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A RARE CASE OF CONGENITAL MIDFACE UNDERDEVELOPMENT (dysgenesis cerebrofacialis mediana)

A. Y. MANSONE

Congenital deformities of the midface manifest themselves in different variants: 1) median cleft of upper lip, 2) median cleft of nose (also called double nose), 3) double nose together with median cleft of upper lip, 4) developmental anomaly with absence of dedian part of upper lip (prolabium) and the premaxilla, the septum nasi, the collumella, the vomer, the lamina cribrosa and the crista galli. This anomaly is the most severe form and is met with relatively rarely. In the literature, only two single cases of this monstrosity have been described (Ernst, 1909; Essbach, 1961; Brucker et al., 1963; Baibak et al., 1966).

These authors pointed out that apart from the anomalies referred to above, underdevelopment of the prosencephalon was also observed. Chiefly the rhinencephalon, i.e. the olfactoric part of the brain, is affected. This is why this anomaly has been given the name arhinencephalia.

In the Soviet Union there was one case of defective development of the midface. It was found in the body of a two-day-old newborn. Unfortunately, the specimen was delivered to the author of this communication only after dissection, when the brain had been removed.

The face was characterized by a flat nose the tip of which was as though retracted. The midsegment of the upper lip and the premaxilla were missing. In the anterior cranial fossa, the crista galli and the lamina cribrosa were absent. This region was filled with loose tissue.

In the oral cavity there was a bilateral penetrating cleft, 15 mm in width. The nasal cavity was divided by the bony septum nasi. The lower edge of the vomer was free and sharp. The auricles were limeleaf-shaped.

The anterior point of the vomer was connected with the tip of the nose by a thin sheet of soft tissue. A small intumescence of soft tissue, 3 mm wide, linked up with the columella, started at the lower edge of the vomer. At the

lower margin of the intumescence there was a suspended structure of cartilaginous consistence and triangular-prism shape.

Ernst and Essbach considered arhinencephalia to be a variant of a median cleft of the upper lip. In 1963, Brucker et al. suggested that such a type of anomaly was linked up with a bilateral penetrating cleft of the upper lip combined with the absence of the midsegment of the lip and the premaxilla.

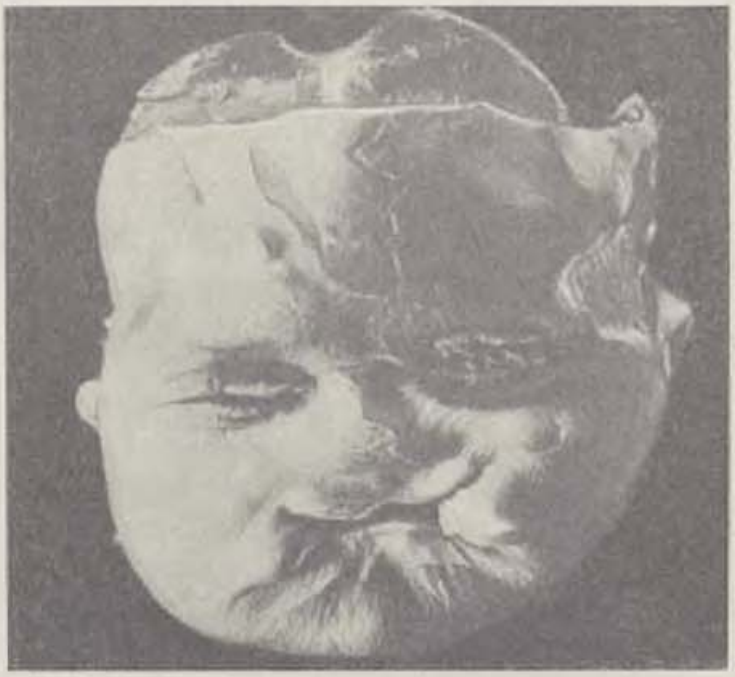

Fig. 1. Head of newborn body with abnormal development of the midface

Brucker et al. based their suggestion on the fact that in median cleft of the upper lip which is frequently accompanied by a double nose, orbital hypertelorism has always been observed.

Great interest has been attached to the question as to the mechanism of median facial cleft development from the point of view of its embryological origin.

In the early stages embryonic development of the midface is closely connected with the development of the forebrain. This is the reason why there is an interconnection between the degree of defect in the structures forming the midface and those of the forebrain. Brucker et al. pointed out that small defects in the face may be reflected in minimal anomalies of the forebrain. Speaking only of the pathogenesis of a facial defect these authors considered that in the embryogenesis of the face, some of its median structures may remain underdeveloped. As a rule, these structures develop from the inner nasal swelling in the face of the embryo. Particularly the nasal swelling develops from the tissues of the primary forebrain.

Summing up the above, Brucker et al. assumed that arhinencephalia was an anomaly in the development of a certain part of the face of an embryo, which corresponded to the primary forebrain and the inner nasal swelling.

From the point of view of embryogenesis, the assumption of Brucker et al. who consider arhinencephalia to be a type of bilateral penetrating cleft rather than a median cleft of the upper lip may be elucidated by the following: In the case of a median cleft, the two inner nasal swellings would not fuse with each other, which in arhinencephalia are either underdeveloped or absent altogether.

The case referred to at the beginning is interesting in that it presents a rare observation of an intermediate stage of development of the midface,

Fig. 2. Lower margin of septum nasi and anterior edge of vomer with swelling of soft tissue are seen through incision on sides of face

i.e. between fully developed arhinencephalia and simple bilateral penetrating cleft of the upper lip.

The specimen has a flat nose, the crista galli and lamina cribrosa are missing, and the auricles rudimentary. However, a developing septum nasi is present as well as the beginning of the developing median segment of the upper lip and premaxilla.

Thus, the above observation may serve as evidence of Brucker's et al. assumption that arhinencephalia is a type of bilateral cleft of the upper lip rather than a median cleft. This is why the author of this communication considers this assumption to be correct and the anomaly of the midface, described above, a case of dysgenesis cerebrofacialis mediana rather than of arhinencephalia.

Children with such an anomaly are most frequently born dead of die in the first hours or days after birth (Essbach). One of the causes of death in these children Brucker et al. considered to be the incapibility of the organism to adapt itself to extrauterine life. It sould be taken into account that underdevelopment of a certain part of the central nervous system goes together with this anomaly of the face. This points to the necessity of paying attention to the psychic and neurological condition of the child in any congenital cleft deformity of the face.

SUMMARY

A case of defective development of the midface with missing median segment of the upper lip and premaxilla was described. Such a deformity is interesting because it represents a rarely observed stage of development of the midface, i.e. the intermediate stage between fully developed arhinencephalia and a bilateral penetrating cleft of the upper lip.

The case described above may serve as evidence for the assumption of Brucker et al. (1963) of arhinencephalia being a type of bilateral cleft of the upper lip rather than a median cleft.

In these deformities, underdevelopment of certain parts of the central nervous system should be expected. It therefore follows that it is necessary to pay attention to the psychic and neurological condition of the child in these deformities. B. K.

RÉSUMÉ

Cas rare de la dysgenèse cérebrofaciale médiane

Mansone A. J.

On a décrit un défaut du développement de la partie médiale de la face avec une absence du segment médial de la lèvre supérieure et de l'os intermaxillaire. C'est un cas très intéressant parce qu'il présente un stade rarement apparaissant du développement de la partie médiale de la face, c'est-à-dire le stade entre l'arinencéphalie totale et la fente pénétrante bilateraire de la lèvre supérieure.

Le cas décrit peut prouver l'hypothèse de Burker et col. (1963) que l'arinencéphalie est une espèce de la fente bilateraire de la lèvre supérieure, mais pas du tout de la fente médiane.

En cas de tels défauts, on doit dompter avec un développement insuffisant des certaines parties des nerfs centraux. Il résulte ainsi qu'il faut faire attention à l'état psychique et neurologique de l'enfant ayant une fente congénitale.

ZUSAMMENFASSUNG

Ein seltener Fall medianer zerebrospinaler Dysgenese

Mansone A. J.

Der Autor beschreibt den Fall einer Störungsentwicklung des mittleren Gesichtsteils mit fehlendem Mittelsegment der Oberlippe und des Zwischenkiefers. Ein derartiger Fall ist deshalb von Interesse, weil er ein Entwicklungsstadium des mittleren Gesichtsteils darstellt, das nur selten zu sehen ist, d. h. das Stadium zwischen vollständiger Arhinenzephalie und beiderseitiger durchgreifender Oberlippenspalte.

Der beschreibene Fall kann als Bestätigung der Hypothese von Burker und Mitarb. (1963) dienen, dass Arhinenzephalie eine Art von beiderseitiger Oberlippenspalte und nicht eine mittlere Spalte darstellt.

Bei derartigen Fehlern muss mit mangehalfter Entwicklung bestimmter Teile des Zentralnervensystems gerechnet werden. Daraus ergibt sich, dass dem psychischen und neurologischen Zustand des Kindes mit angeborener Spalte Aufmerksamkeit geschenkt werden muss.

RESUMEN

Un caso raro de la disgenesía cerebrospinal mediana

Mansone A. J.

Fue descrito un caso de un defecto en la evolución de la parte central de la cara con falta segmento central del labio superior y del hueso intermaxilar. Tal caso es interesante porque representa el estadio de la evolución de la parte central de la cara, el que se puede ver rara vez, es decir el estadio entre la arinencefalia completa y una fisura penetrante bilateral del labio superior.

El caso descrito puede servir de comprobación de la hipótesis de Burker y col. (1963) de que la arinencefalia es una variedad de la fisura bilateral del labio superior y no de la fisura central.

En tales defectos hay que contar con una evolución insuficiente de ciertas partes del sistema nervioso central. De ello se desprende que es necesario fijarse en el estado psíquico u neurológico del niño con una fisura congénita.

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KENACORT A IN THE TREATMENT OF HYPERTROPHIC SCARS AND KELOIDS IN CHILDREN

S. JAWORSKI

Hypertrophic scars and keloids have long been a subject of interest to several medical disciplines, chiefly surgery, dermatology and radiology.

The problem of differentiation between these two types of lesions is regularly discussed in the literature. Despite numerous investigations on tissue culture (1, 2), microstructure of collagen (3) or enzymatic content of the tissues (4), there are as yet no definitively established criteria for differential diagnosis.

Pediatric surgeons have often dealt with this problem. Children are referred to the out-patient clinic because of abnormally abundant scar tissue following burn, mechanical injury or vaccination. Their parents are usually alarmed of the unsightly lesion and demand its radical excision. Such operations are frequently performed, sometimes with the use of cutaneous grafts to cover the skin defects. These procedures produce initially a favourable cosmetic effect in both hypertrophic scars and keloids. A few months later, however, the lesions will commonly recur and may be even larger than was the one previously removed.

Radiotherapy has been used with occasional good results but it should be postponed because of the hazards of radiation upon the developing child.

A significant therapeutic progress was achieved due to the introducement of local administration of corticotropine and adrenal cortical steroids (cortisone, hydrocortisone, cortisol).

Action of corticosteroids:

1. Reduction of cellular response — inhibitory effect on proliferation of fibroblasts (5).

2. Control of oedema and exudative reaction — blocking the formation of new blood vessels and inhibition of capillary permeability.

