EFFECT OF GENERAL ANAESTHESIA ON THE MAGNETIC RESONANCE IMAGING SIGNAL FROM THE BRAIN

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Medical magnetic resonance imaging (MRI) systems usually measure the activity of hydrogen nuclei (protons). As most body tissues are composed mainly of water, MRI is, in effect, a method of determining water content and changes in the distribution and molecular environment of water molecules in the body. The measurements used are \( T_1 \), the spin lattice relaxation time (or longitudinal magnetic relaxation time) and \( T_2 \), the spin-spin lattice relaxation time (or transverse magnetic relaxation time). \( T_1 \) is dependent on the interaction of protons with surrounding nuclei and molecules, that is, the lattice. \( T_2 \) depends on the interaction of protons with one another. Areas in the body with low water content have short \( T_1 \) values, whereas areas with high water content have long \( T_1 \) values.

Mander and colleagues [1] found a significant increase in \( T_1 \) in patients immediately after electroconvulsive therapy (ECT). However, five volunteers in the study who received the same anaesthetic but without ECT showed non-significant decreases in \( T_1 \). This raised the possibility that general anaesthesia may itself alter the MRI signal from the brain. The present study has explored this possibility.

METHODS AND RESULTS

We studied 10 women (ASA I or II, aged 26–50 yr (mean 39.9 yr)) scheduled for cone biopsy of the cervix, following an explanation of the procedure. All patients gave written consent to the study, which was approved by the local Ethics Committee. Patients receiving any medication other than the contraceptive pill, or with a history of epilepsy, were excluded.

Each patient had an MRI brain scan on the afternoon before surgery. Cone biopsy was carried out using a standard anaesthetic technique. Premedication with temazepam 20 mg by mouth was given 1 h before operation. Anaesthesia was induced with thiopentone 4–7 mg kg\(^{-1}\) i.v. and diamorphine 2.5 mg i.v., and maintained with nitrous oxide, oxygen and enflurane administered via a facemask and a circle absorber system with 3 litre min\(^{-1}\) fresh gas flow. After anaesthesia, when the patients were recovered sufficiently, they underwent a second scan. This was carried out 2.5–6.5 h (mean 4.4 h) after induction of anaesthesia.

An M & D Technology Ltd 0.08 Tesla resistive magnetic resonance imaging system was used. For each patient \( T_1 \) and \( T_2 \) measurements were made on 12-mm thick transverse sections of the brain at a level 10 mm above the widest point of the lateral ventricles. \( T_1 \) and \( T_2 \) measurements were made also from sagittal sections in small areas of the pons, midbrain and corpus callosum. The \( T_1 \) and \( T_2 \) values of individual pixels are calculated automatically by the scanner's computer, but first the margins of the relevant brain areas have to be defined. This was done independently by two raters, using a manual cursor. The means of the

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SUMMARY

Magnetic resonance images of the brain were obtained in 10 female patients before and 2–6 h after a standard general anaesthetic. The longitudinal magnetic relaxation times (\( T_1 \)) showed a non-significant decrease, whereas the transverse magnetic relaxation times (\( T_2 \)) increased in all scans, with significant increases in the pons and corpus callosum. This suggests that general anaesthesia may be associated with structural changes in the brain at a molecular level.

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two sets of relaxation times derived were used in all subsequent calculations. The significance of the mean changes in $T_1$ and $T_2$ values were assessed using Wilcoxon’s rank test for matched pairs.

The mean $T_1$ value decreased after anaesthesia and surgery in the different sections examined, apart from the left transverse section, in which there was no change (table I). However, none of these changes was significant.

The mean $T_2$ increased in all sections measured, significantly in the pons and corpus callosum (table I).

**COMMENT**

The main difficulty in studying patients before and after anaesthesia and surgery is the delay caused by the patient’s need to recover from the procedure before re-scanning. In Mander’s study and another by Scott (unpublished observations) the subjects underwent ECT, which is one of the few procedures that can reasonably be undertaken in the MRI suite; the time taken before re-scanning was approximately 30 min. The mean time between induction of anaesthesia and re-scanning in this study was 4.4 h because, in addition to the need to allow the patient to recover, the MRI scanner was not always immediately available. It is possible, therefore, that the observed changes would have been greater had re-scanning taken place sooner.

$T_1$ values reflect the water content of different tissues [2] and changes in $T_1$ may reflect shifts in the distribution of water molecules within the brain. The ECT studies showed an increase in $T_1$, which may have been offset partially by the effect of the anaesthetic, as $T_1$ in the volunteers (anaesthesia alone) showed a small decrease. As ECT is known to produce a temporary breakdown of the blood–brain barrier [3], Mander and colleagues suggested that the increase in $T_1$ which they observed after ECT was caused by osmotic changes secondary to changes in the blood–brain barrier. The mean $T_1$ values decreased in four of the five brain areas examined, and in both cerebral hemispheres in the five volunteers, although none was significant ($P < 0.05$). These changes accord with the findings in the present study, so it may be that a genuine change is occurring and that, with larger numbers of patients, a statistically significant change would be obtained. Although the precise cause of this change is unclear, it probably indicates an alteration in either the density or the molecular environment of water molecules in the tissue. Mander’s group [4] showed that ingestion of 14 units of alcohol (half a bottle of spirits) by normal volunteers was associated with a significant decrease in $T_1$ in several parts of the brain, consistent with the observation that alcohol promotes diuresis and dehydration. As alcohol resembles general anaesthetic agents in many pharmacological aspects, including lipid solubility, the mechanism involved may be the same.

The cause of the increase in $T_2$ (which was significant), which was not measured in the earlier study by Mander and co-workers [4] also cannot be explained at present. We believe that this is the first study in human subjects of changes in $T_2$ associated with anaesthesia. Animal studies have been concerned largely with the effect of experimental physiological insults [5], or with the uptake and elimination of anaesthetic agents, using a variety of different resonating nuclei (for
example phosphorus-31, carbon-13, fluorine-19 and sodium-23, in addition to hydrogen-1) [6]. Reid (unpublished observations) has also confirmed the findings of Mander's group that electrically induced convulsions in rats produce a temporary increase in T₁, but he did not measure changes in T₂. We suggest that the changes may result from structural alterations which have taken place at a molecular level, and this study describes the changes found using one method of anaesthesia. It seems clear, however, that the phenomenon merits further investigation with other anaesthetic agents and, in particular, study of the time course of the changes produced.

REFERENCES