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Magnesium in pregnant women and the newborn

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Summary: In this article the importance of Mg for pregnant women and fetal outcome is reviewed. The physiological changes of Mg-containing body fluids and of tissues are discussed. Mg supplementation during pregnancy seems to be necessary and the efficacy on maternal health and on the newborn are reported. Serum Mg levels decrease during pregnancy and there is a 25% increase of renal Mg excretion. Mg supplementation has a positive effect, with reduced incidence of hospital admission and preterm labour, while the gestational age of the fetus is longer. Convulsions may occur in newborns with hypomagnesaemia. Hypermagnesaemia of the newborn following $MgSO_4$ infusions to toxaemic mothers has been reported.

Key words: Magnesium, magnesium supplementation, newborn, pregnancy, review.

Introduction

The assumption that the placenta provides protection against chemical insult has been seriously challenged, not least by the thalidomide disaster of the early 1960s; hence during pregancy, special caution is necessary in the administration of drugs that might adversely affect the uterus or fetus. In obstetric practice, the recommendation "to avoid the use of all drugs except those essential to maintain pregnancy or the health of the mother"[1] often results in therapeutic nihilism. Our first concern with oral magnesium (Mg) supplementation arose when it proved effective against pregnancy-induced calf cramps (in which, for example, quinine-containing drugs are contraindicated[2], and calcium supplements are ineffective[3]. In a woman receiving 15 mmol Mg orally, not only did the painful cramps stop within 24 h, but the clinical symptoms of preterm labour were also obviously attenuated[4]. These observations led to the start of controlled clinical tests, the results of which are reviewed in the following article, together with the relevant literature and basic data.

Magnesium in pregnancy

Magnesium metabolism in pregnancy

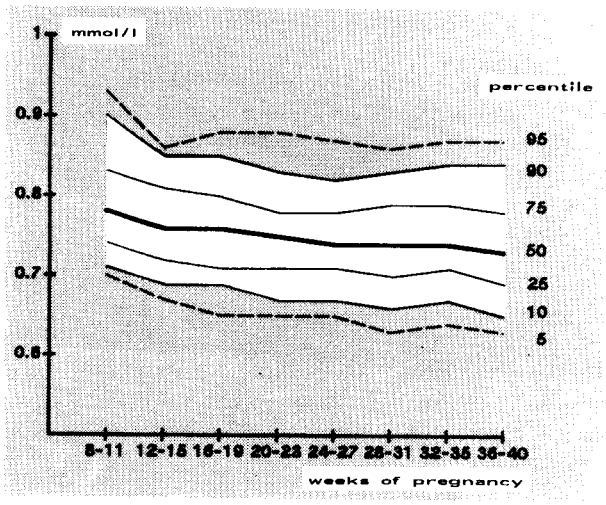
Magnesium concentration in body fluids and tissues — Under physiological conditions, serum/plasma Mg concentrations average 0.9 mmol/litre in adults, ranging from 0.75 to 1.0 mmol/litre in 90% of the cases[5,6]. Durlach reported levels of 0.88 ± SD 0.005 mmol/litre in a population of healthy, nontetanic persons[7]. Of the total body magnesium, only 55-60%[7,8] is present in the ionized, biologically active form, the remainder being bound mainly to albumin and partly complexed to anions. Since Mg is concentrated intracellularly, tissue levels are generally higher. In the myometrium, for example, levels amount to 16.6 ± 2.5 mmol/kg dry weight (Table 1).

Table 1. Mg content (mmol/litre, or mmol/kg where indicated) in different body fluids and tissues[9-11,19]. Values are means ± SD.

Tissue body fluid	Mg content (mmol/litre) Non-pregnant women	Pregnant women
Plasma	0.9 (0.75-1.1)	
	0.83 (± 0.005)	0.71 (± 0.07)
Erythrocytes	2.3 (± 0.17)	
Whole blood		1.19 (± 0.09) (n=30)
Uterine tissue	6.58 mmol/kg (wet weight)	
Myometrium	16.6 (± 2.5) mmol/kg (dry weight)	13.3 (± 2.9)
Placenta		21.35 (± 3.74) (dry weight) (n=30)
Urine Mg	3.81 (± 0.92)	4.78 (± 1.10)
Breast milk		
(colostrum)	1.59 (± 0.43)	
(mature)	1.41 (± 0.30)	
		range (1.22 to 1.46)
Full-term neonate (4373g)	11.1 mmol/kg free body tissue	

