A systemic approach to the oral problem of mandibular resorption

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SUMMARY

It is argued that mandibular resorption can be considered as osteoporotic in nature. This calls for a systemic approach in its treatment. One aspect mostly neglected is the tendency of bone to bind calcium and phosphate ions. A direct measure for this tendency is the difference in pH between the bone extracellular fluid and blood plasma. This pH difference is directly reflected by the degree of saturation of blood plasma with octocalcium phosphate. Calculation of these degrees of saturation of about 2000 data of blood plasma compositions given in the literature indicates that calcitonin injection, estrogen replacement and magnesium supplementation increase the tendency of bone to bind calcium and phosphate ions and hence should be used in the therapy of mandibular resorption and of bone resorption in general.

KEY WORDS:

Mandibular resorption - Osteoporosis - Calcitonin - Estrogen - Magnesium

RÉSUMÉ

Il est prouvé que la résorption mandibulaire peut être considérée comme étant de nature ostéoporotique. Ceci veut dire que le traitement doit être systémique. L'un des aspects souvent négligés est la tendance qu'a l'os à fixer les ions calcium et phosphate. Une mesure directe de cette tendance est obtenue par la différence de pH entre le fluide extracellulaire de l'os et le plasma sanguin. Cette différence de pH est directement révélée par le degré de saturation du plasma sanguin par rapport au phosphate octocalcique. Le calcul de ce degré de saturation sur environ 2000 données de compositions plasmatiques fournies dans la littérature indiquent que des injections de calcitonine, l'équilibration en oestrogènes et un apport de magnesium augmentent la tendance de l'os à fixer les ions calcium et phophate et, par conséquent, devraient être utilisés pour la thérapie de la résorption mandibulaire et de la résorption osseuse en général.

MOTS-CLÉS:

Résorption mandibulaire - Ostéoporose - Calcitonine - Oestrogenes - Magnesium

INTRODUCTION

The bone mineral content of mandibles decreases with age from the age of about 45 years (von Wowern and Stoltze, 1980). The rate of mineral loss is higher for women than for men (von Wowern, 1988). Many authors have drawn special attention to atrophy of the alveolar ridges after extraction of teeth. This is most pronounced within the first 6 months of the edentulous period and then decreases slowly through the next 36 months; but slight resorption can occur throughout life (Atwood, 1971; Tallgren, 1972). It can even be accelerated by wearing the denture and for that reason the mandible overdentures seem to be preferable (Jozefowicz, 1970; Whinery, 1975; Crum and Rooney, 1978; de Baat et al., 1988). On the other hand, variations

in bone mass of the mandible as a whole do not seem to be clearly related with functional activity within the mandible (absence or presence of teeth) (von Wowern 1976). Moreover, there is a high variation in the state of alveolar ridge resorption among different long-term denture wearers (Bergman and Carlsson, 1985). These facts indicate that loss or diminution of mechanical function plays only a secondary role in the loss of bone from the mandible with age.

Several studies have been made, in which the bone mineral content of mandibles was compared with those of other bones (Rosenquist et al., 1978; von Wowern and Melsen, 1979; von Wowern, 1985; von Wowern et al., 1988). The results indicate that in the normal individual, variations in the mandibular bone mass can to some extent be explained by variations in the bone mass in other skeletal sites which like the mandible consist primarily or entirely of cortical bone; only weak correlations were found to skeletal sites consisting mainly of trabecular bone (von Wowern, 1986). The existence of different trends of age-related diminutions of bone mineral content in different parts of the skeleton has also been established (Schaadt and Bohr, 1988). Again, this variation could be related to changes in the patterns of mechanical function of the bones with age. However, the factors determining that there is a loss of bone mineral with age, are more than mechanical: they are systemic, they are related to ageing in general (Garn et al., 1967). In that context this ageing process is called osteoporosis.

