ganglia. These highly suggestive correlations were further supported by pharmacological manipulation (table 2). 5,6-DHT, which destroys 5-HT neurons[20] in the ganglia has a concomitant effect on the acidic PL, especially in the rich 5-HT containing CG. The minimal changes seen in the VG parallels its relatively smaller content of 5-HT. Furthermore, our previous studies[14] have shown a reciprocal relationship between DA and 5-HT but it is possible that in the reverse situation, i.e., 5-HT effects on DA, a more directly proportional relationship may exist as suggested by the 5,6-DHT effects on lyso PC (table 2). The destruction of dopaminergic neurons by 6-OHDA produced an increase (about 73% in the pedal ganglia[14]) in the 5-HT content. These previously reported changes[14] paralleled the PL changes (table 2) in that there was a marked decrease in lyso PC matched with a significant increase in the acidic PL.

The data presented suggests a functional relationship between specific phospholipids and monoamines in ganglia of *Mytilus edulis*. More direct evidence such as that shown for the stimulation of enzymes of monoamine metabolism by specific phospholipids[21] are needed to confirm these suggestions. Portions of this work have been presented previously[22].

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Effect of electroconvulsive treatment on serum dopamine-beta-hydroxylase activity in man

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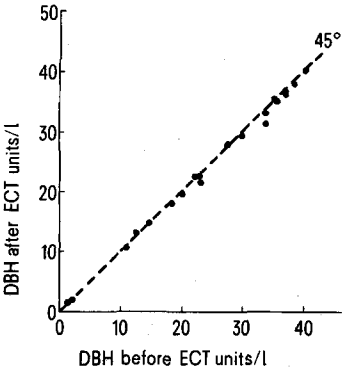
Summary. The activity of dopamine-beta-hydroxylase was measured in the serum before and immediately after electroconvulsive treatments. No significant difference was observed, suggesting that the seizures did not cause an increase in the peripheral sympathetic tone.

Effective electroconvulsive treatment (ECT) causes major convulsions and thus can be clinically and biochemically paralleled with epileptic major convulsions. Spontaneous grand-mal seizures are accompanied by autonomic changes but direct evidence of sympathetic activation is meagre. Heart rate and blood pressure are slightly changed but these changes may be secondary to other changes (e.g. respiratory). ECT, like spontaneous grand mal seizure, is believed to cause a simultaneous discharge of (all?) brain neurons. ECT was found to increase serum prolactin level[3]. Ohman et al.[3] discuss the possibility that the effect on serum prolactin is due to a general nonspecific CNS activation, and since ECT was not found to cause a significant increase of thyroid stimulating hormone[4], they concluded that the effect of ECT was more or less specific. There is evidence that ECT activates central adrenergic mechanisms[4]. The present study was carried out to determine whether sympathetic activation occurs in the periphery in humans. The levels of dopamine-beta-hydroxylase (DBH) in serum before and after ECT were used as a measure of peripheral sympathetic activity[5]. This enzyme is responsible for the last step in the synthesis of noradrenaline with which it is released from sympathetic nerve endings.



Serum DBH activity following ECT plotted against pre-ECT activity. Each point represents the activity values preceding and following a single treatment.

Materials and methods. Patients. The study included 8 patients, 6 males and 2 females, ranging in age from 14 to 65 years. 7 patients were diagnosed as schizophrenic and 1 as involutional depressive. Each patient underwent several ECT treatments and DBH was examined in 2 or 3 of these in each patient. 2 of the patients were not receiving drugs and the others were on antipsychotic (haloperidol, fluphenazine or chlorpromazine) or antidepressant (imipramine or chlorimipramine) drugs.

ECT procedure. Following bed-rest for 30 min, the patient was premedicated by thiopental, 4 mg/kg, and succinylcholine, 0.5 mg/kg i.v. Electroshock treatment was immediately delivered by a Siemens Konvulsator 2077 with continuous pulse sequence at an intensity of 400–550 mA peaked current referred to a patient resistance of 300 ohms and current flow duration of 0.8–1.6 sec. In all patients at least a minor seizure was observed in response to the ECT.