Especially towards the end of pregnancy a substantial decrease of Mg concentrations in body fluids and various tissues has been demonstrated. According to Seelig[10] lowered serum concentrations were first reported by Krebs & Briggs[12] and Bogart & Plass[13] in 1923. From Seelig's extensive review of the literature it can be concluded that, independently of different methods and reference values, hypomagnesaemia with an incidence averaging ~ 17% has been detected in 75% of all studies. Accordingly, Aikawa[14] concluded: 'There may exist a subclinical deficiency of Mg even in normal pregnancy'. In our investigations, also, Mg concentrations were found to be reduced, from 0.83 ± 0.05 (non-pregnant women) to 0.71 ± 0.07 mmol/litre at week 40 of pregnancy[15]. The time course, resembling the data of Baltzer & Daume[16], is depicted in Fig. 1.

Fig. 1. Serum Mg (mmol/litre) in pregnant women: median (fat line) and different percentiles (number of women in each period of pregnancy = 77-284).



In this context it must be noted that plasma volume expands in normal gestation (dilution effect) whereas it is markedly constricted in pre-eclampsia (concentration effect); hence a haematocrit values, eg, should be used for the correction of haemodilution[10] (for discussion see[17]). Furthermore, since serum protein content usually decreases somewhat during pregnancy, total Mg should be corrected for protein according to the following formula:

$$Mg_c = \frac{Mg_m}{0.76 + PP/30}$$

where Mg_c = corrected total magnesium (mmol/litre); Mg_m = measured total magnesium (mmol/litre); and PP = measured total protein (g/dl).

However, the magnitude of correction is usually small[18], and Seelig[10] concluded that pregnancy-induced hypomagnesaemia is real, despite different techniques employed.

It is generally agreed that intracellular electrolyte determinations are a more reliable parameter for evaluating the actual Mg status[7,10,14]. Controversy exists about whether erythrocyte Mg is a suitable index[17]. During pregnancy a significant decrease in Mg content of the myometrium has also been observed: related to dry weight, myometrial Mg concentrations decreased from 16.6 to 13.3 mmol/kg in the last 10 weeks of pregnancy; related to wet weight a reduction from 3.40 ± 0.51 to 2.82 ± 0.64 mmol/kg could be demonstrated[15]. Taking numerous balance studies into account[10] there is no doubt that pregnancy is prone to lead to Mg depletion because of increased demand and renal losses (see later). Hence, Mg intake will be largely decisive in determining whether or not a Mg deficit develops.

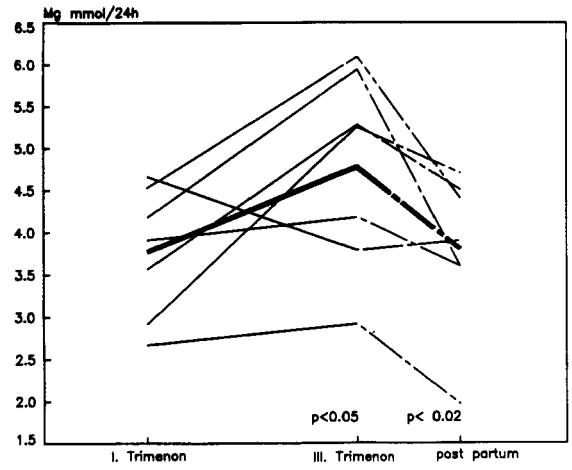
Renal magnesium excretion — Urinary Mg excretion was studied as a possible reason for the hypomagnesaemia described, correlating it with diminished Mg levels in the myometrium[19]. Twenty four hour urine and blood samples were taken from seven pregnant volunteers once every two weeks (beginning at 6-12 weeks of pregnancy) (Fig. 2). Mg was determined in the urine and plasma using the AAS technique. A 25% increase in Mg excretion from 3.81 to 4.78 mmol/24 h ($P < 0.02$) (Table 1) was shown. Simultaneously, plasma Mg levels decreased by 15% from 0.82 ± 0.05 to 0.70 ± 0.03 mmol Mg/litre ($P < 0.01$). Mg clearance

increased by 41% from 3.23 ± 0.76 to 4.57 ml/min ($P< 0.01$). Hence, increased urinary Mg excretion is probably one of the main reasons for the resulting magnesium deficit. The relatively low renal reabsorption may be due to the following changes associated with pregnancy:

(1) Increase in extracellular fluid
(2) Increase in glomerular filtration rate
(3) Reduced Mg reabsorption due to increased sodium reabsorption.

