CLEARANCE OF INDOCYANINE GREEN AS AN INDEX OF LIVER FUNCTION DURING CYCLOPROPAANE ANAESTHESIA AND INDUCED HYPOTENSION

A. R. Abdel Salam, G. B. Drummond, H. W. Bauld and D. B. Scott

SUMMARY

The rate of decrease in the plasma concentration of indocyanine green (ICG) following a bolus i.v. injection has been studied in two groups of patients before and during anaesthesia. In eight patients anaesthetized with nitrous oxide and oxygen, and who received a neuromuscular blocking drug, induction of anaesthesia was not associated with a significant change in ICG half-life (from 4.59 to 3.85 min) but the subsequent administration of cyclopropane was associated with a statistically significant increase to 5.61 min. In a second group of anaesthetized patients, breathing spontaneously a mixture of oxygen, nitrous oxide and halothane, the induction of arterial hypotension with either lumbar extradural block or sodium nitroprusside was not associated with significant changes in ICG half-life.

Clearance of bromsulphthalein (BSP) from blood is accepted generally as a quantitative index of liver function (Rosenthal and White, 1925), but it requires up to 45 min to perform a single test. Therefore it is unsuitable for the measurement of rapid changes in liver function. The method depends on the assumption that BSP does not leave the vascular compartment following i.v. injection other than by hepatic extraction. This assumption may be valid also for the dye indocyanine green (ICG), which is removed more rapidly by the liver than is BSP (Caesar et al., 1961).

After the i.v. injection of ICG in an aqueous solution, the dye is bound to plasma albumin, and extracted by the liver until the plasma concentration has decreased to about 4% of the original maximum value (Cherrick et al., 1960). Clearance of ICG after an i.v. bolus dose has been used, in man, as an index of hepatic disease, based on the principle described by Dobson and Jones (1952). This correlates well with results obtained using BSP (Cherrick et al., 1960; Caesar et al., 1961). However, the method has not been used widely in anaesthesia. ICG has been used for the measurement of liver blood flow and function, during anaesthesia, with a constant infusion technique (Bradley et al., 1945), but the method requires sampling of hepatic venous blood by catheterization under radiographic control and is, therefore, unsuitable for widespread use.

We have assessed the feasibility of using ICG clearance from peripheral blood following an i.v. bolus dose to detect acute changes in liver function before and during anaesthesia. Using a constant infusion method with sampling from the right hepatic vein, Price and others (1965) established that cyclopropane anaesthesia was associated with a reduction in ICG clearance and a reduction in hepatic blood flow. Similar changes were reported by Shackman, Graber and Melrose (1953) using a constant BSP infusion method. Using the simpler technique of measuring the rate of decrease in peripheral blood concentration after the administration of an i.v. bolus dose of ICG, we have measured liver function during cyclopropane anaesthesia to see if the same changes can be demonstrated.

These measurements showed that acute changes in liver function could be demonstrated in this way. Subsequently we employed the method to determine the effects of arterial hypotension, during anaesthesia, induced by means of either sodium nitroprusside infusion or lumbar extradural block.

METHODS

A dose of 50 mg of dye in 10 ml of aqueous solution was chosen as this produced plasma concentrations which could be measured accurately using the apparatus available and is similar to the doses employed by other workers (Cherrick et al., 1960; Caesar et al., 1961). The changes in blood concentration of ICG were measured using an earpiece densitometer (Waters XP-302) and recorded on a potentiometric flatbed recorder (Servoscribe RE511).
at a paper speed of 30 mm/min for a total duration of about 12 min. Thus the method is quick and simple, with the only discomfort to the patient being that of an i.v. injection. In a preliminary study, sampling and spectrophotometric analysis of peripheral venous blood showed that the densitometer output was directly proportional to the plasma concentration of dye.