That bone loss is primarily determined by systemic factors, also for the mandible, has been indicated by several studies (Baxter, 1981; Bays and Weinstein, 1982; Bras et al., 1983; Sones et al., 1986; Habets et al., 1987). In these studies a correlation was found between osteoporosis and mandibular atrophy. Shapiro et al. (1985) studied a group of postmenopausal patients and found inadequate amounts of bone in the mandible, loss and/or mobility of teeth, edentulism and poorly fitting dentures. Von Wowern (1982) made a histomorphometric analysis of mandibles of different age and found the same changes as those called typical for other osteoporotic bones. Hence, age-related loss of bone mineral from the mandible can also be classified as being for the most part «osteoporotic» in nature. This calls primarily for a systemic approach in the prevention and treatment of mandibular bone loss, which differs considerably from the «implantology» approach that gains more and more popularity among the dental

profession nowadays, although both approaches may work complementary to the benefit of the older dental patient. In the systemic approach the dentist depends admittently largely on the patient's physician.

A complete systemic approach to the problem of osteoporosis is quite extensive. The diagram of Fig. 1 is illustrative for the calcium homeostasis of a man of average weight and it may help to limit the number of factors on which we should concentrate in this study. From Fig. 1 it can be derived that the following conditions may lead to a negative calcium balance:

(a) insufficient renal reabsorption
(b) malabsorption through the intestines or insufficient nutrition
(c) an imbalance between bone resorption and bone new formation
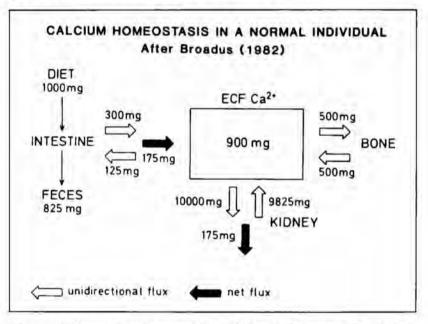(d) a combination of these factors.

Fig. 1: Schematic representation of the daily systemic calcium homeostasis in a healthy individual of about 70 kg weight in zero external calcium balance according to Broadus and StewartSimilar (1983). Similar schematic representations are known for the phosphate homeostasis, but these are more complex due to the participation of intracellular compartments (Nordin, 1976).
Fig. 1: Représentation schématique de l'homéostasie calcique journalière chez un individu sain de 70 kg environ, en situation de balance calcique externe nulle (selon Broadus et Stewart, 1983). On trouve également des représentations schématiques de l'homéostasie du phosphate, mais elle sont plus complexes à cause de la participation des compartiments intracellulaires (Nordin, 1976).

Starting with factor (a) as a possible cause of bone loss, it must be noted that renal osteodystrophy does exist as a disease (Reitz et al., 1987) but it is distinctly different from osteoporosis. It has been widely accepted in the USA that factor (b) is the main cause of osteoporosis: the American diet would contain too less calcium and too much phosphate (Baxter, 1981; Spencer et al., 1984; Shapiro et al., 1985; Ramazzotto

et al., 1986) and, therefore, supplementation of the diet with calcium and vitamine D is advocated, also to diminish alveolar ridge resorption (Wical and Brussee, 1979). However, evidence is pilling up (Anon, 1986; van Beresteijn, 1989) that this does not hold, at least not for the European situation. Hence, we are left with factor (c) as the most probable cause of postmenopausal and ageing osteoporosis. It is known since long that parathyroid hormone promotes bone resorption and calcitonin promotes new bone formation. Therefore, it is not strange that calcitonin therapy is advocated to increase new bone formation (Gonzalez et al., 1987). In postmenopausal osteoporosis estrogen replacement seems to have success (Shapiro et al., 1985). It is striking how much interest is focussed on calcitonin and estrogen therapy nowadays (e.g. Abstracts of Sixth International Workshop on Bone and soft Tissue Densitometry, Baxton, England, 1987).

The aim of the present study is to show that in the formulation of factor (c) one important aspect is mostly neglected or overlooked: one might call it the tendency of bone to «bind» calcium and phosphate ions. It will be shown that this tendency can vary and it will be analyzed on which factors it depends. Important in this context are the following data. In previous studies it was proved that the solubility controlling phase of bone mineral in vertebrates is octocalcium phosphate OCP (Driessens and Verbeeck, 1986; Driessens et al., 1986a). There is a necessity by physico-chemical laws that the bone extracellular fluid BECF is constantly kept in equilibrium with that octocalcium phosphate of bone mineral. Therefore, the ion activity product of OCP in BECF reading