In addition, the fraction of ultrafiltrable Mg may be increased[10]. The renal Mg excretion is shown in Fig. 2.

Fig. 2. Mg excretion (mmol/litre) in 24 h urine during pregnancy. The collective regression line is fat[20].



As already mentioned, Mg requirement is markedly increased during pregnancy[10] and we have no precise information about whether the choice of nutrients may be changed, ie about whether meals with low Mg content may be preferred. In pregnancy the additional requirement of energy is only small, but the additional requirement of Mg is high. The increased food energy required amounts to only ~ +15%. Hence it is difficult to provide additional Mg. Doyle *et al*[21] found a significant correlation between dietary Mg intake towards the end of the first trimester and prenancy outcome.

Magnesium supplementation during pregancy

Effect on the mother — Even though Mg has had its place in obstetrics for the management of pre-eclampsia and eclampsia since the beginning of this century and is still recommended as 'the drug of choice in this situation'[1], its significance for preterm labour was only recognized in the 1960s[22]. Ten years later a reduction of preterm labour by low dose Mg infusion (1-2 g/day) was described[23]. General interest in the role of Mg in obstetrics was reactivated by our observation that supplemental Mg medication allowed a considerable reduction of the dose of β-adrenegic agents used for tocolysis of preterm labour[4]. Based on these findings it could be demonstrated, mainly in retrospective studies, that the frequency of fetal growth retardation and pre-eclampsia was reduced in association with Mg supplementation[24].

To ascertain these observations a double blind study was performed in 568 unselected women included in the trial as early as possible in pregnancy but not later than 16 weeks of gestation. Magnesium, 15 mmol as magnesium aspartate hydrochloride, was given orally per day. The placebo group received aspartic acid[25].

In the placebo group significantly more women were admitted to hospital (65 *vs* 44). Concerning the indications of admission to hospital, haemorrhage during pregnancy (17 *vs* 4), incompetent cervix (17 *vs* 8), and preterm labour (26 *vs* 12) occurred more frequently in the placebo group. The median gestation was significantly longer in the women treated with Mg (40 weeks *vs* 39 weeks and 6 days), although the difference between the medians was not more than 1 day. No specific, undesirable effects were reported in the treatment group (Table 2).

Table 2. Indications for hospital admission during pregnancy in women supplemented orally with 15 mmol of Mg or placebo.

	Magnesium-treated group (number of women)	Placebo group	Significance
Hospitalized women	44	65	$P< 0.05$
Indications coded			
Haemorrhage	4	17	$P< 0.01$
Incompetent cervix	8	17	$P< 0.05$
Preterm labour	12	26	$P< 0.05$
Other indications	20	5	

These results have been completely confirmed in another recently published Hungarian study in which 985 pregnant women were included. They also received 15 mmol Mg per day (as the asparta-te), or placebo. In addition, these authors observed a reduction in the incidence of pre-eclampsia (5.6% *vs* 3.7%)[26]. Indirect support for these data is given in a large scale prospective follow-up study of 7870 pregnancies during the years 1964-1970 in which 20 German centres have participated.

Among 500 variables evaluated it turned out that women with stomach complaints taking (Mg-containing!) antacids had a surprisingly low rate of early and late abortions and their babies exhibited low perinatal mortality[27].

Serious drug interactions with Mg supplements are not expected and no clinical data have been published up to now. It may be that the pharmacological initiation of labour with oxytocin is modulated by plasma Mg levels, since women with higher Mg concentrations needed a significant increase in the use of this drug to augment labour[28]. Furthermore enteral iron absorption is supposed to be disturbed in the presence of Mg. This might be due to Mg:Fe interactions and to gastric pH alterations, since many Mg salts are used as antacids. Preliminary data obtained with chloride-containing Mg compounds (Mg-Asp-HCl) have shown, however, that in this combination no significant interactions occur[29].

