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Oxygen Saturation during Electroconvulsive Therapy

Oxygen saturation was measured during anaesthesia and electroconvulsive therapy (ECT) using ear oximetry. Significant hypoxia occurred during some treatments; desaturation related to the number of ventilations performed after muscle relaxation and prior to the shock. Desaturation did not correlate significantly with seizure duration. These findings highlight the need for adequate oxygenation during ECT.

The major goal of anaesthesia for ECT is prevention of complications such as discomfort, fractures, aspiration of gastric contents, bradycardia and hypoxia. Monitoring of oxygenation is important during anaesthesia and, as clinical signs are often unreliable, ear oximetry is recommended as a more precise method of detecting hypoxia. The present study measured haemoglobin saturation during routine anaesthesia and ECT to assess the incidence of hypoxia under normal circumstances.

Method

Local Ethical Committee approval was obtained for the study and only patients unable or unwilling to consent were excluded. Four separate ECT sessions were studied, involving 40 patients who were grouped according to which anaesthetist was responsible for them. Anaesthetists were asked not to deviate from their usual practice and did not know what was being recorded; they were not allowed to see the oxygen saturations during the study. The apparatus was unobtrusive and did not interfere with the routine of the list. All patients were anaesthetised using methohexitone and suxamethonium without atropine. A flow of 6–10 litres per minute of 100% oxygen was invariably administered, using a Mapleson C circuit, without a carbon dioxide absorber. Positive pressure ventilation was performed following the administration of the muscle relaxant, both before and after the seizure. The number of positive pressure ventilations given prior to the shock and the time at which oxygen was administered after the seizure were recorded. The seizure duration was measured using the isolated arm technique (Fink & Johnson, 1982).

Oxygen saturation was measured using a Hewlett-Packard ear oximeter, which measures saturations in the range of 65–100%. The patient's ear was 'arterialised' by brisk rubbing for 15–30 seconds, and the probe was applied to the pinna. A potentiometric recorder made a continuous trace of the oxygen saturation during anaesthesia and ECT. The change in oxygen saturation was calculated by subtracting the lowest saturation reached after seizure and before the administration of oxygen from the baseline value.

Data were analysed using Pearson's Product Moment Correlation Coefficient and analysis of variance, followed by the Tukey test, where appropriate.

Results

Four anaesthetists were responsible for 10 patients each; 33 patients were female and 7 were male; the median age was 49 and the age range 21–78 years. The doses of methohexitone and suxamethonium used by each anaesthetist did not differ significantly (Table I).

TABLE I
The doses of anaesthetic drugs used in the four groups of patients

	Anaesthetist			
	1	2	3	4
Methohexitone: mg				
Mean	66	64	51	74
SD	24	22	17	28
Suxamethonium: mg				
Mean	45	30	35	35
SD	10	8	6	6

The range of oxygen saturations recorded during ECT was 79–100%. 16 subjects demonstrated a fall in oxygen saturation of more than 5% from the baseline value, lasting from 15–75 seconds. Prior to anaesthesia there were no significant differences in oxygen saturation between the four groups, and the degree of hypoxia was unrelated to initial oxygen saturation (Table II). Patients in group 1 remained better oxygenated than those in groups 2 ($P<0.01$), 3 ($P<0.01$) and 4 ($P<0.05$) (Table II). The only difference in anaesthetic technique between the groups was the number of ventilations performed after the suxamethonium, but prior to the shock; anaesthetist 1 ventilated more than the others ($P<0.01$, Table II). The change in oxygen saturation correlated with the number of ventilations given prior to ECT ($r=0.65$, $P<0.001$). All anaesthetists commenced oxygenation within 60 seconds after the seizure had finished, and the oxygen saturation invariably returned to baseline levels or higher.

The seizure duration was significantly less in group 2 compared with group 1 ($P<0.05$, Table II). The duration of seizure did not correlate significantly with the change in oxygen saturation ($r=0.21$).