3. Limitation of nonspecific responsiveness of the skin to irritating agents (reddening, pain).

4. Atrophy and degeneration of the collagen stroma of the skin.

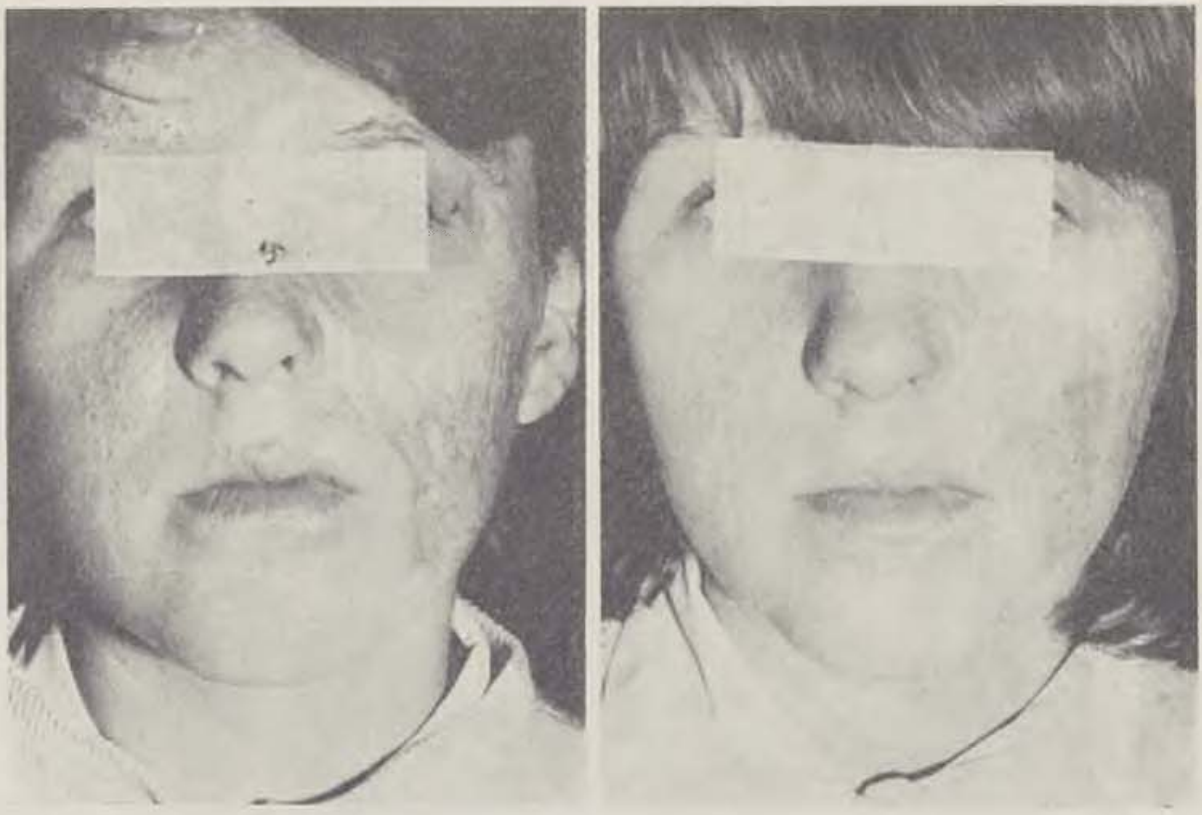

Fig. 1. Post burn hypertrophic scars on the face. — a. 12 years after burn, b. 4 years after treatment with Kenacort A

Action of corticosteroids on the connective tissue has been investigated in tissue culture (6). Among other findings, the number of fibroblastic mitoses was found to be significantly reduced and the hormones exerted catabolic influence upon various tissues (bones, skin, tendons). Comparative studies revealed similar results in vitro and in vivo.

The mechanism of action of these hormones is not clear. It has been assumed that they inhibit the synthesis of aminoacids and proteins during the process of fibroplasia (5). This theory explain in relatively easy way the therapeutic effect upon young scars and keloids as well as the preventive action against their further growth.

METHODS

The drug used in this study was Kenacort A, Squibb's aqueous suspension of triamcinolone acetonide 10 mg/ml, adapted to intradermal administration. Its local action was found to be 20 per cent more effective than that of prednisolone and ten times stronger than that of hydrocortisone (7). The only untoward side effects are local transitory atrophy of subcutaneous tissue and the appearance of teleangiectasia (8). According to numerous authors (5, 8, 9, 10), there are no systemic complications even with the use of a very high dose. For example, 1800 mg in one series of injections was not accompanied by

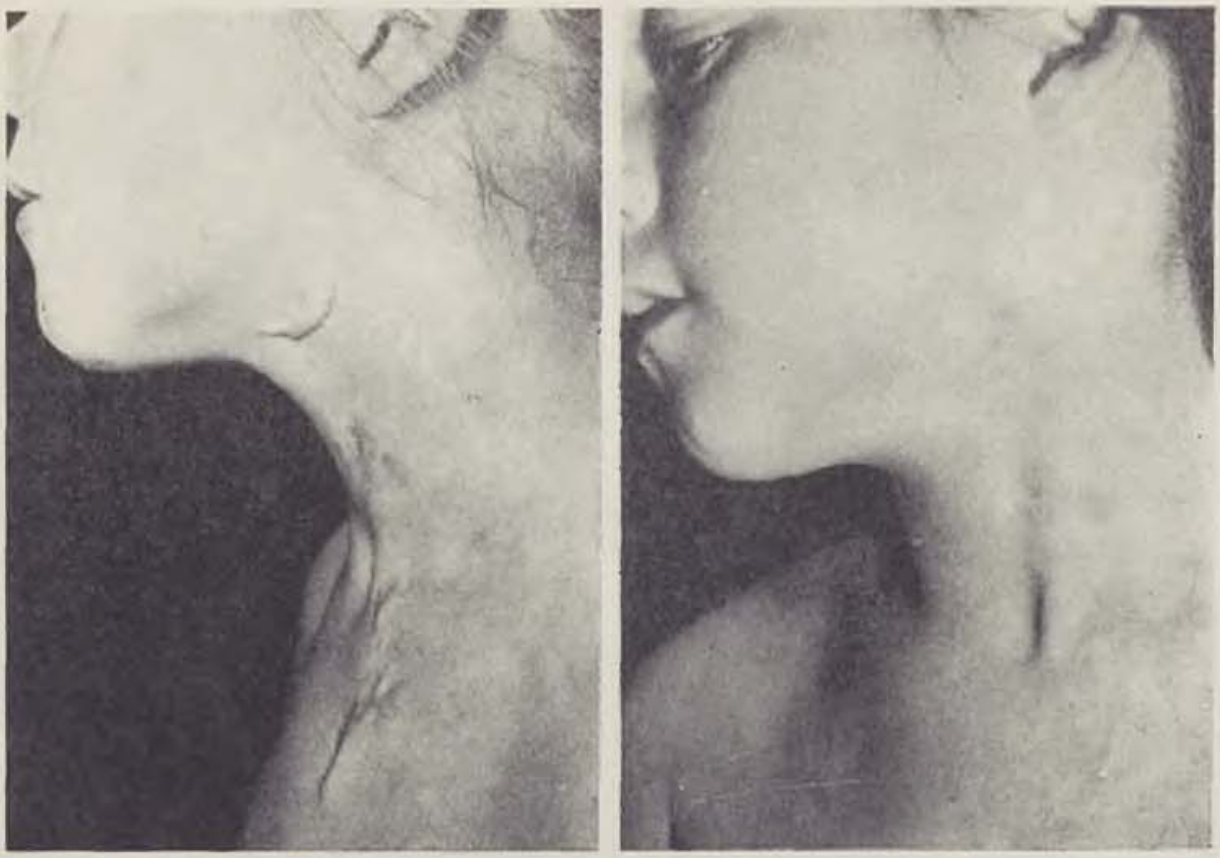

Fig. 2. Post burn hypertrophic scars on the neck. — a. 8 months after burn, b. 4 years treatment

a fall of urinary steroids (11). The convincing evidence of clinical value of triamcinolone is its strong local activity and the lack of any harmful influence upon the adrenal cortex.

A tuberculin-type syringe was used in order to inject the drug under high pressure and in a precisely measured dose. Kenacort A was administered into cicatricial tissue by several injections. A single dose was 5 to 15 mg. This procedure was repeated 3 times every 3 weeks. The total amount of the drug used was 15 to 45 mg, depending upon the size of the lesion. In the case of failure to obtain a satisfactory effect, the above treatment was repeated after 2 to 3 months.

The parents of children were asked for the following data: the time and type of trauma, the duration and course of wound healing, the time of onset of the scar formation, local complaints and previous treatment.

On examination, attention was paid to the location and size of the lesion, the degree of its elevation above the normal skin, pigmentation, temperature, consistency, mobility and contractures and other secondary deformities. Each child was photographed in two projections.

The lesions which qualified as keloids demonstrated significant growth potential and solid consistency. They were distinctly elevated above the surrounding tissues, whitish, opalescent, and were covered with thin, shiny epi-

dermis. Traumatic injury was usually disproportionately small as compared with the size of the resulting keloid. Keloids showed a high recurrence rate following surgery.

The diagnosis of hypertrophic scar was made when the lesions were much larger, of irregular density, only moderately elevated and covered with shranken skin. Young scars were pink and pruritic. In a later stage, they were the color of normal skin and the itching was less annoying.

The above differentiation was based only up on the clinical appearance. It is by no means adequate because of the great number of lesions on the borderline between the two types.

Tab. 1. Summary of 60 cases treated with Kenacort A

	Scars	Keloids	Total
Post-burn	30	7	37
Post-traumatic	10	2	12
Post-vaccinal	—	11	11
Total	40	20	60

Biopsy was not performed for preliminary histologic evaluation since the routine analysis of specimens is not helpful in the differentiation between hypertrophic scars and keloids.

Kenacort A was used in the treatment of small lesions as well as of those with disturbed function or unsightly appearance on the exposed areas of the body.

MATERIAL

In the years 1966—1971, 60 children were treated. The most numerous group (37 cases) represented post-burn lesions. In 12 patients the lesions had resulted from traumatic injury, and in 11, from vaccination. The age of the children ranged from 6 months to 14 years.

Tab. 2. Results of treatment with Kenacort A in 49 cases

Scars/Keloids	Result of treatment			Total
	good	moderate	poor	
Post-burn	14/1	12/—	3/1	29/2
Post-traumatic	3/1	4/—	—/—	7/1
Post-vaccinal	—/3	—/2	—/5	—/10
Total	17/5	16/2	3/6	36/13

Post-burn lesions.

The healing of the wounds had always been prolonged (1 to 6 months, 2 months, average), and was complicated by abundant suppuration. In 5 children of this group, cutaneous grafts were used in the treatment of the wounds. Thirty patients developed hypertrophic scars and the remaining 7, true keloids. Most lesions were located on the face, neck and anterior surface of the chest.

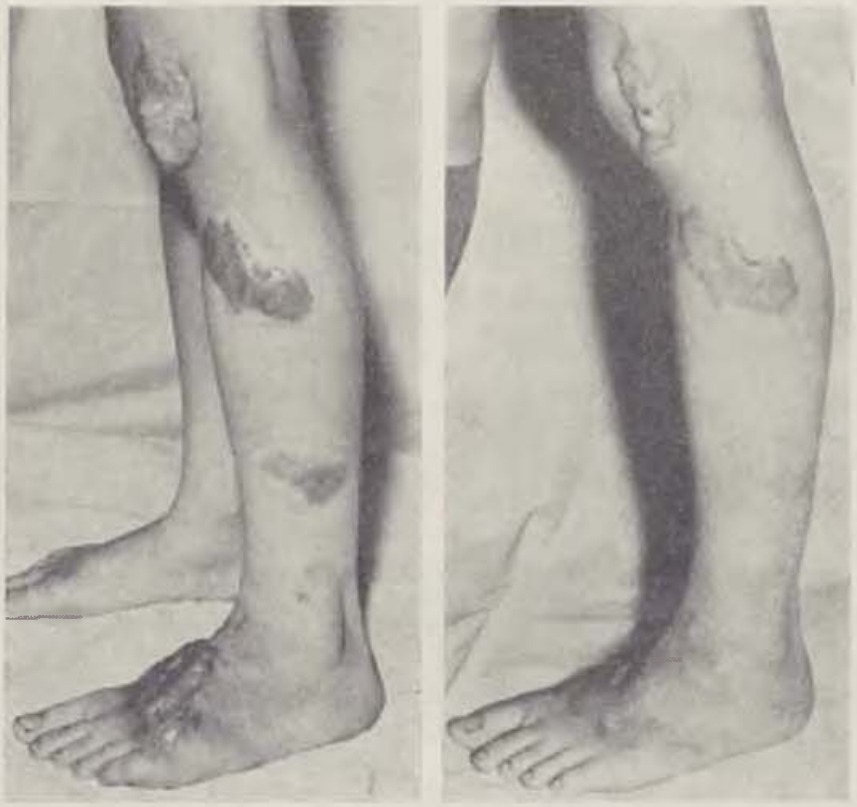

Fig. 3. Keloids following burn on the foot and leg. — a. 6 months after burn, b. 2 years after treatment — dissolution of keloid on the dorsal side of the foot. No signs of regression in other keloids which were left untreated

According to the information obtained from the parents, the onset of the hypertrophic scarring was usually one month after the healing of the wounds, but was prolonged for 3 to 12 months in 11 patients. Growth of the scar or keloid progressed generally for 3 to 6 months, in sporadic cases up to the second year. Lesions were mostly limited to the skin. In 5 cases the scars were deep, fixed, and adherent to the underlying bone. Subjective symptoms (itching and pain) appeared in about half of the patients with both young and old lesions.

In 13 children, the treatment was started in the first year after wound healing. In that time, scars and keloids were still progressing and were defined as young lesions. In the remaining 24 patients, the mean time of the starting treatment was 18 months and only in one case it was as late as 12 years after the healing of the wound.

Post-traumatic lesions.

Twelve children were treated in this category. In half of them hypertrophic scars resulted from accidental injury, in 3 from excision of haemangiomas and in 3 following operation upon cleft lip, undescended testis and congenital ring constrictions. Wound healing was uncomplicated and in only 5 cases of longer duration — 3 to 4 weeks. The hypertrophic process showed great variation in its durations — 2 months to 2 years (3 months, average). Only in 2 cases were the lesions keloids. The treatment with Kenacort A was carried out generally 6 to 12 months after the termination of healing.

Post-vaccinal lesions.

Of the total 11 children, all affected with keloids, 8 had been vaccinated with BCG, and 4 against small pox. One child had two keloids following both types of vaccinations. The site of the reaction healed usually in 1 to 4 weeks, in one patient, 4 months after vaccination against small pox.