Effect on the fetus — The positive effect of maternal Mg supplementation seems to contribute to a longer gestational age. In our study mentioned already (see Table 2) admission of neonates to the intensive care unit was significantly less common in the treatment than in the placebo group (36 *vs* 20). In the subgroup of mothers regularly supplemented with Mg the incidence of babies below 2500g could be reduced from 8.2% to 2.8%. They had a significantly greater crown-heel length (50 *vs* 49) and head circumference and their Apgar scores were less often below 8 (0 *vs* 5)[25]. In addition, a 10% reduction in the incidence of intrauterine growth retardation could be demonstrated[26] (Table 3).

Table 3. Fetal outcome in women treated with magnesium or placebo during pregnancy.

	Magnesium group (n=217) (Centile)			Placebo group (n=220) (Centile)			
Variable	5th	50th	95th	5th	50th	95th	Significance
Bithweight (g)	2610	3340	4285	2245	3300	4110	$P< 0.05$
≤ 2500g (n)		6*			18*		$P< 0.05$
≤ 1500g (n)		0*			6*		$P< 0.05$
Infant length (cm)	46	50	53	44.5	49	53	$P< 0.05$
Head circumference (cm)	33	35	37	31	34	36.5	$P< 0.05$
10 min Apgar score <7 (n)		0			5		$P< 0.05$

*Related to total numbers in the magnesium group and in the placebo group, there were only 2.76% of infants with birthweight < 2500g and 0% < 1500g in the magnesium-treated group *versus* 8.18% with birthweight under < 2500g and 2.72% < 1500g in the placebo group.

It would be worthwhile studying whether the development of newborn and small children is improved after magnesium supplementation during pregnancy, because offspring of rats supplemented with magnesium during pregnancy showed a better learning behaviour than others exposed to a Mg deficit during pregnancy[30]. Conversely, retrospective studies on 58 mothers with premature deliveries showed significantly more frequent clinical and electromyographic signs of hyperexcitability than 51 mothers with full term infants[31]. In this context, a recently published study of Sibai, Villar & Bray[32] needs to be discussed briefly. One hundred and eighty five primigravid women between 13 and 24 weeks gestation received oral supplements of 15 mmol Mg (as Mg-aspartate-hydrochloride) daily and were compared to 189 placebo-treated patients. Although mean diastolic blood pressure did not exceed 78 mm Hg in any group, the principal concern of the study was directed at detecting any bloodtpressure lowering effects of Mg supplements. This was, of course, impossible, although serum Mg levels increased significantly from 0.642 (placebo group) to 0.691 mmol/litre (supplement group) ($P< 0.01$). It is not very surprising that pregnancy and natal outcome revealed no significant treatment effects, since the population under study consisted of indigent, mostly black volunteers, ie of rather unreliable patients, whose basal dietary daily Mg intake (estimated to amount to 260 mg) was so low that hypomagnesaemia persisted even in the supplemented group. In view of this obvious underdosage of Mg these data appear inappropriate to cast serious doubts upon the two other studies which clearly show beneficial effects of Mg supplementation dring pregnancy.

Some major differences in study design have also to be outlined. The study of Sibai *et al.*[32] compares a low dosage *versus* a high dosage magnesium supplementation, while in our study[25] patients with no magnesium supplementation were compared with those on a high dosage. The study group was restricted to young primigravidas in order to include patients at higher risk of pre-eclampsia. Furthermore magnesium supplementation was started at a later stage in pregnancy, 17.8 weeks *versus* 13.3 weeks in our study.

It should be clearly stated that in their study Sibai *et al.* did not find an effect of increasing magnesium supplementation from 100 mg to 465 mg daily with intake started after 13 weeks in young primiparas in preventing the development of pre-eclampsia. They certainly have not disproved the finding of beneficial effects on overall outcome of early onset high dose magnesium supplementation in an unselected group when compared to no magnesium supplementation.

High dose parenteral magnesium treatment

Pre-eclampsia — In 1905 Meltzer & Auer[33] had already demonstrated a muscle-relaxing effect of subcutaneous $MgSO_4$ in animals. The first use of Mg in the treatment of eclampsia was published by Rissmann in 1916[34]. While in German-speaking countries this therapy at first did not gain acceptance, $MgSO_4$ became the routine therapy of eclampsia in the United States after the contributions of Lazard[35] and Dorsett[36].

