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PENETRATION POWER OF ANTISEPTIC CREAMS („IN VITRO" MODEL WITH PIG SKIN)

V. Garcia Torres, R. Herruzo, F. X. Santos Heredero, F. Lenguas, S. Martinez Ratero, C. del Fresno

INTRODUCTION

Local treatment of burns is controverted, mainly due to the great number of effective substances, according to different authors (MOLESKI (1978), MODAK (1981), RICHARDS (1981), LAWRENCE (1982), PEGG (1982)). The most frequent of these products are Silver Nitrate, Mafenide Acetate, Silver Sulfadiazine, Chlorhexidine, Iodine Povidone, Nitrofurazone and some local antibiotics (McMANUS, GOODWIN, PRUITT (1983)). None of these substances meets the requirements of the ideal local treatment of burns (MOLESKI (1978), ZELLNER, BUGYI (1985)). The ideal product should: be able to destroy both superficial and deep located microorganisms, be toxicity-free for the host, have a wide spectrum and its repeated application be easy and painless.

In this work we try to determine, through an "in vitro" method, the penetration power of different antiseptic creams in order to value the first of the above mentioned criteria of a good local chemotherapy.

MATERIAL AND METHODS

1. Material

a. Antiseptic creams:

1% Silver Sulfadiazine, 1%Silver Sulfadiazine with 2.2% Cerium Nitrate, 2.2% Cerium Nitrate, 10% Iodine Povidone, 0.2% Nitrofurazone, 0.1% Chlorhexidine, 0.5% Chlorhexidine, 1% Chlorhexidine,

b. Microorganisms: 17:

P. aeruginosa, K. pneumoniae, S. marcescens, S. aureus, Enterobacter cloacae, E. coli, S. faecalis, Sercina lutea.

All of them isolated from burn wounds.

E. coli K12, J5, control.

c. Lyophilized pig skin (Lyoderm).

2. Methods (Fig. 1).

After hidratation of the pig skin, it is fragmented with sterile scalpel in 1.5 cms. side squares. These pieces are disposed on sterile Petri plates with 20 ml. of Agar AOAC until no water is seen on or surrounding them.

Afterwards, a barrier of sterile cream is placed in the borders and pores of each skin fragment to avoid diffusion of the antimicrobial agents which are then placed in the middle of each fragment. The drugs (100 microl.) are placed, other pure, other diluted (from 1/2 to 1/128).

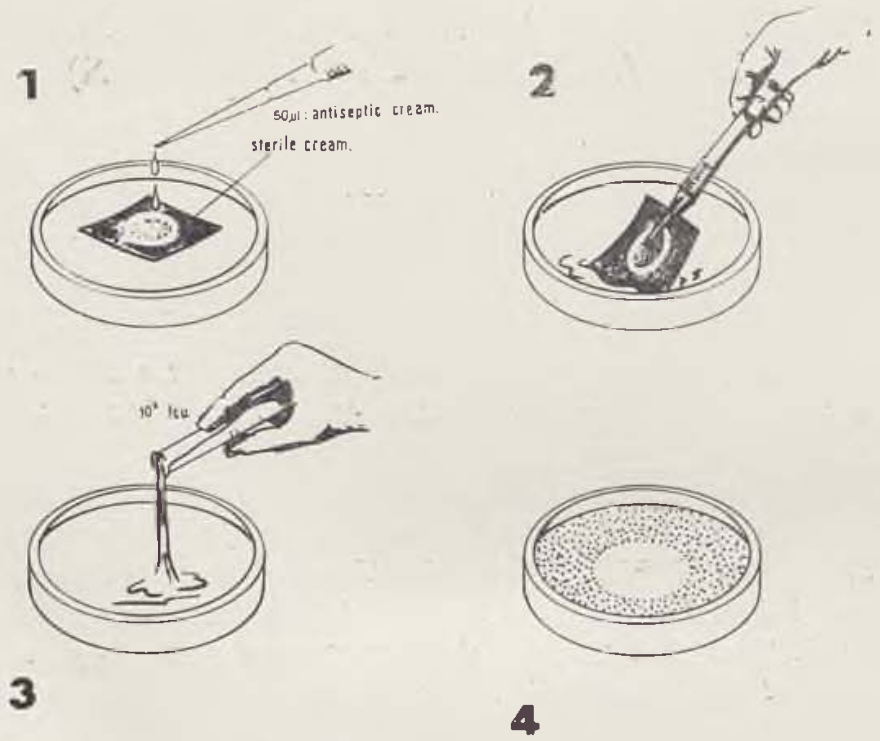

Fig. 1. Steps of the experimental model.

After 24 hours at 37 C grades in an humidified ambient, the skin squares are removed leaving in each plate only the amount of cream that could have passed through the skin thus penetrating in the agar. Then the plates are cultured, by immersion, with one of the microorganisms studied at a concentration of 100.000 fcu/ml. The plates are then dried in a stove and incubated at 37 C during 24 hours.

The last step of the method is to read the inhibition halos of the creams that passed through the skin. This reading is compared with the MIC (minimal inhibitory concentration) of these creams against the different microorganisms. This is done in the classic way: the creams are diluated, from 1/10 to 1/10240, and are included in Agar plates. Using the Steer replicator, 100.000 ufc of the 17 microorganisms are seeded on the plates. They are incubated in a stove at 37 °C and results are read 24 and 48 hours later. MIC is the lower agent concentration at which microorganisms growth is inhibited. The quotient between

the MIC obtained with the classic method and the MIC after passing through the skin is what we call "penetration fraction".

RESULTS

Table 1, 2 and 3 show the penetration fraction (PF) of the antiseptic creams used. It can be observed that 1% Silver Sulfadiazine, 1% Silver Sulfadiazine with 2.2% Cerium Nitrate, 2.2% Cerium Nitrate, 10% Iodine Povidone and 0.1% Chlorhexidine evidence no penetration with this method. On the other side, 0.5% and 1% Chlorhexidine have spare penetration against Gram positive Cocos and some very sensible enterobacteria like E. coli and Klebsiella, but no penetration was detected against others more resistant like Enterobacter, Serratia or Pseudomonas. At last, 0.2% Nitrofurazone is the antiseptic cream with greatest penetration power although it has no activity against Pseudomonas.

TABLE 1. Penetration Fraction (PF) of 1% Silver Sulfadiazine (AgS), 1% Silver Sulfadiazine with 2.2% Cerium Nitrate (AgS + CeN) and 2.2% Cerium Nitrate (CeN).

	AgS	AgS + CeN	CeN
Pseudomonas aeruginosa (1, 2, 3)	0	0	0
Klebs, pneumoniae (1, 2, 3); Serratia marcensens (1, 2);		0	0
Staphylococcus aureus (1, 2, 3)	0	0	0
Streprococcus faecalis	0	0	0
Escherichia coli	0	0	0
Sercina lutea	0	0	0
Escherichia coli-K12 J5 (control)	0	0	0

TABLE 2. Penetration Fraction (PF) of 10% Iodine Povidone (IP) and 0.2% Nitrofurazone (N).

	IP	N
Pseudomonas aeruginosa (1, 2, 3)	0	0
Klebs. pneumoniae (1, 2, 3); Enterobacter cloacae; K.E.S. 0 Serratia marcen ens (1, 2);		1/4
Staphylococcus aureus (1, 2, 3)	0	1/4—1/2
Streptococccus faecalis	0	1/4
Escherichia coli	0	1/2
Sercina lutea	0	1/4
Escherichia coli-K12 J5 (control)	0	1/4

In a parallel study with 4% Chlorhexidine the penetration fraction increased in direct relation with concentration against all microorganisms except Pseudomonas.

The results are summarized as follows:

— Silver Sulfadiazine, Cerium Nitrate, Iodine Povidone and 0.1% Chlorhexidine show no penetration at all with this model.

— 0.5% and 1% Chlorhexidine show mild penetration.

— 0.2% Nitrofurazone is the most effective from the penetration point of view.

DISCUSSION

Although the activity of antiseptic creams in direct contact with microorganisms is good (similar with what occurs in the surface of the scar), it varies considerably when the penetration fraction is quantified (equivalent with what occurs under the scar).

1% Silver Sulfadiazine, 1% Silver Sulfadiazine with 2.2% Cerium Nitrate and 2.2% Cerium Nitrate (Table 1): they have spare penetration and no bacterial activity. This lack of penetration has been reported by various authors. MODAK (1981) found that "in vitro" sensibility of P. aeruginosa for Silver Sulfadiazine has no relation with "in vivo" resistance. RICHARDS (1981) and PRUITT (1983) showed no penetration at all across the burn lesion. PEGG (1982) reports a 5% penetration but in our experience it should be much lower because, although it's MIC (classic method) against the majority of germs is high, no amount of substance could be detected across the skin. It should be noticed that none of the products with Cerium demonstrates penetration capacity in our study.

10% Iodine Povidone (Table 2): it has no no penetration in our experimental model as previously reported by PEGG (1982) and KOCH (1985). ZELLNER (1985) and BALOGH (1985) measured Iodine blood levels after treatment with this drug. They found amounts of Iodine not high enough to alter thyroid function. These levels are not active against microorganisms, due to the high MIC of 10%. Iodine Povidone and its rapid inactivation by organic environment (HERRUZO (1981)).

STEFANIDES (1976), by means of an "in vitro" test, found that this product has good penetration through the scar. But he uses a glass sphere on the scar to obtain a good contact with the agar, and this artefact may alter the scar permeability. This can be the reason why all products studied by the author, except Silver Nitrate, present a great penetration through the scar, in opposition with the results obtained by other authors and by ourselves.

0.2% Nitrofurazone (Table 2): shows the greatest penetration fraction: 25—50%, that means a 1/2 or 1/4 reduction in the value of its MIC. Nevertheless this activity is not seen against the most resistant microorganisms (Pseudomonas) (MIC < 1/10), so another drug must be selected when these germs are found (MUNSTER (1984)).

Chlorhexidine (Table 3): has very spare penetration at low concentration (0.1%) but it becomes apparent when used at 0.5% against entero-

bacteria and Gram positive cocci. There are no significant differences between 0.5% and 1% concentrations although we have found a tendency to increase its effect with the 4% concentration.

Considering only the penetration power of the antiseptic creams, we conclude that the election order should be:

1st Nitrofurazone (except in those patients who dont tolerate its side-effects).

2nd. 10% Iodine Povidone and 1% Silver Sulfadiazine.

At this point is should be mentioned that none af the products show subscar efficence against Pseudomonas, the most frequent microorganisms in Burn Units. This means that local therapy is only able to control superficial proliferation of microorganisms thus facilitating the unique actual local treatment: surgical debridement (Table 4).

TABLE 3. Penetration Fraction (PF) of 0.1%, 0.5% and 1% Chlorhexidine (C).

	0.1% C	0.5% C	1% C
Pseudomona aeruginosa (1, 2, 3)	0	0	0
Klebs. pneumoniae (1, 2, 3); Enterobacter cloacae; K.E.S. 0 Serratia marcen ens (1, 2);		0—1/256	0—1/256
Staphylococcus aureus (1, 2, 3)	0	1/512—1/256	1/512—1/256
Streptococcus faecalis	0	1/256	1/256
Escherichia coli	0	1/257	1/256
Sarcina lutea	0	1/256	1/256
Escherichia coli-K12 J5 (control) 0		1/256	1/256

TABLE 4. Penetration fraction (PF) of antiseptic creams (RESUME)

1%	Silver Sulfadiazine	No penetration
1%	Silver Sulfadiazine +	
2.2%	Cerium Nitrate	No penetration
2.2%	Cerium Nitrate	No penetration
10%	Iodine Povidone	No penetraztion
0.2%	Nitrofurazone	PF = 1/2 — 1/4 (except Pseudom. aerug.)
0.5%, 1%	Chlorhexidine	mild penetration: 1/128—1/512

It the patient is treated within the first 24 hours with some of the above mentioned drugs (mainly Nitrofurazone or 0.5%, 1% Chlorhexidine) the microbial contamination can be maintained at a very low level until surgical debridement and coverage.

SUMMARY

By means of an "in vitro" method using pig skin, the authors determine the penetration power of some antiseptic creams in order out the most effective one from this point of view in the treatment of subscar-located infections.

The following antiseptic creams were studied:

1% Silver Sulfadiazine
1% Silver Sulfadiazine with 2.2% Cerium Nitrate
2.2% Cerium Nitrate
10% Iodine Povidone
0.2% Nitrofurazone
0.1%, 0.5% and 1% Chlorhexidine.

These products were faced with 17 microorganisms isolated from burn wounds and a control one.

The minimal inhibitory concentrations (MIC) obtained after passing through the penetration power of some antiseptic creams in order to find out the most effective one from this point of view in the treatment of subscar-located infections.

RÉSUMÉ

Pouvoir pénétrateur des crèmes antiseptiques — expérience "in vitro" sur la peau porcine

Garcia Torres, V., Herruzo, R., Santos Heredero, F. X., Lenguas, F., Martinez Ratero, S., Fresno, del C.

Les auteurs ont examiné "in vitro" le pouvoir pénétrateur de quelques crèmes antiseptiques sur la peau porcine et ont cherché une thérapie plus efficace des infections de localisation sous-cutanée. On a examiné les crèmes antiseptiques suivantes: sulfadiazine d'argent 1%; sulfadiazine d'argent 1% + nitrate du cérium 2,2%; nitrate du cérium 2,2%; iode-Povidon 10%; nitrofurazone 0,2%; chlorhexidine 0,1%, 0,5% et 1%. Ces substances ont été confrontées à 17 espèces de microorganismes, isolés des plaies de brûlures et des plaies de contrôle. Les concentrations d'inhibition minimales (MIC), obtenues après la pénétration dans la peau porcine, ont été comparés aux MIC, obtenues sans insertion de la peau, dont résultait la fraction de pénétration (PF).

ZUSAMMENFASSUNG

Penetrationsfähigkeit antiseptischer Kreme — ein Versuch in vitro mit Schweinehaut

Garcia Torres, V., Herruzo, R., Santos Heredero, F. X., Lenguas, F., Martinez Ratero, S., Fresno, del C.

Die Autoren erforschten in vitro die Fähigkeit einiger antiseptischer Kreme, die Schweinehaut zu durchdringen, und suchten eine effektive Therapie einer unter der

Haut lokalisierten Infektion. Es wurden die folgenden antiseptischen Kreme erprobt: 1 % Silbersulfadiazin, 1 % Silbersulfadiazin + 2,2 % Serinnitrat, 2,2 % Zeriumnitrat, 10 % Jod-Povidon, 0,2 % Nitrofurazon, 0,1 %, 0,5 % und 1 % Chlorhexidin. Sie konfrontierten diese Stoffe mit 17 Arten von Mikroorganismen, die aus Verbrennungs- und Kontroll-wunden isoliert wurden. Die minimale Inhibitionskonzentration (MIC), die beim Durchdringen der Schweinehaut erziel wurde, verglich man mit der MIC, die man ohne Einleitung der Haut gewann, wodurch man dann die Penetrationsfraktion (PF) erhielt.

RESUMEN

La capacidad de penetración de de la cremas antisépticas — el experimento in vitro con la piel porcina

Garcia Torres, V., Herruzo, R., Santos Heredero, F. X., Lenguas, F., Martinez Ratero, S., Fresno, del C.

Los autores examinaban in vitro la capacidad de algunas cremas antisépticas de penetrar en la piel porcina y buscaban la terapéutica más efectiva contra la infección subcutánea. Fueron investigadas estas cremas antisépticas: 1% sulfadiazina de plata + 2,2% nitrato de serina; 2,2% nitrato de cerio; 10% yodo-Povidone; 0,2% nitrofurazone; 0,1%, 0,5% y 1% clorhexidina. Estas substancias fueron enfrentadas con 17 tipos de microorganismos aislados desde las heridas causadas por quemaduras y las de control. Las concentraciones inhibidoras mínimas (CIM) obtenidas a base de la penetración en la piel porcina fueron comparadas con las concentraciones de CIM obtenidas sin ninguna peneración; de tal manera se obtuvo la fracción de penetración (FP).

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AETIOLOGICAL, MODIFYING AND LETHAL FACTORS IN CLEFT LIP AND PALATE

M. Černý, M. Fára, J. Hrivnáková

INTRODUCTION

Cleft lip and palate can serve as a model object of prenatal semi-lethal, multifactorial conditioned congenital defect. While often successful in experimental models (8, 10), the search for possible aetiological factors in man remains inconclusive as to the aetiology of the defect except for the generally recognized genetic disposition derived from familial incidence, the presence of microforms of the anomaly, from twin studies and from population or racial differences.

One of the causes of this aetiological uncertainty may be in the large number of the factors involved, in a low degree of effect and in but a small part of the cases or in interaction with other low-effect factors. What seems more important, however, is that the lethal factor is present in every embryo toxic factor co-responsible for the aetiology of cleft. As a result, this factor appears among the group of cleft-affected embryos while on the other hand, there is progressively less of it under the effect of lethality. Hence, in a group is cleft affected childrem compared with controls, we may not find any major (statistically significant) differences in the proportion of the suspected factor, or even the more lethal factor may be represented in the group less prominently than the general population (2, 6).

If the embryo has crossed the threshold of teratogenesis, its developmental homeostasis has been impaired and, apparently, even a minor impulse may alter the degree of the cleft defect. A weak exogenous factor is then more likely to affect damaged embryos (or embryos where the sum of embryotoxic factors exceeds the threshold of teratogenesis) than in healthy embryos. In our own material we noted changes which could be explained by a shift of subtotal cleft lip (CL_s) into the group of total clefts (CL_t) or by a shift into the CLP group (cleft lip and palate), (3, 4, 6, 9). These changes which change the degree of the defect are called modifying factors. The modifying factors appear to act

in both ways, i.e. in terms of the degree of the cleft some factor may exercise a "protective" influence while others may aggravate the defect (embryotoxic factor). For example, female embryos exposed to the protective influence may not develop CLP (which would have developed without this "protection') so that only cleft palate (CP) may develop. In this way, the sex ratio in the population is altered, and that reciprocally with a high negative correlation between CP and CPL.

Every embryotoxic factor exercises an obvious aetiological, modifying and lethal influence, possibly in different proportions, though post-natally it is primarily the modifying influence that is most noticeable and that takes the form of a change in the proportion of the active factor in cleft subtypes, or a change in the spectrum of cleft defects.

Different types of cleft need impulses of different magnitude for their onset and development. The distance to the treshold of lethality is the opposite of the distance of the cleft type from the treshold of teratogenesis; CLP being the closest to lethality. Lethality is another factor capable of changing the spectrum of cleft defect. Given a weak impulse, CLP may more often develop CL; new CL cases arise less frequently because the "dose" will not do to cross the treshold of teratogenesis so that the factor becomes accumulated in CLP and reduced in CL. Vice versa, if the factor is stronger, new CL cases may arise and fewer cases of CLP will be registered because of the effect of lethality (4, 6, 9).

Changes in the representation of a factor in some groups classified by the type of cleft may lead to the conclusion that an active factor is involved, though it remains uncertain whether it exerts a "protective" or "embryotoxic influence". Studies of the interaction of two or more factors offer one of the possibilities. It the factor under study is found to have increased the effect of a known embryotoxic factor, is is likely to be embryotoxic itself. A decrease in the share of primiparae in the familial incidence of the cleft and the presence of blood group B may be an example (6, 9). Another indirect possibility is the detection of correlation between the rates of incidence of two factors concerning cleft subtypes. A higher positive correlation is a sign of a similar distribution and, thereby, also a similar (modifying) effect of both. For example, there is a positive correlation between the rate of cleft incidence in first-degree relatives and the proportion of the B blood group in females probands (6, 9). Comparisons of the situation in subgroups of men and women with cleft defect is another possibility as will be shown next (3, 4, 8).

MATERIAL AND METHODS

Probands with cleft lip (CL), cleft lip and palate (CLP), and isolated cleft palate (CP) were selected from patients of the Department of Plastic Surgery, Prague, from the catchment area of Bohemia (western part of Czechoslovakia, a genetically Central European population of 6 million, a high rate of cleft incidence — 1:520). The probability of detection at the Department is $p = 0,9$; the statistics ignore the stillborn and children who had died prior to coming

to the Department, about 2% of the children had undergone surgery at other centres. The number of the groups is different and stated in each particular chapter as are the methods of diagnosis.

Six subgroups were created in accordance with the degree of the defect:

CL_s — subtotal cleft lip
CL_t — total cleft lip
CLP_m — unilateral (monolateral) cleft lip and palate
CLP_b — bilateral cleft lip and palate
CP_d — isolated cleft of hard (durum) and soft palate
CP_m — isolated cleft of the soft (molle) palate

In some cases six subgroups were created in accordance with the basic cleft types (CL CLP, CP) and sex.

FAM 1° — coefficient of the rate of probands with cleft-affected parents or siblings (not the rate of the cleft defects in the parents). For example FAM 1° = 0.035 means that 3.5% of the probands have one or both parents with clefts or cleft-affected siblings.

Sr — sex ratio as the share of men calculated from the whole group where the whole group equals 1. (For example, Sr = 0.600 or 60% of the group are men).

Kp — coefficient of primiparae = share of primiparae calculated from the whole group, where the whole group equals 1.

R — correlation coefficient established most frequently between FAM 1° and the factor under study.

The differences in the rate of incidence were tested with the chi-square test in accordance with the customary criteria.

RESULTS

Familial incidence (FAM 1°)

Family history was taken in 2 764 probands without other associated defects and without syndromes as registered at the Department in the years 1964—1984 (Groups 1). The total number of cleft-affected children thus found was 3 399. Here we refer solely of the relative frequency of probands with cleft-affected relatives of the first degree, i.e. parents and siblings (FAM 1°), (3). The second group taken from nearly the same detection effort (up until 1984) was constituted in an ABO/blood groups study. Only the syndromes were singled out there, not probands with associated defect (n = 2 614) (6). For unknown reasons the two groups differ from each other only in the CP_m (Table 1). Statistical data for CL and CLP show in good agreement that women have about 1/3 higher FAM 1° and that both sexes have FAM 1° values proportional to the morphological gravity of the defect. For example, the values for bilateral total cleft lip and palate (CLP_b) in female probands are about three times as high as in cleft lip (CL_s) and twice as high as in total unilateral clefts. As regards clefts of the hard palate men and women show very much the same values, which is at variance with the idea of women being more susceptible to CP. We hypothetize that using the FAM 1° values we test the indirect necessary "dose" of

Table 1. Relative proportion of probands with cleft in parents or siblings (FAM 1°), rated by cleft defect type, M = males, F = females. Group I (n = 2 764) is without associated defects and syndromes. Group II (n = 2 614) — only probands with syndrome not with associated defect were excluded (Group ABO).