Four children had 5 recurrences after surgical excision of their keloids, the primary lesion was treated in all others. The growth of the keloid was found to be delayed and slower than in post-burn lesions. The parents observed progressive increase in size of postvaccinal scar after 6 to 12 months, even after 3 years. On the other hand, the recurrence after surgical excision developed rapidly — during one month following operation. Its final size was much larger than that of the primary lesion.

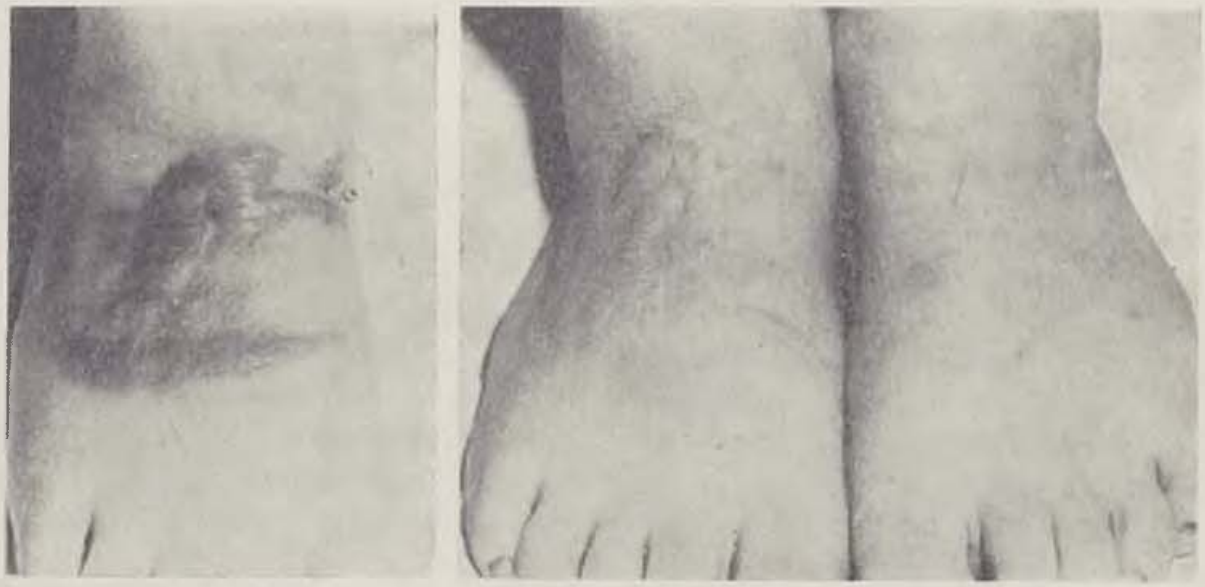

Fig. 4. Post burn hypertrophic scar on the dorsal side of the foot. a. 4 months after burn, b. 2,5 years after treatment

The patients came for treatment of old keloids an average of 5 years after vaccination. Two children of this group showed an inherent predisposition to abnormal scarring. This was evidenced by the formation of keloids following uninfected surgical wounds far distant from the initial lesion.

RESULTS

An analysis of results was performed in 49 patients followed for from one to 5 years. The remaining 11 children had to be excluded since the time of observation of the therapeutic effects was too short.

The result of treatment was noted as good if the lesions flatened out and became much softer, contractures were released, and local complaints receded completely. This estimation was not disqualified by the presence of such complications as local subcutaneous atrophy or teleangiectasia which are known to disappear spontaneously.

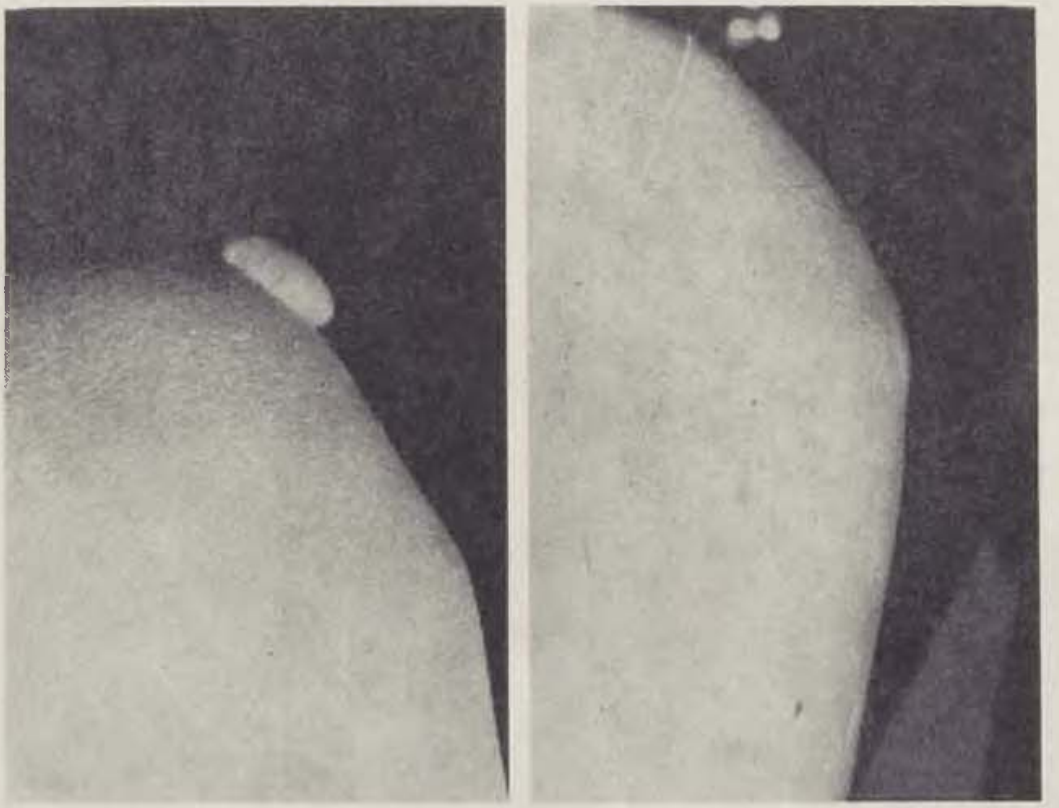

Fig. 5. Keloid following BCG vaccination. — a. Prior to the treatment, b. 3 years after treatment

The result was regarded as moderate in 18 patients in whom the treatment was found to be less effective. For example the lesion was quite soft and pliable, but not flattened out at all, or the contracture persisted despite softening of the scar.

It should be noted that in the group of post-traumatic, mostly uncomplicated lesions, a favourable result was achieved in all children.

Six patients showed no improvement at all. Two of them, who have already been mentioned, were congenitally predisposed to excessive scarring. In the other 3 children, the direct effect was satisfactory, but a few months later it was considered poor.

One fact deserves attention. Among 9 patients with unfavourable results of treatment, 6 were affected with keloids. It must be added, however, that in

the time of treatment of all these children the lesions were already mature and of several years duration.

In a couple of patients whose postvaccinal keloids had been treated with good results, the lesions were surgically removed because of persistent hyperpigmentation. Excision was performed with caution to minimize traumatization of tissues. There was no recurrence during over one year's observation.

One series of treatment with Kenacort A was found to be adequate to obtain positive results in 33 children. In those who were not helped the treatment was repeated. Two series were administered to 13 and three series to 3 patients. Two initial injections gave an idea of the effectiveness of the treatment — the scar was softer and flattened out. So that subsequent administration of the drug was facilitated.

If no distinct improvement is obtained after the first series of injections — the prognosis of further treatment is poor.

SUMMARY

Treatment with Kenacort A is indicated in hypertrophic scars and small-sized keloids resulting from burns, surgical and accidental injuries as well as from vaccinations. It should be started in the stage of active growth of the lesion.

Kenacort A administered locally in children is a safe drug as it does not produce any systemic reactions. Local complications (atrophies, teleangiectasia) recede spontaneously.

In those cases in which complete regression of the lesion is unobtainable, Kenacort A produces a better local condition for plastic surgery and reduces the recurrence rate.

RÉSUMÉ

Utilisation de Kenacort en traitant les cicatrices hypertrophiques et les chéloïdes chez les enfants

Jaworski S.

Le traitement par Kenacort A est indiqué en cas de cicatrices hypertrophiques et de chéloïdes moindres apparaissant après les brûlures, après les interventions chirurgicales ou les accidents occasionnels, même qu'après la vaccination. Il doit être appliqué dans la période du stade actif de la prolifération pathologique.

Kenacort A localement utilisé chez les enfants est un remède sans danger ne provoquant aucune réaction générale. Les complications locales (atrophie, téléangiectasie) disparaissent spontanément.

En cas où la disparition totale ne peut pas être obtenue, Kenacort A peut contribuer à une amélioration des conditions locales pour l'intervention plastique et pour la prévention des récidives.

ZUSAMMENFASSUNG

Die Anwendung von Kenacort in der Behandlung hypertrophischer Narben und Keloide bei Kindern

Jaworski S.

Die Behandlung mit Kenacort A ist angezeigt bei hypertrophischen Narben und kleineren Keloiden, die sich nach Verbrennungen, chirurgischen Leistungen oder Zufallsunfallen gebildet haben, sowie auch nach Impfungen, und sie soll zum Zeitpunkt des aktiven Stadiums des pathologischen Wachstums eingesetzt werden.

In lokaler Applikation bei Kindern ist Kenacort A ein verlässliches Mittel und ruft keine Allgemeinreaktionen hervor. Komplikationen lokalen Charakters (Atrophie, Teleangiektasie) ziehen sich spontan zurück.

In Fällen, wo ein vollständiges Verschwinden der Läsion nicht erzielt werden kann, kann Kenacort A zur Besserung der lokalen Bedingungen für den plastischen Eingriff und zur Verhütung von Rezidiven beitragen.

RESUMEN

Aplicación de Kenacort en la terapia de cicatrices hipertróficas y de queloides en los niños

Jaworski S.

La terapia por Kenacort A es indicada en los casos de cicatrices hipertróficas y en queloides menores aparecidas después de quemaduras, intervenciones quirúrgicas o accidentes ocasionales, así mismo que después de vacunaciones y debe ser aplicado en el período del estadio activo de la proliferación patológica.

Kenacort A en aplicación local en los niños es un medio no peligroso y no provoca ninguna reacción general. Complicaciones de carácter local (atrofia, teleangiectasias) retroceden espontáneamente.

En los casos, en los cuales desaparición completa de la afección no se puede conseguir, Kenacort A puede ayudar a mejorar las condiciones locales para una intervención plástica y prevenir recidivas.

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THE ROLE OF MITOTIC ACTIVITY IN THE FORMATION OF THE SECONDARY PALATE

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Our cognition sometimes lacks in adequate comprehensiveness in that the scientists' attention tends to concentrate on the dramatic stages and crucial points of morphogenesis such as are the natural outcome of a series of processes going on in an uninterrupted and inconspicuous way. The insufficiency of some of them only becomes apparent in the points of interlacement, too late, as a rule, to try and discover the causes. Being unable to make use of even the simplest judgement of "post hoc, propter hoc", we come out with hypotheses. The present state of the knowledge about the pathogenesis of cleft palate, where the most reputable of theories puts emphasis on the delayed horizontalization of the palatal shelves (Fraser, 1965) is a good enough example of this.

Proof of the cleft palate in mice appearing as a result of corticoid treatment even when the palatal shelves begin to horizontalize at a stage of development well within the standard (Dostál, Jelínek, 1973) made us undertake a systematic study of those elementary morphogenetic mechanisms which have a role to play in the development of the secondary palate. The first to come into consideration was the intensity of proliferation processes the importance of which in the formation of the secondary palate and its disorders is not only emphasized by several authors (Andersen, Matthiessen, 1967, Aronov, 1970) but even indirectly proved (Mott et al., 1969, Andrew, Zimmerman, 1971). Since to seek the causes of the pathological state means, in the first place, to know the normal processes well, we decided to employ the most common experimental model — the mouse embryo — and on it to record in space and time the pattern of proliferation processes in the course of the growing, horizontalization and fusion of the palatal shelves.

MATERIAL AND METHOD

In order to be able to follow the proliferation we employed the well-tried technique of direct colchicine application adapted for use in mammalian embryos. Colchicine, it is generally known, has a capacity of arresting in the

metaphase all the cells that enter mitosis since the moment of application. The proliferation intensity is then gauged by the number of arrested mitoses for a given period of time which — compared with the simple count of mitotic figures — offers the advantage of being able to intercept also changes taking place in the course of the mitosis which in itself undergoes as many ontogenetic changes as to distort the results.

