CALCIUM INFUSION IN THE DETECTION OF BONE DISEASE IN PARATHYROID DISORDERS

By

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ABSTRACT

1. The retention of an infused load of calcium was determined under standard conditions in 25 patients with various parathyroid disorders, in 12 normal control subjects, and in 3 patients with idiopathic hypercalciuria.

2. A normal range of 40–60 per cent calcium-retention was found, and there was some support to the thesis that hypercalciuria per se may lower the retention of calcium.

3. Patients with primary hyperparathyroidism showed a wide range of calcium retention reflecting on one side probably hypercalciuria (low calcium retention) and on the other osteitis fibrosa generalisata (high calcium retention). In detecting early bone involvement in parathyroid hyperfunction, the calcium retention test was of equal or greater value than alkaline phosphatase determination in the serum.

4. In secondary hyperparathyroidism due to severe renal insufficiency, a high calcium retention was seen pointing either to delayed calcium excretion (low GFR) or increased avidity of the skeleton for calcium as a consequence of an admixture of osteomalacia and osteitis fibrosa.

5. All hypoparathyroid patients retained large quantities of calcium. In three of these cases, an elevated alkaline phosphatase level indicated osteomalacia, possibly following inadequate calcium absorption from the gut, while in two patients a low filtered load of calcium accounted for the apparent high calcium retention.


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Calcium infusions have been used primarily to study the suppressability of parathyroid activity by raising the serum calcium level (Howard et al. 1952, 1953; Kyle et al. 1954, 1962; Pronove & Bartter 1961). In this study, calcium infusions were carried out to detect possible bone involvement in a variety of parathyroid disorders, and the per cent retention of an infused load of calcium was used as an index for the ability of the skeleton to attract calcium.

**Patients and Methods**

Studies were undertaken in 16 patients with proven primary hyperparathyroidism, in 3 with secondary parathyroid hyperfunction due to renal insufficiency, and in 6 patients with hypoparathyroidism. A group of 12 subjects with no demonstrable disturbance in calcium-phosphorus metabolism and with radiologically normal bones served as controls. In addition, 3 hypercalciuric patients with no provable parathyroid or skeletal disease were examined to gain some information on the influence of increased calcium excretion (as is seen so frequently in primary hyperparathyroidism) on calcium retention.

Each patient ingested a low constant calcium diet (in most instances containing either 150 or 300 mg calcium per 24 hours) for 3 to 5 days prior to study. 24-hour urine collections from 8 A.M. to 8 A.M. were started at least 2 days prior to the day of infusion (Fig. 1). On the day of infusion, all patients received an intravenous load of calcium gluconate containing 10 mg calcium per kg body weight in 500 ml of normal saline, over a 3 hour period (9 A.M.-12 Noon). All but two patients (one of whom a child) were given a minimum of 600 mg calcium. 24-hour urinary calcium on two days of control and on the infusion day was determined by the Kramer-Tisdall method (1921) as modified by Clark & Collip (1925). The calcium retention was calculated by the following formulas:

\[
\begin{align*}
(1) \quad & uCa_{id} - uCa_{cd} = uCa_{net} \\
(2) \quad & iCa - uCa_{net} = ret.Ca \\
\text{ret.Ca} \times 100 & = \% ret.Ca, \\
\end{align*}
\]

Fig. 1.
Method for determination of calcium retention: Urinary calcium depicted by hatched areas below the zero line.
i.e. the net amount of calcium excreted from the infused load (\(u\text{Ca}_{\text{net}}\)) was obtained by subtracting the mean urinary calcium on control days (\(u\text{Ca}_{\text{ctl}}\)) from the urinary calcium on infusion day (\(u\text{Ca}_{\text{inf}}\)), giving the retained amount of calcium (\(\text{ret. Ca}\)), which is expressed as a percentage of the infused load. Faecal calcium excretion was neglected, assuming an insignificant rise in stool calcium following calcium infusion, as demonstrated by McCance & Widdowson (1939); Baylor et al. (1950) and Canary et al. (unpublished data). The serum alkaline phosphatase was determined by the Bodansky method (1933) or the Bessey-Lowry technique (1946). Bessey-Lowry units being converted for comparison into Bodansky units by multiplying with the conversion factor 1.8, as given in the original publication by Bessey et al. (1946) and confirmed by Fraser & King (1957) and Südhof et al. (1962). Converted values are indicated in Tables 1–4 by an asterisk.