The half-life of ICG in the blood was determined from the recorded decay curve of the dye concentration. A typical record is shown in figure 1. For up to 3 min after injection a rapid decrease in the blood concentration was recorded as a result of distribution of dye throughout the intravascular compartment. After 10 min, the densitometer reading had decreased to a point at which accurate measurement had become difficult. In addition, there is evidence that, after this time, the rate of decrease of plasma concentration becomes less (Cherrick et al., 1960). Therefore, densitometer readings obtained between 3 and 9 min after injection are used for analysis. Between these times, the rate of reduction of plasma dye concentration was assumed to be a single exponential process. The logarithms of the densitometer readings at 20-sec intervals from 3 to 9 min were plotted against time (fig. 2), and a straight line of best fit for this period was calculated by the method of least squares. Estimation of the plasma dye concentrations at such frequent intervals by means of the earpiece was advantageous in the analysis and fitting of the data, compared with the smaller number of values that could be obtained from blood sampling. In all instances the correlation coefficient for this least squares line was greater than 0.98, indicating that, for these data, the assumption that the process may be described by a single exponential function was valid. From the slope of the line, the half-life of the dye in the circulation is calculated easily as the slope is log 2/half-life.

Although small concentrations of dye could be detected spectrophotometrically in blood samples taken 20 min after a bolus injection, the deflection of the earpiece densitometer was small and changed very slowly and thus the clearance test could be repeated.

**Cyclopropane anaesthesia**

To assess the method during anaesthesia, cyclopropane was chosen as this has been shown previously to influence liver blood flow (Price et al., 1965). After a full explanation of the procedures involved, eight patients (age range 21–45 yr), who were free from a history or clinical evidence of liver disease, agreed to take part in these investigations.

Before the administration of premedicant drugs a clearance test was performed with the patient fasted. One hour after i.m. premedication with an opiate and atropine, anaesthesia was induced with thiopentone 200–300 mg i.v. followed by tubocurarine 30–45 mg, endotracheal intubation, and maintenance of anaesthesia by artificial ventilation with nitrous oxide (3 litre/min) and oxygen (1 litre/min) using a volume-cycled circle system ventilator with carbon dioxide absorber (Cape-Waine).

The minute volume used was calculated using Nunn’s predictions (Nunn, 1967) to maintain $P_{aCO_2}$ within the normal range. When the patient appeared clinically settled, and the arterial pressure (measured by sphygmonanometer and auscultation) and heart rate were stable, 50 mg of ICG was given i.v. and the changes in blood concentration of the dye were detected by the earpiece densitometer. Arterial pressure and heart rate were measured and recorded at 5-min intervals.

After 15–20 min, when the residual dye was barely detectable with the densitometer, the fresh gas flow was changed to cyclopropane (600 ml/min) and oxygen (300 ml/min) for 4 min, and then to cyclopropane (200 ml/min) with oxygen (300 ml/min) until the experiment ended (14 min after cyclopropane administration). No changes were made in
the ventilator settings. Five minutes after cyclopropane administration had commenced, the ICG dose was repeated, and the densitometer readings were recorded. Although it is unlikely that a steady state had been achieved, it was felt that cyclopropane in the concentrations used would have a marked pharmacological effect during the period of ICG clearance. Surgery did not start until this last test was completed. For each patient the ICG half-life was calculated, between 3 and 9 min after ICG administration for (1) the fasting conscious state, (2) anaesthesia with nitrous oxide and (3) anaesthesia with cyclopropane.

Capillary blood samples were taken from a thumb 10 min after the start of cyclopropane administration, and analysed for \( P_{O_2} \), \( P_{CO_2} \) and pH, using an IL 213 apparatus. One sample could not be analysed for technical reasons.

**Induced hypotension**

Eleven patients undergoing major gynaecological surgery were studied during hypotensive anaesthesia, which is employed frequently in this centre as a routine technique. After a full explanation of the procedures involved, all gave their consent for the study. The ages ranged from 27 to 47 yr, and no patient had any history or clinical evidence of hepatic or cardiorespiratory disease.

Each patient had an ICG clearance test performed on the day before surgery, 4 h after a light breakfast. On the day of operation, each received premedication with diamorphine 5 mg and atropine 0.6 mg i.m. One hour later, anaesthesia was induced with thioptenone 400–500 mg and maintained with nitrous oxide (3 litre/min) and oxygen (1 litre/min) using a circle system with carbon dioxide absorption. Ventilation was spontaneous throughout, from a well-fitting face mask. Halothane 0.5% was added usually to achieve quiet spontaneous ventilation, and arterial hypotension was not induced unless there was complete satisfaction with the patency of the airway. The care of the patient was in the hands of an anaesthetist who took no part in the investigations.