$$\log I_p^{OCP} = 8 \log (Ca^{2+}) + 2 \log (HPO_4^{2-}) + 4 (PO_4^{3-})$$

in which (X) is the activity of ion X in the aqueous solution, is kept constantly at the value for its solubility $\log K_{SP}^{OCP}$ which is -69.6 (Neuman, 1975; Driessens et al., 1986a). However, there is a small pH gradient between BECF and the other body compartments (Nordin 157; MacGregor, 1965; MacGregor and Brown, 1965) whereas there is no active transport of calcium and phosphate ions between BECF and the other body compartments (Scarpace and Neuman, 1976). This makes that the sign of the pH gradient between BECF and the other body compartments determines whether bone has the tendency to bind calcium and phosphate or to expell those ions to the other body compartments: when BECF has a higher pH, bone will try to bind

calcium and phosphate. However, when BECF has a lower pH, it will try to expell calcium and phosphate ions (Driessens et al., 1987b). The pH of blood plasma and most other body compartments is fixed practically at the value of 7.4 by renal and lung function. Temporary deviations in the pH of BECF are the main reason why blood plasma is not exactly in equilibrium with OCP in the different vertebrates and in man (Driessens et al., 1988). As OCP is the solubility controlling phase in bone mineral, the uptake or expulsion of calcium and phosphate ions by bone happen at a constant ratio of 1.33 as the formula of OCP is

$$Ca_8 (HPO_4)_2O (PO_4)_4 \cdot 5H_2$$

In this study, it will be determined what is the rate at which bone is able to correct changes in the $\log I_p^{OCP}$ and what is the contribution of intestinal absorption and renal excretion in this correction process.

The physical chemistry of this system determines that the $\log I_p^{OCP}$ of blood plasma increases when the pH of BECF becomes lower, and the reverse (Driessens et al., 1987b). Hence, the stationary-state value of $\log I_p^{OCP}$ of blood plasma is a direct indication for the level of the pH of BECF, and thus for the tendency of bone to bind calcium and phosphate ions. This tendency becomes higher when the $\log I_p^{OCP}$ of blood plasma becomes lower. The second purpose of this paper, is to determine whether the tendency of bone to bind calcium and phosphate ions is affected by different states of disease. The third purpose is to determine which agents can increase the tendency of bone to bind calcium and phosphate ions. The fourth purpose is to determine whether in man there are diurnal variations in the tendency of bone to bind calcium and phosphate ions. These tendencies can thus be derived directly from the corresponding $\log I_p^{OCP}$ values for blood plasma.

MATERIALS AND METHODS

Appropriate literature data (in total, about 2000 values) about the calcium and inorganic phosphate concentrations of blood plasma of animal vertebrates and man were collected and used for the calculation of $\log I_p^{OCP}$. It was assumed that inorganic phosphate was not bound to the blood proteins, as the literature studies showed that all inorganic phosphate was ultrafilterable. Further, it was assumed that 45% of the total calcium of man and

animals was ionized calcium (Driessens et al., 1989; Nordin, 1976). Other assumptions used in the calculations were pH 7.4, Na 140, K 4, Cl 105, Mg 1 and carbonate 24 mmol L^{-1}, unless stated otherwise in the literature. Activity coefficients were approximated by the extended Debye-Hückel formula (Robinson and Stokes, 1970), dissolution and complexation constants were taken from Driessens (1982) and ion-size parameters from Kielland (1937).

RESULTS

In neonates the exchange of calcium and phosphate ions between BECF and blood plasma can be assumed to be normal (it exists already since bone formation), but the contribution of intestines and kidneys to the calcium and phosphate homeostasis is not yet normalized. In the literature (Snodgrass et al., 1973; Tsang et al., 1973) it appears that the blood plasma of neonates can contain very differing concentrations of calcium and phosphate. However the data are scattered over a rather distinct area between some extreme values. See Table I. From this table it appears that the calcium/phosphate ratio of blood plasma of neonates can vary quite dramatically. However, at a certain age the degree of saturation log I_p^{OCP} is practically constant. For the variation of log I_p^{OCP} with age see Driessens et al. (1989).

TABLE I.
Extreme concentrations of calcium and phosphate (mmole l^{-1}) and the corresponding degrees of saturation of blood plasma with respect to OCP in human neonates.