Tab. 1

Stage	Area between palatal shelves (planimetrical units)	Index of proliferation activity		apical / basal
		apical	basal	
12/24	260	32.6		—
	220	54.5		—
	240	124.2		—
13/12	340	66.1		—
	390	65.9		—
	400	56.4		—
	360	59.5		—
13/24	470	238.8	113.0	2.11
	430	30.7	29.0	1.06
	490	125.3	58.9	2.12
	530	127.7	93.3	1.37
14/12	350	57.4	54.6	1.05
	430	38.2	32.5	1.18
	440	56.4	68.9	1.22
	520	97.5	59.8	1.63
	570	77.5	40.4	1.92
	620	86.1	55.5	1.55
14/24	630	26.2	20.2	1.32
	650	39.7	36.1	1.09
	730	41.2	32.1	1.28
	700	33.8	23.7	1.43
15/12	20	19.7	15.9	1.24
	50	16.7	13.9	1.20
	20	22.6	16.2	1.40
15/24	0	15.6	13.3	1.17

Females of randombred mice H-Velaz were allowed to mate overnight, the day of the appearance of the vaginal plug was put down as the 1st day of gestation. Microgram amounts of colchicine, dissolved in saline, were injected straight into the amniotic sac between the 12th and 16th days of gestation. After two hours of the cytostatic effect, we collected the embryos, examined them histologically and on the basis of fixed criteria we evaluated the number of arrested mitoses within the scope of the ocular grid focussed

at the transversal section of the palatal shelf (A) and the mesenchyme of the maxilla in the immediate vicinity of its insertion part (B). Thus we examined 25 embryos taken from 14 females.

CONCLUSIONS OF OBSERVATIONS

a) the overall proliferation activity in the palatal shelves reaches its peak at the turn of the days 13 and 14 of the embryonic development, i.e. at least 24 hours before horizontalization (Tab. 1, Fig. 1).

b) the growth activity is invariably the highest in the apical parts (i.e. medial margins) of the palatal processes.

c) the distribution of the growth centres along the antero-posterior axis of palatal shelves depends on the stage of development (Tab. 2). At the beginning of the palatal process formation (end of the 12th, beginning of the 13 th day) we find the peak of the proliferation activity in the rostral regions (behind the premaxilla) just before horizontalization (end of the 13th, first half of the 14th day), another maximum appears in the central or as far as the posterior parts of the palatal shelves.

d) the period of the horizontalisation and fusion of the palatal shelves along the midline is characteristic for its relatively low standard of proliferation.

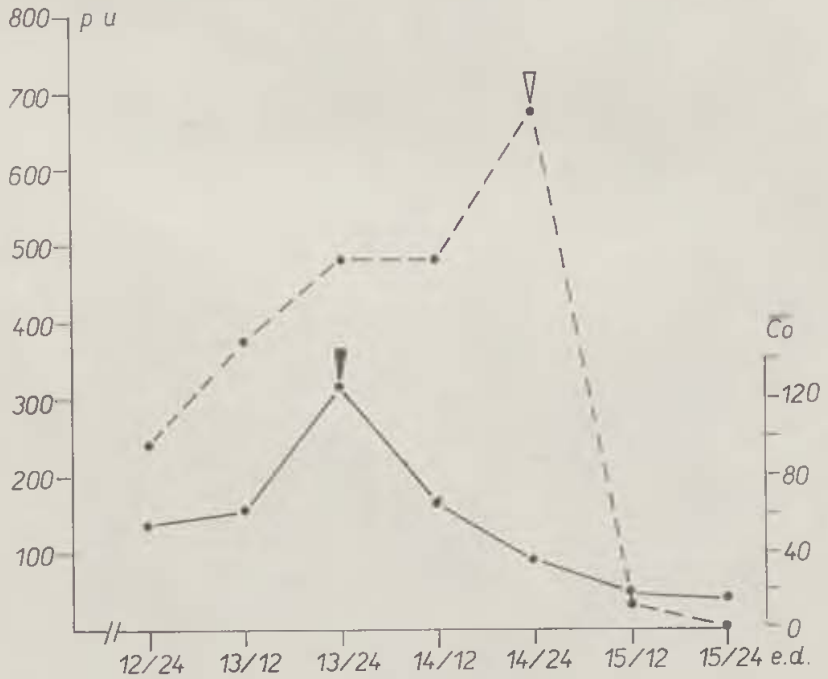

Fig. 1: Development of proliferation activity in the mouse palatal shelves in relation to time. Abscissa: day/hour of embryonic development, left ordinate: projection area of gap between palatal shelves in planimetric units (p. u.), right ordinate: index value of proliferation activity (Co) in the apical part of the palatal shelves. Dashed line: index of secondary palate development, empty arrow — point of palatal shelves horizontalization. Full line: changes in the index of proliferation activity, full arrow — absolute peak of proliferation. Curves link the values of medians.

Tab. 2

Stage	Position of proliferation centre			No proliferation centre
	anterior	middle	posterior	
12/24	+++			
13/12	++++			
13/24	+	+++		
14/12	+	++	+++	
14/24		+		+++
15/12				+++
15/24				+

In other words, the proliferation intensity during the formation of the secondary palate reaches its absolute maximum before horizontalization in the region where the palatal shelves will establish their first contact.

DISCUSSION

The relatively long interval between the maximum of proliferation activity (st. 13/24) and the mean point of palatal shelves horizontalization (st. 15/8 — Dostál, Jelínek, 1972) suggests that the cell proliferation itself is neither an essential nor immediate cause of horizontalization. On the contrary, it is quite obvious that in order to complete the morphogenetic movement of the palatal shelves a collusion of factors is necessary at that moment, most of them being more significant, such as release of the tongue from in between them. However, this in no case means that a disturbance in the proliferation processes cannot play a substantial part in the formation of cleft palate. Two facts can be quoted in support of this suggestion:

1) The period of maximum proliferation activity in the palatal shelves coincides with the peak of the sensitive period (to be more precise: with the period of the anticipated maximum concentration of corticoids within the embryo — Dostál, Jelínek, 1971) which, as will be generally known, inhibit proliferation (Seifert, Hilz, 1966).

2) Having compared the curve of the projection area between the palatal processes during the embryonic development following corticoid application with that of the control group, we find the values of the cleft-palate group to show significant differences only after horizontalization when the palatal shelves of the embryos likely to develop cleft palate leave a narrow gap between them to grow progressively in the course of further development (Dostál, Jelínek, 1973). Unlike most of the control group (95 %) the palatal shelves of the cleft-palate group fail to close in on horizontalization, a fact which suggests an absolute insufficiency. It would appear then that the mass of the palatal shelves is bound to assume a certain minimal volume by

Fig. 2: Progressive changes of the gap between the palatal shelves occurring in the course of normal development.

a certain critical point of development and to be used up after horizontalization in order to establish their contact along the midline. This contact appears to be facilitated by a conformation of the palatal shelves margins in between the vault of the nasal cavity, the septum and the dorsal part of the tongue. Unless the shelves have developed adequately, contact fails to take place by the time of the threshold point and with the continuing lateral growth of the head the gap between them goes on being irreversibly widened. Seen from this point of view, cleft palate can appear not only as a result of delayed horizontalization, as claimed by F r a s e r (1965), but also as a result of absolute insufficiency resulting again from a disturbance of the proliferation processes long before this point. Naturally enough, the "threshold" theory remains unaffected by that. Based on our experience, delayed horizontalization would require a much violent intervention and, as a result, would be a less frequent affair.

There is one great advantage to our hypothesis. It can be verified experimentally by measuring the proliferation activity in embryos with cleft palate, which is intended to be the subject another report. J. H.

SUMMARY

The colchicine method was used to record the course of proliferation activity in the palatal shelves and the adjacent mesenchyme of the maxilla in 25 embryos of H-Velaz mice during the period of formation of the secondary palate. The absolute maximum of the proliferation activity precedes the horizontalization of the palatal shelves by at least 24 hours which makes a direct and essential part of proliferation in this critical phase of palate formation highly improbable. In view of the fact that maximum proliferation activity takes place in the region of the future first contact of palatal shelves we believe an undisturbed course of proliferation processes prior to horizontalization to be an essential condition for developing a critical minimum volume of the palatal processes and thus enabling their fusion.

RÉSUMÉ

Rôle de l'activité mitotique au cours de la formation du palais secondaire

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Chez 25 embryons des souris H-Velaz, nous avons représenté à l'aide de la méthode de colchicin — dans la période de la formation du palais secondaire — le cours des actions prolifères dans les lamelles palatines et dans le mésenchyme voisin du maxillaire supérieur. Le maximum absolu de l'activité prolifère précède la formation horizontale des lamelles palatines le moins de 24 heures ce qui rend extrêmement improbable la participation directe et déterminante de la prolifération pendant cette phase critique en développement du palais. Du moment que le maximum de l'activité prolifère existe dans la région du contact prochain des lamelles dans la ligne médiale, nous supposons que le cours tranquille des actions prolifères avant la formation horizontale soit la condition indispensable pour atteindre le volume critique minimum des apophyses palatines qui permet leur réunion.

ZUSAMMENFASSUNG

Die Rolle der mitotischen Aktivität bei der Gestaltung des sekundären Gaumens

Jelínek R., Dostál M.

Bei 25 Embryonen von Mäusen des H-Velaz-Stammes haben wir während der Gestaltung des sekundären Gaumens mittels der Kolchicinmethode den Verlauf der Proliferationsvorgänge in den Gaumenscheiben und in dem unmittelbar anliegenden Maxillenmesenchym abgebildet. Der absolute Höhepunkt der Proliferationsaktivität erscheint um mindestens 24 Studen früher als die Horizontalisierung der Gaumenscheiben, wodurch die direkte und entscheidende Teilnahme der Proliferation in dieser kritischen Phase der Gaumenentwicklung höchst unwahrscheinlich wird. Angesichts der Tatsache, dass das Maximum der Proliferationsaktivität im Bereich des nächsten Kontaktes der Scheiben in der Mittellinie vorkommt, nehmen wir an, dass der ungestörte Verlauf der Proliferationsvorgänge vor der Horizontalisierung eine unerlässliche Bedingung für das Erreichen des kritischen minimalen Volums der Gaumenausläufer bildet, die ihre Verschmelzung ermöglicht.

RESUMEN

Papel de la actividad mitótica durante la formación del paladar secundario

Jelínek R., Dostál M.

El transcurso de las acciones prolíferas en las láminas palatales y en el mesenquimo inmediatamente adyacente del maxilar fue representado por el método con colchicina en 25 embriones de ratones H. Velaz. El máximo de la actividad prolífera precede a la formación horizontal de las láminas palatales de 24 horas por lo menos, lo que hace la participación directa y decisiva de la proliferación en ésta fase crítica de la evolución del paladar sumamente improbable. Tomando en cuenta que el máximo de la actividad prolífera ocurre en el área del contacto futuro de las láminas en la línea central, suponemos que un transcurso no perturbado de las acciones prolíferas antes de la formación horizontal es condición imprescindible para alcanzar el volumen mínimo crítico de las apófisis palatales que hacen posible su fusión.

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PLASTIC OPERATION ON URINARY BLADDER IN NEUROGENIC DISORDERS OF MICTURITION (Communication I)

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Neurogenic disorders of micturition are a collective syndrome of functional disorders which develop due to damage to the brain and spinal cord, the peripheral ganglia, nerves and the intramural nervous plexus.

The syndrome is based on an interruption of the reflex arch of the act of micturition, which may develop along any of its parts, starting at the peripheral receptors and finishing in the cortical analyzers, and is usually connected with an injury, burns, tumours and congenital disorders of the spinal cord or the peripheral nerve structures.

Thus the aetiology of neurogenic disorders of micturition is polyhedral, while the mechanism of these disorders may be arranged into quite a simple scheme: injuries affecting nerve routes, intraspinal and extraspinal ganglia and nerves.

From a practical point of view it is essential that in the interruption of urinary-bladder innervation, the reservoir and evacuatory function of the urinary bladder is interrupted immediately, no matter the primary factors which have led to the disorder.

This is met with quite frequently, a pathogenic plan of the treatment of neurogenic disorders of micturition, which would ensure renewal of the reflex arch of the act of micturition, however, has not yet been elaborated.

In the authors' opinion, restoration of urinary-bladder innervation by organopexy, the principles of which have been laid down by the Belorussian morphologists headed by Golub, may be the way to elaborating such a pathogenically founded treatment of neurogenic disorders of micturition. These authors have shown that new nerve routes form across the contact area between the small intestine and the urinary bladder sutured to it, which are capable of ensuring the roundabout connections of the urinary bladder with the central nervous system. In other words, a true reinnervation of the urinary bladder takes place. These studies on the reinnervation and quite numerous investigations on the revascularization of intraabdominal organs (Kirillov; Pytel; Makshanov; Doletsky et al.) have created a real foundation for the pathogenic treatment of neurogenic disorders of micturition.