Group		Type of cleft defect					
		CL_s	CL_t	CLP_m	CLP_b	CP_d	CP_m
I	M	0.035	0.033	0.058	0.104	0.071	0.050
	F	0.047	0.051	0.080	0.159	0.080	0.051
II	M	0.028	0.032	0.053	0.097	0.080	0.105
	F	0.050	0.051	0.074	0.130	0.082	0.035

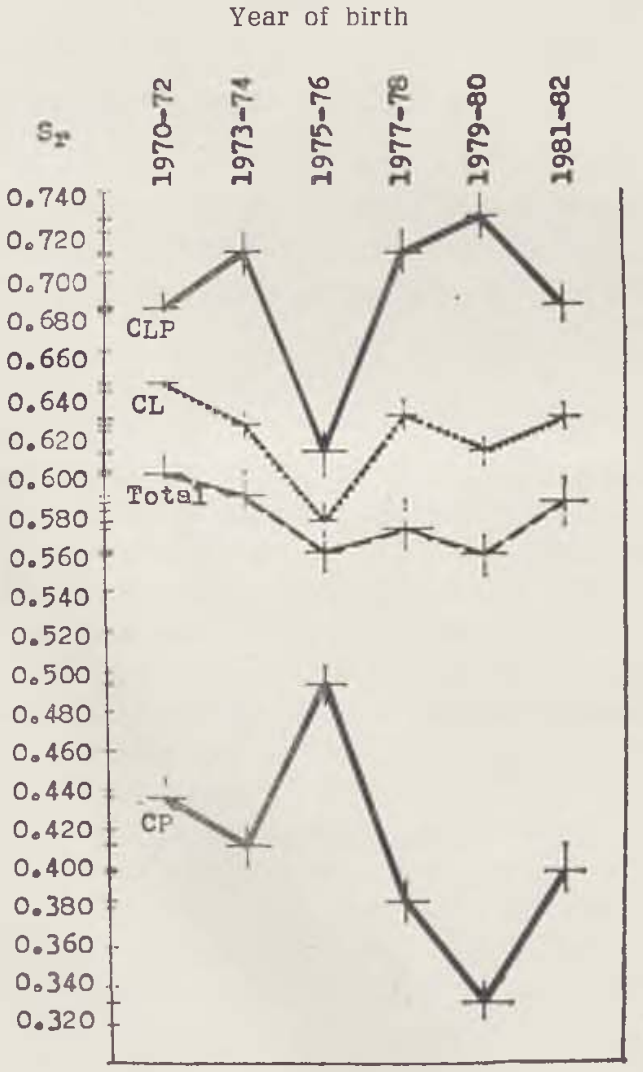
teratogenic agents for different forms of cleft. A measure of evidence is also in that the emperical risk values for the probands' children, so long as they are known, show the same kind of trend, being the lowest in CL men and the highest in CLP women (3, 6).

Sex differences, sex ratio

In the group of clef-affected children there are some well-known great differences in the sex ratio for the types and subtypes of cleft showing the female sex as being more resistant to CL and CLP. Graph No. 3 was designed with regard to the FAM 1° value found in the basic types of the defect (CL, CLP, CP) by the age of the embryo at the time of their onset and with a representation of the possible CL → CLP modification. (FAM 1° is given in per cent here). Apart from the increased resistance of the female sex the graph also shows the important fact that a total cleft in male patients "requires" about as much of an impulse as cleft palate in women. This, in our view, accounts for the "reciprocal balance", in this case in the sex ratio which we found repeatedly in our group (1, 4, 6, 9). As an example, we can refer to the sex ratio found in probands classified by the year of birth, n = 2 374. The negative correlation between the CLP and CP sex ratios is R = —0.905 (1). The explanation is in that a certain impulse (reaching FAM 1° = 8%) acts on both female and male embryos. In the latter there will be more CLP (the sex ratio will increase) while in the former there will be "only" more CP (the sex ratio will decrease). In this case there will not be more CP in men as it is included in CLP. A study of the prevalence of CLP and CP in men and women will show very much the same results. This phenomenon of "prenatal reciprocal balance" has to be taken into account in any study of active factors in cleft defects, though this calls for investigation of large series of probands.

Regional differences

A comparison of six Czech regions (including Prague) shows differences as to the diverse characteristics of clefts though there are no significant differences in the incidence of the defect as a whole, (n = 2 879). It appears that regional differences represent a complex, multifactorially conditioned phenomenon. Out of all the possible active factors we are able to analyze the share of primiparae (e.g. low birth rate in Prague or high share of primiparae (2) reduce the values of FAM 1°) or the distribution of the ABO blood groups (6) but we can make only indirect estimates of the effects of environmental factors. Again, there is a straightforward negative correlation between the sex ratios for CLP and CP. Similar negative correlations are found regionally



Graph 1. Sex ratio (Sr) in subgroups rated by the year of birth and type of cleft. There is a striking negative correlation between the values of Sr in CLP (cleft lip and palate) and in CP (isolated cleft palate); throughout the group, Sr shows relative stability — a case of prenatal reciprocal equilibrium.

between primiparity and familial incidence (as shown further), between CLP and CP prevalence, between the rates of incidence of the A and B blood groups and so on. The fact that there are no major changes in the total prevalence but only in the spectrum of the cleft defects and other characteristics is again in good agreement with our hypothesis of prenatal population equilibrium. The changes seen in regions with propably the worst-affected environment (north Bohemia, Prague) can best be explained by differences in the birth rate, lethality of the cleft-affected embryos, particularly those with CLP, and by the lethality of cleft-affected male embryos (8).

ABO blood groups

The ABO blood groups were identified in 2 614 probands of our principal group. 6 432 pregnant women from Prague 5 served as controls. Compared with the general population no significant differences in the distribution of blood groups were found in the group as a whole. Significant differences were found in the CL and CP sex ratio classified by blood groups, the value being relatively low in the A group, high in the B and AB groups (Table 2). Owing to the low number of the AB group we chose to consider solely group B for our further study. In our view, the lower group A sex ratio is due to the "protective factor" which is responsible for the fact that no clefts develop in the male embryos whereas in the female embryos where a stronger impulse is necessary for CL to develop this "protective influence" is insufficient. The opposite is true with the B blood group. Concurently with this explanation, incomplete cleft lip is more frequently found in men in the absence of group A and in the presence of group B. The opposite is the case with women. In such a case there will be more group A women and fewer group B women (X^2, $p < 0.01$) while men show the opposite situation ($p < 0.01$).

Given a positive family history up to the coefficient on kinship of 1/16, there was a lower proportion of primiparae we put down to lethality. The same result is also in this ABO group (X^2, $p < 0.02$). The A blood group, howe-

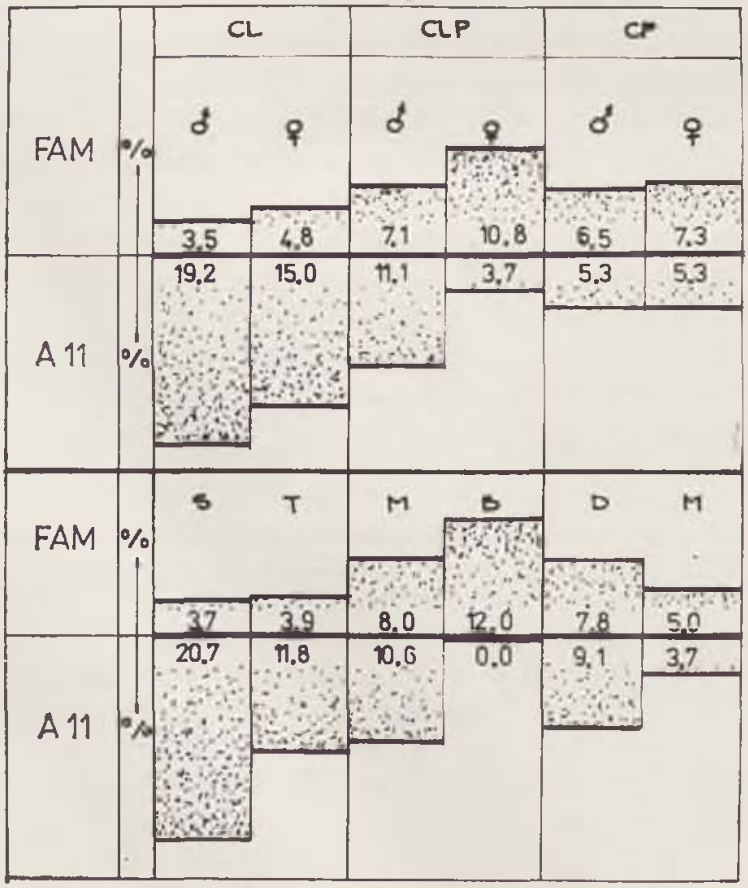
Tab. 2. Sex ratio (Sr). i. e. proportion of men as a whole, when the whole equals 1, the total number of probands (n) in principal types of cleft (CL, CLP, CP – see the text), in ABO blood groups.

Blood groups:	CL		CLP		CP		Σ	
	Sr	n	Sr	n	Sr	n	Sr	n
A	0.554	276	0.682	515	0.339	333	0.567	1124
O	0.597	196	0.686	373	0.446	258	0.590	827
B	0.646	113	0.689	222	0.462	119	0.619	454
AB	0.660	50	0.633	98	0.508	61	0.603	209

ver, shows a similar share af primiparae for familial and non-familial incidence, whereas in the other blood groups, primiparity in the presence of positive history is on the decline, the lowest being in the B group (X^2, $p < 0.02$); the greatest differences were found in the subgroup of women with familial incidence (X^2, $p < 0.01$). We attribute this phenomenon to decreased lethality of embryos affected by clefts in the simultaneous presence of familial incidence in blood group A — as distinct from group B.

The correlation coefficient between FAM 1° and the ratio of the ABO blood groups in six subgroups rates by the degree of the cleft (CL_s ... CP_m — see Methods) was highly negative in women with blood group A ($R = 0.859$) and positive for group B ($R = \div 0.931$).

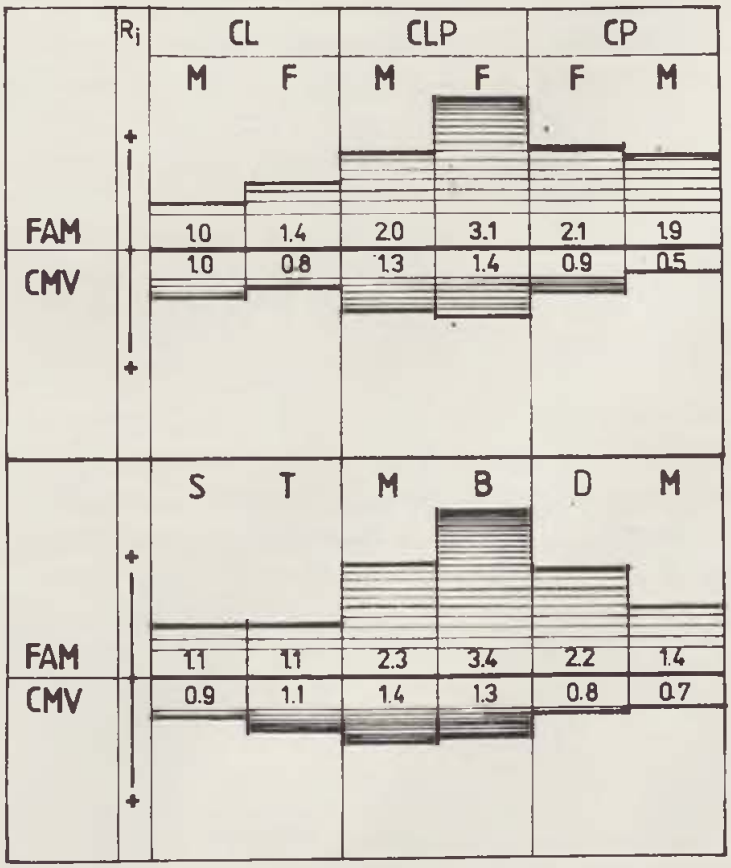
These results inspired the preliminary hypothesis that group A hampers the development of the more serious forms of cleft in women (hence it remains in CL), thus decreasing embryo lethality, and that in men it can still prevent the development of a cleft defect (CL). Conversely, the blood group B in women enhances the tendency towards the more serious forms, thus increasing lethality, too, while in male embryos it reduces also the threshold for aetiological factors (6, 9).



Graph 2. FAM 1° values and incidence of HLA A11 antigen (both in per cent) in subgroups rated by type of defect and sex (M = male, F = female) and by six subtypes of cleft (SL = cleft lip, S = subtotal, see Methods).

As for cleft defects, Bonner et al. found linkage between disposition and the H-2 locus H-2 in the mouse and, in a group of 133 probands, a probable association of the antigen HLA-A2 ($p < 0.005$) with cleft defects in Caucasians and an increased rate of antigens Aw24 and A28. Japanese authors studied 109 probands with all types of cleft to find a significant difference in Cw7, particularly in women, and markedly increased values in B7. There was no evidence of a genetic linkage between clefts and HLA (4).

We ourselves tested 147 mothers of children with cleft defects, in particular, 7 antigens of locus A (A1, 2, 3, 9, 10, 11, 28) and 14 antigens of locus B (B5, 7, 8, 12, 13, 14, 15, 17, 18, 21, 22, 27, 35, 40). An assessment of the incidence of antigens throughout the group as well as in the subgroups rated by the type of cleft, sex and other factors using Fischer's exact method could not rule the zero hypothesis. There were, however, major negative correlations between FAM 1° and the incidence of antigens A9 and A11 ($R = -0.815$ and -0.814) and a positive correlation in B17 ($R = +0.737$) in 6 subgroups rated by the degree of the cleft (see Methods). In subgroups rated by the main types of cleft (CL, CLP, CP) and sex, those particular antigens exhibited higher correlations,



Graph 3. Relative values of FAM 1° with CL values for men taken as one unit (= 1) similarly as in the frequency of positivity CFR against CMV. Subgroups as in Graph 2. A case of positive correlation of two factors.

too (A9, R = —0.743, A11, R = —0.784, B17, R = +0.817) to FAM 1°. This appears to involve a "protective influence" in the case of antigens A9 and A11 and an "embryotoxic" influence in antigen B17. We described these antigens as probably active in cleft defect (4).

Maternal parity (order of probands' birth)

The order of the cleft-affected probands' birth was identified in 3 029 children of our group (or 2 804 children) of the pilot group. The coefficient of primiparity of established (—K_P), (see Methods).

In the case of primiparity, positive family history (up to the coefficient of kinship = 1/16) was less frequent than in multiparity (X^2, $p < 0.01$). In cases of positive history, the K_p was significantly lower (X^2, $p < 0.01$), in cases of cleft lip and total cleft (CLP). In cases of incomplete clefts (whether already CL or CLP) the K_p value was lower in CL and higher in CLP (X^2, $p < 0.02$). This phenomenon appears to be due to the influence of the primipara where CL_s (subtotal) is transformed into total CL and harelip develops into a total cleft (CL → CLP), i.e. a modifying influence. As for primiparae, CLP appears to be more susceptible to prenatal selection (miscarriage). In cases of isolated of cleft palate (n = 980). The K_p value is higher in the more severe forms and rises in the sequence of: CP submucosum → molle → durum → Robin's anomaly. We assume that in this case primiparity worsens the degree of the cleft (2). Families in the indicidence of clefts in siblings were not included in the groups with K_p assessment.

The correlation coefficient between K_P and FAM 1° in subgroups though rather low (R = +0.324), probably due to the fact that lethality has a role to play in subgroups ith CLP_m and CLP_b, thus reducing the K_P values against FAM 1° (2).

Twins

The pilot group was found to include 62 pairs of twins; the theoretical expectation was 60 probands from twins, there were 68. Against expectations, positive family history was less frequent in twins (X^2, $p < 0.05$), priminently so in CL and CLP (X^2, $p < 0.02$). The probands' sex ratio was reduced to Sr = 0.485 against the expected Sr = 0.588. This appears to be due to the increased lethality in twins similarly as in the case of primiparity there is elimination of twins with positive histories and cleft-affected male embryos, though multiparity acts as a "substitute" for genetic disposition (being "embryotoxic") (2).

Maternal age

Maternal age was assessed in only 402 probands. The point of this exploratory fact finding was to show the effect of higher maternal age as women over 30 years of age have double the usual spontaneous miscarriage rate. In this particular series, mothers over 30 are more frequent that in the general po-

pulation (X^2, $p < 0.01$). This appears to suggest that cleft defects are more likely to develop in the children of older mothers; similarly there are fewer primiparae than it would be expected in the general population (X^2, $p < 0.02$), the probands' sex ratio is non-significantly lower in older women. There is a low and negative correlation between FAM 1° and the proportion of older mothers (30 +, $R = -0.383$). This is probably due to the fact that owing to embryo lethality older mothers are more likely to be associated with the less severe forms of the defect, i.e. the opposite of FAM 1° (6).

Table 3. Correlation coefficients (R) between the incidence of antibodies against Epstein-Barr virus (EBV), cytomegalovirus (CMV), cleft incidence of the proband's parents (FAM 1°) and some HLA antigens in six subgroups rated by the type and degree of cleft defect (CL_s ... CP_m — see Methods). The underlined correlation coefficients have the value of $p < 0.05$.

Correlation		MOTHERS		CHILDREN	
between:	FAM 1°	CMV	EBV	CMV	EBV
FAM 1°	—	+0.624	−0.749	+0.509	−0.824
CMV	+0.624	—	−0.368	—	−0.043
HLA A9	−0.815	−0.583	+0.246	−0.782	−0.344
A11	−0.814	−0.182	+0.416	−0.071	+0.890
B5	+0.708	+0.844	−0.479	+0.571	−0.537
B17	+0.737	+0.371	−0.950	+0.073	−0.827
B18	−0.624	−0.547	+0.822	−0.482	+0.523
B35	−0.822	−0.123	+0.730	−0.021	+0.795

Cytomegalovirus (CMV)

Complement-fixation (CF) antibodies of the IgG type were tested in 345 cleft-affected children and in their 245 mothers at their first visit at our Department, repeated sampling was in 2—4 months.

Within the group of children there was a difference (X^2, $p < 0.05$) in the positivity values for CL (43%), CLP (55%) and CP (34%). In subgroups rated by the child's age, the CLP group was found different even at the age of 201 days against the general population (X^2, $p < 0.05$) when we no longer presume the presence of transplantacelly transmitted CF antibodies. In the mothers' group, positivity was found more frequently in mothers of CLP children, particularly in titres of 1:16 and more and at the age of over 29 years (X^2, $p < 0.02$).

The correlation between FAM 1° and the proportion of positivity in cleft-affected children of six subgroups rated by the degree of the defect (see Methods) was found positive ($R = +0.509$), as was in their mothers ($R = +0.624$). Graph 6 shows a positive correlation in the children's group, FAM 1° and the proportion of positivity being expressed in related units where the value for CL men is equal to 1. In the upper part of the graph there are subgroups rated by the principal types of cleft (CL, CLP, CP) and the probands'

sex. (F = women, M = men). In the lower part of the graph we give the cleft subtypes, for example, CL S = subtotal cleft lip, T = total CL (see Methods).

The above results have led us to the preliminary hypothesis, according to which CMV infection may exercise an aetilogical or modifying influence, which is why we regard as useful to perform more detailed tests for CMV (IgM, culturing) as part of preoperative tests and in the planning of yet another pregnancy (5).

Epstein-Barr virus (EBV)

The teratogenic effect of EBV is seen as rarely as its transplacental transmission.

The presence of antibodies against the capsid (VCA) antigen of EBV of type IgG was tested in 132 infants with clefts aged 6 weeks up to 6 months, in 133 mothers of cleft-affected children, and in 53 CP children aged to five years. In the children's group the lowest rate of positivity was seen in CLP, as was in the mothers also in titres of 1:80 and more.

The correlation coefficient between FAM 1° and the rate of positivity in subgroups rated by the degree of defect was highly negative, in children R = —0.824, in mothers in titres of 1:80 and more R = —0.749). The correlation between the incidence of EBV, CMV antibodies and HLA antigens are shown in Table 4.

A loss of positivity against EBV was found in CLP and CP, hence also the negative correlation to FAM 1°. This might be due to the lethality of cleft-affected phoetuses in the presence of maternal EBV infection (6, 7).

DISCUSSION

The purpose of our communication was to summarize briefly our theoretical assumptions and results of a study of cleft defects. We realize that no interpretation of the effect of "active factors" can be unambiguous and that the results require verification at other centres and for different populations. The complexity of human cleft research arises from the interpretation of aetiological, modifying and lethal factors, from interaction between active factors and from the laws of embryonic organogenesis. As such research requires large groups taken from different populations international cooperation is essential there.

SUMMARY

Prenatal factors influencing cleft lip (CL), cleft lip and palate (CLP) and isolated cleft palate (CP) may account for the development of the defect (aetiological factor), for a change in the degree or type of the defect (modifying factor), or for the death of the ambryo (lethal factor). Each active factor appears to act in all three directions, all be it at different ratios. This is used for analysis of groups of cleft-effected individuals registered at the Department of Plastic Surgery, Prague, from the catchment area of Bohemia.

Table 4. Relative proportion (total = 1) of factors suspected of participation in the teratogenesis of cleft defects in the general population (column 2), in the whole group of cleft-affected children or in their mothers (column 3) and in subgroups rated by the type of cleft defect (columns 4—9). CL_s — incomplete cleft lip, CL_t — total. CLP_m — monolateral complete cleft, CLP_b — bilateral. CP_d — hard and soft palate cleft, CP_m — soft palate cleft. Column 10 — correlation to FAM 1° — see the text. Column 11 — number of persons in the group. In lines: FAM 1° proportion of probands with cleft incidence in first-degree relatives (3). ABO-F — proportion of blood groups A and B in female probands, correlations to FAM 1° in this group (6). HLA antigens were tested in mothers of cleft-affected children (4). CMV — cytomegalovirus antibodies CFR (5), EBV — against BCA antigen of Epstein-Barr virus (7). Mothers 30+ — proportion of multiparae over 30 years of age in the group of multiparae. K_p — total coefficient of primiparae (firstborn) throughout the group (2). K_p FAM+ — proportion of firstborn with positive family history up to the coefficient of kinship of 1/16 (2).