Author/Time	Ca	P	Ca/P	log I_p^{OCP}
Snodgrass et al. (1973)	2.75	1.77	1.55	−68.0
6 - 8 days	1.48	3.55	0.42	−68.1
Tsang et al. (1973)	2.75	1.30	2.12	−68.6
at birth	1.38	3.55	0.39	−68.6

Moore et al. (1985) nephrectomized lambs and analyzed their blood plasma before and some time after the operation. See Table II. It appears that the operation changed the calcium/phosphate ratio of their blood plasma drastically in the course of 10 days post operation but the degree of saturation log I_p^{OCP} remained unchanged.

Infusions of either calcium or phosphate salts give also interesting results. See Table III. The response

TABLE II.
Effect of nephrectomy on the calcium and phosphate concentrations (mmol l^{-1}) and the degree of saturation of blood plasma in lambs (Moore et al., 1985).

Nephrectomy	Ca	P	Ca/P	log I_p^{OCP}
Before	3.00	2.25	1.33	−67.63
After	1.85	3.85	0.48	−67.75

time of log I_p^{OCP} to these changes was shorter than 1 hour in dog and man. However, the response time of the calcium/phosphate ratio of their blood plasma was of the order of a day. This is further corroborated by the data of Fig. 2, obtained from cows.

In Table IV the average deviations from normal values in the degree of saturation of blood plasma with OCP in several states of disease and health are summarized. For the prevention of bone loss and pathological calcifications it is favourable to keep the degree of saturation of the pool of extracellular fluids on the low side. Perhaps the low values during pregnancy and lactation give the impression of a natural protection of the body of the mother against bone loss. Further, it may be noted that most diseases which are accompanied by a high degree of saturation of blood plasma are those which in the long run result in bone loss, pathological calcifications or both.

In Table V results for the change in log I_p^{OCP} for different forms of acidosis and alkalosis are shown. Alkalosis decreases the degree of saturation of blood plasma, whereas acidosis increases it. These results are corroborated by those of Fig. 3 for dietary acidic phosphate acidosis and calcium carbonate alkalosis in rats. In this figure it is observed that during excessive acidosis and alkalosis not only the degree of saturation of blood plasma, but also the normal calcium/phosphate ratio is disturbed.

TABLE III.
Concentrations of calcium and phosphate (mmole^{-1}) and the corresponding degrees of saturation of blood plasma before and after infusion of calcium or phosphate salts.

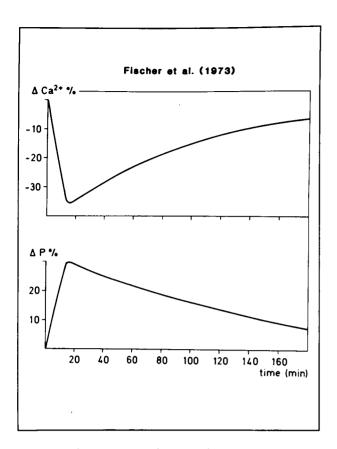| Infusate	Infusion	Ca	P	Ca/P	log I_p^{OCP}
CaCl$_2$ in dogs (Massry et al., 1970)	Before	2.80	1.61	1.74	−68.59
	After*	3.18	1.35	2.36	−68.60
Na$_2$HPO$_4$ in men (Coburn et al. 1969)	Before	3.25	1.45	2.24	−68.45
	After*	1.38	4.19	0.33	−68.60

* data taken after a time of about 1/2 hr.

Fig. 2: Changes in calcium phosphate concentrations of blood plasma during and after infusion of phosphate in cows (Fischer et al., 1973).

Fig. 2: Changements dans les concentrations en calcium et phosphate du plasma sanguin pendant et après injection de phosphate par intraveineuse chez la vache (Fischer et al., 1973).
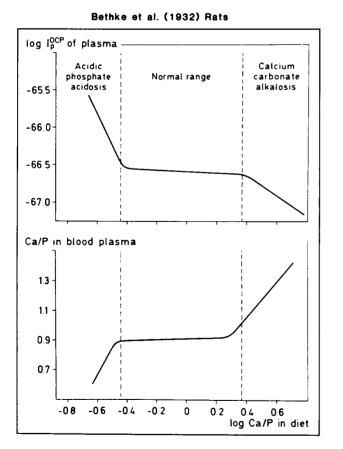

Fig. 3: Degree of saturation $\log I_p^{OCP}$ and molar calcium/phosphate ratios in blood plasma of rats as a function of the logarithm of the calcium/phosphate ratio of their diet. Source Bethke et al. (1932).