Arterial pressure was measured by a sphygmomanometer and auscultation, and the mean pressure was calculated as the diastolic pressure plus one-third pulse pressure. When ventilation, heart rate and arterial pressure were stable, an ICG clearance test was performed. After 20 min, hypotension was induced using one of two methods.

In seven patients, sodium nitroprusside 0.005% was administered as a continuous i.v. infusion, and the rate was adjusted to maintain the systolic arterial pressure at 60–70 mm Hg.

In four patients, before the induction of anaesthesia, a catheter was inserted into the lumbar extradural space through either the L2–3 or L3–4 interspace. Extradural blockade was produced using 20 ml of 2% lignocaine solution. This caused a less rapid reduction in arterial pressure than the nitroprusside infusion, but to systolic pressures similar to those in the nitroprusside-treated group.

After a stable hypotensive state had been induced, a further dose of ICG was administered, and the clearance of the dye was followed. Surgery did not commence until this second clearance test had been completed.

**Analysis of results**

The small amount of data available in the groups did not allow testing for a normal distribution of the variable of ICG half-life. Therefore, statistical tests for significance that do not depend upon this assumption were employed. Circulatory changes (arterial pressure and heart rate) were analysed using a paired Student’s \( t \) test.

**RESULTS**

**Cyclopropane**

In the conscious fasting state, the eight patients had a mean ICG half-life of 4.59 min (range 2.83–8.83 min). During nitrous oxide anaesthesia, the mean half-life decreased to 3.85 min (range 2.83–5.33 min). Six patients showed a decrease and two an increase in half-life. During cyclopropane administration, the mean half-life increased to 5.61 min (range 3.5–7.83 min). Seven of the eight patients showed an increase in half-life from the conscious state to anaesthesia with nitrous oxide (\( P < 0.05 \)). The decrease in half-life from the conscious state to anaesthesia with nitrous oxide was not significant (table I).

The circulatory effects of cyclopropane were of interest. Mean arterial pressure increased and heart rate decreased, both changes being significant at the 5% level.

The mean \( P_{CO_2} \) of the capillary specimens was 40 mm Hg (SD 5 mm Hg), mean pH 7.34 (range 7.27–7.46), and \( P_{O_2} \) 157 (SD 43) mm Hg, in the seven subjects in whom these values were determined.
TABLE I. Effects of cyclopropane. I = test period before anaesthesia, II = anaesthesia with nitrous oxide, III = anaesthesia with cyclopropane

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>ICG half-life (min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Heart rate (beat/min)</th>
<th>Pco₂ (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
<td>II</td>
</tr>
<tr>
<td>1</td>
<td>F</td>
<td>36</td>
<td>4.08</td>
<td>3.25</td>
<td>4.67</td>
<td>80</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>32</td>
<td>3.67</td>
<td>2.83</td>
<td>6.02</td>
<td>94</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>44</td>
<td>3.00</td>
<td>2.83</td>
<td>6.00</td>
<td>129</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>21</td>
<td>3.50</td>
<td>4.67</td>
<td>3.50</td>
<td>87</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>30</td>
<td>6.83</td>
<td>4.33</td>
<td>4.50</td>
<td>100</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>25</td>
<td>4.00</td>
<td>4.75</td>
<td>5.83</td>
<td>92</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>28</td>
<td>8.83</td>
<td>5.33</td>
<td>7.83</td>
<td>85</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>45</td>
<td>2.83</td>
<td>2.83</td>
<td>6.50</td>
<td>80</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>33</td>
<td>4.59</td>
<td>3.85</td>
<td>5.61*</td>
<td>93</td>
</tr>
<tr>
<td>SD</td>
<td></td>
<td>6</td>
<td>2.11</td>
<td>1.03</td>
<td>1.34</td>
<td>16</td>
</tr>
</tbody>
</table>

* Significant compared with preceding period (P < 0.05).