It must be pointed out that this therapy makes use of pharmacological effects of high parenteral magnesium doses[7], blocking dose-dependently the release of acetylcholine at the neuromuscular junction for example[37], as well as the release of catecholamines from the adrenal medulla[38], and enhancing the production of prostacyclin by vascular epithelium[39].

At the present time, two therapeutic procedures are carried out. In case of an eclamptic seizure or a life-threatening seizure a 'loading dose' of 4g $MgSO_4$-7-H_2O (~ 16 mmol Mg) is injected slowly intravenously. Then a continuous infusion of 1g $MgSO_4$-7-H_2O/h (~ 4 mmol/h) is recommended[40]. The other schedule also starts with a loading dose of 16 mmol Mg intravenously; then a second dose of 40 mmol is injected intramuscularly, followed by further injections of 20 mmol Mg intramuscularly in alternate buttocks 4 hourly[41]. To reduce pain 1ml of a 2% lignocaine solution may be added[42].

In numerous cases it could be shown that high dose $MgSO_4$ is a reliable therapy for eclampsia or pre-eclampsia with few side effects; only in case of extreme hypertension is combination with hydralazine recommended[42]. $MgSO_4$ has a wide therapeutic range, but it is necessary to know the side effects which may occur during increasing serum Mg concentrations, which include reduction in blood pressure, nausea, vomiting, CNS depression, hyporeflexia, ECG changes, depression of respiration, coma and finally cardiac arrest, as the serum concentration increases to ten times the initial value[7,43].

During effective therapy with parenteral Mg, serum Mg levels are usually found to range between 1.5 and 4.0 mmol/litre[44]. The effects on the fetus and the newborn are discussed below.

Preterm labour — Kumar and coworkers have proven under *in vitro* and *in vivo* conditions that magnesium reduces the contractility of the human myometrium[45]. This is in agreement with data of Popper, Batra & Akerlund[46] showing that the uptake of ^{45}Ca in strips of human myometrium was considerably decreased by high extracellular Mg. Magnesium exhibited better tocolytic effects than ethanol[47].

In cases of insufficient tocolytic effects, Mg may be combined with infusions of β-adrenergic agents[48]. To reduce fetal respiration deficiency in case of delivery lung maturation must be induced by glucocorticoids during tocolysis with β-adrenergic agents. During this procedure pulmonary oedema is an uncommon but nevertheless known complication. The same complication may occur when Mg is used instead of β-adrenergic agonists[49]. Based on one case report, Snyder & Cardwell recommend that caution should be exercised when synthetic Ca antagonists like nifedipine are combined with Mg since neuromuscular blockade developed under these conditions[50].

Other aspects

Calf cramps

In a retrospective analysis it turned out that 6% of pregnant women suffer from calf cramps. In 19 of 21 women these cramps were alleviated after oral Mg therapy (10 mmol/day)[51]. Since other drugs like quinine must not be prescribed to pregnant women, Mg supplementation (see Introduction) is a beneficial alternative in these cases. As mentioned already, Ca is ineffective under those conditions[3].

Cardioprotection during tocolysis

During Mg deficiency, calcium influx into the cell is significantly facilitated, to the extent that the application of catecholamines to Mg-deficient laboratory animals caused extensive necrosis of myocardial tissue. This effect is amplified by simultaneous medication with 9-α-fluorocortisol. On the other hand the development of necrosis was prevented by feeding diets which were highly enriched with Mg, such as Mg-Asp-HCl[52]. From extensive available data, pregnancy can be assumed to present a Mg-deficient situation. During tocolysis with β-adrenergic agents, an induction of fetal lung maturation by corticosteroids is often necessary. Cardiotoxic effects of these substances may therefore also result. Hence we proposed cardioprotection with Mg in the form of 50 mmol Mg-Asp-HCl/day in addition to β-adrenergic therapy[53].

Magnesium in the newborn

Physiological aspects

A Mg gradient across the placenta is assumed since fetal plasma Mg levels are higher than maternal concentrations. This gradient, which is not due to differences in protein binding between mother and fetus[54], seems to be sufficient to protect the fetus against severe maternal Mg deprivation. A hypothetic active transport system from the

mother to the fetus may explain the gradient, but its existence remains to be proven (for further reading see[55]).