Fig. 3: Degré de saturation $\log I_p^{OCP}$ et rapports molaires calcium/phosphate dans le plasma sanguin des rats en fonction du loarithme du rapport calcium/phosphate de leur régime (Bethke et al., 1932).

TABLE IV.
Average deviations from normal values in the degree of saturation of blood plasma with OCP in several states of disease and health in humans.

Reference number(s)	Patients (n)	State	Average $\Delta \log I_p^{OCP}$
3	7	Hypophosphatemia	− 4.8
16	3	Hypoparathyroidism	− 0.8
44	40	Pregnancy (20-40 weeks)	− 0.5
42	62	Lactation	− 0.3
21	4	Mseleni Joint Disease	0
53	101	Postmenopause + estrogen	0
53	101	Postmenopause	+ 0.3
10, 61	126	Osteoporosis	+ 0.4
7, 26	11	Paget	+ 0.4
26, 83	24	Hyperparathyroidism	+ 0,4
54	30	Tetanus	+ 0.7
26, 85	7	Metastasis	+ 1.7
45, 55, 93,94	139	Renal failure, hemodialysis	+ 2.0

TABLE V.
Effect of acidosis and alkalosis on the degree of saturation of blood plasma.

Influence	Form	Source	$\Delta \log I_p^{OCP}$	Species
Alkalosis	Dietary NaHCO³	Ballina et al. (1985)	− 0.5	rat
Acidosis	Work	Tibes et al (1974)	+ 1.6	man
		Aloja et al (1985)	+ 0.7	man
	Shock	Carpenter et al (1978)	+ 2.2	baboon
	Renal	Johnson et al (1978)	+ 2.4	man
	Insuffi-	Varghese et al (1973)	+ 2.0	man
	ciency	Raman et al (1972)	+ 2.2	man
		Fuss et al (1976)	+ 1.6	man

TABLE VI.
Effect of PTH and calcitonin injections on the degree of saturation of blood plasma*.

Hormone	Source	$\Delta \log I_p^{OCP}$	Species
PTH	Schneider et al (1975)	+ 0.7	dog
	Heaton (1965)	+ 0.4	rat
	Bethune et al (1968)	+ 0.6	man
Calcitonin	Haas et al (1968)	− 0.7	man
	Bijvoet et al (1968)	− 1.4	man
	Kenny et al (1965)	− 0.8	rat
	Chan et al (1968)	− 0.2	eel

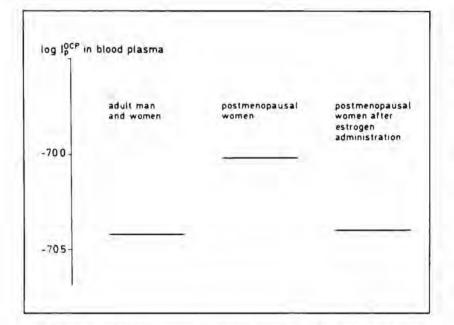* Response time less than 1 hr.

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Fig. 4: Effect of estrogen administration on the log I_p^{OCP} of blood plasma of postmenopausal women. Source Hodgkinson (1982).
Fig. 4: Effet de l'administration d'oestrogenes sur le log I_p^{OCP} du plasma sanguin chez la femme postménopausée (Hodgkinson, 1982).
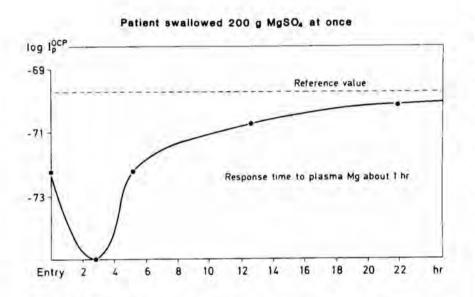

Fig. 5: Effect of an oral overdose of magnesium sulfate on the log I_p^{OCP} of blood plasma as a function of time. Source Garcia Webb et al. (1984).
Fig. 5: Effet d'un surdosage de sulfate de magnesium par voie orale sur le log I_p^{OCP} du plasma sanguin en fonction du temps (Garcia Webb et al., 1984).