TABLE II. Effects of hypotension induced with sodium nitroprusside. I = test period before anaesthesia, II = anaesthesia, III = anaesthesia with induced hypotension. Two tests before anaesthesia were technically unsatisfactory

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>ICG half-life (min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Heart rate (beat/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>1</td>
<td>27</td>
<td>3.42</td>
<td>3.67</td>
<td>3.67</td>
</tr>
<tr>
<td>2</td>
<td>33</td>
<td>4.17</td>
<td>4.00</td>
<td>3.17</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>3.67</td>
<td>3.65</td>
<td>5.00</td>
</tr>
<tr>
<td>4</td>
<td>27</td>
<td>—</td>
<td>3.00</td>
<td>3.67</td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>3.25</td>
<td>3.83</td>
<td>3.33</td>
</tr>
<tr>
<td>6</td>
<td>27</td>
<td>—</td>
<td>3.67</td>
<td>3.17</td>
</tr>
<tr>
<td>7</td>
<td>47</td>
<td>3.17</td>
<td>3.40</td>
<td>4.40</td>
</tr>
<tr>
<td>Mean</td>
<td>35</td>
<td>3.54</td>
<td>3.61</td>
<td>3.77</td>
</tr>
<tr>
<td>SD</td>
<td>11</td>
<td>0.40</td>
<td>0.32</td>
<td>0.69</td>
</tr>
</tbody>
</table>

* P < 0.01; † P < 0.001.

Mean bicarbonate concentration (derived using the Siggaard-Andersen nomogram) was 21 m-mol/litre (SD 1.0).

Induced hypotension

In the patients who received nitroprusside, the mean half-life for ICG was 3.54 min when conscious, 3.61 min during general anaesthesia before the induction of hypotension, and 3.77 min after the induction of hypotension. These changes in half-life were not statistically significant (table II, fig. 3).

The mean values for the extradural group were 5.22, 5.54 and 5.67 min, for the before-anaesthesia, anaesthesia and anaesthesia-with-hypotension periods respectively. No statistical significance could be attributed to the changes (table III, fig. 3).

Of the total of 11 patients studied, the induction of hypotension caused an increase in the ICG half-life in four, a decrease in four, and no change in three patients.

During sodium nitroprusside infusion, the mean arterial pressure decreased from an average of 77 mm Hg to 52 mm Hg, and with extradural blockade from 79 to 57 mm Hg. The heart rate increased from 83 to 125 beat/min with sodium nitroprusside, and decreased from 77 to 70 beat/min after extradural blockade. These changes in mean arterial pressure and heart rate were all significant, at the confidence levels shown in tables II and III.
INDOCYANINE GREEN AS AN INDEX OF LIVER FUNCTION

Fig. 3. ICG half-life, mean arterial pressure and heart rate for the nitroprusside group (seven patients) and the extradural group (four patients). For each group, the three periods shown are before anaesthesia, during anaesthesia and during anaesthesia with hypotension.

TABLE III. Effects of hypotension induced with lumbar extradural block.
(Annotation as for table II)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>ICG half-life (min)</th>
<th>Mean arterial pressure (mm Hg)</th>
<th>Heart rate (beat/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>I</td>
<td>II</td>
<td>III</td>
</tr>
<tr>
<td>1</td>
<td>34</td>
<td>3.67</td>
<td>5.00</td>
<td>5.00</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>7.67</td>
<td>6.83</td>
<td>6.17</td>
</tr>
<tr>
<td>3</td>
<td>46</td>
<td>3.08</td>
<td>5.17</td>
<td>5.19</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>6.67</td>
<td>5.17</td>
<td>6.30</td>
</tr>
<tr>
<td>Mean</td>
<td>36</td>
<td>5.27</td>
<td>5.54</td>
<td>5.67</td>
</tr>
<tr>
<td>SD</td>
<td>7</td>
<td>5.8</td>
<td>6.36</td>
<td>0.68</td>
</tr>
</tbody>
</table>

* P < 0.05; † P < 0.01.