1	2	3	4	5	6	7	8	9	10	11
Factor	Incidence in:		subgroups rated by cleft type						Correlation to	n
	Population	Group	CL_s	CL_t	CLP_m	CLP_b	CP_d	CP_m	FAM 1° R_s =	Group
FAM 1°	0.002	0.069	0.037	0.039	0.080	0.120	0.078	0.050	—	2770
ABO-F A	0.423	0.453	0.478	0.469	0.432	0.409	0.421	0.531	−0.859	1082
B	0.179	0.160	0.161	0.143	0.166	0.209	0.161	0.117	+0.931	1082
HLA A9	0.170	0.272	0.310	0.294	0.234	0.188	0.364	0.296	−0.815	147
	0.190									
A11	0.120	0.102	0.207	0.118	0.106	0.000	0.091	0.037	−0.814	147
	0.090									
B5	0.130	0.102	0.069	0.118	0.106	0.188	0.091	0.074	+0.708	147
	0.150									
B7	0.260	0.252	0.345	0.235	0.255	0.125	0.185	0.364	−0.758	147
	0.270									
B17	0.100	0.068	0.069	0.000	0.043	0.188	0.074	0.074	+0.737	147
	0.090									
B35	0.160	0.068	0.069	0.118	0.085	0.000	0.091	0.037	−0.822	147
	0.140									
CMV proband	?	0.450	0.362	0.441	0.560	0.514	0.320	0.273	+0.509	345
mother	0.620	0.593	0.568	0.615	0.591	0.667	0.579	0.500	+0.624	245
EBV proban	?	0.159	0.278	0.286	0.173	0.000	0.118	0.091	−0.824	132
mother	0.189	0.083	0.074	0.154	0.111	0.000	0.046	0.100	−0.749	133
Mothers 30 +	?	0.238	0.327	0.214	0.382	0.160	0.294	0.357	−0.383	252
K_p total	0.454	0.451	0.445	0.468	0.471	0.460	0.453	0.399	+0.324	3029
K_p FAM +	0.454	0.387	0.252	0.442	0.373	0.426	0.445	0.407	+0.356	696

In terms of cleft defect teratogenesis (aetiology, modification, lethality) a "protective influence" appears probable in female embryos, in blood group A (ABO system), in HLA antigens A9, A11, B35.

An "embryotoxic influence", i.e., an increase in all the three influences is seen in male embryos, regional influences, in the B and AB blood groups, in HLA antigens B17, in primiparae, in multiple pregnancies, in older mothers and in cytomegalovirus infections. The Epstein-Barr virus seem to increase, in particular, CLP embryo lethality.

The activity of the factors was rated by means of correlations between the cleft frequency in first-degree relatives and the frequency of the factor in six subgroups classified by the cleft defect subtypes, by means of interactions between two and more factors and by a study of the differences between male and female probands. A model of the treshold of CL and CP teratogenesis was proposed, a model in agreement with prenatal reciprocal equilibrium.

Key words: cleft lip and palate, protective factors, embryotoxic factors.

RÉSUMÉ

Influences étiologiques, modificatrices et létales chez la fente labiale et palatine

Černý, M., Fára, M., Hrivnáková, J.

Les influences prénatales chez la fente labiale (CL), chez la fente totale (CLP) et chez la fente palatine isolée (CP) peuvent mener à la genèse de la malformation (influence étiologique), à la mutation du degré ou du type de malformation (influence modificatrice), où à la mort de l'embryon (influence létale), Chaque facteur actif agit évidement dans tous les trois sens, mais en proportion différente. Ces faits sont utilisés dans les analyses des groupes de sujects avec les fentes, de la région de la Bohème, interceptés à la Clinique de la chirurgie plasticque à Prague.

"L'influence protectrice" — dupoint de vue de la tératogenèse des fentes (diminution d'influences étiologiques, de modification où de létalité) est vraisemblable chez le groupe sanguin A (système ABO), chez les antigène HLA A9, 11, B 35 et chez les embryons du sexe féminin.

"L'influence embryotoxique" est-à-dire augmentation des trois influences citées — s'est manifestée chez les embryons masculins, chez les influences régionales, chez les groupes sanguins B et AB (ABO système), chez l'antigène HLA — B 17, chez les primipares, chez les multipares, chez les mères plus âgées et chez l'infection cytomégalovirales. Le virus d'Epstein-Barr devrait augmenter la létalité de l'embryon avec CLP.

L'activité des facteurs a été évaluée selon les correlations entre la fréquence des fentes chez les parents de ler degré et entre la fréquence du facteur de six sous-groupes qui étaient triés d'après les sub-types de fentes, les interactions entre deuz ou plusieurs facteurs et l'observation de différences des sujets masculins et féminins. On a proposé le modèle de seuil de la tératogenèse CL et CP correspondant à l'équilibre réciproque prénatale.

ZUSAMMENFASSUNG

Ätiologische, modifizierende und letale Einflüsse bei einer Spaltung der Lippe und des Gaumens

Černý, M., Fára, M., Hrivnáková, J.

Pränatale Einflüsse bei einer Spaltung der Lippe (CL) einer Gesamtspaltung (CLF) und einer isolierten Spaltung des Gaumens (CP) können zur Bildung einer Defekts (ätiologischer Einfluss), zu einer Veränderung des Grades oder des Typs des Defekts (modifizierender Einfluss) oder zum Absterben des Embryos (letaler Einfluss) führen. Jeder aktive Faktor wirkt offentsichtlich in alle drei Richtungen, jedoch in verschiedenen Verhältnis. Dies wird zu einer Analyse der Gruppen mit Spaltungen an der Klinik für plastische Chirurgie in Prag aus dem Gebiet Böhmens ausgenutzt.

Ein „schützender Einfluss" vom Gesichtspunkt der Teratogenese der Spaltungen aus (Verminderung der ätiologischen Wirkung, Modifizierung oder Letalität) ist bei der Blutgruppe A (ABO-System), bei HLA Antigen A 9, 11, B 35 sowie bei dem weiblichen Geschlecht des Embryos wahrscheinlich.

Ein „embryotoxischer Einfluss", d.h. eine Steigerung der erwähnten drei Einflüsse, machte sich bei männlichen Embryos, bei regionalen Einflüssen, bei der Blutgruppe B und AB (ABO-System), bei HLA Antigen B 17 bei Primiparen, bei vielfacher Gravitat, bei älteren Müttern sowie bei einer Zytomegalovireninfektion bemerkbar, wobei der Epstein-Barr-Virus die Letalität des Embryos mit CLP wahrscheinlich steigert.

Die Aktivität der Faktoren wurde durch Korrelationen zwischen der Häufigkeit der Spaltungen bei Verwandten ersten Grades und der Häufigkeit der Faktoren in sechs Untergruppen bewertet, die nach den Subtypen der Spaltungen, den Interaktionen zwischen zwei und mehr Faktoren und der Beobachtung der Unterschiede zwischen den Probanden männlichen und weiblichen Geschlechts gebildet wurden. Es wurde ein Modell der Schwelle der Teratogenese bei CL und CP vorgeschlagen, das mit den pränatalen reziproken Gleichgewicht in Einklang steht.

RESUMEN

Las influencias etiológicas modificadoras y letales en la hendidura del labio y paladar

Černý, M., Fára, M., Hrivnáková, J.

Las influencias prenatales en la hendidura del labio (HC), en la hendidura total (HLP) y en la hendidura del labio aislada (HP) pueden resultar en un defecto (la influencia etiológica), en un cambio en el grado o tipo del defecto la influencia modificadora/o en la muerte del embrión (la influencia letal). Cada factor activo probablemente está activo en todas estas tres direcciones pero en una proporción diferente. Este hecho se utiliza para efectuar el análisis de los grupos con la hendidura registrados en la Clínica de la Cirugía Plástica en Praga, la región de Bohemis.

"La influencia protectora" desde del punto de viesta de la teratogénesis de las hendiduras (la disminución del efecto etiológico, él de modificación o de la letalidad) probablemente existe en el grupo sanguíneo A (el sistema ABO) en los antígenos de histocompatibilidad A9, 11, B35 y en el embrión femenino.

"La influencia embriotóxica", es decir el aumento de los dichos tres influencias apareció en los embriones masculinos, en las influencias regionales, en los grupos sanguíneos B y AB (el sistema ABO), en el antígeno B 17 de la histocompatibilidad, en, en primíparas, en los embarazos múltiples, en las madres más viejas y en la infección

por citomegalovirus; el virus Epstein-Barr propablemente aumenta la letalidad del embrión con HLP.

La actividad de los factores fué avalorada por medio de las correlaciones entre la frecuencia de las hendiduras en los parientes del primer grado y entre la frecuencia del factor en seis subgrupos clasificados según el subtipo de las hendiduras, por medio de las interacciones entre dos y más factores y por el estudio entre el propositus masculino y femenino. Los autores propusieron un modelo del umbral de la teratogenesis de HL y HP el que está en acuerdo con el equilibrio recíproco prenatal.

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EXTERNAL EAR RECONSTRUCTION

G. V. Kruchinski

The history of external ear reconstruction closely related to the achievements of plastic surgery as a whole is marked by years of search, failure and disappointment. Just recently a new revolutionary era began. After years of mistrust, if not downright rejection, the practical possibility of reconstruction of the external ear resembling in shape the natural form has become reality. This amounts to a major success for present-day plastic surgery.

The generally recognized works by Tanzer and Converse are seen as the most important acts of initiative to have laid the foundations for the present-day possibilities of external ear reconstruction.

In the following few years this initiative was augmented by some Soviet and especially foreign authors (1—5).

Thanks to all this, the basic stages, methods and techniques of external ear shaping are fairly well defined now. There is almost a universal agreement on the main source of the material best suited the planned reconstructed external ear: autogenous costal cartilage. At the same time, Cronin (4) and other surgeons who used plastics (Silastic) had gathered experience which, however, some other authors found a little disappointing. Now, more precision is required in determining the indications and the child's age, at which plastic surgery on the auricle should be performed.

Quite apart from the undisputed achievements already made, total external ear reconstruction continues being a very difficult and complex operation such as only few clinics in the world can afford. The results of plastic surgery on the auricle have yet to be made more stable. There are still a number of unresolved problems, different views of the methods and techniques of making the groundwork for the planned pinna. The method is still rather a complex one, the elevation of a block of costal cartilage is still a traumatic affair for the patient. In children aged 6—7 years such a block of cartilage is very small in size, hence the need to postpone treatment.

In our present communication we want to report our experience which in our view offers acceptable answers to the above listed questions. The point is to find a substitute for inborn subtotal defects of the external ear, defects often associated with unilateral incomplete development of the maxilla, mandible, facial and soft tissue (syndrome of branquial arche I and II), with hemofacial microsomia or atomandibular dysostosis. In such cases plastic operations on bones, mandibular elongation, and orthodontic treatment usually preceded the plastic operation on the external ear proper. In minor changes in the facial bones, only plastic surgery on the auricle was performed.

During the past seventy years (1971—1988), the following three main sources for the external ear cartilage were used at the clinic of the Byelorussian Institute for Post-Graduate Medical Education, in particular: re-inforced cadaverous ear cartilage, ear cartilage from living donors, and autogenous costal cartilage for the external ear structure to be modelled from.

Comparing those methods after the elapse of so many years it is possible to conclude with certainty that the method of shaping the structure from costal cartilage elevated from the patient himself or herself is the most reliable method despite all of its shortcomings.

It also appeared that a structure made of a living donor ear cartilage and stiffened with autogenous costal cartilage keeps slowly deforming until the reconstructed external ear loses its original satisfactory shape. The structure itself may occasionally break down thus causing deformation of the reconstructed external ear. Observations of this kind are described in our previous books.

METHODS

Step by step we improved the method of cartilage structure formation as well as the other stages of plastic surgery on the auricle. This method differs from previously described techniques. So far we have used the new method in a total of 28 patients. Six of them were found to have the syndrome of branquial arches I and II, the rest suffered from a congenital subtotal defects of the external ear in the form of microtia of the 3rd degree. The patients' age ranged from 7—25 years; 17 were of the male sex, 11 of the female sex, and 3 patients were over 12 years of age.

The size and shape of the planned auricle were determined according to an intact normal external ear using a wire form made of aluminium wire, 2—3 mm thick. The form was shaped according to the helix of a normal pinna so as to make its margin recede inside by 2—3 mm all over its circumference, i.e. to make the form smaller than the helix circumference roughly by the thickness of the skin and subcutaneous connective tissue covering the helix of a normally shaped external ear.

We bent the form from the crus helicis along its rising portion and further on according to its shape up to the caudal part of the helix (Fig. 1 a, b). The form was made for each patient separately one day before the operation, and sterilized similarly as the rest of the instrumentarium.

Using the wire form we marked on the skin the contours of the planned pinna making of the supraorbital and tragoorbital lines and also of the vertical line coinciding with the posterior margin of the mandible.

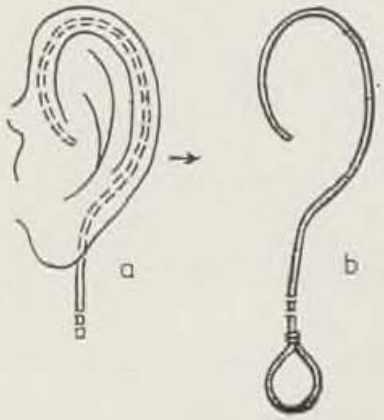

Fig. 1. Shape of normal external ear wire form made to fit outline. Dash line shows that the form should be smaller than the contours of the helix (a). Wire form is shown separately (b).

The incision for the implantation of the cartilaginous structure was made anteriorly from the rudiments according to Tanzer, at the same time placing the ear lobe in the correct position, or in the shape of a straight line. The skin above the rudiments and in all directions was mobilized on a much broader scale than the outlines of the planned external ear.

We proceded with an almost total removal of the cartilaginous rudiments transferring one of the most suitable portions under the skin to the site of the planned future tragus, making sure that it remained in contact with the underlying connective tissue.

Having severed the particular attachments, we packed the large subcutaneous capsule for the implantation of the external ear cartilage structure with wet gauze tampons to stretch the skin and add to the supply of skin.

Making an arch-shaped incision, if possible subperichondrially, we removed all of the cartilagenous portion of the 7th rib and, if needed, also the 6th rib, as a rule, from the side of the external ear defect.

From the cartilaginous portion of the 7th rib, which is longest of all the other ribs, we cut several stripes for different purposes. Thus along the external margin of the rib we cut a narrow and long strip (5—6 mm thick) of the cartilage for the planned helix (Fig. 2 a) rounding its edges and tapering them carefully to make the strip copy the shape of the wire form.

At the broad end of the remaining portion of the costal cartilage we cut 3 thin flat stripes about 3—3.5 cm long (Fig. 2 b) to use them subsequently for the flat structure of the planned external ear.

From the remaining portion of the cartilage we cut a thin stripe, bifurcated at one end, for the construction of anthelix and crura anthelicis (Fig. 2 c) and,

in addition to that, a thick (0.5 cm or more) slightly curved strip for shaping the hollow (Fig. 2 d). Should this amount of cartilage prove too little, an extra part of the cartilage of the 7th rib should be elevated for the purpose.

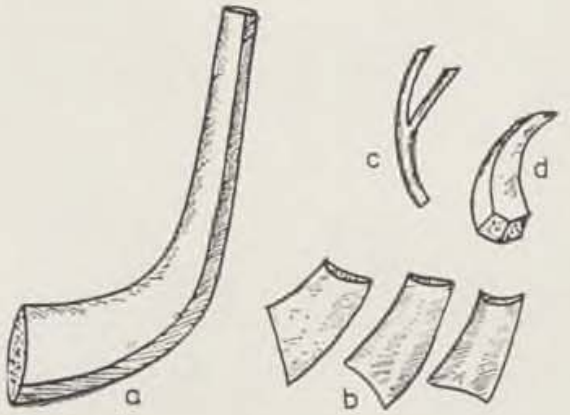

Fig. 2. Diagram of exposed 7th rib and elements excised from costal cartilage to form the auricular structure: a) strip for helix, b) flat and broad stripes for the structure groundwork; c) bifurcated strip for anthelix, d) thick, slightly curved strip for the concha.

Using the wire form we construct from the above-described stripes a cartilaginous structure containing elements distributed in the form of two succesive steps in the horizontal and vertical planes similarly as in a normally shaped external ear; for that purpose we place the broad flat stripes successively so as adjoin each other, placing on them stripe for the planned helix, bending in to the shape of the wire form and fixing the stripes to one another (Fig. 3 a).

The distribution of the flat stripes has a major role to play. The flat stripe was found to have a tendency to curve to the side of the damaged surface and vice versa. Hence we place transversely in the middle of the structure two flat stripes (1, 2) with one surface intact and facing the inside of the structure. In such a case, the stripes, in the process of subsequent inevitable deformation,

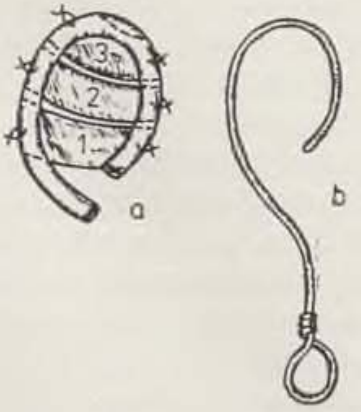

Fig. 3. Diagram of shaping the flat groundwork and the helix using (a) wire from (b) metal hooks and polyamide suturing material.

will curve towards the side of the underlying tissue, as a result of which the hollow will preserve its depth.

The first stripe (1) designed to keep the helix in the required position is fixed with metal hooks, and the rest of the stripes with polyamide sutures. In this way, the flat part of the structure with the contours of the helix is shaped to resemble the outline of a normal undeformed auricle (Fig. 3 a, b).

The structure should then be turned with the convex internal side up and fitted a stripe designed to give shape to the hollow (Fig. 4 a, b). This stripe ought to be thick enough and, if possible, broad enough (Fig. 4 b), and subsequently fixed with wire hooks.

The diagram shows a separate representation of this stripe as well as a cross-section of the structure with its elements (helix, flat groundwork and a stripe for shaping the hollow) localized in the horizontal and vertical planes in the form of two successive steps (Fig. 4 c).

Fig. 4. General view of the inside of the structure and (a) strip for shaping the concha in situ, (b) conchal strip shown separately, (c) diagram of cross-section of the structure with localization of constituent element in the form of two successive steps.

Then we turn the structure again placing the bifurcated strip for the anthelix on the external side of the structure and subsequently fix it together with the hollow-shaping strip to the flat groundwork and to each other. This is followed by a thorough check of the completed structure: we smooth the edges, remove exces cartilage and meticulously bend the hook ends (froms the inside of the structure).

We place the structure under the skin into the capsule on the site of the planned external ear localizing it according to the skin markings and fixing it to the underlying connective tissue by means of a few single catgut sutures. In cases where the earlobe was immediately placed in its proper posititon, we split its uper portion to attach it to the free caudal end of the external ear structure. In this way a good union with the whole reconstructed part of the auricle was achieved.

We close the wound leaving the drain in position and distributing the skin so as to cover the convex and concave elements of the relief of the auricle and the hollow. Wet gauze pads were used to press the skin down to the base.

The second stage of the plastic operation takes place in four-months time. It consists in the separation of the auricle thus formed and, should that be found, necessary, in correcting the position of the earlobe. The wound is

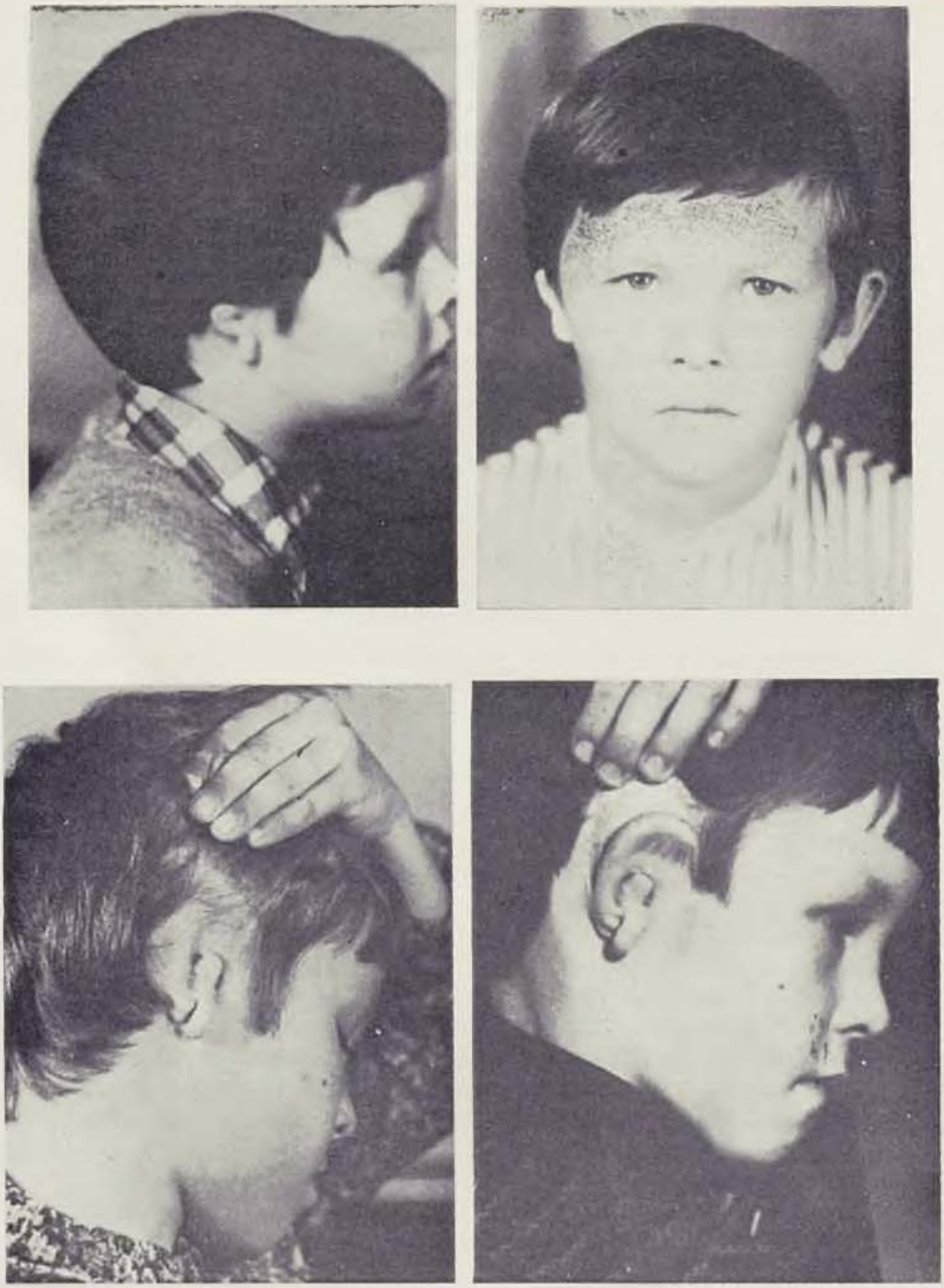

Fig. 5. (a—d) Patient L., 7.5 years, third degreee microtia on the right. State prior to treatment — profile and en face (a, b), stages of plastic surgery (c, d)