TABLE VII.
Effect of Mg on the degree of saturation of blood plasma with OCP.

Author(s)	Diet or Infusate	$\Delta \log I_p^{OCP}$	Species
25 studies (see Driessens et al (1988)	Magnesium deficient diet	+ 0.5 + 0.5	mouse, rat, dog, cow, man
Massry et al (1970)	MgCl₂ infusate	− 1.0	dog
Cruikshank et al (1981)	MgSO₄ infusate	− 0.7	man

In Table VI changes in log I_p^{OCP} are shown which were induced by injections of parathyroid hormone PTH or calcitonin. PTH appears to increase the level of saturation, whereas calcitonin decreases it. The response time of the blood plasma to these injections appeared to be less than 1 hour.

In Fig. 4 the change in log I_p^{OCP} in postmenopausal women occurring due to oral estrogen administration (Hodgkinson, 1982) is shown.

Table VII shows the changes in log I_p^{OCP} due to either magnesium deficient diets or infusates with magnesium salts. Magnesium deficient diets induce (as a first reaction) an increase in the degree of saturation (Driessens et al., 1987b), whereas magnesium infusates result in a decrease. These results are corroborated by those of Fig. 5, in which it appears that the response time to changes in the magnesium status is of about 1 hour.

The pH of blood plasma in healthy persons is known accurately. Diurnal variations occur between 7.37 and 7.43 whereby arterial blood has a slightly higher pH than venous blood (Boning et al., 1974; Drop et al., 1978; Kurtz et al., 1983; Tibi et al., 1982). The total calcium, ionized calcium and inorganic phosphate concentrations in human blood plasma of healthy volunteers and their diurnal variations are also known with great accuracy (Markowitz et al., 1985; Perry et al., 1986; Mellerup et al., 1976). The results for the calculated values of log I_p^{OCP} as a function of the time of the day are given in Fig. 6. It appears that on the average the blood plasma of
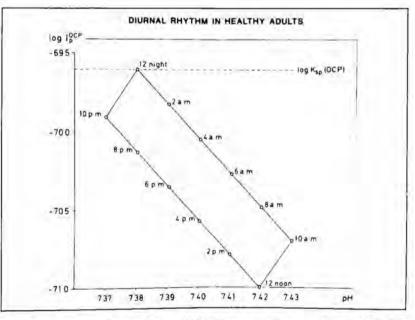

Fig. 6: Average logarithm of the ion activity product of OCP versus pH as a function of time of the day in the blood plasma of healthy adults.
Fig. 6: Moyenne logarithmique du produit de l'activité ionique de l'OCP contre le pH en fonction du moment de la journée dans le plasma sanguin d'adultes sains.

healthy adults remains just slightly undersaturated with respect to OCP. However, at night the degree of saturation appears to be higher than during a large part of the day.

DISCUSSION

The extracellular pool of calcium and phophate is kept in homeostasis by exchange via intestines, kidneys and bone. The data obtained in this study underline the importance of bone for the maintenance of a certain degree of saturation in the extracellular fluids. The response time of blood plasma to changes in the degree of saturation by this ion exchange with bone is shorter than 1 hour.

By its nature the above process of ion exchange with the crystals of bone is not able to correct disturbances in the calcium/phosphate ratio of the extracellular pool. This must happen by changes in the net fluxes from the intestines and through the kidneys. The response time of blood plasma to disturbances in the calcium/phosphate ratio due to these net fluxes through intestines and kidneys appears to be of about 1 day.

