåhlin, 1964). Depression of neuromuscular transmission may also be at least contributory. Tests using isolated organs have unfortunately been limited to the twitch response but inhibition was found in human intercostal muscles exposed to 4–8 per cent halothane (Sabawala and Dillon, 1958) and an analogous effect with 1.5 per cent halothane in frog sartorius preparations was ascribed to desensitization of the post-junctional membrane (Gissen, Karis and Nastuk, 1966).

It is known that 1–3 per cent halothane potentiates paralysis by tubocurarine and gallamine but not by suxamethonium in man (Burns et al., 1957; Katz and Gissen, 1967; Baraka, 1968), and in cats anaesthetized with chloralose (Burn et al., 1957). The present findings in cats for tubocurarine and gallamine are in agreement and attention is drawn to negative correlation between mean arterial blood pressure at the time of giving these drugs and the ensuing intensity and duration of paralysis they produced. Thus, a lower arterial blood pressure was associated with a more intense and prolonged paralysis. Whereas blockade by suxamethonium was virtually unaltered by c.0.5 per cent halothane in the present tests, Burn and associates (1957) found that the higher concentration of 1.35 per cent was antagonistic.

**Thiopentone.**

Despite slow infusion at 1.25 mg/min, thiopentone lowered arterial blood pressure and heart rate. Though its ganglion blocking effect is negligible in cats (Larrabee and Holaday, 1952; Norman and Löfström, 1955), the anaesthetic increased the hypotensive effect of tubocurarine as in man (Price, 1960). Hypotension in man has been attributed to postarterial pooling of blood and the consequent reduction of cardiac output (Price et al., 1952).

Infusion of thiopentone in cats increased the responses of the gastrocnemius muscle to indirect stimulation with bursts of tetani, leaving the twitch responses unaffected. The effect may depend upon species, dosage, route of administration and/or frequency of stimulation. Thus, potentiation of the twitch responses, as well as tetanic contractions of the tibialis muscles, followed 1–2 mg/kg thiopentone intra-arterially to dogs anaesthetized with urethane (Kraatz, Gluckman and Shields, 1953). In rats anaesthetized with urethane, intra-arterial injection of 2 mg thiopentone (a relatively high dose for this species) caused a rapid augmentation and then a decline of the twitch response (Quilliam, 1955). Analogous effects have been reported to occur in isolated rat diaphragms whether the muscle was stimulated directly or indirectly (Secher, 1951; Sirnes, 1954). Quilliam (1955) commented that the increase in the twitch tension with thiopentone is consistent with a slower propagation of the muscle action potential associated with a slower and more prolonged contraction; in a simple twitch there is insufficient time for the full tension of the muscle to be developed before relaxation has set in.

Infusion of 8–10 mg/kg thiopentone to cats lightly anaesthetized with chloralose, significantly increased neuromuscular blockade by tubocurarine or gallamine but did not modify the response to suxamethonium. Similar intensification of the paralyzing effect of tubocurarine was observed in rabbits by Sirnes (1954), who also found that paralysis by decamethonium was increased by large intravenous doses of thiopentone, 22–44 mg/kg. This would presumably have been associated with severe hypotension and may have little relevance to the usual human dosage of approximately 4 mg/kg.

**ACKNOWLEDGEMENTS**

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**References**


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**SOMMAIRE**

Une ventilation avec 75 pour cent de protoxyde d'azote n'affecta pas, chez des chats légèrement anesthésiés au chloralose, la pression sanguine ni la fréquence cardiaque ni la réaction du muscle gastrocnémius après stimulation indirecte; le blocage par tubocurarine et gallamine s'intensifia significativement mais celui par suxamethonium demeura inchangé. La ventilation avec environ 0.5 pour cent d'halothane causa de l'hypotension et bradycardie, déprimant légèrement la réaction musculaire après stimulation tétanique indécente mais ne modifia pas les convulsions individuelles. L'intensité et la durée de la paralysie par tubocurarine augmentèrent significativement tandis que la modification ne fut pas significative en ce qui concerne gallamine. L'infusion de thiopentone, 8-10 mg/kg i.v. diminua la pression sanguine et la fréquence cardiaque et intensifi la légèrement les contractions tétaniques des muscles, sans modifier les réactions convulsives; le blocage par tubocurarine et gallamine s'intensifia significativement et celui par suxamethonium ne changea pas. Halothane et thiopentone potentiérent l'effet hypotenseur de la tubocurarine.
ANAESTHETICS AND INTERACTIONS WITH BLOCKING AGENTS