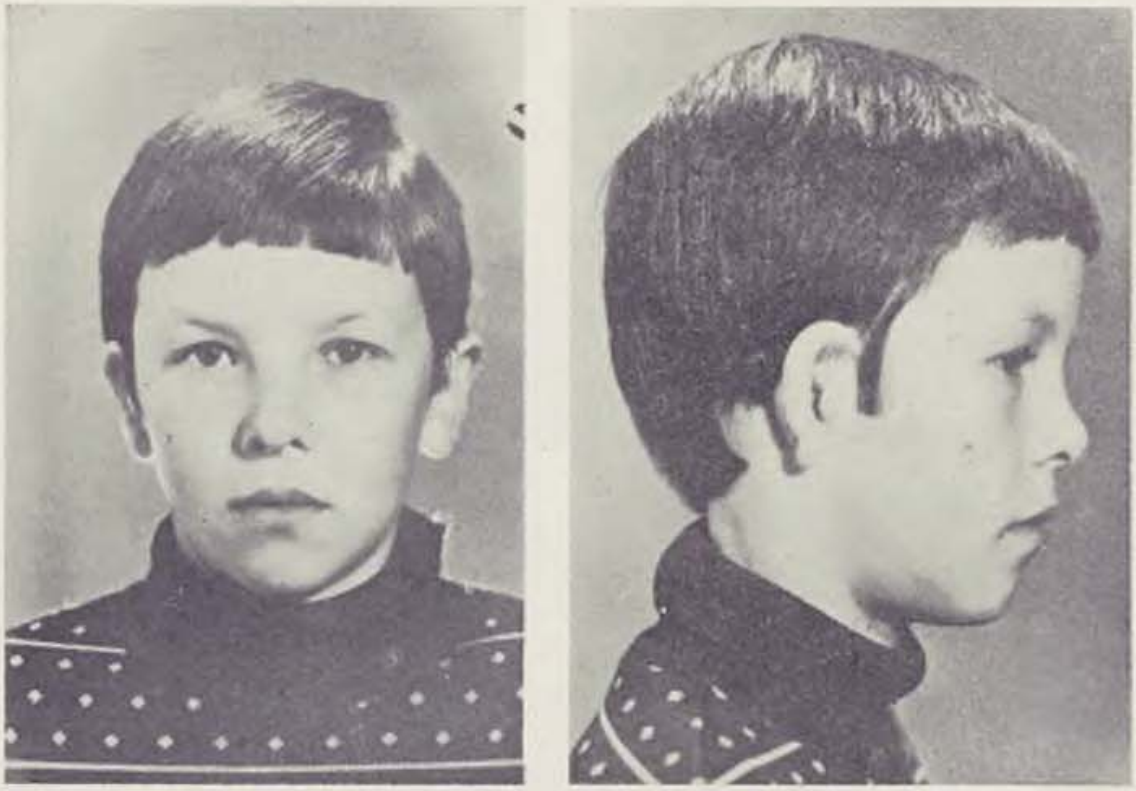

Fig. 5. (e, f) 3 years after end of plastic surgery — en face (e) and profile (f).

covered with a free skin transplant elevated from the chest next to the costal cartilage donor site.

The hollow and the tragus are shaped in the third stage. For that purpose a skin flap is excised at the site of the future hollow with the pedicle turned anteriorly to the site of the planned tragus, removing cicatricious and cartilaginous tissue from the wound. To form the tragus protrusion we double the skin flap to create a fold in the shape of a tragus, leaving a little bit of the rudimentary cartilage within its mass. We remove wedge-shaped portions from the free skin transplant to make it cover freely the bottom and the walls of the hollow. In this way, we give shape to the hollow of the acoustic meatus and the tragal projection.

RESULTS

All our operations ended successfully and the external ear gradually developed a distinct relief, stable shape and position.

The above described auricular structure provides a solid flat groundwork for the shaping of the helix, anthelix and concha which are situated in the horizontal and vertical planes in the shape of two successive steps similarly as in the cartilage of a normal undeformed external ear. This is an important circumstance as it helps to stabilize the shape and brings better aesthetic effect

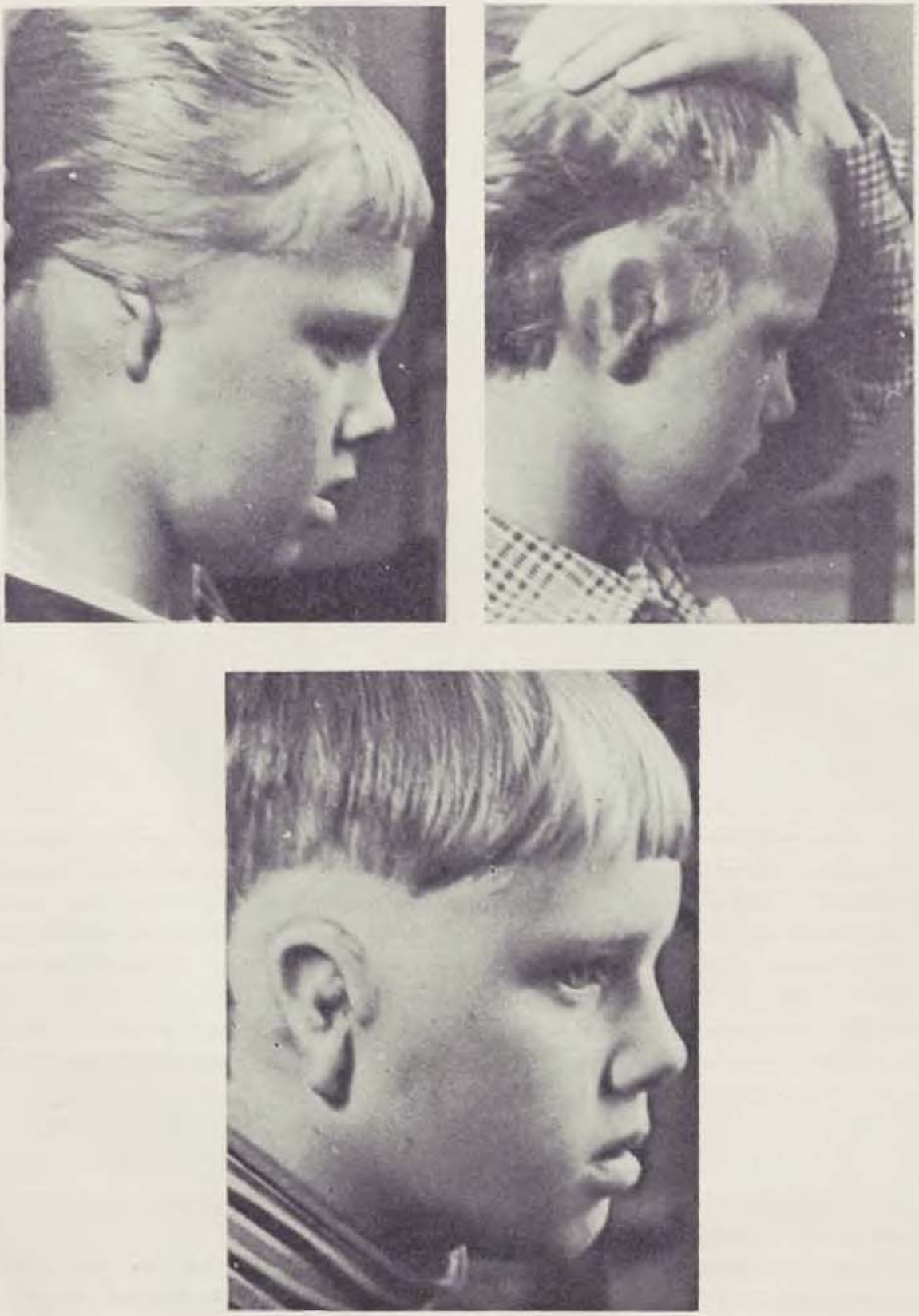

Fig. 6. Patient D., 7 years, third-degree microtia on the right. State before treatment (a), during treatment (b) and one year after end of plastic surgery (c).

of plastic surgery for external ear construction. In addition to that, there is no need to elevate a block of costal cartilage, which helps to simplify the operation and to go ahead with auricular plastic surgery in children aged 7—8 years.

There was evidence to show that a successful plastic operation for reconstruction of the external ear was possible in cases where rudiments of sufficient size had been preserved, for example, in the form of a cutaneo-cartilaginous tubercle providing enough skin to cover the newly developed structure.

Occasionally it proved necessary to include a corrective operation in order to improve the shape of the helix, to correct a protruding auricle, to remove unnatural skin fold on the inside of the auricle and so on.

The whole process of the plastic operation took about one year to complete with an interval of 3—4 months inbetween the individual stages.

Rare as they were, the complications included suppuration of the underlying cartilage, exposure of the metal hooks, detachment of part of the skin on the inner side following the transplantations or the shaping of the concha, etc. Despite those complications the treatment invariably ended in a success.

Examples of plastic surgery for external ear reconstruction in cases of third-degree microtia are given in Fig. 5 and 6.

CONCLUSIONS

1) External ear reconstruction permiting to attain a near-natural shape of the ear has become feasible despite the fact that only a few clinical centres can venture it.

2) Autogenous costal cartilage has been found the most reliable of all the known materials used for the construction of the auricular structure.

3) The proposed new method for external ear reconstruction involves the making of a flat cartilaginous groundwork on which to form the helix, anthelix as well as the concha situated in the horizontal, vertical and saggital planes in the form of successive steps similarly as in the cartilage of a normal auricle.

4) The new technique eliminates the need to elevate the block of cartilage from two ribs, hence is is less traumatic and better suited to go ahead with external ear reconstruction at a younger age (7—8 years).

SUMMARY

Plastic reconstruction of the external ear designed to achieve a near-natural shape is now a practical proposition even though only few clinical centres can afford to perform it. The present communication is based on experience in the practical application of this new otoplastic technique in 28 patients aged 7 to 25 years.

The method involves the making of an auricular structure on a flat cartilaginous groundwork on which to form the helix, anthelix as well as the concha situated in the horizontal, vertical and saggital planes in the shape of two successive steps similarly as in the cartilage of a normal external ear.

The proposed technique eliminates the need to excise the block of cartilage from two ribs, and for that reason is less traumatizing and better suited for going ahead with the otoplasty in children of younger age (7—8 years).

Given the present rate of incidence of auricular deformity and incomplete development of facial bones and jaws, osteoplasty precedes otoplasty.

Key words: reconstruction, external ear, subtotal congenital defect, wire form, costal cartilage, planes, cartilaginous structure, helix, anthelix, tragus, auricular concha.

RÉSUMÉ

Reconstruction du pavillon d'oreille

Kruchinskij, G. V.

Reconstruire la forme du pavillon d'oreille de telle façon qu'elle rapellerait la forme naturelle, c'est désormais la réalité, quoique accessible à quelques cliniques peu nombreuses. Cette communications est fondée sur le matériel rassemblé lors de l'application de la nouvelle méthode d'otoplastie chez 28 patients, en âge de 7 à 25 ans.

La méthode consiste en formation de la charpente à la base du cartillage plat, sur laquelle on forme, du côté supérieur, l'hélix et l'anthélix et, du côté inférieur, le creux, tout étant situé dans le plan horizontal, vertical et sagital en forme de deux gradins succédants, comme chez le cartilage du pavillon d'oreille normal.

La technique proposée élimine la nécessité d'enlèvement d'un bloc cartilagineux de deux côtes ce qui la rend moins traumatisante et ce qui permet d'effectuer l'otoplastie aussi chez les enfants (âgées de 7 à 8 ans).

Si la malformation du pavillon d'oreille est combinée avec l'évolution incomplète du squelette facial et de maxillaires, on exécute, dans le premier plan, les interventions ostéoplastiques, et seulement après la plastie du pavillon d'oreille.

ZUSAMMENFASSUNG

Rekonstruktion der Ohrmuschel

Kruchinskij, G. V.

Die plastische Rekonstruktion der Form der Ohrmuschel, die der natürlichen ähnelt, ist heute eine Tatsache, wenn sie auch vorläufig nur einigen wenigen Kliniken zugänglich ist. Das Material für diese Mitteilung sind die Erfahrungen, die bei der Anwendung der neuen Art der Otoplastik bei 28 Patienten im Alter von 7 bis 25 Jahren gewonnen wurden.

Das Wesentliche der Methode liegt in der Bildung eines Gerüsts auf dem flachen knorpeligen Grunde, auf dem man von oben die Helix und Anthelix formt und von unten eine Vertiefung, alles in der horizontalen, vertikalen und sagitalen Ebene in der Form zweier aufenanderfolgender Stufen wie im Knorpel der normalen Ohrmuschel.

Die vorgeschlagene Technik beseitigt die Notwendigkeit einer Abnahme eines knorpeligen Blocks aus zwei Rippen und ist daher weniger traumatisierend und gestattet auch, bei Kindern (im Alter von 7—8 Jahren) eine Plastik der Ohrmuschel auszuführen.

Beim gegenwärtigen Erscheinen von Defekten der Ohrmuschel und einer unvollständigen Entwicklung des Skelets des Gesichts und der Kiefer zuerst knochenplastische Operationen vorgenommen und erst dann die Plastic der Ohrmuschel.

RESUMEN

La reconstrucción del pabellón de la oreja

Kruchinskij, G. V.

Hoy es posible efectuar la reconstrucción de la forma del pabellón de la oreja el que se parece al pabellón natural aunque esta técnica puede ser realizada solamente en algunas clínicas. Este hecho lo muestran las experiencias obtenidas a base del empleo de un nuevo método de la otoplastica efectuada en 28 pacientes en la edad de 7—25 años.

El principio de este método consiste en la formación de un esqueleto sobre la base cartilaginosa, sobre la cual se forma desde arriba el hélice y antehélix y desde abajo una contavidad que están localizados en el plano horizontal, vertical y sagital en la forma de dos estrados sucesivos de la misma manera como en el cartílago del pabellón de la oreja normal.

El método propuesto elimina la necesidad de sacar el bloque cartilaginoso desde dos costillas y por eso este modo es menos traumático y asi hace posible efectuar la operación plástica del pabellón de la oreja en los niños (en la edad de 7—8 años).

En caso de los defectos contemporáneos del pabellón de la oreja y en casos de un desarrollo incompleto del esqueleto facial se efectuan, en el primer lugar, las operaciones otoplásticas y después ua plástica del pabellón de la oreja.

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UNEXPECTED GASTROINTESTINAL COMPLICATIONS IN SEVERELY BURNED CHILDREN

V. Poláček, L. Brož, J. Kripner, I. Bouška, E. Liška

INTRODUCTION

Extensive burn trauma is almost invariably accompanied by more or less serious gastrointestinal complications (1, 8, 7). There is evidence of involvement of the small intestine, reduced nutrient transport in the intestinal villi, reduced DNA synthesis in the mucosal cells, as well as other functional or organic disorders (4, 5, 11). Some of those disorders are rather inconspicuous and unexpected and consequently difficult to diagnose. In our report we wish to present one case of such complications co-responsible for a fatal course of a major burn trauma affecting a child patient. The difficulty of diagnosing childhood disorders is aggravated by the child's poor cooperation, impossibility to assess muscle contraction, resistance and tenderness in cases of abdominal skin burns, as well as by the fact that medication with analgetics, narcotics completely suppresses both subjective reactions and objective findings (3). Also, a number of laboratory test results tend to be affected by the underlying disease and concomitant complications frequently present, where especially wound, systemic or generalized infection may obscure an inflammatory abdominal emergency.

CASE REPORT

We present the case of a three and a half year-old boy who suffered II-IIIrd-degree burns involving 60 % of the body surface as a result of a stove explosion, apart from major inhalation injury.

The boy was a gipsy child from a socially underprivileged family. According to the family history the boy came from secundigravidity, and was treated for hyperbilirubinaemia phototherapeutically after birth. During his first seven months of life he was hospitalized five times, i.e. three times for dyspepsia (one case of toxic dispepsia?) and twice for obstructive

bronchitis. Also diagnosed were hepatopathy, pyelonephritis, anaemia and general hypotrophy. The patient also underwent an operation for right-sided inguinal hernia.

He was referred to the Burns Department from his regional hospital three and a half hours after the accident. On admission the child was in a state of shock aggravated by hypothermia (for reasons of cooling the affected body surface the boy was transported in wet sheets). On the other hand, the patient was relatively well secured for (venous system, cannulation, urinary catheter, intubation in the presence of spontaneous ventilation). During the transport, sanguinolent sputum was evacuated first from the endotracheal cannula and later from the laryngeal part of the burned mucosa. With regard to the state of severe shock, all that could be done on admission was placing a sterile dressing on the areas affected, introducing a tube into the stomach and placing the patient on a tempered air fluidized bed at the intensive care unit. Artificial pulmonary ventilation was initiated followed by comprehensive anti-shock and subsequent care consisting of infusion therapy, transfusion of plasma, blood and subsequently human albumin. Antibiotics in accordance with sensitivity, i.v. immunoglobulins, heparin, alpha-blockers, H2 receptor blocker, steroid hormones, vitamins and other drugs depending on the momentary needs and conditions of the child. Sufficient diuresis was restored 17 hours after the injury, and, judging by the clinical symptomatology, blood circulation became stabilized within 24 hours. During the first two days, relieving incisions on both arms and the trunk were necessitated by generalized oedema. What was first a discrete pulmonary finding worsened markedly on days 4 and 5, with an X-ray showing hyperaemia and infiltrates on the right as well as cardiac