With the aim of elaborating a clinical variant of the operation, the authors of this communication carried out seven series of experiments on 71 animals (dogs). In the first two series, models of spinal and peripheral denervation of the urinary bladder were elaborated, in series I the spinal cord was severed at the level between the XIIth thoracic and Ist lumbar vertebrae, in series II the peripheral nerves and vessels of the urinary bladder were severed. In the following series (III to VII), the different variants and sources of urinary bladder reinnervation were studied, as well as the functional competence of the newly formed routes of reinnervation. In series III, an integral part of the small intestine was sutured to the urinary bladder, in series IV, the urinary bladder was wrapped around with omentum, in series V it was wrapped into the mesentery of the small intestine. In series VI and VII, vesicopexy was carried out with an isolated segment of small intestine stripped of its mucous membrane, actually a segment of ileum, 40—50 cm. away from the ileocaecal angle. The way the intestine was stripped of its mucous membrane was different in the two series. In series VI, the mucosa was scaped off the isolated small-intestine segment after slitting it open by a longitudinal incision and spreading it out. In series VII, the isolated segment of intestine was stripped of its mucosa by the method elaborated by the authors of this investigation, which is based on the peeling-off of the membrane at the level of the submucous layer without opening the lumen. Such aseptic preparation has the decisive advantage of making it possible to connect homogeneous muscle layers of intestine and bladder and avoid the danger of the formation of mucous cysts.

The results of series I and II (controls) showed that spinal as well as peripheral denervation of the urinary bladder leads to severe septico-neural changes in the organs of the urinary system and may finally cause death of the animal from urosepsis.

The physiological and morphological findings in the remaining series (III to VII) of experiments showed that suturing of an isolated and laid-out segment of small intestine stripped of its mucosa by the authors' method to the urinary bladder gave the highest indices in cytometry and that intravesicular pressure rose when the mesentery of the isolated intestinal segment was stimulated with electric current. This was evidence of high physiological properties of the newly formed nerve routes. Apart from this, in the zone of contact between the intestinal segment and the urinary bladder, a denser vascular network was found than in the animals of the other series of experiments, and also a larger number of various nervous elements, such as nerve endings, nerve fibres, bundles and cells. Usually, nerve elements are found equally frequent in the entire area of union. All this is undoubtedly evidence of the advantage of vesicopexy using a laid-out segment of small intestine stripped of its mucosa by the authors' method over the other methods of reinnervation.

The final solution of the problem of introducing the method into clinical practice required elucidation of the compensatory possibilities of nerves and vessels of the area of union under conditions of urinary-bladder denervation.

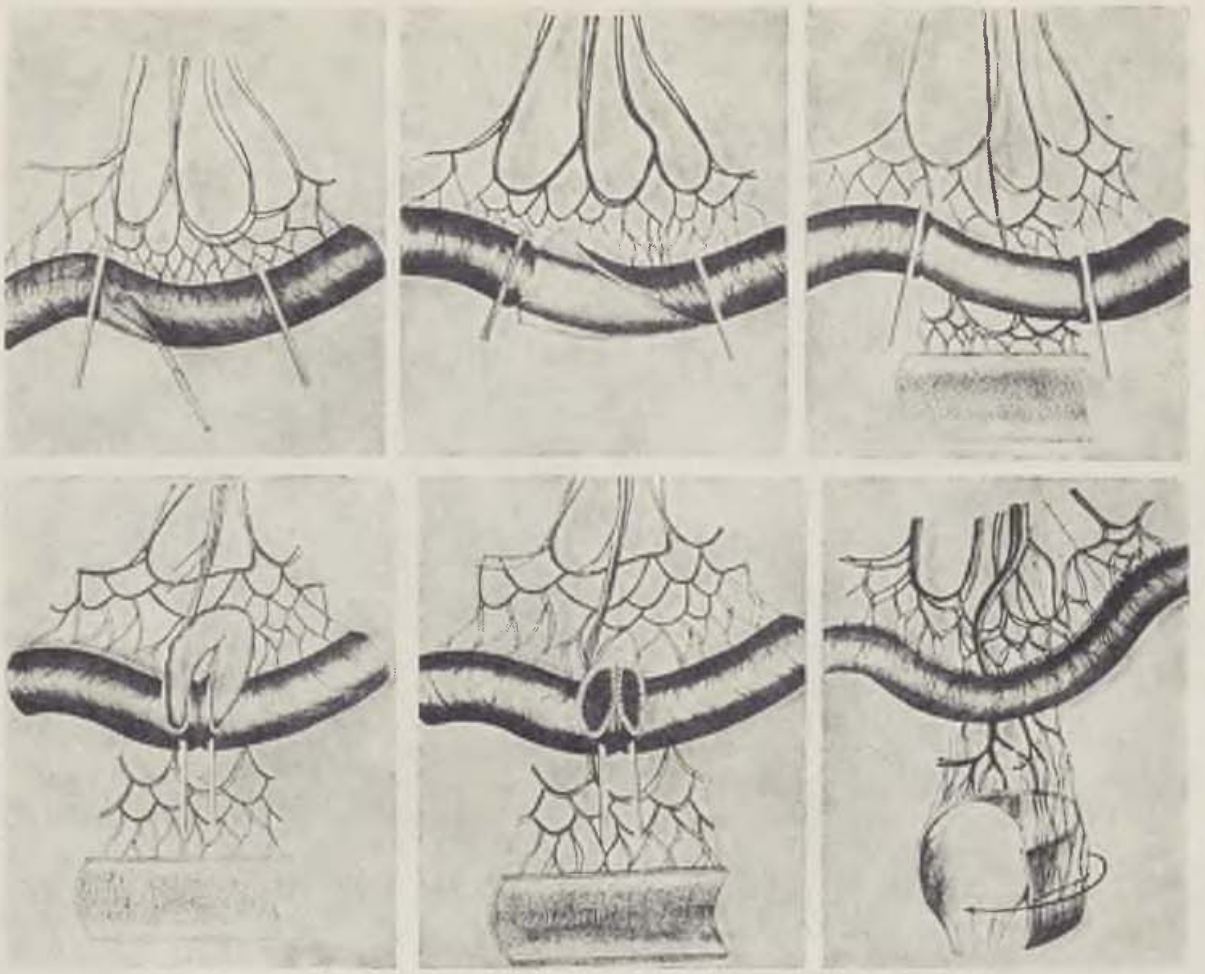

Fig. 1. Diagram of ileovesicopexy

For this purpose, the function of the urinary bladder after suturing various organs and tissues to it was investigated after subsequent denervation. It was ascertained that suturing of a laid-out segment of small intestine stripped of its mucosa led to results which best met the aims the authors had set themselves at the beginning. In the method, the rich source of additional innervation is combined with the blood supply of the large contact area of the donor with that of the recipient organ. This is evidently the reason why trophic changes in the denervated bladder developed much less than in the animals of the other series of experiments, restoration of bladder function was more complete and the rate of animal survival much higher. Apart from this, an important index of the advantage of the method over all other methods of vesicopexy is presented by the highest findings in cytometry, as carried out after denervation of the urinary bladder. It should be pointed out that the intestine flap which had been sutured to the urinary bladder, was not only a source of reinnervation and revascularization, but also included muscle tissue which might have been the source of reinforcement of the detrusor urinae. This ileovesicopexy as carried out in experiments has led to restoration of urinary-bladder function in dogs with subsequent peripheral denervation, which justified the authors

to try and elaborate a surgical method for clinical application in the treatment of patients with neurogenic disorders of micturition.

The surgical procedure of ileovesicopexy as employed in patients is based on the following (Fig. 1): The abdominal cavity is opened by an incision be-

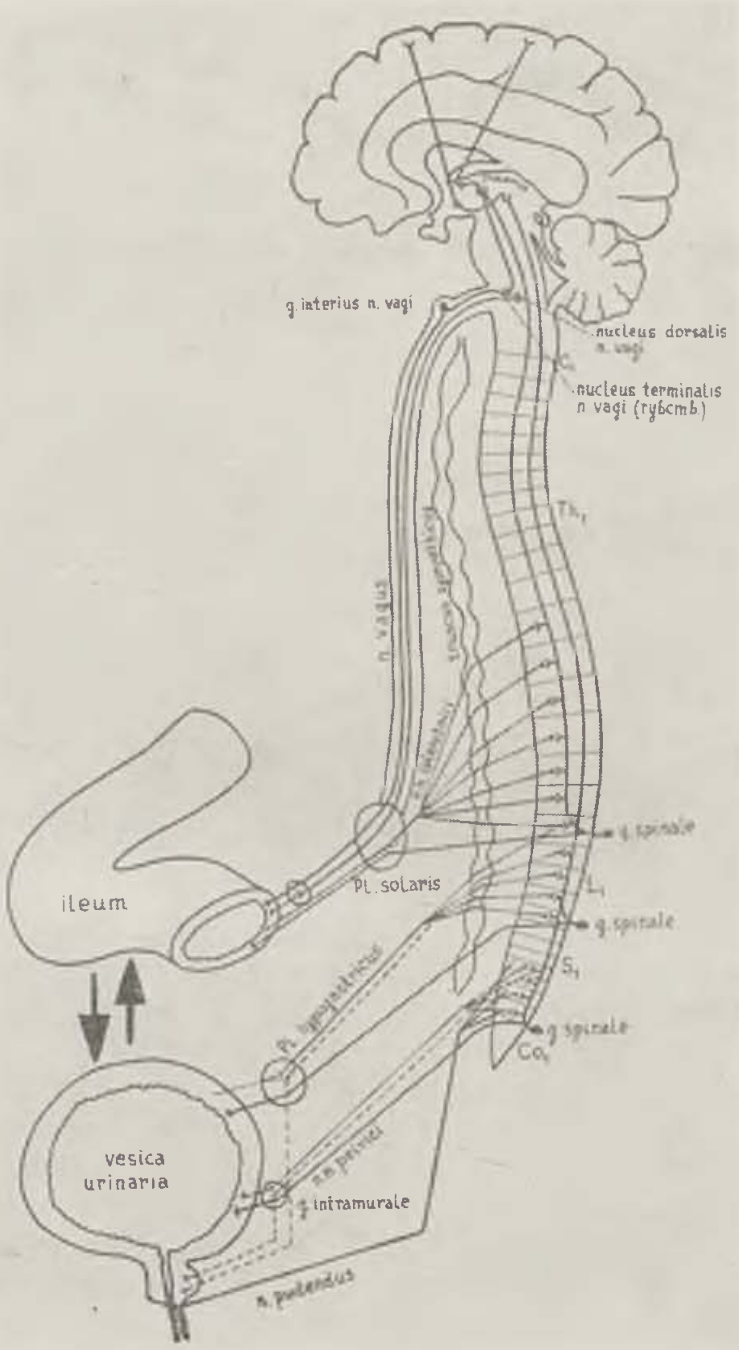

Fig. 2. Diagram of urinary-bladder reinnervation

tween the mons pubis and umbilicus, 40—50 cm. in length, a segment of small intestine is chosen from the ileocaecal angle, 15 cm. in length, with a well developed central vessel and a long enough mesentery. Two small windows are made in the mesentery close to the intestine and the latter is clamped with

soft intestinal clamps. A mesenteric pedicle of the isolated segment of intestine with a central vessel is carefully dissected. A longitudinal incision is made on the free margin of the isolated intestine, which penetrates the serosa and muscle layers, avoiding injury to the mucous membrane or opening the intestinal lumen. The intestinal wall is then divided into two layers by partly blunt and partly sharp dissection. The mucous tube blocked up from two sides is removed without opening the intestinal lumen, and the vessels are ligated with thin catgut. After complete separation of the muscular layer, the serosomuscular flap with the mesenteric pedicle is transferred to the back of the small intestine. The window in the mesentery is carefully closed by a silk suture down to the mesenteric root. The clamps are then approximated and the two stumps of intestine are anastomosed end-to-end. The device for mechanical suture of intestine may also be used for the purpose.

The urinary bladder is stripped of its peritoneum, and completely emptied, because fixation of the transplant must be carried out on the empty bladder. The union of the intestinal flap with the bladder, which takes place over a large area, would otherwise diminish bladder capacity. The mesentery of the transplant is fixed to the posterior parietal peritoneum and the transplant itself led out into the perivesicular space. The peritoneum is sutured around the pedicle and the flap is clapped over the empty bladder like a helmet, its edges sutured to it, and scattered sutures laid in a chessboard manner over the entire area of contact. The perivesicular space is drained with chlorvinyl tubes. Urine is drained through a retaining catheter or a suprapubic drainage tube.

Between 1962 and 1972, ileovesicopexy was carried out by the authors of this communication in 43 patients who had suffered from neurogenic disorders of micturition. The results of the experiments and the clinical experience permit us to determine the indications for the operation as disorders in contraction of the urinary bladder and the loss of the urge to urinating.

However, interruption of the reflex arch in the afferent or efferent sections is common to all these forms of disorders in which the operation is indicated. So it is in patients with neurogenic disorders of micturition based on affections of the spinal cord, provided the damage has not involved the centres of small intestine innervation, and also disorders of micturition based on injuries to extraspinal nerves or ganglia of the bladder. The diagram of reinnervation is shown in Fig. 2.

In 41 of the 43 operated on patients, the postoperative period was uneventful, but in two there were complications; one had to submit to another operation for peritonitis three days after ileovesicopexy, and after that the postoperative period was no more complicated. The patient eventually became well and was discharged home in good condition. In the second patient, ileus developed four days after ileovesicopexy, which required a by-pass anastomosis and a temporary jejunostomy. This patient, too, became well and was discharged home in satisfactory condition. However, it must be stated that these complications were observed in the first stage of employing the operation in clinical practice, which probably may be connected with first getting acquainted with many details of the method.