In rats experimental maternal Mg deficiency exerted profound adverse effects on various aspects of reproduction. When severe Mg deficiency existed during the whole gestation, 100% of the implanation sites of the fetus showed resorption. When Mg-deficient diets where offered during the second half of gestation about 20% of the offspring showed a wide variety of malformations[56]. Under similar conditions high neonatal mortality and pathological brain alterations of the newborns, such as necrosis and reduction in tissue thickness, could be shown[57]. Fetal anaemia was already obvious at a relatively mild degree of Mg deficiency, involving a reduction in haemoglobin and erythrocyte count as well as macrocytosis, associated with numerous microcytes and red cell fragments. A noticeable growth retardation was also observed when newborn rats were nursed by mothers receiving a Mg-deficient diet. A period of Mg deficiency of more than three months resulted in sterility of both males and females[58]. Interactions between low dietary Mg and high Ca on pregnancy outcome have recently been discussed by Pinkham & Kubena in this Journal[59]. In view of these experimental data it seems necessary to initiate epidemiological studies in man.

Hypomagnesaemia

In 1937 Nothmann[60] described fits in newborn babies and related them to hypomagnesaemia. In a child of a mother with primary hyperparathyroidism neonatal tetany was found to be associated with hypomagnesaemia[61]. If neonatal convulsions associated with hypocalcaemia are not accompanied by hyperphosphataemia and when it turns out to be impossible to normalize Ca concentrations unless the patient is treated with Mg, hypomagnesaemia may be the reason of these convulsions[62]. The origin of neonatal hypocalcaemic tetany may be differentiated by two symptoms: hypomagnesaemia and oedema. Babies in whom there is a positive correlation between serum Ca and Mg levels, showing a rise of serum Mg during Ca infusions, had no pitting oedema on the feet. On the other hand babies with oedema failed to show this phenomenon. Hence, secondary aldosteronism is suggested as a contributory factor in the production of neonatal hypomagnesaemia, favouring the renal excretion of Mg[63].

Another type of neonatal tetany seems also to be possible: late onset of neonatal tetany occurs in primarily healthy babies, full-term and with normal birthweight, born mostly in winter or spring, and belonging to a lower socioeconomic class. They were usually fed cow's milk formula with a low Ca content and a low Ca:P ratio. Half of these patients exhibited hypomagnesaemia and 60% had hyperphosphataemia[64]. Therefore, Mg deficiency may play a central pathogenic role.

While irritability is the main symptom of late neonatal tetany, early neonatal tetany is associated with hypotonia and reduced responsiveness occurring in the course of perinatal disorders such as prematurity, respiratory distress and hyperbilirubinaemia[64]. With the exception of prematurity, Mg is scarcely involved in these conditions.

It should be noted that (primary) hypomagnesaemia can also be transferred genetically. It is known that daughters of women with latent tetany frequently suffer from this disease as well[7,65]; and a case of primary hypomagnesaemia with secondary hypocalcaemia in a girl of consanguineous parents has been described in the literature and an autosomal recessive and X-linked inheritance is suggested[66]. Up to now, ~ 30 similar cases have been described[10].

Beside this, other symptoms less impressive than fits may be related to Mg deficiency. Full-term newborn babies with the clinical impression of hyperexcitability had significantly lower serum Mg on the first day after delivery, normalizing by day five[67]. In studies on young children polygraphic tracings showed a relationship between serum Mg and the pattern of sleep. After Mg injection 'quiet sleep' increased and 'active sleep' decreased[68].

Lower serum Mg levels were found in infants who were small for gestational age[69]. However, conflicting results are also reported[70].

Hypomagnesaemia

Mg is easily transported through the placenta even against the concentration gradient. Therefore, high serum Mg concentrations are expected in the fetus following standard therapy for pre-eclampsia/eclampsia, when high quantities of Mg are given to the mother intravenously[71].