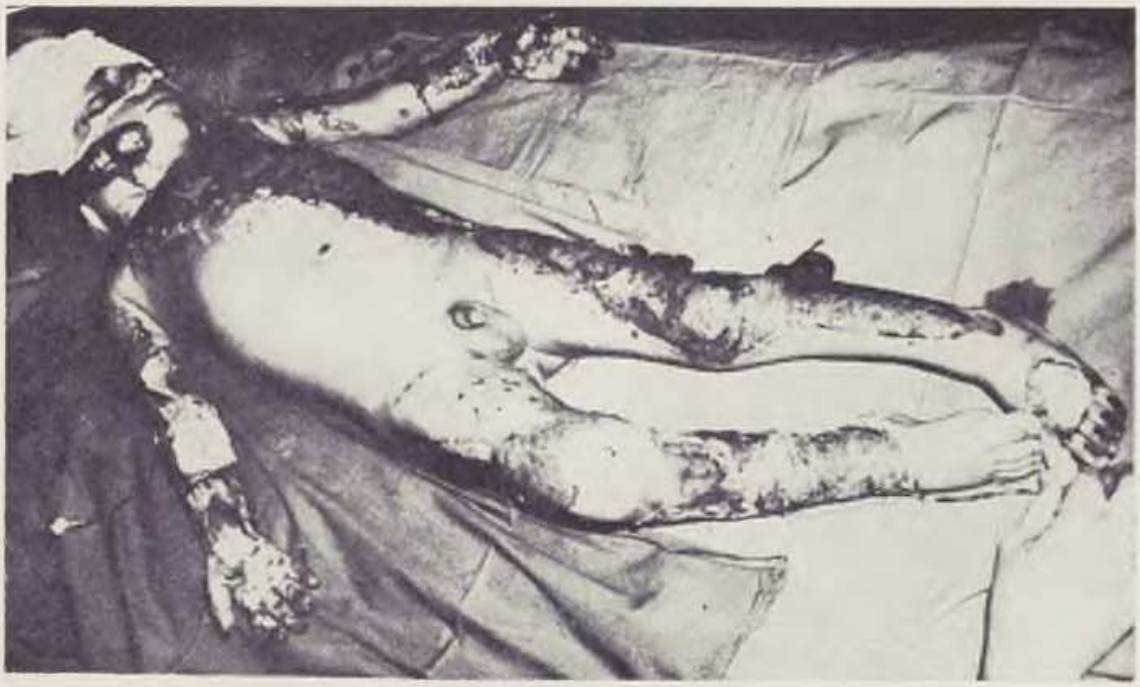


Fig. 1. General anterior view of the patient

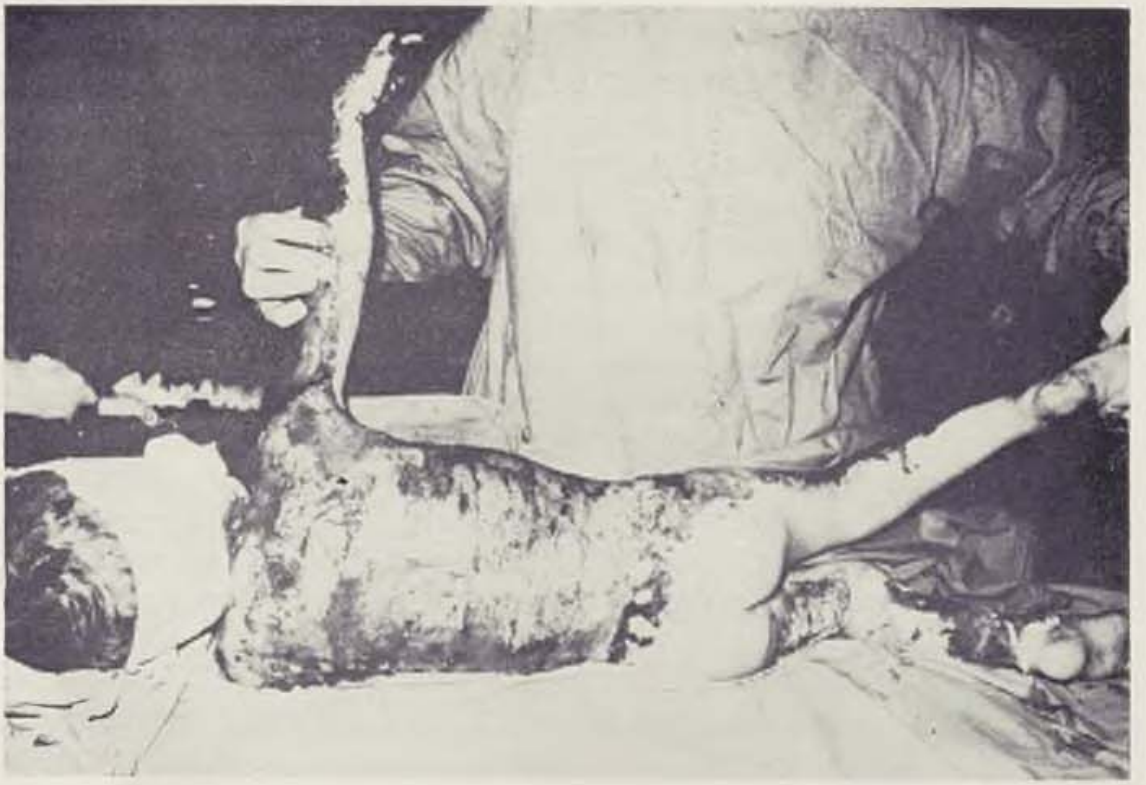


Fig. 2. General posterior view

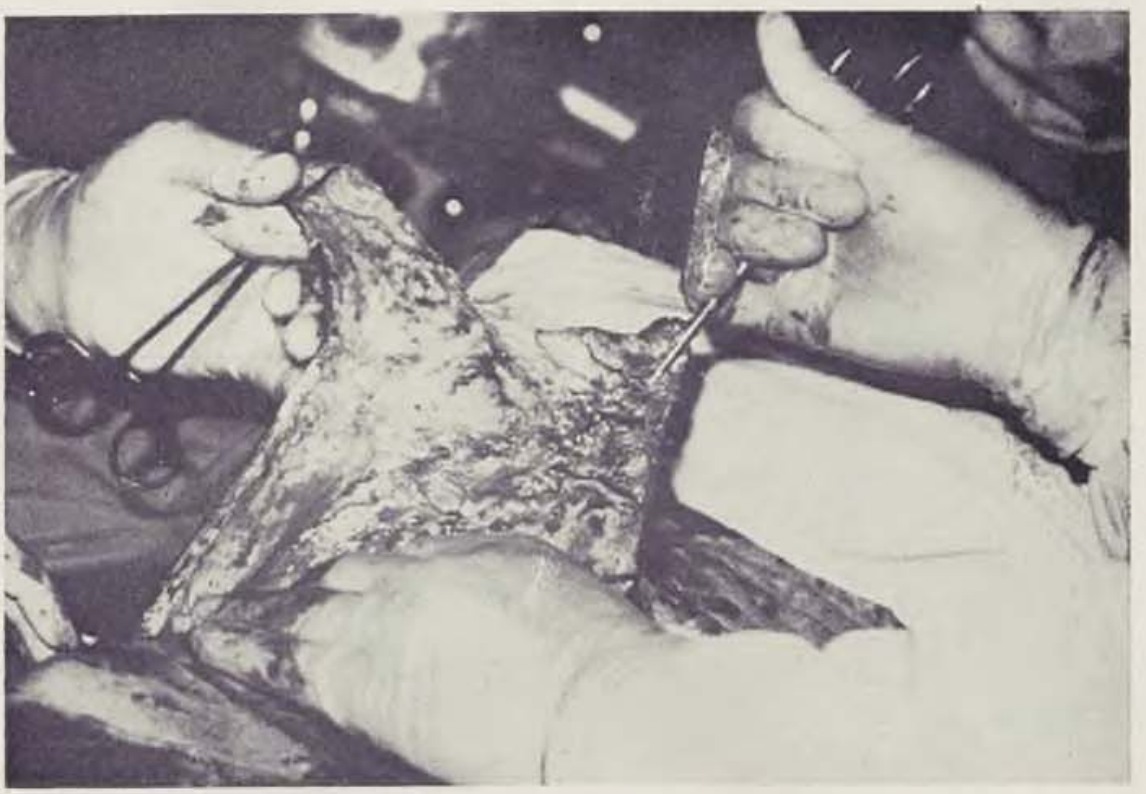


Fig. 3. Avulsion on the trunk

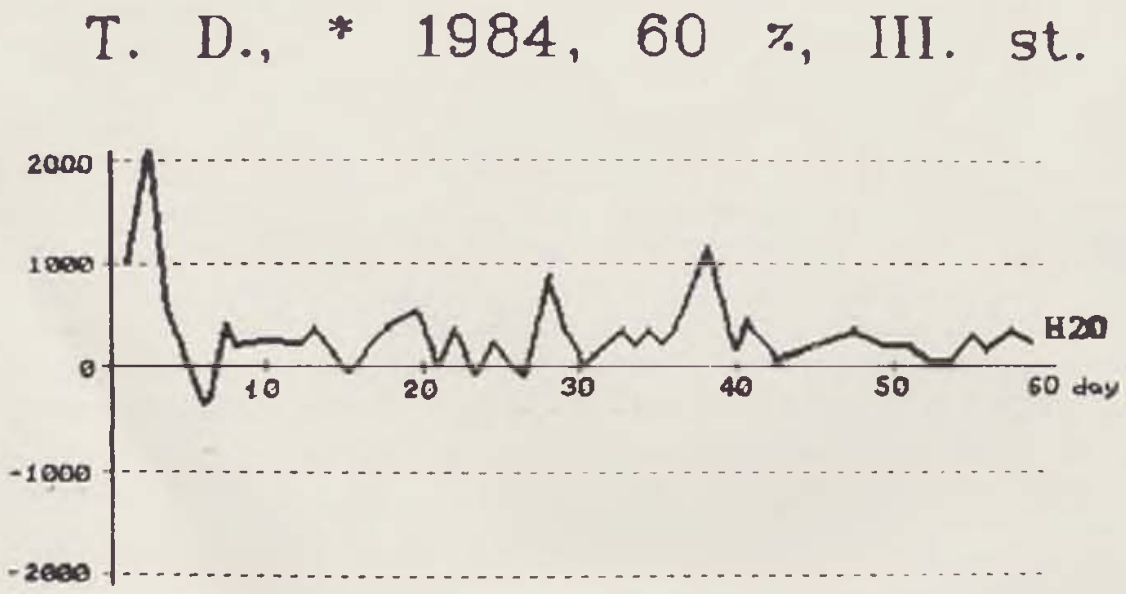
dilatation. On day 6 tracheostomy was performed for reasons of long term arteficial ventilation, and on day 8 following partial improvement in the pulmonary situation, necrectomy — avulsion on the trunk and lower extremities (Fig. 1, 2, 3).

The surgical procedures were performed while the boy was in a poor general condition. The wound areas were gradually covered with xenografts and starting from the day 13 till 37 with autographts and allographts. At that time the whole area was covered with the exception of 5% BSA. Also the general patient's condition showed improvement, especially between the days 32 to 51. Respiratory insufficiency following inhalation injury seemed to be over, blood gases, X-ray and also bronchoscopy revealed normal findings. Several attempts were made to re-start spontaneous breathing but each time such attempt was followed by manifestation of respiratory insufficiency, running temperature etc. There was a temporary restoration of bowel function. At the same time the alimentation per os was attempted. Nevertheless, the feeding tube had to be reintroduced in spite of repeated vomiting in a few days (Fig. 4).

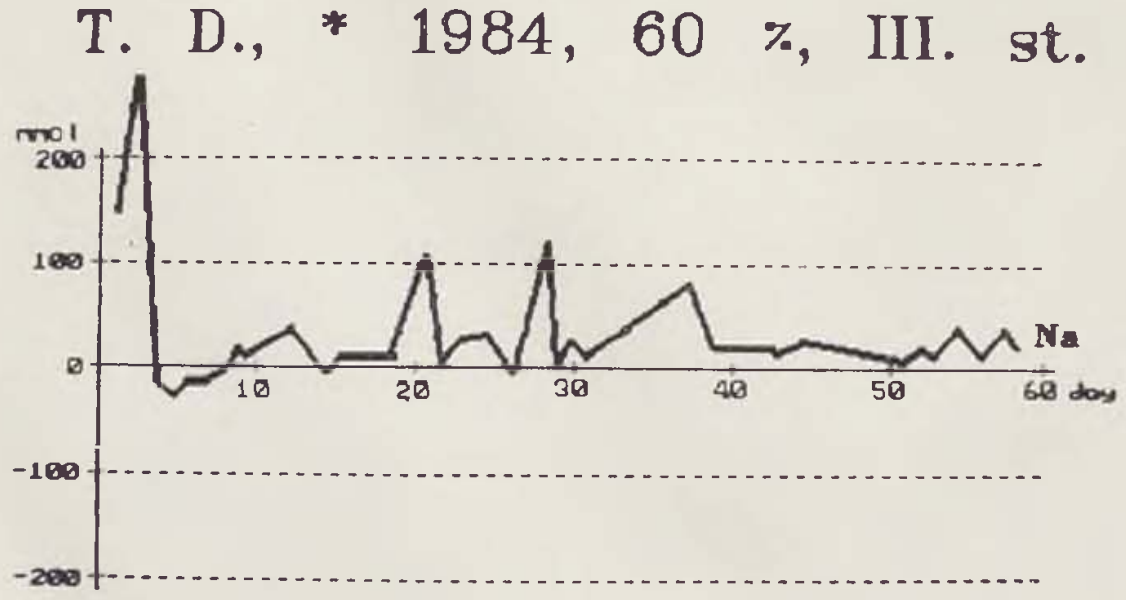
Throughout the period of hospitalization the boy had a tendency to tachykardia and hypertension with variable or even septic temperatures culminating in the region of 39—40 ^{0}C. A critical, albeit, transient situation, was noted on post-injury day 44. Following the application of steroids there was a temporary stabilization with signs of metabolic balance (Graph I,

Fig. 4. Plain picture of the abdomen (horizontal projection)

II, III). On day 48 there were elevated aminotransferases observed (AST 2,5, ALT 4,5) and transitory adynamic state. On day 53 we found what were already healed areas in a state of disintegration spread over some 15 % of body surface, on day 55 Astrup's tests showed a deterioration of pulmonary blood gas exchange, although this was still corrigible by changing the ventilation parameters on the Siemens — Elema set. However, on day 59 the quite discernible clinical and biochemical signs of respiratory insufficiency

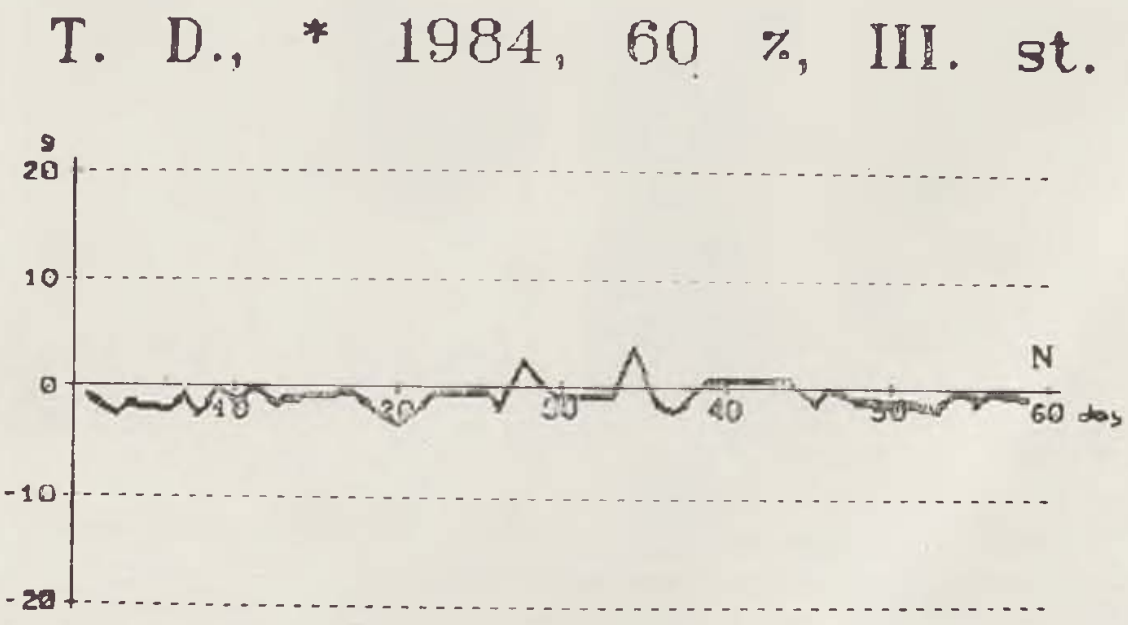


Graph I — H_2O balance



Graph II — Na balance

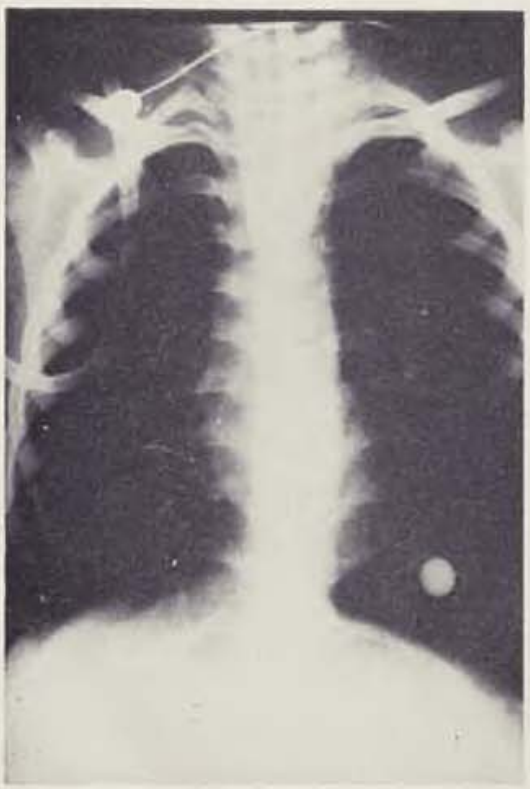
(pCO_2 14,0, pO_2 4,5 pH as much as 7,05) were no longer controllable and ultimately resulting in cardiac arrest. Bleeding into the gastrointestinal tract (blood in tube) and the discharge of a small quantity of mucous stools were noted in the terminal stage. Cardio-pulmonary resuscitation lasting 30 minutes proved unsuccessful.



Graph III — nitrogen balance

This case report is an example of multiple organ failure (cardio-pulmonary, renal, gastrointestinal failure and metabolic breakdown in a state of sepsis). The immediate cause of death (because of the dominant signs of asphyxia) was in our view due to bronchopneumonia, to which we also attributed the presence of leukocytosis found on day 51 and increasing until the point of death (14.25×10^3 .... 19.55×10^3), (Fig. 5, 6, 7, 8).

The post-mortem examination showed a deep thermal injury of the skin and subcutis on the trunk and extremities extending over 60% of the body surface. Autopsy and bacteriological examinations showed the presence of sepsis (in the spleen — Anterococcus, Staphylococcus pyogenes aureus) but also mycosis. There was also acute pulmonary oedema with the production of asphyctic or also hyaline membranes bordering the lumen of alveoli as seen in cases of "shock lungs", and also ulcerous post-intubation laryngitis. Quite unique were findings in the abdominal cavity where the subhepatic region showed signs of suppurative peritonitis more or less bordered by fibrin developing in this localization. The gall bladder revealed the presence of chronic productive cholecystitis with conspicuous connective tissue proliferation so that the gall bladder wall reached 3—4 millimeters in thickness.



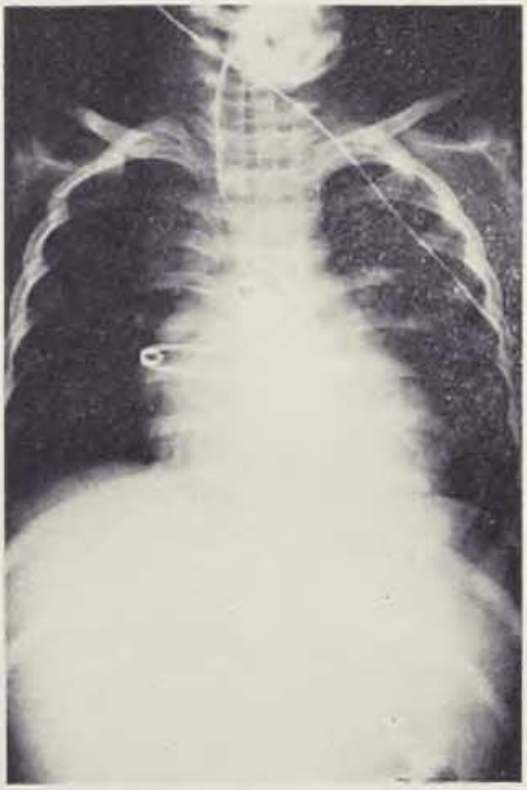
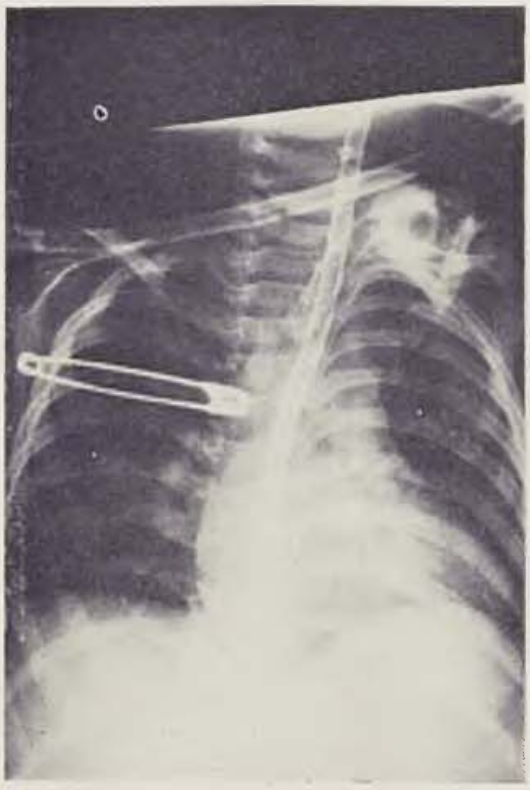


Fig. 5. X-ray of the heart and lungs — post-injury day 1. — Fig. 6. X-ray of the heart and lungs — post-injury day 29



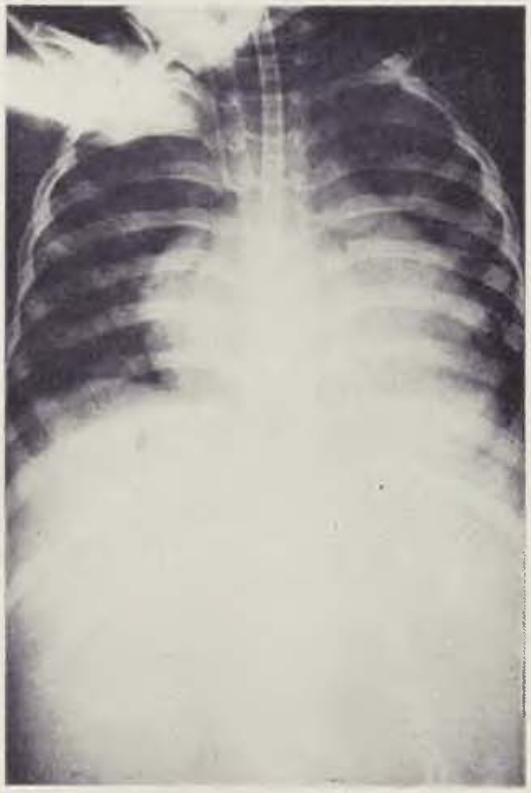


Fig. 7. X-ray of the heart and lungs — post-injury day 53. — Fig. 8. X-ray of the heart and lungs — post-injury day 57 (signs of progression)

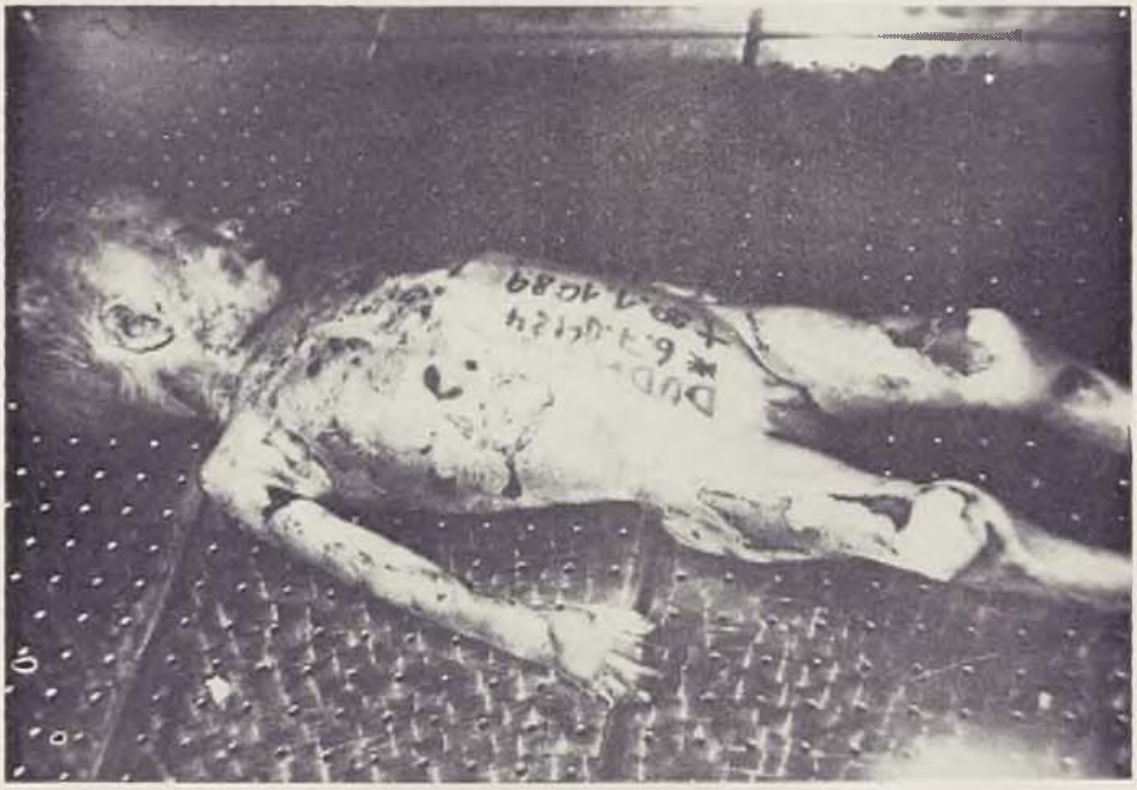


Fig. 9. General view of the dead child — following transplatations there are only minor areas on the lower extremities

Fig. 10. Post-mortem finding — chronic cholecystitis perforating into the liver

The gall bladder showed the presence of a mucopurulent inflammation leading to perforation into the liver and to an acute suppurative hepatitis in the vicinity. Histological signs of marked fibroproduction in the gall bladder wall indicated gall bladder involvement already at the time of the accident so that the septic condition following the burn only facilitated the further development of acute exacerbation (Fig. 9, 10, 11).

