

SECRETION OF PARATHYROID HORMONE AFTER SURGICAL TRAUMA

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Parathyroid hormone was measured in nine patients subjected to surgery under general anaesthesia not involving operative bone trauma. Total calcium concentration, ionized serum calcium and serum albumin was also analyzed pre- and postoperatively. An increase in serum parathyroid hormone was registered postoperatively without any accompanying decrease in ionized serum calcium. The measured changes are at present unexplained, but can be caused by other mechanisms than bone trauma.

Key words: parathyroid hormone; surgical trauma

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Operative bone trauma under general anaesthesia has been reported to cause a postoperative elevation of circulating parathyroid hormone (PTH) levels (Hulth & Johnell 1979). This increase was accompanied by a decrease of the total serum calcium (S – calcium), but when the calcium concentration was corrected for the simultaneous fall in the serum – albumin (S – albumin) this so-called “corrected S – calcium” remained unchanged.

The purpose of this study was 1) to examine whether the changes in PTH – levels were due to the operative trauma per se, or if it was a reactive response to the specific bone trauma, and 2) to examine whether the ionized S – calcium was a better parameter than the albumin corrected S – calcium for describing the changes in the calcium homeostasis.

PATIENTS AND METHODS

The patients, six men and three women aged 45–66 years (mean 59 years), were all subjected to a surgical

trauma (endarterectomy of the carotid artery (four patients), iliac – femoral arterial by-pass (four patients) and pneumonectomy (one patient). All had normal serum creatinine and were anaesthetized using halothane. The operations lasted from 1 to 3 hours. Three patients received citrated blood transfusions during surgery, respectively 500 ml, 500 ml and 1500 ml.

Blood samples were taken after an overnight fast preoperatively, the 1st postoperative day, the 3rd day, the 6th or 7th day and the 10th to 14th postoperative days. Total S – calcium, ionized S – calcium and S – albumin was determined immediately. Samples for determination of PTH were centrifuged within 2 hours, and the serum was stored at –20°.

The total S – calcium was determined by atomic absorption spectrophotometry.

The ionized S – calcium was determined by a Ca²⁺ sensitive electrode (Radiometer, Copenhagen) after equilibration to fixed pH and p_{CO₂}.

The S – albumin was determined using an immunochemical method (Laurell 1966).

The S – PTH was analysed at Medicinsk Laboratorium, Copenhagen using a radioimmunoassay method standardized to Medical Research Council, England No. 71/324 = 1 (Rasmussen et al. 1978).

Total S – calcium was corrected for S – albumin according to the formula: “corrected S – calcium” = total

$S - \text{calcium} + 1.1 \times (0.700 - S - \text{albumin})$ (Dent 1962).

The results were registered for each patient as the percentage of the preoperative value. Statistical evaluation was done using the Wilcoxon test of paired data.

RESULTS

The results are shown in Figure 1. PTH was significantly elevated on the 1st and 3rd postoperative day ($P < 0.001$ on day 3) tending to normalise towards the 10th to 14th day. Total S - calcium and S - albumin decreased significantly postoperatively ($P < 0.01$ on day 3), whereas ionized S - calcium showed no decrease ($P > 0.1$). When total S - calcium was corrected for the decrease in S - albumin a decrease was seen on the 1st postoperative day ($P < 0.05$) returning to preoperative levels on the 3rd day.

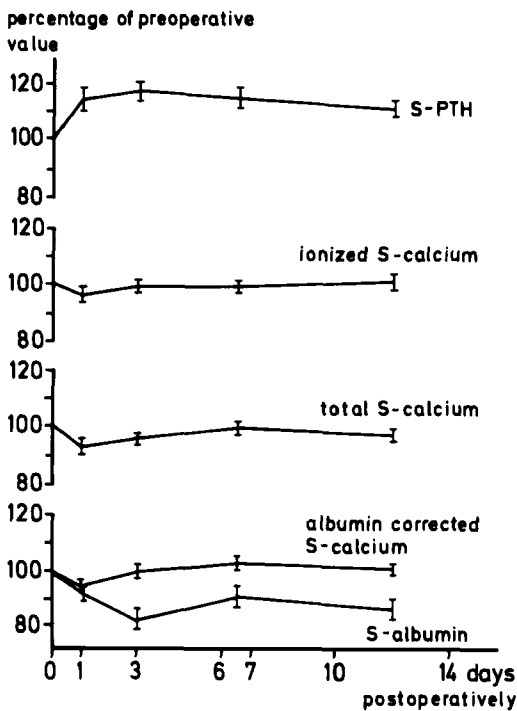


Figure 1. Postoperative values expressed as the percentage of the preoperative values of PTH, ionized S - calcium, total S - calcium, S - albumin and "corrected S - calcium" in nine patients subjected to general surgery. The values are expressed as the mean \pm s.d.

The three patients receiving blood transfusions during surgery did not differ from the other six patients, nor did the duration of the operation influence the results.

DISCUSSION

The present study confirmed the observations by Hulth & Johnell (1979) with respect to the elevation of PTH after surgery. Using the same assay for PTH as in the present study their patients had higher PTH levels than our patients did. Whether this is due to an additive stimulation of the bone trauma is unclear. Several other explanations exist. The patients in the present study were older, only a few patients received blood transfusions and the tissue damage was probably less following carotid surgery than after Harrington's operation.

The study by Hulth & Johnell (1979) did not demonstrate any change in the albumin corrected S - calcium, but the method of calculation was not mentioned. In the present material a decrease was seen on the 1st postoperative day. Furthermore we included measurements of the ionized S - calcium, and this parameter remained unchanged during the postoperative course. Only postoperative values were registered, so a preoperative fall in the ionized S - calcium as the physiological basis for PTH stimulation can not be ruled out. Rodriguez et al. (1976) and Hinkle & Coopermann (1971) have shown a significant fall in ionized S - calcium preoperatively. Both authors ascribed this change to low P_{CO_2} values. Animal experiments have confirmed, that alkalosis is a potent depressor of ionized S - calcium (Kaplan et al. 1971).

In the study by Rodriguez et al. (1976) a transient increase in PTH was seen, returning to normal 1 hour after surgery. The explanation for this might be that the assay used for measuring PTH also measures biologically active N - terminal PTH, which has a short half-life (Arnaud et al. 1971, Potts et al. 1971). In the present material predominantly C - terminal biologically inactive fragments of PTH were measured. Thus it is possible that the increase in PTH was due to a preoperative fall in ionized S - calcium, which

had returned to normal before the C – terminal biologically inactive fragments had been cleared from the blood.

Several other possibilities exist. Decreased triiodothyronin (T_3) due to hemodilution following peroperative infusion of 1000–2000 ml saline might stimulate PTH – secretion (Mosekilde 1979). Hemodilution can also lead to a decreased concentration of vitamin D metabolites, which in turn can cause a transient normocalcaemic hyperparathyroidism (Fischer et al. 1973a).

Another explanation might be that the PTH change is due to an increased adrenal stimulation. Catecholamines have been shown to stimulate PTH secretion (Fischer et al. 1973b), and surgical trauma under general anaesthesia is known to stimulate the secretion of catecholamines as well as several “acute phase proteins”. Probably several of the above mentioned mechanisms or even others are responsible for the postoperative elevation of PTH. The exact mechanism can only be clarified through further studies, but bone trauma per se does not seem to be the only cause of PTH stimulation. Ionized S – calcium is recommended rather than albumin – corrected S – calcium for describing changes in the calcium homeostasis.

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