Early postoperative results were appraised according to the following criteria: restoration of the urge to urinating, micturition at will, change in the amount of residual urine, influence of the treatment on inflammatory complications in the kidneys, their function and the general condition of the patient.

Impairment of general condition and of micturition have not been observed after operation.

A full clinical success (restoration of the urge to urinating and micturition at will) was achieved in 31 patients, partial effect (restoration of the urge to urinating and micturition after the bladder had been filled to the maximum) was registered in nine patients. In three patients, their conditions remained unchanged after operation. The first signs of restoration of the urge to urinating became manifest already eight to ten days after operation, which coincides with the time, when copious vessels are bridging the site of union between the transplant and the bladder wall. Restoration of the urge to urinating is completed by the 20th to 25th day after operation.

Two patients died ten to 36 months after operation, in spite of restoration of the urge to urinating, from progressive chronic kidney defficiency and nephrogenic hypertension.

In these patients, the operation proved too late and could not arrest the progressive complications developing in other organs. The histological examination carried out in these patients post mortem showed numerous vessels and nerves without any signs of degeneration at the site of union between the transplant and the bladder wall.

Thus experimental as well as clinical observations have furnished evidence that the nerve elements at the site of union are of full value, because both the afferent (urge to urinating) and efferent (micturition at will) innervation is restored. The findings referred to above bear witness of true reinnervation of the urinary bladder by ileovesicopexy, which, therefore, is recommended by the authors of this communication for the treatment of neurogenic disorders of micturition.

B. K.

SUMMARY

With the aim of elaborating a pathogenic foundation for the treatment of neurogenic disorders of micturition, the authors have carried out seven series of experiments on 71 animals. The result of investigating the function of the urinary bladder under conditions of peripheral and central denervation and after suturing to it an isolated segment of ileum stripped of its mucosa has shown that nervous routes capable of ensuring the function of the denervated bladder can be restored. As a result of the morphological and physiological findings, the method of ileovesicopexy was elaborated and introduced into clinical practice.

The operation has been carried out in 43 patients suffering from neurogenic disorders of micturition. Complete clinical success characterized by restoration of the urge to and control of micturition was achieved in 31 patients, in nine patients success was partial and in three patients the conditions after operation

remained the same as they had been prior to it. These findings have furnished evidence of the possibility of true reinnervation of the urinary bladder via the method of ileovesicopexy, which, therefore, is recommended by the authors for the treatments of neurogenic disorders of micturition.

RÉSUMÉ

Opérations plastiques sur la vessie pendant les troubles neurogènes de la miction

Savtchenko, N. J., Mokhort V. A.

Pour établir le plan du traitement des troubles de la miction, les auteurs ont fait sept séries d'expériences sur 71 animaux. Le résultat des études de la fonction de la vessie dans les conditions de la dénervation périphérique et centrale et après la fixation à celle-ci du segment isolé de l'iléon privé de la muqueuse a montré que les connections nerveuses se renouvelaient et devenaient capables d'assurer la fonction de l'organe dénervé (la vessie). Sur la base des faits morphologiques et physiologiques constatés on a établi les méthodes opératoires de l'iléovesicopexie et on les a introduites dans la pratique clinique.

Cette opération a été entreprise sur 43 malades souffrant de troubles neurogènes de la miction. Au point de vue clinique on a réussi — en renouvelant l'impulsion à la miction et en réglant cette fonction — chez 31 malades totalement, chez 9 partiellement, tandis que chez 3 malades leur état ne s'est pas changé après l'opération. Ces faits prouvent la possibilité d'une reinnervation effective de la vessie par l'iléovesicopexie. C'est pourquoi les auteurs la recommendent pour traiter les troubles neurogènes de la miction.

ZUSAMMENFASSUNG

Plastische Operationen an der Harnblase bei neurogenen Harnstörungen

Sawtschenko N. J., Mochort V. A.

Zwecks der Zusammenstellung eines Planes für die Behandlung der neurogenen Harnstörungen unternahmen die Autoren sieben Serien von Versuchen an 71 Tieren. Das Ergebnis der Untersuchung der Harnblasenfunktion unter den Bedingungen der peripheren und zentralen Denervation und nach Sutur des isolierten und von der Schleimhaut befreiten Iliumsegmentes hat gezeigt, dass sich die Nervenverbindungen wiederherstellen und fähig werden, die Funktion des denervierten Apparates (Harnblase) zu sichern. Auf Grund der ermittelten morphologischen und physiologischen Tatsachen wurde die Operationstechnik ausgearbeitet und in die klinische Praxis eingeführt.

Die Operation wurde bei 43 Kranken mit neurogenen Harnstörungen unternommen. Ein voller klinischer Erfolg, der durch die Wiederherstellung des Harnzwanges und Besserung dieser Funktion bestätigt wurde, wurde bei 31 Kranken erzielt, ein Teilerfolg bei 9 Kranken und bei drei Kranken blieb der Nachoperationszustand wie vor der Operation. Diese Tatsachen bestätigen die Möglichkeit reeller Reinervation der Harnblase durch Ileovesikopexie, die deshalb von den Autoren zur Behandlung der neurogenen Harnstörungen empfohlen wird.

RESUMEN

Operaciones plásticas de la vejiga urinaria en perturbaciones neurógenas de la micción

Savchenco N. J., Mojort V. A.

Con el fin de establecer el plan de la terapéutica de las disturbaciones neurógenas de la micción los autores llevaron a cabo siete series de experimentos en 71 animales. El resultado del estudio de la fonción de la vejiga urinaria bajo las condiciones de una desnervación periférica y central y después de haber fijado a la misma un segmento aislado del íleon desprovisto de la mucosa mostró que las conexiones nerviosas se restituían y se hacían capaces de suplir la fonción del órgano desnervado (de la vejiga urinaria). En base de las condiciones morfológicas y fisiológicas constatadas fueron establecidos los métados operativos llamados íleovesicopexia e introducidos en la práctica clínica.

La operación fue performada en 43 pacientes con disturbaciones neurógenas de la micción. Un suceso clínico total, que fue comprobado por la restitucion del deseo de orinar y por el arreglo de esta fonción, fue conseguido en 31 pacientes, un suceso parcial en 9 y en 3 pacientes el estado después de la intervencion fue igual como antes de la misma. Estas realidades comprueban la posibilidad de una reinervación efectiva de la vejiga urinaria por íleovesicopexia, la cual por lo tanto es recomendada por los autores para la terapia de las disturbaciones neurógenas de la micción.

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MORTALITY IN BURN INJURY OVER A PERIOD OF TEN YEARS

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INTRODUCTION

In spite of up to date views and the advancement of management, burns are still the cause of a high percentage of deaths. Sprechler and associates cite in their statistical survey a mortality of 3,3 %, Tumbusch 20,5 %, and Hayes 32,0 % (Birke) (1).

The mechanism of death in burn injury has a variety of causes: heart arrest, oligemic shock, acute respiratory or renal insufficiency, high fever, bacterial septicaemia, pulmonary embolism, haemorrhages, necrosis of the liver, and so forth. It is considered today that the outcome of burns depends on a number of factors: the localisation, extent of burned surface, depth of burn, general condition prior to injury, age, individual resistance, and therapy.

The occurence of death in burn injury can be divided into early and delayed, early death taking place during the first 24 hours, or 3—4 days, whilst a delayed one can occur after several days, weeks or even years.

Deadly outcome in our patients

Of 329 patients treated at the department of Surgery in Zagreb in the period of ten years (1958. to 1968.), 17 died (5,16 %). Our youngest patient whose death was a result of burns was aged 1 year, the oldest 85 years. In cases of death the percentage of burnt body surface amounted from 21 % to 73 %. In all of these patients the injuries were deep (Tab. 1).

Tab. 1. Relation between age and percentage of burnt body surface

Age of patient	Burnt body surface in %			
	up to 30 %	31—50 %	51—70 %	above 70 %
Under 10 years	0	2	1	0
11—20	0	0	0	0
21—40	1	4	1	0
41—60	0	1	0	1
Above 61 years	1	4	1	0

In our patients the most frequent cause of burns was flame, and in one case the burns of the upper respiratory airways were due to gasoline vapours catching fire (Tab. 2).

Death occured earliest in patients with a greater percentage of burnt body surface, and in cases of deeper burns. The number of days of survival was lowest in children up to 4 years and in persons of more than 70 years (Tab. 3).

Tab. 2. Causes of burns and scalds in our patients

Flame	13
Hot water	1
Hot iron	1
Caustic soda	1
High-voltage electricity	1
Total	17

Death was due, in the majority of cases, to one or several complications. All cases of death were transfered to the Institute of Forensic Medicine, where autopsies were performed, except in three cases where the family was opposed. The result of the autopsies is shown in Tab. 4.

DISCUSSION

During the first hours or days following burn injury, death may occur due to acute heart failure or to periphereal circulation failure-shock (2, 3).

Heart failure is encountered in older persons (4) or in cardiac or pulmonary patients. The pathologic finding in the heart consists of fatty or hyalin degeneration with smaller haemorrhages into the muscle. Forster (5) describes a finding of myocarditis, as we found in one case, and Pack (6) underlines right heart dilatation, as autopsy revealed in four of our patients. Two patients died with signs of burn shock. Burn shock used to be the major cause of death,

Tab. 3. Age, percentage, and days of survival in our cases of death

Age	%	Days of survival	Age	%	Days of survival
1	40	4	40	40	163
3	50	6	58	40	28
6	55	64	65	30	4
25	40	179	65	30	3
29	70	5	68	45	81
32	40	10	71	40	67
33	30	5	84	50	2
37	40	29	85	50	4
39	73	3			

with onset 6—12 hours following burns, sometimes up to 3—4 days. It is encountered in deep burns with a percentage of burnt body surface above 10 % (in children), or 15 % (in adults) (7). The mortality due to burn shock shows a decline in the last 20 years thanks to current anti-shock therapy. In superficial burns the development of shock is due to pain, while in deep ones it is due to the loss of fluid. The greatest loss of fluid takes place in the interval of the first 12 hours after which transudation decreases. Transudation is the result of augmented capillary permeability of the burnt skin area. Consequently for the loss of plasma, oedema develops, resulting in hemoconcentration. Cardiac output decreases, blood flow is diminished, and there is a drop in oxygen transport due to vasoconstriction. Doubtless, the circulatory function is affected by autonomic neural factors. Oxagen transport is all the lower in cases of acute anaemia. Ullmann (8), and Matthewes (9) indicate the maximal irritability of sensitive nerve endings in the burnt area as being responsible for shock. Prinzmetal and Bergmann (2) consider it to be due to fluid loss, while other authors mention a variety of unknown factors. Green (10) sums all three factors together, burn shock beeing caused by: fluid loss, normal and abnormal metabolic products, and reflex neurogenic mechanisms.

Tab. 4. Direct cause of death caused by burns

1. Inflammative changes (lungs or similar)	7
2. Cardiac failure	3
3. Loss of Blood	2
4. Shock	1
5. Acute failure of suprarenal gland	1

Acute respiratory insufficiency is most often encountered in children where pulmonary oedema develops because of fluid overloading during treatment, or due to the disbalance of electrolytes. In some cases death takes place suddenly in a state of acute respiratory disturbance, but it more often occurs after several days following infection and state of bronchopneumonia. This was the finding revealed by autopsy in more than 50 % of our cases. In acute death the lungs present a purple hue, they are congested, oedematous to a certain extent, and contain almost no air at all. In other cases there was greater oedema, as we found in five deaths due to burns.

When there is a fire in a closed space, burns can be caused by inhaling flames, smoke, or hot air. Such was the case of one of our patients who had acquired burns due to ignited gasoline vapours, and the autopsy performed revealed a muco-purulent tracheobronchitis and confluent bronchopneumonia of the left lung. Inhalation of poisonous gases and vapours formed during incomplete combustion, leads to severe tracheobronchitis which may develop into pneumonia, or may be the cause of death due to an acute pulmonary oedema (3).

In 50 % of our deaths, acute renal insufficiency was clinically determined. In three of seven cases the pathologic substrate was confirmed by histology,

determining it to be parenchymatous changes or granular atrophy of the kidney. In the majority of cases, acute necrosis of the proximal and distal convoluted tubules is found, which does not necessarily have to be the cause of renal failure. Decreased glomerular filtration is brought about by acute hemodynamic and renal vascular effects, caused by oligemia and hemoglobinuria. It is considered, today, that the cause of renal insufficiency is septicaemia and toxaemia by gram-negative microorganisms. It is necessary to mention that the prognosis is grave even when hemodyalisis is applied.