**RESULTS**

1. *Normal range.* – The mean calcium retention in the control group with no demonstrable disturbance in mineral metabolism was 49.6% (Fig. 2) with a standard deviation of ± 4.5. We have therefore defined our normal range as 40–60% retention of an infused load of calcium.

2. *Effect of hypercalciuria on calcium retention.* – In agreement with Albright & Reifenstein (1948), we define hypercalciuria on a low calcium diet as being present if the 24-hour urinary calcium exceeds the daily calcium intake. On a 150 mg calcium diet, urinary calcium values in excess of 150 mg are highly suspicious – values exceeding 200 mg definitely diagnostic for hypercalciuria. In our small group of patients with idiopathic hypercalciuria (Table 1), the one with the highest calcium excretion on a 150 mg calcium diet retained only 36% of an infused calcium load. This result supports the view that hypercalciuria *per se* may eventually lower calcium retention, but this thesis needs further testing.

3. *Primary hyperparathyroidism.* – The group of 16 patients with primary
hyperparathyroidism can be divided into 3 cases with and 13 without significant renal damage (Table 2).

(a) Cases without renal damage. – Two of the 13 patients had overt bone disease by X-ray, high alkaline phosphatase levels, and correspondingly high calcium retention (cases C. C. and T. W.) (Fig. 3). In another 6 patients (W. C., E. D., R. G., K. H., G. J., G. L.), the alkaline phosphatase was normal or insignificantly increased, and there was no evidence for skeletal changes by X-ray. These patients retained normal or low amounts of calcium, 2 with overt hypercalciuria (W. C. and R. G.) showed no retention. Patient G. F. had normal calcium retention despite normal or border-line elevation of alkaline phosphatase (A. M., A. S., L. S., P. S.). On routine examination, no bone involvement was reported either by X-ray technique or histologic examination. But in a 60 year old woman with radiologically normal bones and an alkaline phosphatase value of 3.7 Bodansky units (normal range: 1.5–4.0) (patient L. S.), who retained 75 % calcium, a careful review of the histologic sections of the bones revealed unquestionable osteitis fibrosa generalisata, previously overlooked. Another patient (A. M.), with a calcium retention of 72 % and an alkaline phosphatase level of 3.4 Bodansky units, showed as the only sign of osteitis fibrosa small bone cysts in the phalanges of one hand. In patient P. S. there was a slight radiological demineralisation of the phalanges, and he retained 70 % of the infused load of calcium and had an alkaline phosphatase value of 4.5 Bodansky units.

![Graph](https://via.placeholder.com/150)

**Fig. 3.**
Relation between retention and alkaline phosphatase in primary hyperparathyroidism: 13 patients without significant renal damage. Normal range for calcium retention (40–60 %) and for alkaline phosphatase (1.5–4.0 Bodansky units) indicated by the broken lines. Of particular interest is the group of 4 patients (heavy black dots) clearly above the upper limit of normal calcium retention, but with practically normal alkaline phosphatase levels.

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In three cases (G. F., A. M., T. W.), postoperative studies were carried out at different time intervals. Two patients showed an increase of calcium retention 1½ months and 7 months after surgery, the third a slight decrease 5 months after removal of the parathyroid adenoma.

(b) Cases with significant renal damage. – In this patient group (endogenous creatinine clearance < 50 ml/min; occasionally elevated BUN), hypercalciuria was noted only in patient M. S., but all three retained large amounts of calcium and showed definite elevation of alkaline phosphatase. In all three cases, osteitis fibrosa generalisata was found by X-ray.