WIRKUNGEN VON ANAESTHETICS UND IHRE AUSWIRKUNGEN MIT ANDEREN NEUROMUSKULAREN BLOCKERN BEI DER KATZE

ZUSAMMENFASSUNG
Katzen, die mit Chloralose leicht anaesthesiert waren, wurden mit 75% Lachgas beatmet, wobei es zu keiner Wirkung auf den arteriellen Blutdruck, die Herzfrequenz oder die indirekte Stimulation des Gastrocnemiusmuskels kam. Blockade mit Tubocurarin und Gallamin wurde signifikant verstärkt, jedoch nicht die Blockade durch Suxamethonium fast unbeeinflusst. Beatmung mit ungefähr 0,5% Halothan verursachte Hypotension und Bradycardie und unterdrückte leicht die Reizantwortung des Muskels auf indirekte tetanische Reize, wobei jedoch die einzelnen Zuckungen nicht beeinflusst waren. Die Intensität und Dauer der Paralyse durch Tubocurarin war signifikant vermehrt, dies traf jedoch nicht für Gallamin zu; die Wirkung von Suxamethonium war praktisch unverändert. Invasion von Thiopenton 8–10 mg/kg i.v. erniedrigte den arteriellen Blutdruck und die Herzfrequenz aber erhöhte leicht die tetanischen Kontraktionen des Muskels ohne Änderung der Zuckungen Blockade mit Tubocurarin und Gallamin war stark vermehrt, während die durch Suxamethonium unverändert war. Halothan und Thiopenton potenzieren die hypotensive Wirkung von Tubocurarin.

CORRESPONDENCE

CARDIOVASCULAR COLLAPSE FOLLOWING INDUCTION WITH PROPANIDID

Sir,—Severe hypotension after injection of propanidid (Gjessing, 1969), or after repeated application of propanidid (Johns, 1970; Bradburn, 1970; Manz and Fank, 1969) may indeed occur. Propanidid is not entirely free from depressant action on the myocardium and peripheral vessels, as has been demonstrated by Sankawa (1965) in experimental studies on dogs, and by Johnstone and Barron (1968) and Radnay (1969) in clinical practice.

Our patient of 63 years was scheduled for transurethral resection for cancer of prostate. Apart from an arterial blood pressure of 160/90 mm Hg which was not treated with antihypertensive drugs, and some evidence of mild hypoxaemia of the myocardium, there were no other complicating diseases. He received atropine 0.5 mg, promethazine 25 mg and pethidine 50 mg intramuscularly for premedication. After oxygen inhalation propanidid 500 mg was given intravenously. Two minutes later red spots appeared over the whole skin and simultaneously cardiac action ceased. After a further minute, breathing movements ceased also and deep cyanosis occurred. External cardiac massage and manual ventilation of the lungs with oxygen by mask were immediately started and soon the pulse became palpable. The blood pressure remained unmeasurable. A rapid drip of Hartmann's solution with mephenytoine 15 mg was followed by a rise of blood pressure to 80/50 mm Hg. Meanwhile the patient regained consciousness. After a second 500 ml of Hartmann's solution with methohexital sodium 20 mg the blood pressure rose to 130/80 mm Hg and the pulse rate to 120 beats/min. There was complete recovery. Spontaneous defaecation and urination occurred. The patient remained amnesic. Previously two general anaesthetics induced with thiopentone had been uneventful.

The ultrashort anaesthetic effect of propanidid may lead to an irresponsible application chiefly by non-anaesthesiologists. It is necessary to bear in mind the possibility of untoward side effects of propanidid especially in patients with lowered cardiovascular reserve and in repeated application of propanidid. As in any other anaesthetic, measures for treating serious side effects must be immediately available.

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REFERENCES