After $MgSO_4$ infusions to toxaemic mothers, hypomagnesaemia in neonates has been described[72]. Serum Mg in the umbilical cord blood reached levels up to 5.75 mmol/litre. The clinical manifestations in the newborn were similar to those in hypomagnesaemic adults: lethargy, hypotonia, respiratory arrest, poor movements, depressed reflexes. But it has also been pointed out that the manifestation of acute or chronic intrauterine hypoxaemia could account for some of these findings[7,72]. In severe cases only exchange transfusion may help to lower serum Mg levels[73]. However, since the role of extracellular Mg in relation to Mg stores in negligible, dialysis constitutes the treatment of choice for severe magne-

sium overload[7]. Recently, Lamm *et al.* have hypothesized that prolonged infusion of $MgSO_4$ especially when initiated during the second trimester, may lead to fetal parathyroid gland suppression with consequent abnormalities resembling rickets. They observed two infants out of five with radiographic bony abnormalities[74]. During the 11th Hohenheimer Mg Symposium (September 1989) Weidinger & Wischnik reported that they have never observed similar cases in their large patient population (in preparation).

The team of Pritchard, most experienced in the therapy of eclampsia with Mg sulphate, surveying about 7000 infants whose mothers had received $MgSO_4$ parenterally, have obtained no evidence that $MgSO_4$ administered prior to delivery to hypertensive mothers according to their schedule and effectively preventing eclamptic convulsions was deleterious to their infants. Pritchard *et al.* give 30-40 g $MgSO_4$/24 h by intermittent intramuscular injections as long as maternal knee-jerk reflexes are demonstrable, urine flow is at least to 100 ml/4 h, and respiration is not depressed. Neonatal serum Mg concentrations remained elevated during the first 72 hours of life (mean at 72 h 1.23 mmol/litre)[75]. In premature babies with birth asphyxia, serum Mg concentrations were found to be higher and may be associated with decreased muscle tone[76].

There are no apparent effects of maternal Mg therapy on the neurological status of the neonate. Neurological performance of the neonate did not correlate with cord Mg levels nor with the total Mg dose administered[76].

Fetal heart rate patterns are helpful in assessing fetal oxygenation. The influence of $MgSO_4$ on fetal heart rate is controversial: for example a loss of variability of fetal heart rate has been described[40,77]. Other studies have reached opposite conclusions[78]. In an extended retrospective study[75], no negative effects on fetal heart rate pattern could be shown during high dose Mg therapy of pre-eclampsia/eclampsia.

Conclusions

The efficacy of high dose parenteral Mg administration in pre-eclampsia and eclampsia is well documented and generally accepted. It makes use of pharmacological (mostly Ca antagonistic) effects of Mg.

Adverse metabolic effects of a pregnancy-induced Mg deficit, which result in multiple disturbances of the mother, the fetus and the newborn, have been noticed by obstetricians only recently. Appropriate Mg supplementation during pregnancy has meanwhile been shown to improve maternal health, the whole course of pregnancy, and fetal outcome.

Mg deficiency seems to influence metabolic hemeostasis negatively, resulting in multiple disturbances. This may be the reason for the impressive effect of magnesium supplementation during pregnancy.

Mg deficiency is also the primary factor in numerous diseases of the newborn and in children. CNS alterations may be most obvious. Psychovegetative disturbances are difficult to prove scientifically. The diseases are as diverse as the role of Mg in metabolism, and may be caused by 'microlesions of metabolism'.

A diversified nutrition with non-purified products — like, for example, whole meal bread — may avoid disturbances of the magnesium homeostasis in healthy people. Situations with greater need, like pregnancy, should be adequately supplemented, and recognized Mg deficiency should be treated with Mg. Well designed studies are necessary to characterize Mg-deficient situations better; thus physicians should become more conscious of the role of Mg in health and disease[7].

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Le magnésium chez la femme enceinte et le nouveau-né

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Résumé: Cette étude constitue une revue générale sur l'importance du magnésium pour la femme enceinte et pour le développement foetal. Une supplémentation magnésique au cours de la grossesse semble être nécessaire. Elle induit des effets bénéfiques sur la santé de la mère et du nouveau-né.

Diverses données majeures se dégagent:

— Les concentrations du Mg sérique diminient au cours de la grossesse.
— L'excretion magnésique rénale s'accroit de 25%.
— Une supplémentation magnésique exerce un effet positif sur la durée d'hospitalisation et le travail prématuré.
— La durée de la gestation est prolongée.
— Des convulsions peuvent survenir chez les nouveaux-nés hypomagnésiques.
— L'hypermagnésémie peut être observée chez des nouveaux-nés dont les mères toxémiques ont été traitées par des perfusion de SO_4Mg.

Mots clés: Grossesse, magnésium, nouveau-né, supplémentation magnésique, revue.

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