4. Secondary hyperparathyroidism. – In 3 patients with secondary hyperparathyroidism due to severe renal insufficiency with chronic acidosis, calcium retention ranged from 93 to 97 (Table 3). Since a delayed calcium excretion was possible because of the very low GFR, the 24-hour urinary calcium, on post-infusion day was also determined in two cases: In patient J. W. it returned to the pre-infusion base-line level (71 mg), in R. B. it stayed somewhat elevated (106 mg). Two of the patients, who had very high alkaline phosphatase levels, revealed severe bone involvement: J. W. had diffuse parathyroid hyperplasia and an admixture of severe rickets and osteitis fibrosa generalisata at autopsy; L. M. showed a border-line elevated serum calcium level (10.9 mg/100 ml) despite a very high serum phosphorus (9.5 mg/100 ml), and by X-ray examination a diffusely demineralized skeleton with bone cysts and subperiostal resorption in the phalanges was detected as well as widespread calcification of the larger arteries.

5. Hypoparathyroidism (Table 4). – Six patients with hypoparathyroidism (5 after removal of all parathyroid tissue and 1 with the idiopathic type) showed high calcium retention ranging from 81 to 91 %. Interestingly enough, in the three cases in which alkaline phosphatase in the serum was determined, the level of this enzyme was clearly elevated. The patient (E. B.) with the highest phosphatase value showed an increased density of the spine after treatment with vitamin D.

**DISCUSSION**

The retention of an infused load of calcium is well defined by a normal range of 40–60 % in control subjects on a low calcium diet. In primary hyperparathyroidism, we found a wide range of calcium retention from zero to very high percentages. A consistently high calcium retention was seen in secondary hyperparathyroidism and in hypoparathyroidism.

Theoretically, two factors may influence calcium retention, provided that the extracellular calcium pool and the intracellular calcium content remain constant (Whedon 1959) or are only temporarily changed following a calcium infusion where the serum calcium returns to the base-line level in less than 24 hours (Kyle et al. 1954):
1. The ability of the skeleton to attract and bind calcium on the mineral-
exchanging surface, and
2. the rate and degree of calcium excretion through the kidneys.

It is conceivable that a balance exists between these two antagonists, in
order to maintain a constant serum calcium level. This concept could best be
illustrated by conditions in which one of the two clearly dominates: On the
one hand in a hypercalciuric subject with normal skeletal function, a decreased
calcium retention should be expected. Unfortunately our group of patients
with idiopathic hypercalciuria is too small, but one of the three patients re-
vealed the presumed low retention of calcium. However, further studies will
be necessary to draw firm conclusions. In the alternative case, patients with
increased avidity of the bones for calcium but with normal kidney function,
\textit{e.g.} vitamin D-deficient osteomalacia, showed a high calcium retention (Haas
\textit{et al.} 1961), revealing the preponderance of the skeletal factor. Primary hyper-
parathyroidism seems to be a condition in which both antagonists, skeleton
(osteitis fibrosa generalisata) and kidneys (hypercalciuria) are overactive.
Theoretically, no calcium could be retained in a case with overt hypercalciuria
but without bone involvement, while a patient with severe osteitis fibrosa but
without hypercalciuria should reveal a high calcium retention. This was true
in our hyperparathyroid patient group where the calcium retention ranged
from 0 to 94\%o. Fig. 4 relates the two factors, osteitis fibrosa and hyper-
calciuria, to the calcium retention. On one extreme, two patients retained no
calcium at all, on the other, the calcium retention exceeded 90 \%o in 4 patients.
But more common was a combination of osteitis and hypercalciuria indicated
by the considerable overlap between the triangular areas representing the two
opponent factors.

Recently a new concept on the action of alkaline phosphatases in the skeleton
was propounded (Fleisch \& Neuman 1960; McLean \& Urist 1961). From a
clinical standpoint, however, determination of alkaline phosphatase activity
in the serum is still of considerable value in estimating osteoblastic activity,
and an elevation of this enzyme is generally considered to be the most sen-
tive index for bone disease in hyperparathyroidism (Albright \& Reifenstein
1948; Dent \& Harper 1962). This was confirmed in our five cases with overt
osteitis fibrosa. But in addition, we found a group of patients with normal
or insignificantly increased alkaline phosphatase activity in which elevated
calcium retention was seen despite overt hypercalciuria. In three of these cases,
we were able to demonstrate osteitis fibrosa which was previously overlooked.
It seems, therefore, that increased calcium retention in primary hyperpar-
athyroidism with or without hypercalciuria reveals osteitis fibrosa generalisata
earlier and more accurately than determination of alkaline phosphatase, while
a low calcium retention (being the consequence of hypercalciuria) does not
exclude bone disease. Since there was an increased retention of calcium in
Calcium retention in hyperparathyroidism (primary and secondary): The two antagonistic factors: skeleton (osteitis fibrosa generalisata) depicted by the hatched triangle, and kidneys (hypercalciuria), indicated by the dotted triangle, show a considerable overlap.

more than half of these patients, the generally accepted view which considers osteitis fibrosa in primary hyperparathyroidism infrequent (Black & Zimmer 1956) seems unjustified.