Hyperpyrexia appears sometimes, usually between the second and the sixth day. It was registered in six of our patients, mostly in children. It's clinical manifestation is a sharp temperature peak, fast pulse rate, vomiting, or signs of an acute inflammatory process, that is, toxaemia. It is most frequently due to septic invasion from the infected wound site, to extensive fluid losses, to toxic disturbances of the thalamic thermoregulatory center, and to loss of functional skin.

We had clinically manifested encephalopathies in 11 patients. It's occurence is more frequent in children, and it's signs were convulsions and coma. In seven cases the pathologic finding was cerebral oedema, in one case tuberculous meningitis and encephalomalacia. Besides hyperemia, oedema with focal necrosis and thrombosis of cerebral arteries and veins was found.

Kolisko (11) mentions certain changes of the adrenal glands in burns, and Berkow (12) describes haemorrhages and haemorrhagic infarction of the core initially, while later it can be found also in the medulla. It is, however, considered a rarity that adrenal insufficiency should be the cause of death. Changes in the adrenal gland were found in three autopsies; in two cases they were haemorrhages, in one case atrophic changes. In one case the cause of death was considered to be the change in the adrenal gland by the performer of the autopsy. It should be mentioned that in cases of death atrophy is rarely found, while it is much more frequent to find hypertrophy because of hypersecretion of the medulla and cortex. Atrophy could be explained by drawn out cachexia, as we found in one patient who died after two years.

Nonbacterial toxaemia often accompanies a bacterial one. Nonbacterial toxaemia is brought about by apsorption of toxic products from the burnt site, and by incomplete proteolysis because of disturbances of oxydative processes due to anoxia and acidosis. As renal functions and that of the liver are impaired, the organism is incapable of detoxication and elimination of toxins, which thus cumulate. Three pathologic findings were parenchimous degenerative changes in the liver, while fat infiltration of the liver was found in two other cases together with parenchimous renal changes.

In the past, death resulting from bacterial infection was less frequent, because oligemia was the cause of death in the majority of cases. As modern shock treatment progressed, the main cause of death was shifted to bacterial infection (Sevitt 63 %) (3). The most frequent bacterial complications are septicaemia, bronchopneumonia, and pyelonephritis. Less often are pyaemia, ulcerous endocarditis, or meningitis. Tetanus is very rare thanks to pro-

phylactic measures. Artz (Birke) (1) presented in 1963 a survey of 1178 cases, in which 50 % of the 224 death cases was due to sepsis. In our material septicaemia was found in four cases, bronchopneumonia in seven, in five of which bilateral. In one case the clinical signs were dominated by tubercoulous meningitis, as was confirmed by autopsy. The main cause of bacterial complications is burnt skin, from where infection spreads by way of lymphatic or blood vessels, mainly during the first two weeks.

The most common causative agents of infection were: Staphylococcus pyogenes aureus, B. coli, Proteus, Klebsiella, and other coli bacteria, which we determined by aid of wound smears and hemocultures. Hemolytic Streptococcus is encountered less often nowadays, due to widespread use of antibiotics (Penicillin).

Of the remaining complications pulmonary embolism is often mentioned (13). Only one of our patients died showing clinical signs of pulmonary embolism, which it was impossible to prove by autopsy. Acute ulceration with bleeding was found in two cases. In one patient we found such contents in the stomach, while in another the mucose was massively eroded together with bleeding and necrotic changes of the terminal ileum and of the ascending colon. Acute ulcers of the digestive tract are more often mentioned by many authors. They are more frequent in the older age groups. In children they are found in the first week following burns, while in adults they appear during the third week.

SUMMARY

Of the 329 treated patients during a ten year period, 17 died, which amounts to 5,16 % of the whole. The burnt body surface extended from 21 to 73 % of the total body surface, and in the majority of cases the burns were estimated as deep ones. Death following burns was dependent of the extent, depth, localisation, age, preexisting diseases, and treatment. The days of survival were markedly shorter in children under 4 years of age, and in older persons above 70 years. In many instances death was due to two or more factors or complications, while in a low number of cases it occured without apparent complications. The complication most often found was bronchopneumonia, extending usually over both lungs. It was most often caused by resistant agents. Second in frequency was heart failure, while acute bleeding was encountered in two cases, due to acute ulcerations of the digestive tract.

RÉSUMÉ

Mortalité par suite des brûlures en l'espace de 10 ans

Vladović-Relja T., Montani D., Zečević D.

Du nombre total de 329 malades traités par suite des brûlures en l'espace de 10 ans, 17 sont morts, c'est-à-dire 5,16 %. D'après l'extension de la zone atteinte, les brûlures occupaient 21 à 73 % de la surface générale du corps et elles étaient appréciées comme profondes. C'étaient l'extension et la profondeur de la lésion, la localisation, l'âge, les maladies précendentes et le traitement qui conditionnaient les décès

par suite des brûlures. Le temps de survie était particulièrement plus court chez les enfants plus jeunes de 4 ans et chez les personnes qui ont passé 70 ans. Chez la plupart des malades le décès résultait des deux ou plusieurs facteurs ou complications, tandis que les décès sans rapport évident avec certaines complications étaient plus rares. Les complications les plus fréquentes des brûlures étaient les bronchopneumonies qui affectaient d'habitude les deux poumons et qui avaient été provoquées — dans la plupart des cas — par les souches microbiennes résistantes. A la deuxième place étaient les insuffisances cardiaques; l'hémorragie aiguë causée par l'exulcération dans le tube digestif était constatée dans deux cas.

ZUSAMMENFASSUNG

Die Sterblichkeit an Verbrennungen während einer Zeitspanne von zehn Jahren

Vladović-Relja T., Montani D., Zečević D.

Von der Gesamtzahl von 329 Kranken, die in der Zeitspanne von 10 Jahren wegen Verbrennungen behandelt worden sind, sind 17, d. h. 5,16 % gestorben. Nach dem Umfang der betroffenen Fläche umfassten die Verbrennungen 21 bis 73 % der Gesamtkörperoberfläche und in meisten Fällen wurden sie als tief beurteilt. Der Tod infolge von Verbrennung war von dem Umfang und der Tiefe der Schädigung, der Lokalisierung, dem Alter und der vorangehenden Erkrankung und Behandlung bedingt. Signifikant kürzer war die Überlebungszeit bei Kindern unter 4 Jahre und bei Personen über 70 Jahre. Bei der Mehrzahl der Kranken wurde der Tod von zwei oder mehreren Faktoren oder Komplikationen verursacht, während die Zahl der Todesfälle ohne offensichtlichen Zusammenhang mit einer Komplikation geringer war. Die häufigste Komplikation bei der Verbrennungen waren Bronchopneumonien, die in der Regel beide Lungen betroffen haben und die meist durch resistente Mikrobenstämme hervorgerufen waren. An zweiter Stelle befanden sich Herzversagungen; akute Blutungen, verursacht durch Geschwürbildung im Verdauungstrakt, wurden in zwei Fällen beobachtet.

RESUMEN

Mortalidad a consecuencia de quemaduras durante un período de 10 años

Vladović-Relja T., Montani D., Zečevic D.

Del número total de 329 pacientes tratados a consecuencia de quemaduras durante el período de 10 años murieron 17, esto es 5,16 %. Según la extensión del área afectada las quemaduras tomaron 21 hasta 73 % de la superficie total del cuerpo y en la mayoría de los casos fueron estimadas como profundas. El fallecimiento a consecuencia de la quemadura fue condicionado por la extensión y la hondura de la lesión, por la localización, edad, enfermedades previas y por terapia. Los niños menores de 4 años y las personas mayores de 70 años sobrevivieron por un período significantemente más corto. En la mayoría de los pacientes la muerte fue causada por dos o más factores o complicaciones, mientras que hubo menos casos de muerte sin conexión algúna evidente con unas complicaciones. Las complicaciones más frecuentes en las quemaduras eran las bronquioneumonías que generalmente afectaron los dos pulmones y fueron provocados por cepas resistentes de microbios en la mayoría de los casos. La insuficiencia del corazón se halló en el segundo lugar; una hemorragia acuta causada por la ulceración en el aparato digestivo fue constatada en dos casos.

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CLINICAL MANIFESTATION OF THE IMPAIRMENT OF THE LIVER IN THE COURSE OF THE BURN DESEASE

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Schjerning (1884) and Bardeen (1897) were the first investigators to give full evidence of certain liver disorders encountered in the course of burn already at the end of the last century (quoted according to 4). The question was later envisaged from the anatomical (5, 6, 12), experimental (10, 13) and clinical points of view in several clinical studies (1, 2, 4, 7, 8, 9, 11).

However, it seems necessary to emphasize that the proper role of the liver in the course of thermic burns could not be correctly evaluated in its right significance before a new approach to the whole problem of thermic injuries of the skin had been developed. A thermic injury is considered nowadays not only as a local process, but as a general disease which affects all vital functions of the living organism. Furthermore, the progress made by the improvement of some basic therapeutic rules and of highly elaborated functional liver tests in the broad sense of the word, has enabled a more profound assessment of the whole problem.

In the following study the authors wish to present the results of their own observations gained on 40 patients treated in the department of intensive care of Burn Unit in Prague from 1. I. 1971 to 31. XII. 1972.

The hepatic lesions were studied from the clinical, laboratory and histological point of view.

Tab. 1. illustrates the distribution of all patients according to their age and sex. The average age of men was 32,7 years, that of women 41.1. The extent of observed skin burns — estimated mostly as injuries of IInd to IIIrd degree and rarely of IVth degree — amounted to 46,5 % of the body surface in men and 54,4 % in women.

12 persons from a total of 40 patients followed up in the present study died mainly as a sequel of septicaemia, toxaemia or circulatory failure, relatively rarely in shock or as a result other complications (Tab. 2).

In this connection it must be emphasized that the extent of the skin burn was clearly larger in deceased persons of both sexes, than in the group of

patients who survived (in deceased men the extent of the skin burn amounted to 50,5 %, in women to 56,0 %).

Special attention was paid to an eventual previous injury of the liver. No anamnestic data could be obtained in one case of a patient transported to the department in a critical state. He died within the first 24 hours. A previous viral hepatitis was reported in 4 instances, diabetes mellitus was present in 3 patients before injury. 11 patients confessed an intermittent or regular alcoholic abuse.

Tab. 1. Distribution of patients according to their age and sex and enumeration of fatalities

	1 to 10	11 to 20	21 to 30	31 to 40	41 to 50	51 to 60	61 to 70	71 to 80	81 to 90	Total	Fatalities
Men	1	6	13	6	1	2	—	2	1	32	8
Women	—	1	1	1	3	1	1	—	—	8	4
Total	1	7	14	7	4	3	1	2	1	40	12

The origin and clinical manifestation on liver damage always coincided with the appearance of well-known complications of the burn, i.e. with the symptoms of shock first of all, then with the occurence of septicaemia or toxaemia. In the initial stage, when symptoms of shock prevailed and stood in the foreground of the whole clinical picture, signs of a developing heapatic

Tab. 2. Causes of death in 12 patients who suffered from burn disease

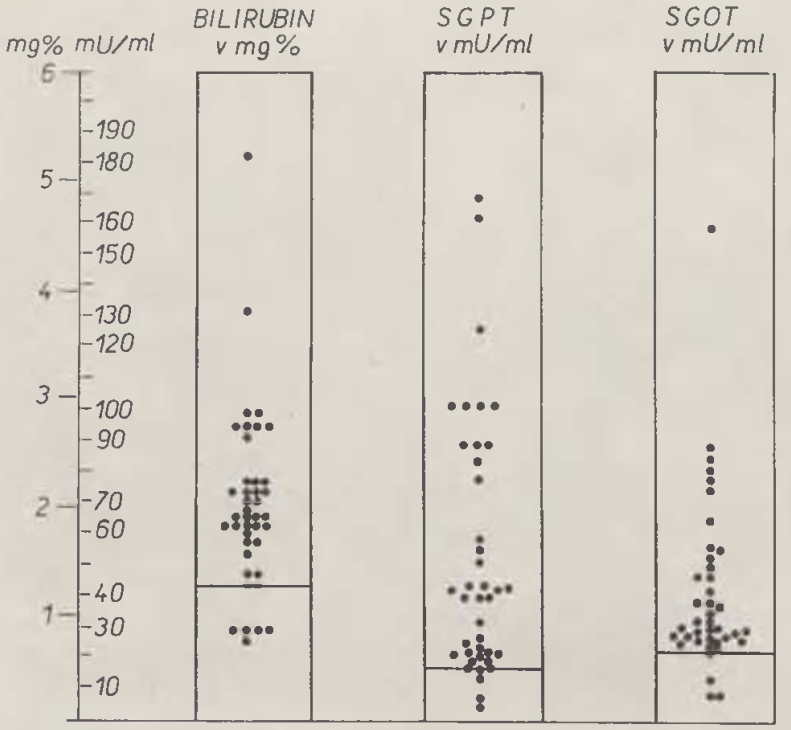
	No of Fatalities
Sepsis or Toxaemia	5
Circulatory Failure	5
Shock	1
Coma hepaticum	1

disorder were usually not clearly marked and could even be missing during correctly directed and performed antishock therapy. In accordance with the given facts the activity of serum-transaminases was relatively rarely found increased, in the initial stage. Such an event took place only in 2 our patients (i.e. 5 %).