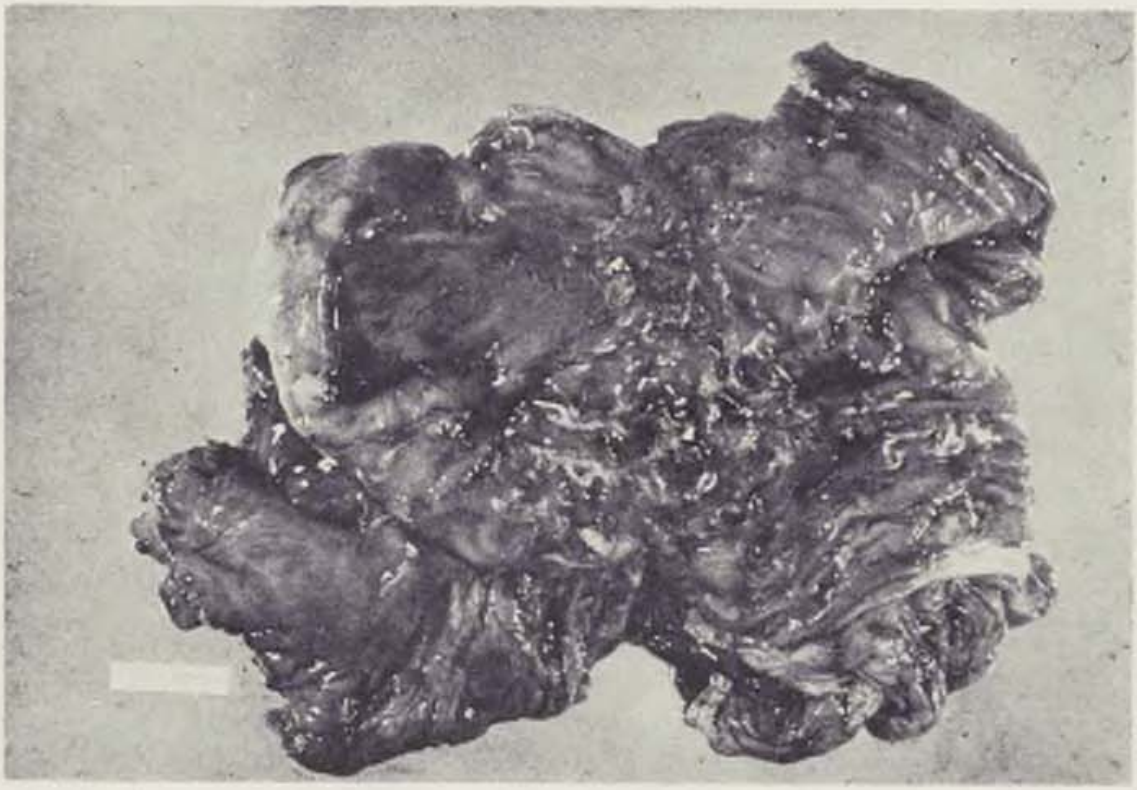


Fig. 11. Post-mortem finding — erosive gastritis

DISCUSSION

Extensive burn injury is often referred to as the gravest accident in human traumatology. The loss of skin as an important organ of the body results in critical local, but mainly, general changes (1). There is critical leakage of body fluids, proteins, electrolytes and hormones; immunocompetent cells and soluble factors of the immune system escape not only through the wound area but, in the presence of generalized oedema, also into the interstitium (2, 6, 9). A proportion of the fluids escape also into what is known the third space, i.e. hollow organs and body cavities, and this, in the case of the alimentary system, is one of the manifestations of early gastrointestinal involvement due to extensive burn trauma. The other frequent complications include adynamic states and bleeding. These are more likely to develop as stress complications, sometimes in re-

sponse to what are seemingly small insulti as well as later on in the acute period (2, 6, 11). Hepatic and biliary complications are, as a rule, discovered later on and tend to be less conspicuous (1, 8, 10). Factors influencing the development of these complications frequently include ischeamia, biliary stasis due to nutrition, or overpressure in the course of artificial pulmonary ventilation, sepsis and others (4, 10). Of late, it has been increasingly evident that ischaemia in the splanchnic region need not be the decisive cause of gastrointestinal complications (4, 5). Acalculous, and in our case even perforating, cholecystitis, is a rare complication with a potentially fatal effect on the clinical course of major burn trauma (10), in our patient, moreover, aggravated by his anamnestic load (including hyperbilirubinemia, repeated dyspepsia, hepatopathy as listed in our case report). Specific, potentially also lethal, late complications of the alimentary tract include pressure source or even tracheooesophageal fistulae due to the gastric tube counter-pressure in chronic artificial alimentation and endotracheal cannula in chronic arteficial pulmonary ventilation (11).

SUMMARY

The authors refer to the frequently difficult diagnosis of gastrointestinal complications of major burns, particularly in childhood, to weigh the factors co-responsible for the development of those complications and to stress the importance of the patient's history. They report on the case of three and a half year-old child to demonstrate the fatal course of a late diagnosed complications — perforating cholecystitis. Autopsy showed the chronic nature of the disease with acute exacerbation in the course of treatment. No case similar to this one has yet been seen at the Prague Burns Centre in any age category.

Key words: major burn trauma, acalculous cholecystitis perforating into the liver, gastrointestinal complications.

RÉSUMÉ

Complication inattendue des voies gastrointestinales chez l'enfant gravement brûlé

Poláček, V., Brož, L., Kripner, J., Bouška, I., Liška, E.

Les auteurs rappelent le diagnostic souvent difficile des complications concernant les voies gastrointestinales chez de vastes brûlures, surtout à l'âge enfantin. Ils prennent en considération les facteurs ayant part à la naissance de ces complications et ils mettent en évidence l'importance des donnés anamnestiques. Dans le cas présenté, ils démontrent l'enfant de 3,5 ans dont le diagnostic de la complication — cholecystite perforative — a été fatalement tardif. L'autopsie a prouvé le charactère chronique de la maladie, avec l'exacerbation aïgue au cours du traitement. Au Centre des brûlures à Prague, on n'a pas encore rencontré un cas pareil, dans aucune cathégorie d'âge.

ZUSAMMENFASSUNG

Eine unerwartete Komplikation des gastrointestinalen Traktes bei einem schwer verbrannten Kind

Poláček, V., Brož, L., Kripner, J., Bouška, I., Liška, E.

Die Autoren erinnern an die vielfach schwierige Diagnostik von Komplikationen des gastrointestinalen Traktes bei einem ausgedehnten Verbrennungsunfall und dies besonders im Kindesalter. Sie erwägen die Faktoren, die am Entstehen solcher Komplikationen beteiligt sind, und machen auf die Bedeutsamkeit anamnästischer Angaben aufmerksam. In der angeführten Kasuistik eines Kindes von dreiundeinhalb Jahren demonstrieren sie die fatal verspätete diagnostizierte Komplikation — die perforierte Cholezystitis. Der Leichenbefund erwies den chronischen Charakter dieser Erkrankung mit akuter Exazerbation im Verlauf der Behandlung. Ein ähnlicher Fall wurde am Prager Arbeitsplatz für Verbrennungen bisher nicht beobachtet und dies in keiner Alterskategorie.

RESUMEN

Complicación gastrointestinal inesperada en niños afectados con graves quemaduraf

Poláček, V., Brož, L., Kripner, J., Bouška, I., Liška, E.

Los autores recuerdan las dificultades que se presentan con frecuencia para establecer un diagnóstico en las complicaciones del aparato gastrointestinal en los casos de amplias quemaduras, en especial en los niños. Consideran los factores que inciden en el surgimiento de esas complicaciones y advierten sobre el significado de los datos de la anamnesia. En la casuística de un niño de tres años y medio demuestran el resultado fatal de una diagnosis de esas complicaciones por haber sido establecida con tardanza: colesistitis perforadora. La autopsia comprobó el carácter crónico de esta dolencia con exacerbación aguda durante su tratamiento. En la praguense clínica de quemaduras no ha sido registrado ningún caso similar en ninguna de las categorias de edades.

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SURGICAL RELOCATION OF RETRACTED EPONYCHION

B. F. Alsbjörn, P. Basse

INTRODUCTION

Burn wound contracture of the dorsal side of fingers often leads to an exposure of the proximal growth zone of the nail. This vulnerable part of the nail can be recovered by a surgical procedure, whereby the retracted and exverted eponychion is relocated. This procedure was first described in 1985 (Alsbjörn et al.). Until now 50 fingers on 31 patients have been operated. Clinical results are reported and commented on.

MATERIAL AND METHODS

50 fingers on 31 patients were operated 6—12 months after the initial burn wound was well healed. Only local anaestetic without noradrenaline was used. The line of incision is illustrated in Fig. 1. The distance between proximal line of incision and the edge of the retracted nailfold is 5—10 mm. Two triangles are excised from the distal part of the incision line. A gentle mobilization distally of the nailfold is performed with inversion of the nailfold. If scarious subcutaneous tissue interferes with the mobilization, it is incised. The relocation and inversion of the nailfold is continued until the triangles are closed. Undermining of the nailfold or total relocation/inversion is avoided as it will compromize the blood flow to the nailfold leading to necrosis. Instead, a subsequent contracture of the cresentshaped scar will, in due time, secure a proper relocation/inversion of the nailfold. Finally, the revealed skin defect is covered with a full thickness skin graft kept in situ by interrupted 6—0 nylon sutures. The locked triangles are also sutured by 6—0 nylon. Sutures are removed on day 8—10.

RESULTS AND DISCUSSION

The predominant reasons for surgical intervention were as follows

— soreness
— nail catching by dressing/undressing leading to
— bleeding and
— infections
— cosmetic reasons

At least 6 months after healing of the initial burn wound must elapse, as burn wound contractures proceed for at least this time. 1—4 fingers were operated per surgical seánce.

1 case (finger) had a partial necrosis of the relocated nailfold. This necrosis was, within doubt, due to a too radical relocation/inversion of the nailfold during the surgical procedure.

Fig. 1. Line of incision. Two triangles are excised at the distal parts of the incision line

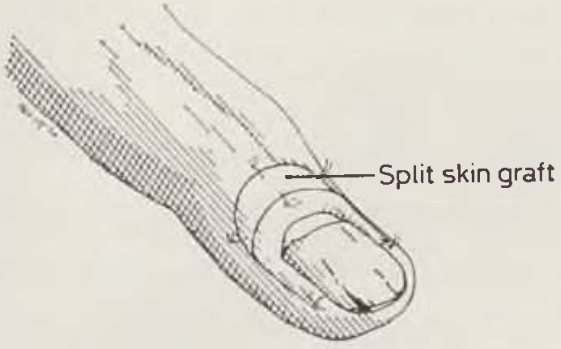

Fig. 2. Insertion of a full thickness split skin

3 cases had insufficient relocation of the nailfolds as judged by clinical evaluation 6 months after relocation procedure. In all three cases, a thin split skin was used. Shrinkage in such thin skin transplants is a well known phenomenon and as such have re-retracted the nailfold.

The remaining 47 cases had optimal surgical results as a normal nail growth was observed after the intervention. None of these 47 cases needed reoperation (observation time of at least 6 months).

SUMMARY

Burn wound contracture of the dorsal side of fingers often leads to retraction of the nailfold with exposure of the growth zone of the nail. Disfigured nails, extremely annoying to the patients, are the results. A surgical procedure and clinical results of 50 cases (fingers) are reported and commented on.

RÉSUMÉ

Réparation chirurgicale de l'éponychion déplacé
Evaluation clinique à propos de 50 cas

Alsbjörn, B. F., Basse, P.

La contracture au côté dorsal des doigts, séquelle de brûlures, mène souvent à la rétraction du recouvrement cutané au dessus de l'ongle, avec la dénudation de la zone de croissance de l'ongle. D'ici résultent les ongles déformés, génant fort la personne atteinte. La description des interventions chirurgicales avec les commentaires sont données, ainsi que les résultats cliniques de 50 cas (doigts).

ZUSAMMENFASSUNG

Der chirurgische Ersatz eines verschobenen Eponychions — Klinische Bewertung von 50 Fällen

Alsbjörn, B. F., Basse, P.

Eine Kontraktur der Wunden der dorsalen Seite der Finger nach einer Verbrennung führt sehr häufig zu einer Retraktion der Hautüberdeckung über dem Fingernagel sowie zu einer Entblössung der Wachstumszone des Nagels. Die Folge sind verformte Fingernägel, was für den Betroffenen äusserst unangenehm ist. Es werden die chirurgischen Eingriffe und klinischen Ergebnisse in 50 Fällen (Fingern) beschrieben und kommentiert.

RESUMEN

Sustitución quirúrgica de eponychion desplazado. Evaluación clínica de 50 casos

Alsbjörn, B. F., Basse, P.

La contracción de las heridas causadas por quemaduras en la parte dorsal de los dedos conduce a menudo a una retracción de la cobertura cutánea sobre las uñas y a la deskorbertura de la zone de crecimiento de las uñas. La consecuencia

es la deformación de las uñas extramadamente desagradable para el paciente. Se descríben y comentan las intervenciones quirúrgicas y los resultados clínicos de 50 casos (dedos).

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REVASCULARIZATION OF AVASCULAR SPONGY BONE AND HEAD OF THE FEMUR IN TRANSPLANTATION OF VASCULAR BUNDLE
(An experimental and clinical study)

M. G. Divakov

The development of methods of controlled revascularization of bones by means of transplantation of vascular tissue complexes in the treatment of aseptic necrosis of unhealed fractures and false joints of bones and their introduction into clinical practice (1—4, 6) called for thorough investigation into the mechanisms of revascularization of bones after similar operations. Qualitative evaluation of the condition of the formed intraosseous vascular network after transplantation of a vascular bundle is not adequate to ensure complete and objective evaluation of revascularization of bones and comparison of this operation with the traditional tunnelization.

To solve this problem, an experimental study was carried out to explore revascularization of avascular spongy bones with morphometric evaluation of the formed intraosseous vascular network.

MATERIAL AND METHODS

Thirty-four adult mongrel dogs of both sexes, 2—10 years old, body mass 15—35 kg, were used as experimental animals. A total of 66 experiments were carried out.

In the first series of experiments (18 dogs), the vascular bundle was transplanted into a massive, free, cylindrical autotransplant (replant) of spongy bone, cut out from the distal epiphysis of the femur by means of a concave cylindrical cutter. In the second series (20 dogs), the vascular bundle was transplanted into the head of the femur 2 months after its avascular disease had been modelled. When modelling this condition, the capsule of the hip joint, the synovial membrane and the whole ligament of the femoral head were incised circularly and the formed cavity in the neck of the femur was packed with paraffin.

In all cases, the subcutaneous artery of the hind leg with two concomitant veins isolated atraumatically along with the surrounding perivascular tissues served as the vascular bundle. The distal end of the vascular bundle was ligated.

Analogous operations, however without transplantation of the vascular bundle, were carried out in the control series (8 experiments to the first series, 20 experiments to the second series).

Times of observation: 2, 3, 4, 6, 12, 16, 24, 48 and 72 weeks. Methods of examination: clinical, roentgenological, histological, morphometrical. After termination of the examinations, the animals were killed under anaesthesia and infusion with a mixture of Indian ink and gelatin was performed. Separated bones were examined roentgenologically, fixed, decalcified and embedded in celloidin. Slices, 15—20 μm thick, were stained with haematoxylineosin and picrofuchsin according to van Gieson. Clarified slices, thickness 100—150 μm, were prepared for the study of the state of blood circulation.

Morphometric examination of the preparations (15—20 μm) was carried out by the planimetric method using an ocular net. The area of arteries, veins, vessels of the microcirculatory bed and the total area of vessels were determined in each preparation. Subsequently, the coefficients of arteries (AC), veins (VC), microcirculation (MC) and total vessels (TC) were established. Each of these coefficients was a relative quantity obtained by dividing the area of the corresponding vessels by the area of the lumen of the canal. Statistical evaluation of the obtained results was carried out on a "Sport-88" personal computer using the methods of variation statistics with dispersion analysis (5).

RESULTS AND DISCUSSION

In the first series of experiments, revascularization of part of the spongy replant was established 2 weeks after transplantation of the vascular bundle. The artery and the veins of the vascular bundle with their branches were found in the drilled canal. The structure of the vessel walls was normal. Intervascular spaces were filled with loose and solid connective tissue and there was a great number of cysts.

After a period of 3 weeks, we observed revascularization of the whole spongy replant due to the growth of vessels into the interstitial spaces from the vascular bed of the perivascular tissues from the centre to the periphery and the growth of vessels from the vascular net of the maternal bed from the periphery to the centre (Fig. 1, a). Distinct anastomoses between the newly formed vascular network and the vessels of the surrounding spongy bone as well as unification of the replant with the maternal bed by means of bony beams occurred after 4 weeks.

Twelve to 24 weeks and more after operation, the revascularized replant was gradually resolved and replaced by newly formed bone. The structure typical of the organ was restored. The walls of the veins became thinner in connection with the change in their function.

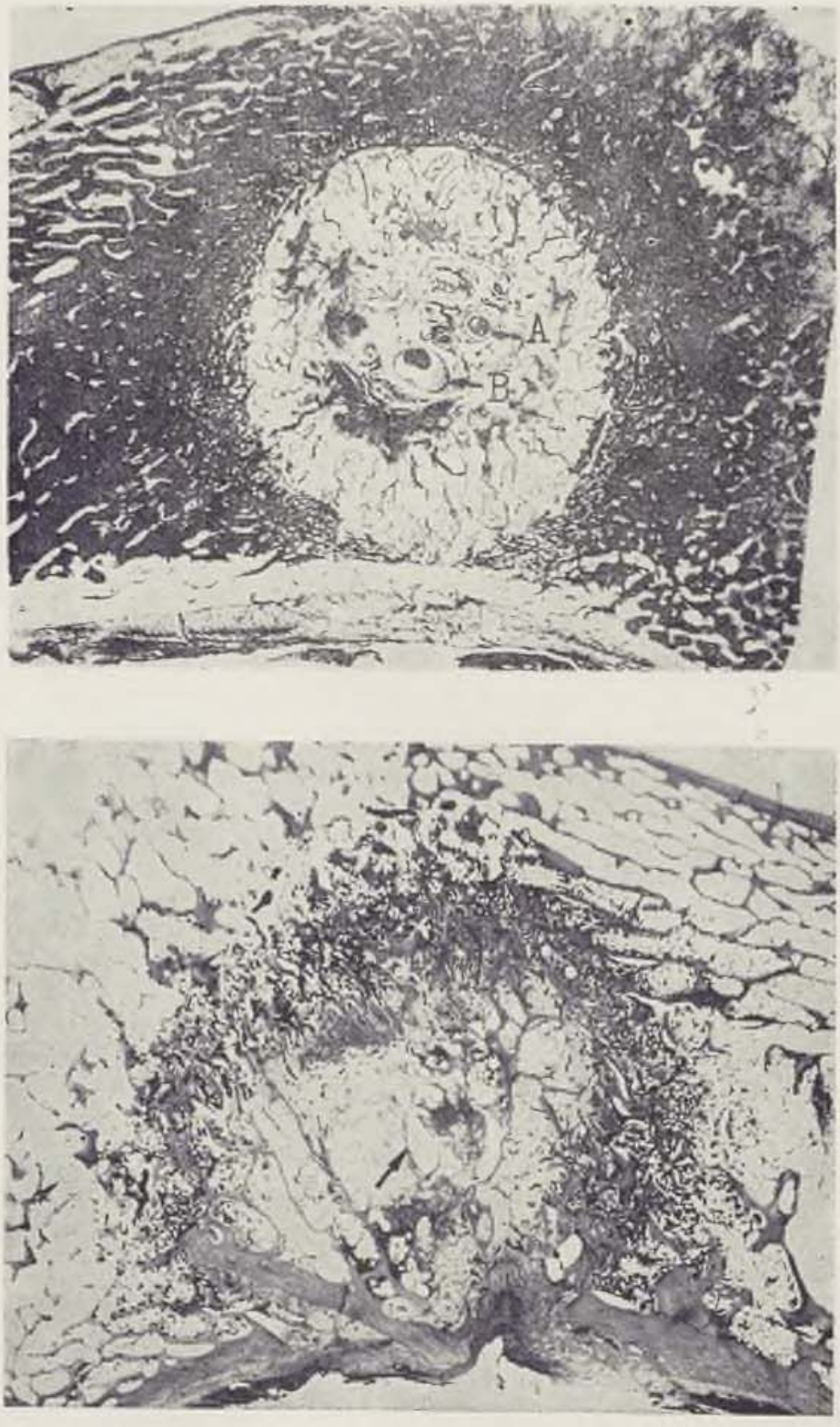

Fig. 1. Histotopograms of the distal epiphysis of the femur 3 weeks after operation (instillation with Indian ink, magn. 5×). a — revascularized spongiosa replant after transplantation of vascular bundle (A — artery, V — vein). Roentgenogram, b — control. Revascularization from the side of the maternal bed. A large number of cysts in the region of the canal (arrow). Haematoxylin-eosin staining.

In the control, revascularization was observed of the external layers of the spongy replant due to vessels growing into them from the bony bed (Fig. 1b). There was a large number of cysts of various sizes in the region of the drilled canal. By the 4th week, the dystrophic changes increased, the bony beams disintegrated, but the zones of revascularization became larger along the periphery. By the 24th week, the structure of spongy bone was restored as a result of complete absorption of the replant and its replacement by newly formed bone.

Second series of experiments. Four weeks after transplantation of the vascular bundle into the head of the femur, reactive processes in the form of bone production in the spongy substance were observed along with the dystrophic changes. In the lumen of the canal, we observed the transplanted vessels (arteries, veins, their branches and vessels of the microcirculatory bed) surrounded by solid and loose fibrous connective tissue along with muscle tissue bundles which were subject to degeneration at later stages. The microcirculatory network of the head of the femur included sinusoids and tissue cysts as a sign of compensatory processes.