Of three patients who were studied before and after removal of a parathyroid adenoma, two showed an increase in calcium retention one and a half and seven months after surgery, the third a slight decrease after five months (Fig. 5). This reflects various phases of healing, being in good agreement with Albright's thesis that osteomalacia may develop after correction of the parathyroid hyperfunction state (Albright & Reifenstein 1948).

In secondary hyperparathyroidism due to renal insufficiency, three factors accounting for the demonstrated high calcium retention can be envisioned:

1. The GFR in all these cases being very low, the calcium excretion can apparently be delayed; this was true in one of our patients with an elevated urinary calcium on the post-infusion day. In such a situation, the 24-hour calcium retention as reported here may be falsely high, and the retention calculated over a longer period of time would offer more adequate information.

2. In many instances, osteitis fibrosa due to parathyroid hyperactivity accounts also for the increased calcium retention as seen in two of our patients.
Table 1.
Hypercalciuria.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>Clinical diagnosis</th>
<th>X-ray of the skeleton</th>
<th>Serum Ca mg/100 ml</th>
<th>Serum P</th>
<th>Serum alk. phosphatase Bodansky units</th>
<th>Urine-Ca mg/24 h</th>
<th>Ca-Retention °/o</th>
</tr>
</thead>
<tbody>
<tr>
<td>W. E.</td>
<td>34 M</td>
<td>Recurrent renal calculi; idiopathic hypercalciuria</td>
<td>Normal bones</td>
<td>9.7</td>
<td>3.0</td>
<td>4.0*</td>
<td>317</td>
<td>50</td>
</tr>
<tr>
<td>W. M.</td>
<td>41 M</td>
<td>Recurrent renal calculi; idiopathic hypercalciuria</td>
<td>Normal bones</td>
<td>10.8</td>
<td>3.3</td>
<td>3.6*</td>
<td>535</td>
<td>50</td>
</tr>
<tr>
<td>A. T.</td>
<td>48 M</td>
<td>Recurrent renal calculi; idiopathic hypercalciuria</td>
<td>Normal bones</td>
<td>10.6</td>
<td>4.4</td>
<td>3.2*</td>
<td>726</td>
<td>36</td>
</tr>
</tbody>
</table>

* Alkaline phosphatase values determined by the Bessey-Lowry method and for comparison converted into Bodansky units by multiplying with the factor 1.8.
**Table 2.**
Primary hyperparathyroidism.