Enzyme assay. Venous blood samples were collected from the patients before premedication and once again 30–150 min following the ECT, while the patients were still prone. The samples were allowed to clot at room temperature, and the serum was separated by centrifugation and stored at −5 °C until assayed. The activity of DBH in serum was measured according to Nagatsu and

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Udenfriend[6]. 1 unit of DBH is the amount of enzyme catalyzing the oxidation of 1 μmole of tyramine to octopamine, in 1 min incubation at 37 °C. The blood samples taken before and after treatment were processed and analyzed simultaneously.

Results. DBH activity values before and after ECT were similar: when plotted one against the other, a straight regression line with a slope of 45° resulted (figure). In the few patients examined more than once, the changes in DBH levels from one ECT to the next were small and not consistent in direction.

Discussion. Our results show that ECT does not elevate DBH activity in the blood, and presumably does not increase sympathetic tone. Most of our patients have been treated with drugs when the ECT were performed. It seems unlikely that these drugs could inhibit sympathetic activation. The 2 patients who were not receiving any drugs also have not demonstrated any elevation of DBH activity after any of the 4 individual ECT sessions. There is ample evidence that ECT increases noradrenergic activity within the central nervous system[4]. Our results suggest that this activation does not occur in the periphery. By analogy, it may be concluded that spontaneous grand-mal seizures do not activate sympathetic pathways directly. This may be due to the fact that cardiovascular control is exerted by structures low in the brain stem, at least in the unconscious state. On the other hand, respiratory function is affected by both spontaneous grand-mal seizures and ECT. The reason for the relative vulnerability of the respiratory control system is not clear. In any case, whenever breathing is affected, sympathetic tone may be changed secondarily.

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Effect of preoperative stress on serum cholesterol level in humans

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Summary. Effect of preoperative stress on serum cholesterol level in 65 patients of different age groups in a surgical ward, has been studied. In all age groups, statistically significant rise of cholesterol in serum preoperatively was noticed as compared with serum cholesterol level at the time of discharge from hospital. The preoperative rise of cholesterol varied from 39 to 56.9% in this series. These findings support previous reports of the effect of mental tension on serum cholesterol level.

Chronic exposure of man to various emotional stressors leads to increased plasma concentration of cholesterol[2–8]. Selye[9] has described a biphasic response of serum cholesterol and lipids in his general adaptation syndrome. Beischer[10] failed to find any significant change in serum cholesterol, phospholipids, and lipoproteins following the subject's first experience of human centrifuge ride, or the experience of simulated failure of low pressure chamber. Mann and White[11] concluded that experimental stress resulted in lowering of blood cholesterol in dogs and rats. Carlson et al.[12] observed increase in plasma-free fatty acids and relatively lesser alterations in cholesterol, and phospholipids in men during experimentally induced emotional stress. Kyle et al.[13] observed lowering of serum cholesterol levels post-operatively as compared with their control values. In view of these observed discrepancies in serum cholesterol values in response to mental tension, it was of interest to study the serum cholesterol levels in patients subjected to preoperative (i.e. psychological) stress.

Material and methods. Subjects selected for preoperative stress investigation were from surgical wards. Patients suspected clinically of lipid-cholesterol metabolic disorders and confirmed by biochemical investigations were discarded. Subjects were classified into 5 groups according to their age. Group A comprized of patients age 11–20, group B of age 21–30, group C of age 31–40, group D of age 41–50, and group E of age 51–60. For each patient, serum cholesterol was estimated on 4 occasions. 1. On the day of admission (morning fasting sample between 08.30 and 09.00 h). 2. On the day of operation just before adminis tering anaesthesia. 3. Postoperatively after the effect of anaesthesia is over (usually between 16.0 and 16.30 h). 4. On the day of discharge (morning fasting sample between 08.30 and 09.00 h).

No anti-stress drugs were administered. Atropine was given to patients who were operated under general anaesthesia. Total cholesterol was determined according to method of Zak[14]. Means and standard error were calculated and results were compared for statistical significance by means of paired 't' test.

Results. The results recorded in the tables clearly indicate a definite pattern, such as rise of serum cholesterol, right from the day of admission, reaching its peak preoperatively, i.e. on the day of operation, falling to a lower level post-operatively and reaching the minimum on the day of discharge. Preoperative rise of cholesterol in all age groups is statistically significant. Percentage rise

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