DISCUSSION

It is possible to analyse the removal of ICG from the blood by calculation of the half-life only if it is assumed that the extraction is a single first-order process. Richards, Tindall and Young (1959) showed that, in the dog, BSP extraction was described by two first-order processes, the slower process probably representing biliary excretion. In man, Barber-Riley and colleagues (1961) showed that analysis of data for BSP in terms of two first-order reactions often proved impossible. ICG is not conjugated before excretion, and enterohepatic recirculation does not occur (Caesar et al., 1961). A reduction in the rate of extraction occurs after about 20 min (Cherrick et al., 1960), and it is possible, therefore, that a slower process may be present, as in the case of BSP. Analysis of our data was not performed after 9 min of clearance, but inspection of the log concentration/time plot beyond 10 min suggested that reduction in the rate of extraction did occur. If this were so, calculation of the half-life from the decay rate over the period from 3 to 9 min after injection might result in the lesser values being disproportionately increased.

ICG half-life in fasting conscious subjects in these two experiments ranged from 2.8 to 8.83 min. This range is similar to that of the half-lives calculated from the data for normal subjects by Caesar and others (1961). These values for ICG half-life indicate that their distribution has a positive skew, which in part justifies the fact that a distribution-dependent test of statistical significance was not employed.

It is possible that the slightly higher values obtained in this study, compared with those calculated from
the data of Caesar and others (1961), have been caused by performing the ICG clearance test while the patients were fasting. We have demonstrated in volunteers (unpublished data) that ICG half-life is significantly reduced after eating a light meal.

Although ICG clearance is used extensively as a liver function test, it has not been used widely in determining changes during anaesthesia. Clearance of a drug from the blood by the liver depends upon two principal factors. First, the efficiency of extraction of the drug from the blood presented to the liver. This may be expressed as the fractional extraction rate and will depend upon the total mass of the liver cells and the activity of the cells (fig. 4). In normal subjects, this extraction ratio has been found to be between 60% and 90% (Cherrick et al., 1960; Reemstma et al., 1960; Wiegand, Ketterer and Rapaport, 1960; Caesar et al., 1961). Second, the liver blood flow controls the amount of drug presented to the liver. It has been suggested that the fractional extraction rate is exponentially related to the liver blood flow, decreasing from a maximum as the blood flow increases (Brauer, 1958).

Assessment of the contribution of each factor to any change in observed clearance requires that they be estimated separately. This necessitates catheterization of the hepatic veins and measurement of the concentration change across the liver. Apart from the technical and possible ethical problems involved, the method may be criticized for use during anaesthesia particularly, because a “steady state” is assumed in the calculation of hepatic uptake, whereas liver function may be changing. These problems are discussed by Epstein and others (1965) and Price and others (1966), and clearly demonstrated, for BSP, in the study by Shackman, Graber and Melrose (1953).

In many circumstances during anaesthesia, especially if light anaesthesia is used and the agents employed are known not to cause gross hepatocellular damage, it might be reasonable to assume that changes in observed clearance of agents such as ICG are primarily caused by changes in liver blood flow.

However, with cyclopropane the situation is complex, as Price and others (1965) found that ICG clearance decreased slightly more than hepatic blood flow, and although hepatic blood flow could be restored with ganglion-blocking drugs, the clearance (and therefore the extraction ratio) did not return to normal. It is possible that cyclopropane may influence both hepatic blood flow and the function of hepatic cells, and studies of hepatic oxygen uptake during anaesthesia (Libonati et al., 1973) support this concept.

The changes in half-life associated with cyclopropane administration in the present study are of the same order as the changes in hepatic blood flow reported by Price and others (1965). The changes observed in the general circulation (increased mean arterial pressure and decreased heart rate) support the suggestion that circulatory factors are associated with the increase in ICG half-life. It was not the purpose of this study to determine the exact nature of these circulatory changes, but it is known that cyclopropane has complex effects, including increased sympathetic activity.

Arterial carbon dioxide tension has a marked effect on liver blood flow (Galindo, 1965; Epstein et al., 1966). In the patients studied, capillary carbon dioxide tensions were almost within the normal range, but some variability between patients was present. As the ventilation for each patient was unaltered throughout the experiment, it is unlikely that gross changes in \( P_{CO_2} \) would occur between the nitrous oxide and cyclopropane periods.