By the 12th week following operation, the zones of newly formed bone became larger, especially in the vicinity of the vascular bundle, and revascularization of the head of the femur continued. After 24 weeks (Fig. 2, a), reconstructive processes could be observed in the femoral head, which conditioned increased X-ray density due to superposition of the newly formed bone on the old osteocyte-lacking bone trabeculae. The structure of the microcirculatory network approached the normal.

Forty-eight to 72 weeks post operation, the structure of the spongy substance of the femoral head was almost normal. There were powerful osteotrabecular formations and vascular network starting from the perivascular tissues around the transplanted vascular bundle. The walls of the veins became thinner, the tissue cysts were preserved. In cases where deformation of the femoral head had taken place, its shape remained unchanged regardless of transplantation of the vascular bundle. In these cases, the lack of correspondence between the articular surfaces in the hip joint enhanced the process of dystrophic changes and development of arthrosis.

In the control, the histological results 4 weeks after operation were in keeping with the dystrophic changes resulting from the disturbance of microcirculation in the head of the femur. The lumen of the drilled canal was filled with loose fibrous connective tissue exhibiting tissue cysts. Twelve to 24 weeks later, the dystrophic changes in the femoral head increased, reconstructive processes set in, foci of rarefaction appeared and so did the cysts which were particularly numerous in the zone of the drilled canal, i.e., the histological data corresponded to arthrosis deformans (Fig. 2, b). Progressive arthrosis deformans was found 48—72 weeks later. The haemocirculatory network was irregular due to the presence of a large number of tissue cysts with predominance of vessels of the microcirculatory type.

According to the results of morphological analysis and the comparison of experiments of the first and second series with the control (the results

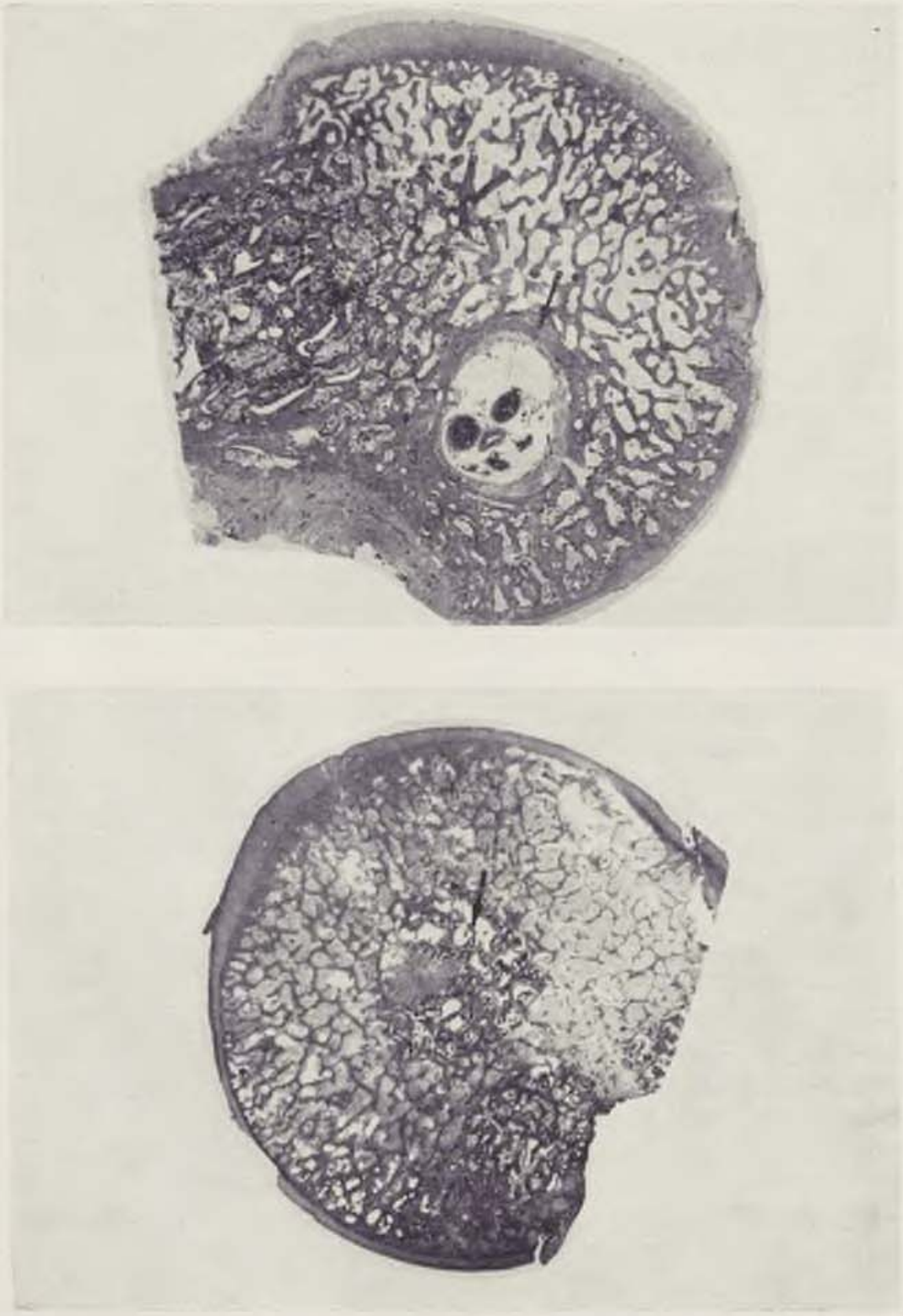

Fig. 2. Histotopograms of femoral head 24 weeks after operation (staining with haematoxylin-eosin, instillation with Indian ink). a — revascularization of the femoral head and formation of osteotrabecular structures (arrow) around the vascular bundle. (Magn. 7×); b — control. Formation of a large number of cysts (arrow) in the region of the drilled canal. (Magn. 6×)

were pooled as they were almost identical in each of the series of experiments) the study of the processes of revascularization of spongy bones revealed a number of certain regularities (Fig. 3). The increase in CA by the 4th week in the first series of experiments is connected, in our

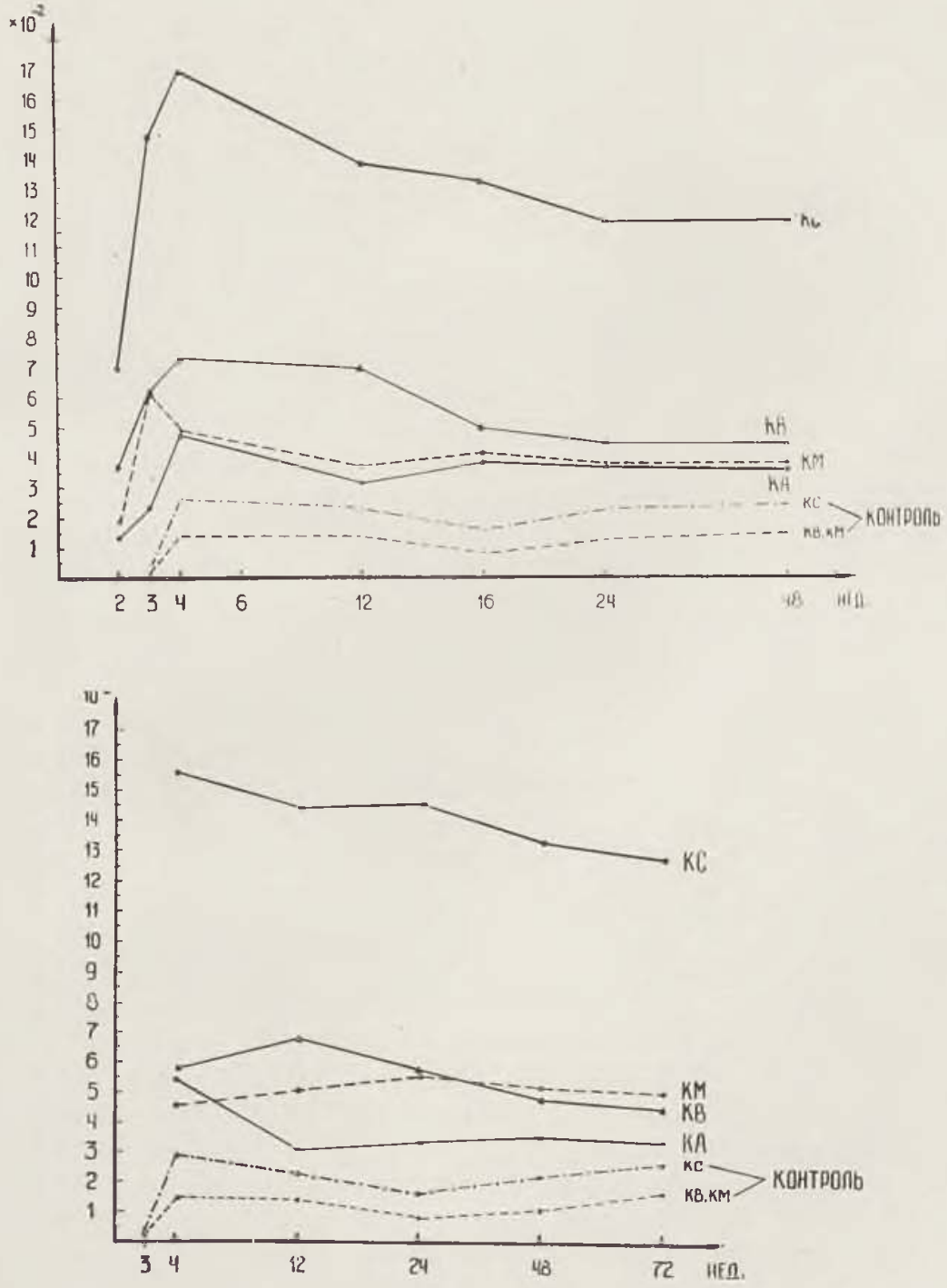

Fig. 3. Changes in the vascular coefficients after transplantation of the vascular bundle and tunnelization (control).
$p < 0.001$; a — series I; b — series II

opinion, with the increase in the capacity of the arterial vessels as a result of complete liquidation of the syndrome of pain following operation, formation of new vessels and increased load on the limb. The increased arterial blood supply induces an increase in VC which is connected with the necessity of more active venous drainage of the revascularized spongy replant. Revascularization of such a replant is finished after 3 weeks, which is manifested by a maximal increase in MC but the given index decreases in proportion with the quantity of anastomoses of the newly formed vessels with the vasculary network of the maternal bone and is stabilized by the 10th to 12th week. As a result of formation of anastomoses between the transplanted vessels and the vessels of the maternal bone, AC decreases as the drainage of blood improves. Complete normalization of the blood flow sets in by the 16th week, which is confirmed by the stabilization of all the coefficients.

In the second series of experiments, AC behaves as in the first series, while VC achieves its maximum by the 12th week, which is connected with the difficult blood drainage due to impaired nutrition of the femoral head. The processes of revascularization continue until the 12th to 16th week, which is confirmed by the increase in MC lasting until then. No considerable decrease in MC occurs in the later periods of observation since the formation of anastomoses between the vessels of the microcirculatory bed and the vascular network of the femoral head is hampered due to the previous impairment of its blood supply. The changes in TC as a whole reflect the process of revascularization of the bones in general, but not so explicitly as the AC, VC and MC.

On the basis of dispersion analysis, we established a fairly high and reliable degree of the effect of an organized factor (transplantation of the vascular bundle) on the vessel coefficients. In the first series of experiments, the index of strength of the effect for AC was 51.7 % ($p < 0.001$), VC — 24.3 % ($p < 0.01$), MC — 28.6 % ($p < 0.01$), TC — 37.2 % ($p < 0.05$; in the second series it was for AC — 82.5 % ($p < 0.001$), for VC — 61.7 % ($p < 0.01$), for MC — 22.9 % ($p < 0.05$), for TC — 42.5 % ($p < 0.01$). The highest index of strength of the effect is typical for AC which testifies to the impossibility of independent formation of arteries.

In the control series, the observations showed a VC equal to zero in all periods. The indices of VC and MC increased by the 4th week, but decreased slightly afterwards and remained at the same level. The low value of VC is connected with the impossibility of independent production of normal veins in the drilled canal. New vessels in the lumen of the canal appeared because of their growing into it from the surrounding tissues to a small depth while the canal was filled with solid and loose fibrous connective tissue. This process was considerably slowed down when the nutrition of the bone or the surrounding tissues was disturbed. According to the results of dispersion analysis, the index of strength of the effect of tunnelization (an organized factor) on VC amounts to 42.8 % ($p < 0.05$), MC to 33.1 % ($p < 0.05$), TC to 60.9 % ($p < 0.01$) with a low value of the vessel coefficients, which confirms

the low effectiveness of tunnelization on the revascularization of bones in aseptic necrosis.

The clinical part of the study was based on the treatment of 12 patients with Kienböck's disease, 16 patients with aseptic necrosis of the femoral head and 38 patients with unhealed fractures and false joints of the navicular bone complicated by various degree of degeneration of the fragments. The duration of disease or remoteness of injury ranged from one to 5 years, the age of the patients from 17 to 45 years. Transplantation of the vascular bundle into the bones of the wrist was carried out according to I. G. Grishin and M. G. Divakov (1), into the femoral head by the method of I. G. Grishin et al. (4).

The examination of results of transplantation of the vascular bundle after 2—3 years demonstrated clinical effectiveness of the method in the early stages of disease. Good results were obtained with the treatment of Kienböck's disease before the falling in of the semilunar bone occurred (index of semilunar bone 0.5 and more) and arthrosis deformans developed.

Transplantation of the vascular bundle into the head of the femur is only effective in absence of falling in or subchondral collapse or signs of dysplasia of the hip joint. In four observations of cases at the stage of settling down, the disease took a progressive course altthough a considerable reduction in the intra-osseous pressure could be observed. In order to restore the congruence of the articular surfaces of the hip joint and liberate the foci of necrosis from the stress of load in such cases, we used transtrochanteric rotational osteotomy of the femur (7) in combination with transplantation of the vascular bundle to create and additional source of blood supply to the head of the femur (Fig. 4). Four such operations were carried out.

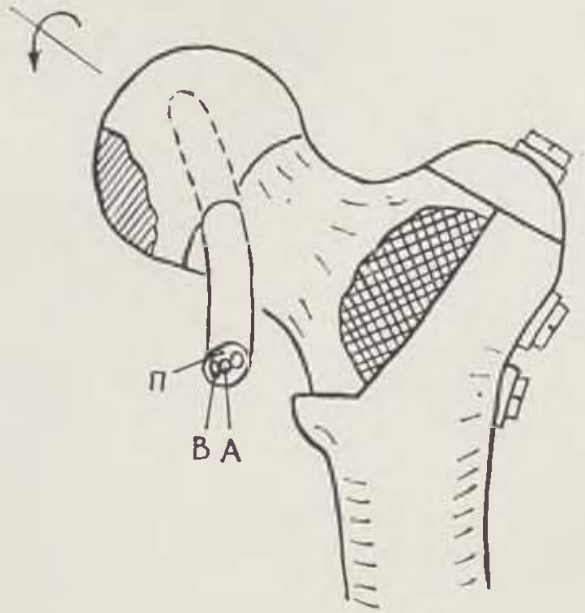

Fig. 4. Schematic drawing of transplantation of a vascular bundle into the femoral head in anterior transtrochanteric rotational osteotomy

Fig. 5. Roentgenogram of a patient with a false joint of the navicular bone in the left carpal joint before (a) and 2 years after operations (b) using transplantation of a vascular bundle

After transplantation of the vascular bundle into the navicular bone, the fragments started healing in 24 out of 38 patients at 2½ to 3½ months (Fig. 5) due to primary formation of an endosteal callus along the course of the vascular bundle.

Transplantation of the vascular bundle was carried out during open reduction of chronic dislocations of the semilunar and navicular bones (3 cases). After 4—6 years, no degenerative changes in the bones or signs of arthrosis deformans were found in these patients. In all cases, the processes of bone reconstruction took their course during 1—1½ years, this being in correspondence with the results of the experiment.

CONCLUSIONS

1. Morphometric analysis permits us objectively to evaluate the process of revascularization of avascular bones and to substantiate postoperative strategy of treatment in aseptic necrosis.

2. Revascularization of an avascular spongy replant following transplantation of a vascular bundle sets in 3 weeks later with complete normalization of blood flow by the 16th week.

3. During experimental disturbance of blood supply to the femoral head, its revascularization following transplantation of the vascular bundle takes place in 12—16 weeks while normalization of the blood flow occurs in 24 weeks. The shape of the femoral head and the congruence of the articular surfaces after a similar operation are not restored in case of their disturbance.

4. Tunnelization of avascular bones does not ensure adequate revascularization, and this also accounts for a low effectiveness of this operation in clinical treatment.

5. Transplantation of a pedicled vascular bundle is indicated in the clinic at the initial stage of aseptic necrosis, i.e., before the development of bone degeneration; for the prevention of bone destruction during open reduction of femoral dislocations, dislocations of carpal and tarsal bones, in reconstructive operations on the hip joint.

SUMMARY

Transplantation of a vascular bundle into an avascular spongiosa replant and the head of the femur after interruption of its blood supply was carried out in experiment (66 experiments on 34 dogs). The periods of observation reached 72 weeks. The mechanisms of revascularization of bones devoid of vessels and the results of morphometric analysis were investigated. It has been established that revascularization of a spongiosa replant sets in after 3 weeks and that of the femoral head after 12—16 weeks.

Clinical observations (69 cases) were concerned with transplantation of a vascular bundle in the treatment of patients with aseptic osteonecrosis.

Key words: aseptic, necrosis, revascularization, transplantation, vascular bundle

RÉSUMÉ

Revascularisation de l'os spongieux avasculaire et de la tête du fémur dans la greffe du faisceau des vaisseaux (Etude clinico-expérimentale)

Divakov, M. G.

Dans une expérience (66 expérimentations sur 34 chiens), nous avons exécuté l'implantation d'un faisceau de vaisseaux sur un implant spongieux avasculaire et sur la tête du fémur, après l'interruption de son alimentation sanguine. Le temps des observations atteignait 72 semaines. Nous avons étudié des méchanismes de la revascularisation des os dépourvus des vaisseaux et effectué une analyse morphométrique. On a constaté que la revascularisation de l'implant spongieux commence après 3 semaines, la revascularisation de la tête du fémur après 12—16 semaines. La circulation du sang devient normale après 16 semaines dans le premier cas, dans le second après 24 semaines.

Les observations cliniques (69 cas) se portaient aux greffes des faisceaux vasculaires dans le traitement des malades avec les ostéonécroses aseptiques.

ZUSAMMENFASSUNG

Die Revaskularisation eines avaskulären spongiosen Knochens und Femurakopfes bei einer Transplantierung des Gefässbündels (Experimental-klinische Studie)

Divakov, M. G.

Als Experiment (66 Versuche an 34 Hunden) haben wir eine Implantation des Gefässbündels in das avaskuläre spongiose Replantat und den Femurkopf nach einer Unterbrechung ihrer Blutversorgung ausgeführt. Die Zeitdauer der Beobachtung betrug bis zu 72 Wochen. Wir studierten die Mechanismen der Revaskularisation der Knochen ohne Gefässe und führten eine morphometrische Analysis aus. Wir stellten fest, dass die Revaskularisation des spongiosen Replantats nach drei Wochen beginnt und die Revaskularisation des Femurakopfes nach 12 bis 16 Wochen. Der Blutkreislauf normalisiert sich im ersten Fall nach 16 Wochen und im zweiten Fall nach 24 Wochen.

Die klinischen Beobachtungen (69 Fälle) betrafen die Transplantation des Gefässbündels bei einer Behandlung erkrankter und aseptischer Osteonekrosen.

RESUMEN

Revascularización de huesos esponjosos avasculares y de cabeza de fémur durante transplante de ramificación vascular (Estudio clínico-experimental)

Divakov, M. G.

De manera experimental (66 experimentos en 34 perros) realizamos implantación de ramificaciones vasculares en tejidos óseos esponjosos avasculares (transplante) y en cabeza de fémur luego de haberse interrumpido su irrigación sanguínea. El período de observaciones se prolongó durante 72 semanas. Estudiamos los mecanismos de revascularización de los huesos a los que se les han extirpado los vasos y procedimos a su análisis morfométrico. Constatamos que la revascularización del

transplante de tejido esponjoso comienza a las tres semanas y la revascularización de cabeza de fémur luego de 12 a 16 semanas. La circulación sanguínea se normaliza en el primer caso a las 16 semanas y en el segundo caso a las 24 semanas.

Las observaciones clínicas (69 casos) estuvieron relacionadas con transplantes de ramificaciones vasculares durante el tratamiento de pacientes de osteonecrosis asépticas.

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CONCENTRATIONS OF SELENIUM AND LIPID PEROXIDES AND GLUTATHIONE PEROXIDASE ACTIVITIES IN PLASMA OF THERMALLY INJURED PIGS

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INTRODUCTION

Lipid peroxide levels are markedly increased in inflammated or burned skin (6, 7). This results from the damage to the cell membranes, consisting mainly from phospholipids, and it is related to the width and depth of lesions (4). Lipid peroxides formed by skin cell damage at the sites of burns are released into the serum and difuse into other organs causing their injury (5). In consequence of superoxide anion generation superoxide dismutase (SOD) activity is stimulated to protect the tissues from lipid peroxides injury (2). Niwa et al. (7) have recently shown that this effct is less pronounced in aged patients, being negligible in the group above 70. The same aged group showed a greater increase in skin lipid peroxide levels than the non-aged group.

The defence of the cell against unwanted oxidative damage is afforded by small molecules (glutatione, tocoferols, carotens) and an array of enzymes (SOD, catalase and glutathione peroxidase-G3HPx) (10). The efficiency of this system depends in large part on selenium concentration because this micronutrient is an essential part of the active site of GSH-Px (3). This enzyme reduces various hydroperoxides to corresponding alcohols, and can also modulate the arachidonic acid cascade (10). A detailed knowledge of behaviour of all parts of protective mechanism against oxidative injury in burn disease should be therefore of major importance. Till now no results of more complex, prolonged studies are available (1, 4, 5, 7), so the intention of this preliminary study was to measure three connected parameters: selenium and lipid peroxide levels and GSH-Px activities in plasma of burned swine during one month post-burn.

MATERIAL AND METHODS

Six female pigs of the Polish breed, at 3 weeks of age, were selected for the study. The weight of the animals varied from 15—17 kg. They were burned under anaesthesia by intramuscular injection of ketamine (2—3 mg/kg body weight) with open flame for 1 min at the right side. The area was about 300 cm^2. The animals were on a standard diet and no medicines were given prior and during the experiment. Blood was sampled from the auvicular marginal vein into heparinized vials before burn infliction, and then on 2, 7, 14, 21 and 28 day after treatment. The samples were immediately centrifuged, the plasma separated and analysed.