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TABLE II

Baseline and change in oxygen saturation, number of ventilations given and seizure duration in the four groups of patients

	Anaesthetist 1	2	3	4
Baseline O_2 saturation: %				
Mean	94.3	92.8	92.2	95.1
SD	2.5	3.7	2.7	2.3
Range	87–98	84–96	87–95	92–98
Change in O_2 saturation: %				
Mean	+4.0*	−7.0	−5.2	−1.1
SD	2.0	5.8	4.0	5.2
Range	0–+6	−13–+1	−11–+2	−10–+4
No. of ventilations				
Mean	18.2**	4.8	7.2	8.8
SD	4.9	1.5	4.5	1.6
Range	11–25	4–9	3–15	7–12
Seizure duration: s				
Mean	34.9	15.6†	31.1	24.7
SD	17.4	4.9	19.5	14.4
Range	15–65	6–23	8–80	15–60

*Group 1 significantly less hypoxic than groups 2 ($P<0.01$), 3 ($P<0.01$) and 4 ($P<0.05$). **Group 1 significantly more ventilations given than groups 2, 3 and 4 ($P<0.01$). †Group 2 significantly shorter seizure duration than group 1 ($P<0.05$).

Discussion

Death during ECT has been ascribed to cardiac problems involving both vagal and ectopic mechanisms (Bankhead *et al*, 1950). The ECT seizure causes widespread physiological changes due to autonomic stimulation. Bradycardia followed by tachycardia usually occurs, with hypotension followed by hypertension, therefore cardiac output falls initially, exacerbating tissue hypoxia, before rising. Low oxygen saturation occurring as heart rate and blood pressure increase predisposes to myocardial ischaemia and arrhythmias. Atropine was recommended to reduce vagal problems, however, its routine use has been challenged as it does not contribute to cardiac stability during ECT (Wyant & Macdonald, 1980).

As early as 1950, Bankhead and colleagues suggested that cardiac ectopic phenomena occur during the 'deepest cyanosis following the convulsion', but the use of oxygen during ECT did not then become routine. In a description of ECT in 1968 the shock was delivered 50 seconds after the relaxant and, following the clonic phase, three hand ventilations were delivered using, 'room air . . . and never oxygen' (Pitts *et al*, 1968). As recently as 1979 it has been claimed that oxygenation during ECT is unnecessary (Joshi, 1979), although apnoea after the shock can last for a few minutes, and will cause significant hypoxia if untreated. The present study was designed to monitor oxygenation in the clinical situation during routine anaesthesia and ECT. Significant hypoxia was demonstrated and desaturation correlated with the number of ventilations performed prior to the shock, a simple variable in anaesthetic practice.

The present study supports the concept that hyperventilation before ECT may prolong the seizure, as the patients who received fewest ventilations (group 2) had the shortest seizure duration, perhaps reflecting hypercapnia. Hyperventilation with oxygen therefore prevents hypoxia, and may also enhance therapy. As hypoxia was not related to the duration of the seizure, it may be that carbon dioxide is a more important determinant of seizure activity.

The occurrence of hypoxia is demonstrated in the present study, but not shown to be detrimental. In normal circumstances transient hypoxia should be of little consequence, but if accompanied by other problems, for example cardiovascular changes or vomiting, may be more serious. A survey of anaesthetic practice in 1980 showed that most patients are now kept well oxygenated, but a few clinics still did not routinely administer oxygen (Pippard & Ellam, 1981). As the same study showed that over 50% of the anaesthesia for ECT is performed by anaesthetists in training, teaching should emphasise the need for adequate oxygenation.

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References

BANKHEAD, A. J., TORRENS, J. K. & HARRIS, T. H. (1950) The anticipation and prevention of cardiac complications in electroconvulsive therapy. *American Journal of Psychiatry*, **106**, 911–917.

FINK, M. & JOHNSON, L. (1982) Monitoring the duration of electroconvulsive therapy seizures. *Archives of General Psychiatry*, **39**, 1189–1191.

JOSHI, V. G. (1979) Modifying ECT with a safe, simple and economical technique. *British Journal of Psychiatry*, **134**, 128.

PIPPARD, J. & ELLAM, L. (1981) Electroconvulsive treatment in Great Britain. *British Journal of Psychiatry*, **139**, 563–568.