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>Clinical diagnosis</th>
<th>X-ray of the skeleton</th>
<th>Serum (mg/100 ml)</th>
<th>Urine-Ca (mg/24 h)</th>
<th>Ca-Retention %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ca</td>
<td>P</td>
<td>alk. phosphatase</td>
</tr>
<tr>
<td>C.C.</td>
<td>59 M</td>
<td>Parathyroid adenoma</td>
<td>Severe general »osteoporosis», subperiost. erosions of phalanges</td>
<td>11.3</td>
<td>2.6</td>
<td>6.5</td>
</tr>
<tr>
<td>W.C.</td>
<td>34 M</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>13.1</td>
<td>3.3</td>
<td>4.8*</td>
</tr>
<tr>
<td>E.D.</td>
<td>54 F</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>11.5</td>
<td>3.3</td>
<td>4.7*</td>
</tr>
<tr>
<td>G.F.</td>
<td>43 M</td>
<td>Parathyroid adenoma</td>
<td>Slight spinal »osteoporosis« ?</td>
<td>12.1</td>
<td>3.7</td>
<td>4.2*</td>
</tr>
<tr>
<td></td>
<td>7 months postop.</td>
<td></td>
<td></td>
<td>10.0</td>
<td>3.3</td>
<td>3.0</td>
</tr>
<tr>
<td>R.G.</td>
<td>20 M</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>11.2</td>
<td>3.0</td>
<td>1.9</td>
</tr>
<tr>
<td>K.H.</td>
<td>58 F</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>12.3</td>
<td>2.2</td>
<td>4.3*</td>
</tr>
<tr>
<td>G.J.</td>
<td>37 M</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>11.5</td>
<td>2.2</td>
<td>0.7*</td>
</tr>
<tr>
<td>G.L.</td>
<td>56 F</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td>12.6</td>
<td>3.5</td>
<td>3.5*</td>
</tr>
<tr>
<td>A.M.</td>
<td>40 M</td>
<td>Parathyroid adenoma</td>
<td>Small bone cysts in phalang., otherwise normal bones</td>
<td>11.4</td>
<td>3.0</td>
<td>3.4*</td>
</tr>
<tr>
<td></td>
<td>5 months postop.</td>
<td></td>
<td></td>
<td>10.2</td>
<td>3.7</td>
<td></td>
</tr>
<tr>
<td>52 M A.S.</td>
<td>Parathyroid adenoma</td>
<td>Normal bones</td>
<td></td>
<td>12.0</td>
<td>2.8</td>
<td>4.5*</td>
</tr>
<tr>
<td></td>
<td></td>
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</tr>
<tr>
<td>L. S.</td>
<td>60 F</td>
<td>Parathyroid adenoma</td>
<td>Radiol. normal bones, but histol. low-grade osteitis fibrosa</td>
<td>12.5</td>
<td>2.5</td>
<td>3.7*</td>
</tr>
<tr>
<td>P. S.</td>
<td>40 M</td>
<td>Parathyroid adenoma</td>
<td>Slight demineralization of phalanges, otherwise normal bones</td>
<td>12.8</td>
<td>2.5</td>
<td>4.5*</td>
</tr>
<tr>
<td>T. W.</td>
<td>37 M</td>
<td>Parathyroid adenoma, malabsorption-syndrome</td>
<td>Severe general. »osteoporosis«, multiple bone cysts, lamina dura absent</td>
<td>13.6</td>
<td>2.3</td>
<td>9.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11/2 months postop.</td>
<td></td>
<td>9.5</td>
<td>2.2</td>
<td>18.0</td>
</tr>
<tr>
<td>Cases with significant renal damage:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G. D.</td>
<td>39 M</td>
<td>Parathyroid carcinoma with metastases ?</td>
<td>Severe general. »osteoporosis« with fractures &amp; bone cysts; histol. osteoclastomas</td>
<td>12.3</td>
<td>2.7</td>
<td>7.9</td>
</tr>
<tr>
<td>M. S.</td>
<td>53 F</td>
<td>Parathyroid adenoma</td>
<td>Severe general. »osteoporosis«, bone cysts, lamina dura absent</td>
<td>12.6</td>
<td>3.1</td>
<td>9.6*</td>
</tr>
<tr>
<td>C. Y.</td>
<td>63 F</td>
<td>Multiple parathyroid adenomas</td>
<td>Slight general. »osteoporosis«, histol. low-grade osteitis fibrosa</td>
<td>11.4</td>
<td>3.2</td>
<td>6.3*</td>
</tr>
</tbody>
</table>

* Alkaline phosphatase values determined by the Bessey-Lowry method and for comparison converted into Bodansky units by multiplying with the factor 1.8.
Calcium retention in primary hyperparathyroidism before and after operation: After surgery, renal calcium excretion is normalized, osteitis fibrosa heals (with an osteomalacic component).

3. The high alkaline phosphatase levels in the two cases indicate an osteomalacic component (which was proven in patient J. W. by histologic examination), may be primarily responsible for the very high calcium retention.

Somewhat unexpected results were obtained in the hypoparathyroid group in which all patients retained more than 80% of the infused calcium. This finding can be explained in two ways:

1. In two cases, the serum calcium rose during infusion, but not to a normal value (from 4.4 to 8.8 mg/100 ml and from 5.4 to 8.5 mg/100 ml, respectively). thus scarcely filling the extracellular calcium pool to a normal level even for a short while. Hence the filtered load of calcium remained low, resulting in a high calcium retention.