According to Broadus and Stewart (1983) the amount of calcium dissolved and repriciated from bone per day in the average healthy man amounts to 500 mg. See Fig. 1. However, Nordin (1976) estimates this to be about 300 mg per day. As precipitation occurs in the form of OCP (Driessens et al., 1986) this latter value means that per day about 0.9 g of OCP is formed. The half-time transformation period of this OCP into the less soluble calcium phophates of bone mineral is about 1 month (Driessens et al., 1986b; 1987a). This means that the skeleton of an adult should contain about 55 g of OCP. The total weight of the mineral in the skeleton of an adult is about 3000 g (Nordin, 1976), which would mean that about 2% of the mineral in the bone of an adult is OCP. Therefore, and for the reason that the particles are extremely small, it is not surprising that OCP is not found, when bone of adults is investigated by physical methods like X-ray diffraction. The only indication for its presence in adult bone can come from solubility studies, in which less than 2% of the mineral is dissolved (Driessens and Verbeeck, 1989). Young bone contains amounts of OCP which can be demonstrated by chemical analysis of the mineral (Driessens et al., 1986, 1987). Yet even in living adult bone OCP is the solubility controlling phase of the mineral, because that phase is more soluble than the other phases.

The general result of Fig. 2 and Tables I through III is thus that bone mineral corrects variations in $\log I_p^{OCP}$ of blood plasma within one hour, but that intestines and kidneys need about 1 day to correct variations in the calcium/phosphate ratio of blood plasma.

The results of Table IV show that diseases like osteoporosis, Paget and hyperparathyroidism are characterized by a high value for $\log I_p^{OCP}$ in blood plasma. As indicated in the introduction, this means that the bone of those patients has a tendency to loose calcium and phosphate ions. Such patients may thus be treated against bone loss by therapy with agents increasing the tendency of their bone to bind calcium and phosphate ions. From Tables VI and VII and from Fig. 5 it follows that a therapy by estrogen replacement, calcitonin administration (either injection or nasal spray) or magnesium supplementation will be of value in this respect. From Fig. 6 it follows that the tendency of bone to bind calcium and phosphate ions reaches a minimum somewhere after dinner. As the action of estrogen, calcitonin and magnesium supplementation starts within an hour after administration and holds on for several hours, it should be recommended that treatments with these agents should be applied right after dinner. Then they will have the maximum effect.

Hopeful results have been reported by d'Angelo et al. (1987) in the treatment of renal bone disease in rats by injections with exogenous calcitonin. Further, estrogen replacement can prevent bone loss in postmenopausal or ophorectomized women (Lindsay et al., 1980; Heancy et al., 1978; Christensson, 1976). However, magnesium supplementation has not yet been tried in the treatment of the above mentioned diseases, although it may be the way which is most easily handled by the patient himself or herself. Thereby, one should keep in mind that magnesium supplementation may only be considered for treatment of patients having a glomerular filtration rate GFR larger than 30 ml min^{-1} (Popovtzer et al., 1969). Otherwise the complications of magnesium excess may appear (Mordes and Wacker, 1977). Another complication of the usual magnesium compounds is that they cause diarrheoa and in this way impair the calcium and phosphate absorption by the intestines. Therefore, we must wait for the development of a magnesium formula which should provide sufficient amounts of magnesium without causing possible disadvantageous side effects.

As far as the mechanisms by which these agents work specifically on the pH gradient between BECF and blood plasma are concerned, it is obvious why calcitonin increases the pH of BECF: it retards the acid production by the osteoclasts. The way of action of estrogen is not clear. However, about magnesium it is known that it activates the ATP-ase which drives the $H^+ - K^+$ pump of the cells in the periosteum and endosteum (Driessens et al., 1988). By this pump H^+ ions are removed from BECF and replaced by K^+ ions. In addition, magnesium seems to suppress parathyroid gland activity (Altenähr and Leonhardt, 1972) and to increase calcitonin levels (Littledike and Arnaud, 1971). Therefore, magnesium acts along at least two specific lines in increasing the pH of BECF. Recent findings (Cohen, 1988) indicate that in post-menopausal osteoporosis a decreased bone magnesium concentration is found and, although many such patients do not show an obvious deficiency of blood plasma magnesium, they show an increased retention of magnesium in the magnesium load test. In conclusion the present data are in favour of daily magnesium supplementation after dinner as a means against mandibular resorption in particular and of systemic bone resorption in general, as advocated earlier (Driessens and Borggreven, 1989). Recently, Steidl and Ditmar (1989) showed that therapy of senile and postmenopausal osteoporosis with magnesium had better results than that with sodium fluoride, which confirms our hypothesis.

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