The concentration of selenium in the plasma was determined spectrofluorometrically with the use of 2,3-diaminonaphtalene as the complexing agent (9). Values are reported as ng Se/ml plasma.

Lipid peroxide levels were assessed according to the method of Yagi (11) with the use of thiobarbituric acid (TBA) and expressed as nmol malondialdehyde (MDA) in ml plasma.

Plasma GSH-Px activity was measured by the method of Paglia and Valentine (8) with the modifications of Hopkins and Tudhope (3), in which GSH-Px activity was coupled to the oxidation of NADPH by glutathione reductase. A unit of enzyme activity was expressed as μmol NADPH oxidized per n/gHb.

The statistical significance of differences between the determined parameters was analysed by the Student t-test.

RESULTS

All the obtained data are presented in Fig. 1. Mean initial plasma selenium levels (84,9 ng/ml) were increased on the second day postburn due probably, at least in part, to the higher hematocrit (more dense blood). Then the concentration of the nutrient decreased steadily to reach to lowest value on 21 day ($p < 0.001$). The last analysis showed higher content of Se but still below the initial level.

The mean concentration of lipid peroxides reached its lowest value on the second day. Afterwards it regularly increased till the 21st day ($p < 0.001$), falling rapidly after another 7 days below the value at the begining of the experiment.

The enzyme activity showed fluctuations, with a drop on the second day, minimal activity was observed by the 14th day. At the end of the study the activity reached the value before burn infliction (0.66 U/ml).

The three parameters were significantly correlated ($p < 0.001$). A positive relationship was noted for the Se vs. GSH-Px $r = 0.7125$, and negative ones for the other two comparisons: MDA vs. GSH-Px $r = 0.944$, Se vs. MDA $r = -0.8007$. The multiparameter coefficient amounts $r = 0.9312$.

DISCUSSION AND CONCLUSIONS

Our findings revealed that the animals chosen for the study had normal plasma Se concentration when compared to the group of experimental animals from the study of Sankari. He observed serum selenium levels higher than in our experiment only after supplementation with Se. It is not easy to compare the results of GSH-Px activity because of variability of analitical methods used. Anyway, this author and Hakkarainen et al. have found similar significant correlation between Se and GSH-Px in plasma of swine, a well-known fact also for other mammals (3).

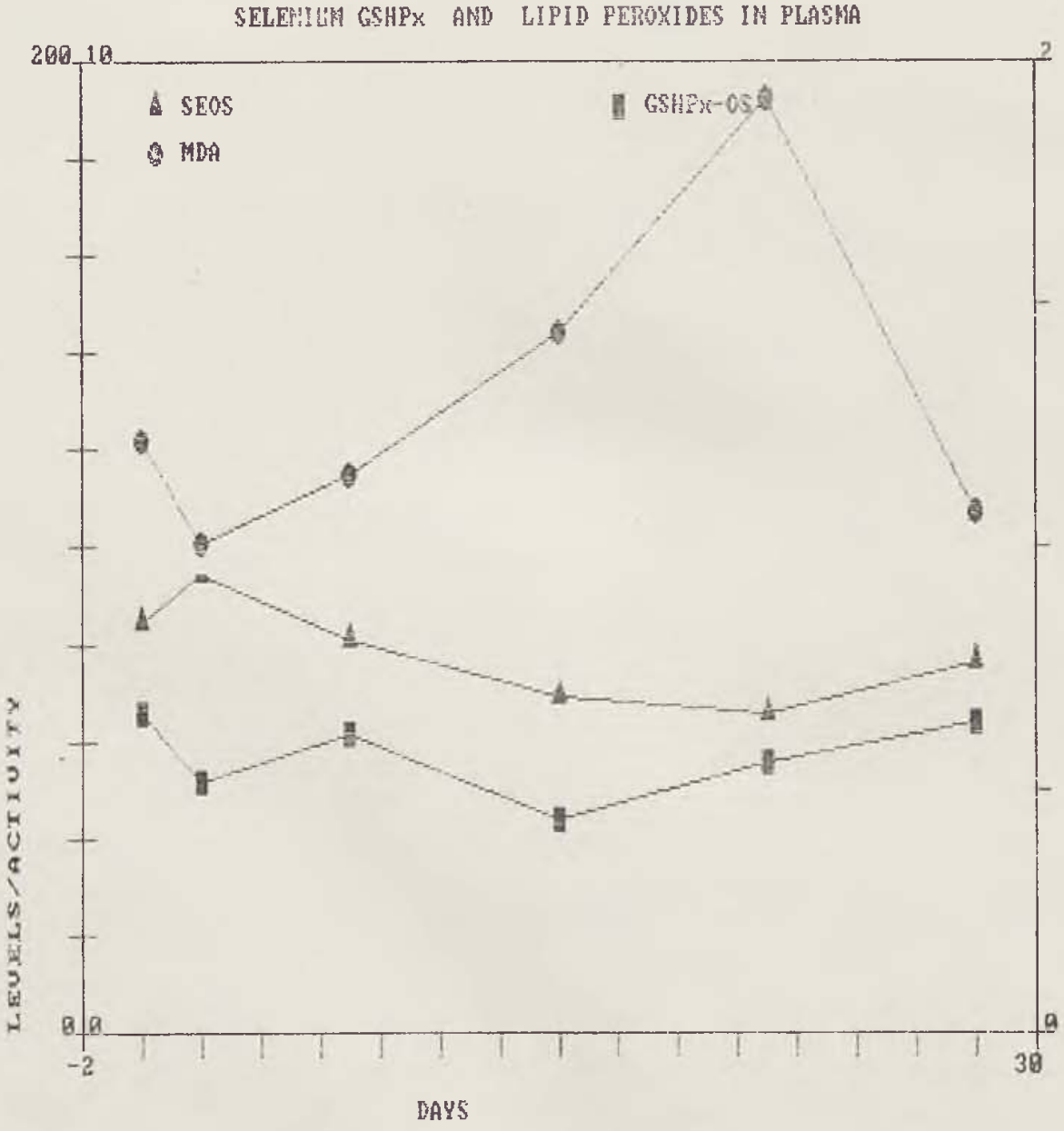

Fig. 1. Plasma selenium (ng/ml) and lipid peroxide (MDA nmol/mg prot.) levels, and glutathione peroxidase activities (U/ml) in thermally injured swine

Until recently only a few studies had measured Se levels in thermal injury (1). Significantly depressed Se levels were reported in patients 2 weeks, during 45 days post-burn, and troughout hospital course. However, we have noted this effect in pigs not earlier than after the second day post burn. Moreower, Boosalis et al. (1) observed a decrease of plasma Se concentration at the moment of an increase in our study. The mechanism of depressed Se levels is unclear. Accelerated Se losses from the wound site or increased Se requirements must be considered. A decrease in carrier protein(s) is an unlikely explanation (1).

Depressed erythrocyte GSH-Px in thermal injury patients have also been observed (1). This tendency was noted in our study, especially on the second and fourteenth day after burn. However, the explanation that patients with thermal injury received multiple drugs which may produce toxicity to the red cells is not good for our plasma enzyme, at least because our pigs were given no medicines. Possibly a weak adaptive capacity mentioned by Nishigaki et al. (5) for SOD in patients with burn is also actual for GSH-Px.

Lipid peroxides can be produced at the cell membranes where chemical or physical damage may occur. This leads to oxidation of polyunsaturated fatty acids presumably through mediation of membrane damage-excited electron transport (6). The activation of xanthine oxidase takes place due to enhancement of purine nucleatide metabolism and granulocyte margination and migration out of the capillary system are induced, which eventually results in lipid peroxidation (7).

Kawai et al. (4) have reported on significantly or markedly increased lipid peroxide levels in the thermally induced skin lesions of mice from the first to the third day. Relase of lipid peroxides from the skin lesions to the plasma should be reflected. Therefore, it is hard to explain a decrease in lipid peroxide levels after two days, which occurred in our study. Further steady increase of the lipid peroxide content up to the third experimental week seems to be the result of growing infection of the wound, i.e. the infiltration of granulocytes activated in the subcutaneous tissue of the burn wound (10).

To summarize trends in our results, we conclude that the three parameters studied are closely correlated in burn disease. There are attempts for applications of antioxidants (SOD, allopurinol) (2, 10) in various infection-dependent inflammations or hypoxia. However we feel that some greater understanding of the role of free radical reactions in disease processes is needed for more rational approach to therapy with the use of antioxidants.

SUMMARY

The mechanism of behaviour of parameters involved in protection of the cell from oxidative damage in burn disease remains unclear. Therefore, selenium and lipid peroxide (MDA) levels, and glutathione peroxidase (GSH-Px) activities have been measured for one month in plasma of thermally injured young pigs. Immediately post-burn mean MDA level and GSH-Px

activity decreased, while Se concentration increased. After 3 weeks lipid peroxide levels reached top concentration. Only the enzym activity returned to initial value at the end of the study, whereas the other two parameters were below the content noted at the begining. The Se concentration was significantly and positively correlated with GSH-Px activity, and negatively with MDA levels. The similar relationship was also showed for the enzyme and lipid peroxides. The results indicate an interesting role of the studied agents in burn disease, but still suggestions of treatment of severely burned patients with antioxidants need to be supported by detailed studies.

RÉSUMÉ

Concentration du sélénium et des bioxydes de lipides et activité de la peroxydase de glutathione dans le plasma des porcs atteints thermiquement

Bieńkowski, W., Gromadźinska, J., Pawłowicz, Z., Struźyna, J., Wąsowicz, W., Szaffer-Marcinkowska, B., Zamorski, R.

Le méchanisme de l'action des facteurs ayant part à la protection des cellules contre l'endommagement par oxydation au cours d'une brûlure reste peu éclairci. C'est pourquoi on a mesuré pendant un mois le taux de sélénium et de bioxyde lipoïde (MDA) et l'activité de la peroxydase de glutathione (GSH-Px) dans le plasma de jeunes porcs brûlés. Immédiatement après la brûlure, le taux moyen de MDA et l'activité GSH-Px baissaient, tandis que la concentration su sélénium montait. Après trois semaines, le taux de bioxyde lipoïde atteignait une concentration maximale, il n'y avait que l'activité de l'enzyme qui rentrait, à la fin de l'observation, aux valeurs initiales, pendant que les deux autres paramètres restaient au dessous du nievau initial. La concentration du séléinum était dans une corrélation significativement positive envers l'activité GSH-Px, mais négative envers le taux de MDA. Des rapports équivalents étaient observés aussi dans le cas de l'enzyme et des bioxydes lipoïdes. Ces résultats témoignent d'un rôle intéressant des facteurs étudiés dans la maladie de brûlure, mais les propositions concernant le traitement de grands brûlés à l'aide des anti-oxydants nécessitent toujours l'appui consistant aux études profondes.

ZUSAMMENFASSUNG

Die Konzentration von Selen und Peroxid von Lipiden sowie die Aktivität von Peroxidasen des Glutathions im Plasma thermal beschädigter Schweine

Bieńkowski, W., Gromadźinska, J., Pawłowicz, Z., Struźyna, J., Wąsowicz, W., Szaffer-Marcinkowska, B., Zamorski, R.

Der Mechanismus des Verhaltens von Faktoren, die am Schutz der Zellen vor einer Oxidationsbeschädigung bei Verbrennungen beteiligt sind, verbleibt ungeklärt. Daher wurden einen Monat lang sowohl der Gehalt an Selen und Peroxiden von Lipiden (MDA) als auch die Aktivität von Peroxidasen des Glutathions (GSH-Px) im Plasma junger Schweine mit Verbrennungen gemessen. Unmittelbar nach der Verbrennung fielen der durchschnittliche Gehalt an MDA und die Aktivität der GSH-Px ab, während die Konzentration des Selens anstieg. Nach drei Wochen erzielte der Gehalt am Peroxid der Lipiden die höchste Konzentration; nur die Aktivität des

Enzyms kehrte gegen Ende der Beobachtungen zu den ursprünglichen Kennziffern zurück, während die beiden übrigen Parameter unter dem Ausgangsniveau blieben. Die Konzentration des Selens stand in bedeutsamer positiver Korrelation mit der Aktivität des GSH-Px, jedoch in negativer Korrelation mit dem Niveau der MDA. Ähnliche Verhältnisse wurden auch im Fall des Enzyms und der Peroxide der Lipide beobachtet. Diese Ergebnisse zeugen von der interessanten Rolle der studierten Faktoren bei Verbrennungserkrankungen, jedoch Vorschläge einer Behandlung schwer verbrannter Patienten mittels Antioxidanten benötigt noch immer ein gründliches Studium.

RESUMEN

Concentración de selenio y peróxidos de lípidos y actividad de la peroxidasis del glutation en el plasma de cerdos afectados termicamente

Bieńkowski, W., Gromadźinska, J., Pawłowicz, Z., Struźyna, J., Wąsowicz, W., Szaffer-Marcinkowska, B., Zamorski, R.

El mecanismo del comportamiento de los factores participantes en la defensa de las células ante el deterioro causado por la oxidación en los casos de quemaduras aún no ha sido esclarecido. Por ello, durante un mes fue medido el contenido de selenio y peróxidos de lipidos (MDA) y también la actividad de la peroxidasis del glutation (GSH-Px) en el plasma de cerdos jóvenes afectados por quemaduras. El contenido promedio de MDA, asicomo la actividad del GSH-Px disminuyeron inmediatamente después de las quemaduras, mientras que la concentración de selenio aumentó. Después de tres semanas el contenido de peróxido de lípidos alcanzó su más alta concentración; sólo la actividad de los enzimas retornó a los índices iniciales al final de las observaciones, mientras que los otros dos parámetros se situaron en los niveles iniciales. La concentración de selenio tenía una correlación significativamente positiva respecto a la actividad del GSH-Px, pero en correlación negativa respecto al MDA. Relaciones similares fueron observadas también en el caso de las enzmimas y peróxidos de lípidos. Estos resultados testimonian sobre el interesante papel de los factores estudiados en los casos de quemaduras, aunque los tratamientos propuestos para pacientes con serias quemaduras utilizando antioxidantes necesitan aún ser apoyados en profundos estudios.

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THE SURGICAL REPAIR OF A DEFECT OF TENDINOUS DISTORTION OF THE PATELLA AND OF THE JOINT CAPSULE DURING AN ARTHROLYSIS OF THE KNEE JOINT

A. A. Grigoruk, A. A. Sokol

The treatment of fractures of femoral diaphysis with the method of maintained bone extension or with open osteosynthesis of bone fragments and a subsequent external immobilization with a plaster bandage leads often to the development of a permanent extension contracture or even to a rigid knee joint. Conservative therapy of these complications sometimes fails to produce the requested beneficial effects because of the cicatrization in the medial part of the femoral quadriceps muscle, or its fixation to the so-called "third point" and an adhesive process in the knee joint.

On the basis of the above mentioned sequelae an attempt was made to restore a sufficient mobility of the knee joint with surgical tendomyolysis of the femoral quadriceps muscle, resection of its degenerated portion and an arthrolysis of the knee joint (2, 3). Peroperatively are exposed the tendons of the medial part of the femoral quadriceps muscle and the patellar ligament with a subsequent resection of the tendinous distortion and of the joint capsule on both sides of the patella. In order to attain satisfactory surgical results the suture of the surgical wound is performed in a position in which the flexion of the tibia attains an angle of 90^0 (2). This position of the tibia leads to the development of a diastasis of the tendinous distortion and of the joint capsule (up to 6—8 cm in length and up to 1—3 cm in width), the developing defect of tissues prevents a suture of the wound. According to the current surgical techniques defects of tendinous distortion and of joint capsule are covered during the suture of the skin wound with a previously detached skin graft. A persisting defect of joint tissues can lead to an unstable tendinous distortion of the patella, a development of synovial fistula, articular infection, prolongation of the required treatment and to

an impairment of therapeutic results. The development of these complications can be promoted by a much too early mobilization of the knee joint.

In the aim to prevent the development of these complications we would like to describe our clinically verified method used for the repair of defects of tendinous distortion of the patella and of the joint capsule after an arthrolysis of the knee joint. For this purpose we dissect from the lateral side of the tendinous distortion and of the joint capsule a split flap consisting of half the thickness of the above mentioned tissues. The dissected oblong flap 1 (Fig. 1) has a wide base situated in front of the defect 2 (Fig. 1) and its dimensions partly overlap the surface of the defect. The dissected flap is turned by 180^0 round its base and extended to cover the defect and subsequently it sutured to the opposite edge of the defect 1 (Fig. 2). The same procedure is used for the repair of the defect on the other side of the patella 2 (Fig. 2).

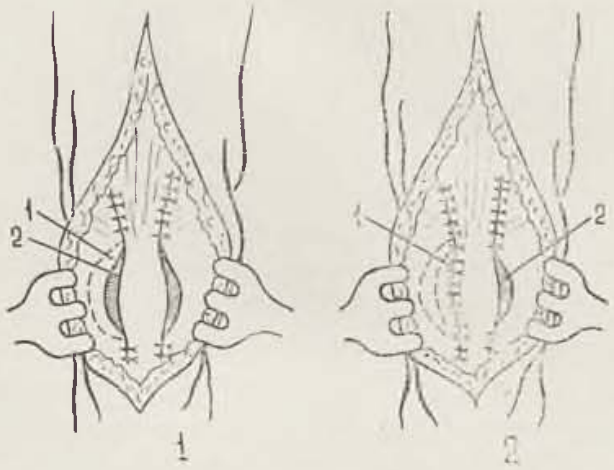

Fig. 1. 1 — the flap dissected from the tendinous distortion of the patella and from the joint capsule; 2 — the defect of the tendinous distortion of the patella and of the joint capsule. — Fig. 2. 1 — the flap turned by 180^0 around its base and sutured to the opposite edge of the defect; 2 — the defect of the tendinous distortion of the patella and of the joint capsule

The technique of the described procedure is not complicated and allows the repair of defects of a tendinous distortion of the patella and of the fibrous joint capsule, a fixation of anterior and lateral joint structures, reduces the risk of complications due to infection and allows an initiation of active kinesitherapy during the early postoperative period.

SUMMARY

The suture of tissues resected during surgical tendomyolysis and arthrolysis of the knee joint required by its extension contracture is carried out during tibial flexion at an angle of 90^0. This can lead to the development

of a diastasis of the tendinous distortion of the patella and of the joint capsule which prevents the suture of the wound. It is suggested to repair the resulting tissue defect with the use of a split flap of local tissues with a wide base situated in front of the defect. The flap is dissected from the lateral part of the tendinous distortion and of the capsule. The flap is turned by 180^0 around its base and extended to cover the defect with its subsequent suture to the opposite edge of the defect. The described supplementary surgical procedure is technically uncomplicated and results in stable anterior and lateral joint structures, in a reduction of the risk of complications due to infection, and allows an early initiation of active kinesitherapy in the early postoperative period.

RÉSUMÉ

Mode de réparation du défaut de distorsion tendineuse de la patelle et de la capsule articulaire dans le cas de l'arthrolyse de l'articulation du genou

Grigoruk, A. A., Sokol, A. A.

En intervenant chirurgicalement dans les cas de la tendomyolyse et de l'arthrose de l'articulation du genou dans le but de réparer la contracture d'extension, on effectue les sutures des tissus réséqués dans la position de flexion du tibia à l'axe de 90^0. A ce moment, survient le diastasis de la distorsion tendineuse de patelle et de capsule articulaire, dont la suture est impossible. Pour résoudre ce défaut tissulaire, on propose son recouvrement plastique par les tissus locaux, à l'aide du lobe bifurqué, avec une large base tournée vers le défaut, le lobe étant prélevé sur la partie latérale de la distorsion tendineuse et de la capsule. Le lobe tourne autour de la base à l'angle de 180^0 et il est suturé après le recouvrement du défaut à son bord opposé. Le complément de l'opération décrit est peu exigeant du point de vue technique, permet de renforcer les segments frontaux et latéraux de l'articulation, diminue le danger des complications infectieuses et donne lieu au commencement de la gymnastique active rééducative dans la période postopératoire précoce.

ZUSAMMENFASSUNG

Eine Methode der Beseitigung eines Defekts der tendinosen Distortion der Patella mit der Kapsel bei einer Arthrolyse des Kniegelenks

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Die Sutura resektierter Gewebe bei einer Operation der Tendomyolyse und Arthrolyse des Kniegelenks wegen einer extensiven Kontraktur wird bei einer Stellung der Schienbeinflexion in einem Winkel von 90^0 ausgeführt. Hierbei entsteht eine Diastase der Sehnendistorsion der Patella und der Gelenkkapsel, deren Sutur nicht möglich ist. Bei der Liquidierung des entstandenen Defekts des Gewebes wird die plastische Überdeckung durch lokale Gewebe mittels eines gespaltenen Lappens vorgeschlagen, dessen breite Basis dem Defekt zugewendet wird, und der aus dem Seitenteil der Sehnendistorsion und der Kapsel ausgeschnitten wird. Der Lappen wird um 180^0 um die Basis gewickelt und nach dem Überdecken des Defekts an dessen entgegengesetzten Rand angenäht. Die beschriebene Ergänzung der Operation ist technisch

nicht anspruchsvoll, gestattet die Befestigung der vorderen und seitlichen Teile des Gelenks, verringert die Gefahr infektiöser Komplikationen und begünstigt den Beginn aktiver Heilgymnastik zu einem früheren Zeitpunkt nach der Operation.

RESUMEN

Procedimiento para eliminar el defecto de distorsión tendinosa de rótula y cápsula articular durante la artrolisis de rodilla

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La sutura de los tejidos reseccionados durante operaciones de tendomiolisis y artrolisis de rodilla se realiza, dada su contracción extensiva, con la pierna en posición flexionada en 90 grados. Al mismo tiempo, se produce diastasis de la distorsión tendinosa de rótula y cápsula articular cuya sutura no es realizable. Para la eliminación del defecto de tejidos surgido se recomienda su cobertura plástica con tejidos locales con la ayuda de un lóbulo escicionado con amplia base volcada hacia el defecto y recortado delaparte lateral de l distorsión tendinosa y de la cápsula. El lóbulo se gira en torno a su base 180 grados y tras cubrirse el defecto se cose a su borde opuesto. El complemento de la operación descripto es técnicamente sencillo, posibilita reforzar las partes anteriores y laterales de la articulación, disminuir el riesgo de complicaciones infecciosas e iniciar una activa terapia gimnástica en el período temprano posoperatorio.

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