2. The remaining patients showed a significant increase in serum calcium during infusion (from a mean of 7.1 to 12.1 mg/100 ml). Two of these patients had elevated alkaline phosphatase levels, and one revealed an increase in bone density by X-ray after treatment with vitamin D. Thus, an osteomalacic component seems to be present in many cases of hypoparathyroidism which can be either attributed to a low Ca × P-product (Albright & Reifenstein 1948), or to an insufficient calcium absorption from the gut because of lack of parathyroid hormone (Kyle, unpublished data).
Table 3.
Secondary hyperparathyroidism (in all patients due to chronic renal failure).

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>Clinical diagnosis</th>
<th>X-ray of the skeleton</th>
<th>Serum</th>
<th>Urine-Ca mg/24 h</th>
<th>Ca-Retention %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ca P alk. phosphatase Bodansky units</td>
<td></td>
</tr>
<tr>
<td>R.B.</td>
<td>42 F</td>
<td>Chron. pyelonephritis with acidosis, recurrent renal calculi, nephrocalcinosis</td>
<td>Slight general. »osteoporosis«</td>
<td>10.5</td>
<td>4.4</td>
<td>2.6*</td>
</tr>
<tr>
<td>L.M.</td>
<td>43 F</td>
<td>Chron. pyelonephritis with acidosis, multiple ectopic calcifications</td>
<td>Severe general. »osteoporosis«, multiple bony erosions</td>
<td>10.9</td>
<td>9.5</td>
<td>20.3</td>
</tr>
<tr>
<td>J.W.</td>
<td>12 F</td>
<td>Chron. pyelonephritis with hydronephrosis; autopsy: hyperplasia of all parathyroid glands</td>
<td>Typical features of rickets, subperiostal erosions of phalanges</td>
<td>9.2</td>
<td>6.1</td>
<td>30.7</td>
</tr>
</tbody>
</table>

* Alkaline phosphatase values determined by the Bessey-Lowry method and for comparison converted into Bodansky units by multiplying with the factor 1.8.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age, sex</th>
<th>Clinical diagnosis</th>
<th>X-ray of the skeleton</th>
<th>Serum Ca mg/100 ml</th>
<th>Serum P mg/100 ml</th>
<th>Serum alk. phosphatase Bodansky units</th>
<th>Urine-Ca mg/24 h</th>
<th>Ca-Retention %</th>
</tr>
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<tr>
<td>B. A</td>
<td>64 F</td>
<td>Sec. to total thyroidectomy, tetany</td>
<td>Normal bones</td>
<td>8.8</td>
<td>5.2</td>
<td></td>
<td></td>
<td>87</td>
</tr>
<tr>
<td>R. B</td>
<td>54 F</td>
<td>Sec. to total thyroidectomy</td>
<td>Slight spinal »osteoporosis«, cranial osteosclerosis</td>
<td>8.1</td>
<td>5.0</td>
<td>6.1</td>
<td>54</td>
<td>81</td>
</tr>
<tr>
<td>E. B</td>
<td>12 F</td>
<td>Idiopathic, tetany</td>
<td>Slight spinal osteosclerosis</td>
<td>8.1</td>
<td>8.6</td>
<td>19.7</td>
<td>79</td>
<td>90</td>
</tr>
<tr>
<td>A. H</td>
<td>44 F</td>
<td>Neck exploration: all parathyroid tissue removed</td>
<td>Slight generalized »osteoporosis«</td>
<td>6.3</td>
<td>6.6</td>
<td></td>
<td>161</td>
<td>87</td>
</tr>
<tr>
<td>S. S</td>
<td>46 F</td>
<td>Sec. to total thyroidectomy</td>
<td>Localized areas of skeletal demineralization</td>
<td>6.0</td>
<td>5.6</td>
<td>8.4</td>
<td>22</td>
<td>87</td>
</tr>
<tr>
<td>C. Y</td>
<td>63 F</td>
<td>Sec. to total parathyroidectomy</td>
<td>Slight generalized »osteoporosis«</td>
<td>5.4</td>
<td>4.9</td>
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REFERENCES


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