As neither sodium nitroprusside nor extradural blockade has any known effect upon hepatic cell function, it is reasonable to assume that in these situations any change in ICG clearance would be caused by a change in hepatic blood flow. This assumption is supported in part by the findings of Kennedy and others (1970) that induction of hypotension by spinal anaesthesia did not cause a change in the extraction ratio for ICG. No significant change in ICG half-life was noted after the induction of hypotension in the present study, and therefore it may be stated that no substantial change in hepatic blood flow had occurred.

Induced hypotension during general anaesthesia using nitroprusside (Wildsmith et al., 1973; Adams et al., 1974), or extradural block (Stephen, Lees and
instance, limb blood flow during anaesthesia is reduced by the induction of hypotension (unpublished data). This is probably because the vessels of the limb are already dilated greatly in the anaesthetized state, and subsequent reductions in arterial pressure cannot be accompanied by further dilatation.

Our finding that no change in hepatic blood flow occurred after induced hypotension contrasts with that of Kennedy and others (1970), who showed that hypotension after high spinal anaesthesia was associated with a reduction in hepatic plasma flow, and of Galindo (1965), who used implanted flow-meters in dogs to demonstrate a reduction in hepatic blood flow after the induction of hypotension by extradural blockade.

From the practical point of view, the importance of adequate function during anaesthesia and hypotension concerns the many drugs which are partly or wholly metabolized in the liver. Whether changes in liver activity are caused by altered flow or altered cellular activity is immaterial, provided that each, if it occurs, is rapidly reversible. Using ICG, the combined effects of the two factors can be discerned, and the acute effects of anaesthesia on the liver can be shown simply, safely and with comparative ease.

REFERENCES


COEFFICIENT D’INDOCYANINE VERTE COMME INDICE DE LA FONCTION HEPATIQUE PENDANT UNE ANESTHESIE AU CYCLOPROPANE ET L’HYPOTENSION PROVOQUEE

RESUME

Le taux dégressif d’indocyanine verte dans les concentrations de plasma à la suite de l’injection intraveineuse du contenu d’un bol a été étudié sur deux groupes de patients avant et pendant l’anesthésie. Sur huit patients anesthésiés...
au protoxyde d'azote et à l'oxygène, ayant reçu une médication de blocage neuromusculaire, l'induction de l'anesthésie n'a pas été associée à une variation importante de la demi-vie de l'indocyanine verte (de 4,59 à 3,85 minutes), mais l'administration ultérieure de cyclopropane a été associée à une augmentation statistiquement significative: 5,61 minutes. Dans le second groupe de patients anesthésiés, respirant spontanément un mélange d'oxygène, de protoxyde d'azote et d'halothane, l'induction de l'hypotension artérielle soit à l'aide d'un blocage lombaire extradural, soit à l'aide de nitroprussiate de sodium n'a pas été associée aux variations significatives de la demi-vie de l'indocyanine verte.

DIE INDOZYANIDGRÜN-KLÄRUNG ALS INDEX DER LEBERFUNKTION WÄHREND CYCLOPROPAIN-NARKOSE UND KÜNSTLICH ERZEUGTER HYPOTONIE

ZUSAMMENFASSUNG

TRANSITO DE VERDE DE INDOCIANINA COMO UN INDICE DE LA FUNCION DEL HIGADO DURANTE LA ANESTESIA DE CICLOPROPANO E HIPOTENSION INDUCIDA

SUMARIO
Se estudió el porcentaje de disminución en la concentración de plasma de verde de indocianina (ICG) después de una inyección intravenosa de bolo. En ocho pacientes anestesiados con óxido nitroso y oxígeno, que recibieron una droga bloqueadora neuromuscular, la inducción de anestesia no estuvo ligada a un cambio significativo en el periodo de ICG (de 4,59 a 3,85 minutos), pero la administración subsiguiente de ciclopropano se asoció con un aumento a 5,61 minutos significativo estadísticamente. En un segundo grupo de pacientes anestesiados, que respiraban espontáneamente una mezcla de oxígeno, óxido nitroso y halotano, la inducción de hipotensión arterial con bloqueo extradural lumbar o nitroprusiido de sodio no fué acompañada de cambios significativos en el periodo de ICG.