PITTS, F. N., WOODRUFF, R. A., CRAIG, A. G., RICH, C. L. & ST LOUIS, A. B. (1968) The drug modification of ECT II. Succinylcholine dosage. *Archives of General Psychiatry*, **19**, 595–599.

WYANT, G. M. & MACDONALD, W. B. (1980) The role of atropine in electroconvulsive therapy. *Anaesthesia and Intensive Care*, **8**, 445–450.

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Anorexia Nervosa and Gastric Rugal Hyperplasia

A case of anorexia nervosa associated with gastric rugal hyperplasia in a 36-year-old male is reported and some speculations are made about the relationship between these two conditions.

Although anorexia nervosa in the male is an uncommon condition, recent studies have shown that males account for 5–10% of all cases (Hogan *et al* 1974; Anderson & Mickalide, 1983). Anorexia nervosa has been associated with a range of medical conditions, including hypothalamic tumour (Heron & Johnson, 1976), pancreatitis (Schoettle, 1979), Gaucher's Disease (Erman & Murray, 1980), Superior-mesenteric-artery syndrome (Froese *et al*, 1978) and pneumoperitoneum (Al-Mufty & Bevan, 1977). Several authors have pointed out that in cases where anorexia-like symptoms co-exist with a physical condition, it is not always easy to decide whether the weight loss is a primary or secondary symptom (Schoettle, 1979). Others have indicated the importance of excluding organic conditions in anorexia nervosa (Heron & Johnson, 1976). The latter authors also comment that even when anorexia nervosa-like symptoms are of organic origin there may be some response to psychological treatment alone, thus highlighting the importance of correct diagnosis before treatment.

Case history

A 29-year-old male wholesale butcher was admitted in an emaciated state measuring 1.765 m but weighing only 55.10 kg. Although overweight as a child, he had weighed 73.00 kg for many years. He gave a 2-year history of gradual weight loss and had lost 25% of his original body weight by the time he was first seen. A previously fit man, during the month prior to admission he had complained of satiety following small meals together with feeling light-headed. He had a 2-week history of being short of energy and, just prior to admission, had collapsed at work whilst carrying a meat carcass on his back. The patient's wife reported that he had always been a loving husband and father who had been quite involved in bringing up their three children. He had often become anxious about his health when developing minor physical illnesses but rapidly returned to his cheerful self on recovery. She had first noticed a problem 2 years previously following the birth of their last child. The patient had again become anxious about his health and, being concerned that his hair might be falling out, had arranged to have a hair transplant. Shortly after that he became concerned about his weight and refused food that she had cooked insisting on providing only high protein meals for himself. He also began to count the calorie content of his meals with the aid of slimming magazines which he hid from the family. By the time of referral he had excluded nearly all carbohydrates from his diet and had begun to abuse laxatives. Although not previously an athletic man, he had recently taken up weight-lifting to the exclusion of all other leisuretime activities. Over the same period he had lost all sexual interest in his wife in spite of their having had a previously satisfactory sexual relationship.

Examination showed marked weight loss, muscle wasting, dry skin and hair and cold cyanosed extremities. His pulse was 60/minute, blood pressure 115/80 mmHg and oral temperature 36.5 °C. The abdomen was scaphoid with no abnormal palpable masses. Even when shown his scaphoid abdomen on video tape he insisted that it protruded. He could not accept that his admission weight was below that expected for a man of his height and build but agreed that he should gain weight though felt that this could only be achieved through muscle development by weight training with reduced food intake.

Investigations revealed a lowered total protein of 53 g/litre with serum albumin of 37 g/litre, raised blood urea of 100 mmol/litre, and raised serum free cortisol of 745 mmol/litre. A barium meal, undertaken because of his complaints of early satiety, demonstrated prominent gastric folds in the body and fundus of the stomach. Although the radiology suggested malignancy, endoscopy and biopsy showed hypertrophic gastropathy with gastric foveolar hyperplasia. There was no evidence of increased gastric secretion even following pentogastric stimulation.

Pending investigation the patient was placed on a regime of bed rest and gradual refeeding which initially resulted in some weight gain. He continued to request laxatives and slimming magazines and, after his wife had refused to co-operate in this, purchased large quantities of a particular brand of mints from the mobile hospital shop which were

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