The involvement of the liver in the clinical picture of burn becomes regular and indubitable as early as on the 3rd to 7th day of the injury. This

fact coincides with the coming stage of sepsis or toxaemia, usually associated with a high body temperature, general malaise and prostration and with some evident changes in the whole internal environment. The above mentioned stage could last sometimes over 3 weeks, rarely up to 6 weeks.

In the list of eventual gastrointestinal polymorphous symptoms anorexia, nausea, rarely vomiting or diarrhoea may apear, occasionally sensation of pressure in the right hypogastrium. These symptoms represent some initial signs of imminent hepatopathy.



Graph 1. Values of serum bilirubin, SGPT and SGOT in patients treated in the Burn Unit

At the same time the authors could very often find a mild enlargement of the liver. Its width exceeded 1 to 2 fingers the right costal border. Very frequently the serum transaminases SGOT and SGPT were found elevated within the range of 25 to 100 m units/ml., but rarely up to 170 m u/ml.

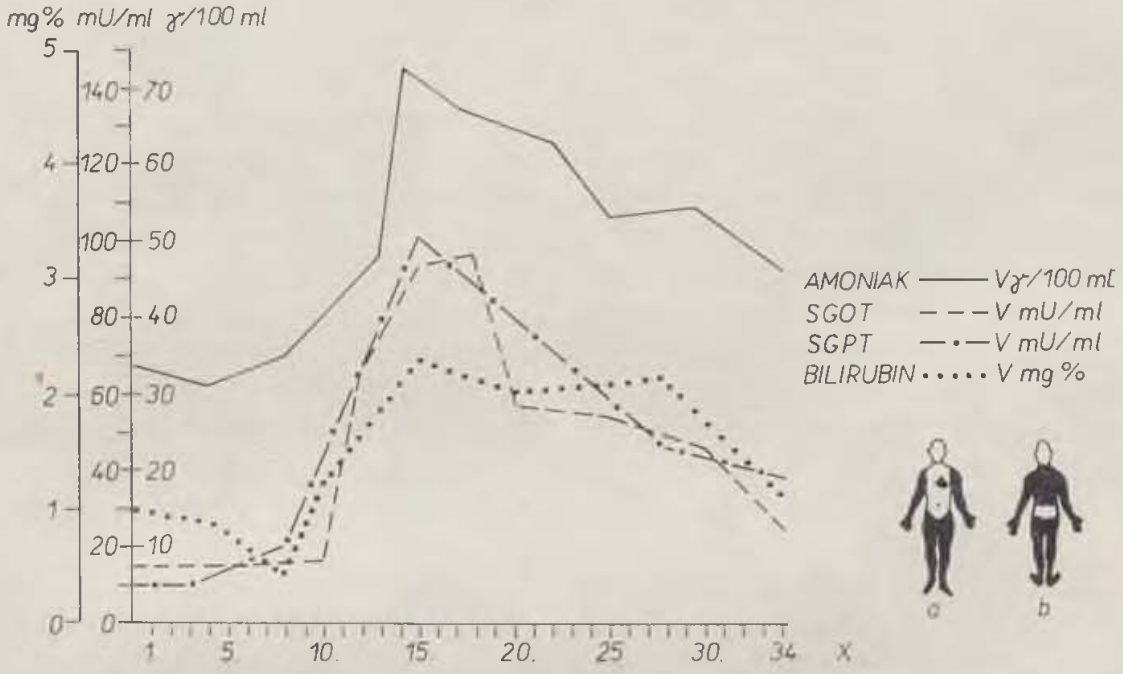
Relatively early after the injury the level of serum bilirubin rose — according to authors' statement in 80 % of all instances. This hyperbilirubinaemia was usually of a mild degree and its values did not exceed the upper limit of 5 mg% (Graph 1).

The levels of serum-ammonia were examined in 8 patients in a long-time study. Mild hyperammonaemia took place in all of them, though with a certain delay compared to other tests used, either at the top of existing toxaemia or at its initial decline. In the later course of the disease the hyperammonaemia did not run parallely with the decreasing activity of serum transaminases and with the lowering level of serum bilirubin (Graph 2).

Specimens of liver tissue were examined in 30 patients, by means of liver biopsy according to the Menghini's technique. However, the puncture of the

liver was relatively rarely made by the authors in the period of the first 48 hours following injury, partly because of the severe general condition of the patient, partly because of the location of the burn in the thoracic and epigastric region. This location did not permit a routine performance of liver biopsy.

Detailed analysis of the histological findings and their closer correlation with the clinical manifestation of the liver damage will be a matter of a future separate study. Nevertheless, the authors wish to point out at this stage that any previous eventual damage of the hepatic tissue must always be taken into consideration, whenever the microscopic structure has to be interpreted.



Graph 2. Z. M., male, born 1927. 75 % of the body surface was burnt (2nd and 3rd Degree) — a = Anterior — b = Posterior. — Sides of the body

The liver damage can be caused by infectious, toxic, nutritional or various other agents. Further — and by no means negligible — factors are the patient's age, repeated narcosis, surgery and chiefly toxaemia. These and other factors may intervene and alter the common course of the disease.

The duration of toxaemia usually depends on covering of the burnt surface by some skin graft.

The observed pathological response of the liver cells seems to be in certain relationship with all factors already mentioned.

Particular emphasis should be laid in this connection on the fact that the above named hepatic disorder did not correspond — neither clinically nor in the laboratory pattern and biopsy — to the well-known picture of a viral lesion. Accordingly with this statement the Au-antigen was found negative in all cases studied.

On the other hand, after a period of 3 to 4 months a manifestation of viral hepatitis may become apparent. It mostly happens in the form of homologous serum hepatitis. Such an event could be ascertained in 7 instances (i.e. in 17,5 %) among our patients studied. A fatal case of severe coma hepaticum ensued from an acute hepatitis in a diabetic female aged 50 years. The patient could not be saved even by appropriate treatment, though she was already in the late stage of recovery from the burn disease.

DISCUSSION AND CONCLUSIONS

Lesions of the liver constitute an integral part of any serious and extensive burn, as confirmed by our present experience based on the observation of 40 patients treated in the department of intensive care of the Burn Unit in Prague.

However, the physical signs of a liver enlargement per se are not reliable evidence of existing liver damage with regard to the localisation of the skin injury and quite often to an insufficient cooperation of the injured person. Therefore it appears to be essential to use some sensitive laboratory tests, especially serum transaminases SGOT and SGPT (7, 9, 11), the level of serum bilirubin (1, 7), or serum ammonia (4, 7).

In the electrophoretic pattern of serum proteins, hypoproteinaemia and hypoalbuminaemia were frequently reported (4, 11), rarely elevated values of the gama globulin fraction (1). Only exceptionally bromsulphthalein clearance has been recommended as a safe evidence of a liver disturbance (4). The level of cholesterol in the blood-serum was also found decreased in the later stage of the burn desease (11).

Secondary liver lesion is established early after the injury. In the stage of initial shock it occurs in a mild form in the clinical picture and morphological structure. The changes of the liver parenchyma become more intensive on the 3rd to 5th day, i.e. they take place with the imminent sepsis or toxaemia.

As already mentioned, a mild liver enlargement commences and in extensive laboratory tests the activity of serum transaminases increases — SGPT more evident than SGOT.

The level of serum bilirubin rises mildly, corresponding to occasional subicteric colouration of the sclera and skin.

The statement of a hyperammonaemia reported by Laborit and ass. as early as 1959 aroused some interest. These authors found hyperammonaemia already in the stage of initial shock, in contrast to our observations, where the elevation of serum ammonia followed belatedly the rise of serum bilirubin and after increased activity of serum transaminases. This period is later accompanied by evident improvement of the general condition of the patient and by a parallel decrease of some laboratory parameters.

Special attention should be paid to the frequently observed hyperbilirubinaemia. It existed in 80 % of all our patients so far studied, already at the end of the 1st week of the burn disease. However, it can appear in any stage and become a source of some differencial diagnostic embrassment.

The existence of jaundice during burn disease war already reported by Wilson, Mc Gregor and Stuart (quoted according to 1). Teodori and ass. observed it relatively rarely. However, if the level of serum bilirubin is constantly examined, some increase of its levels is a quite common finding as confirmed by our experience.

Antonov investigated carefully 44 patients with burn disease accompanied by the appearence of jaundice. He tried to present an explanation and classification of existing jaundice. Generally speaking, it was an early type of jaundice, rightly considered as an integral part of the burn disease, or it was a real viral hepatitis manifesting itself not earlier than after 2 to 3 months following injury, thus in the late stage of recovery or even in the period when the patients were already dismissed from the hospital to home care.

According to our experience it appears to be more useful to leave all patients suffering from early jaundice — established as an evident sequel of a hepatic disorder during the burn desease — in the special care of a trained plastic surgeon with his associated staff of collaborating experts. On the other hand, those patients, who disclosed signs of a late jaundice of the infectious type, benefited if they were treated and supervised by some experienced infectionist. Inocculated viral hepatitis needs special attention and care, while it is superimposed on an altered terrain of liver parenchyma recently injured by a burn disease and threatened by its usual complications.

Hence, it is no wonder, that the disease sometimes takes a less favourable and prolonged course (3), or even when it leads to fatality (12). Such an event was the case in one instance observed by ourselves.

A further important fact must also be taken into consideration: a transition of viral hepatitis into its chronic form could be stated in 2 out of 7 patients clinically controlled and examined by liver biopsy.

All these facts advocate for special attention to be paid to the secondary involvement of the liver tissue and of commonly impaired hepatic functions in the clinical picture and treatment of the burn disease. The above mentioned lesions of the liver cells may undoubtedly and unfavourably influence or even deteriorate the proper manifestation of the burn disease.

Various noxious agents may be involved in the etiology and pathogenesis of the hepatic disturbance. The list of such agents is hitherto incomplete and the interpretation of their effect not precise (10).

The deleterious effect of shock upon the living organism can be reduced nowadays to the least possible measure thanks to recent antishock therapy. The unfavourable influence of ischaemia and anoxia on the liver cells is now generally acknowledged (4, 10, 13) and interpreted as a result of a circulatory impairment affecting the splanchnic and intrahepatic regions (13). Beside that loss of fluid and protein, profound disorder of some important electrolytes and disturbed acid-base balance with a tendency towards acidosis constitute further accessory factors.

At the same time the metabolic energetic rate subsides (13). Furthermore the strain imposed upon the detoxicating hepatic ability (4, 10, 13) and upon

the hepatic RES is much augmented as a consequence of resorbed catabolic cell-products and toxins. The RES seems to be extraordinary sensitive to oxygen lack and acidosis. In the maintenance or accentuation of homeostatic and defensive mechanisms outstanding significance must be attributed to infection, especially if caused by Gram-negative germs (13).

In the list of possible noxious agents some of them should be reconsidered: the stress elicited by repeated anaesthesia and surgical treatment, sometimes even an unfavourable hepatotrophic effect of some highly effective antibiotics, because without them no succesfull combat against sepsis — which as a rule accompanies every extensive thermic injury of the skin — can be assumed and realised.

From all that has been said some therapeutic lines can be derived: The basic principles of any complex treatment consist advantageously in a rapid control of the shock, then in shortening the exposure to unfavourable effects of toxaemia and sepsis by suitable antibiotics, in early cuting off and removing of the skin necroses and covering the wounded skin surfaces by skin transplants under a protective veil of suitable anaesthesia. General care of fluid replacement and of correct electrolyte balance as well as an effective realimentation seem to be the additional main features of any therapeutic scheme.

Suitable treatment by potent medicaments which are able to support some essential liver functions forms a necessary supplementing therapeutic base.

From all medicaments hitherto applied, administration of arginine-malate in the form of intravenous infusions establishes in authors' experience the most useful sort of therapy, which proved to be even more powerfull than the administration of high doses of vitamin B_{12} and thioctic acid. J. S.

SUMMARY

The clinical manifestation of the disorder of liver cells constitutes an integral part of any severe and extensive burn. The derangement of the liver parenchyma occurs as a rule in the stage of toxaemia or sepsis, less frequently in the stage of initial shock. The appearance of jaundice is relatively a common event and accompanies the burn disease up to 80 % of all affected patients. It occurs early after the injury and is relative to the clinical picture of the burn disease.

Etiology and pathogenesis of the secondary hepatic lesion has been discussed and some main principles of a modern complex treatment have been pinpointed in the entire framework of the actual therapy.

RÉSUMÉ

Tableau clinique de la lésion du foie en cas d'une maladie de brûlure

Chlumský J., Vrabec R., Hynčík V., Mareček B., Chlumská A.

On décrit le tableau clinique de la lésion du foie qui suit chaque maladie de brûlure étant considérable et plus étendue. Sa manifestation est moins fréquente dans son stade de choc, mais régulière dans le stade toxémique ou septique. C'est aussi l'ictère qui est probablement fréquent et qui se présente jusqu'en 80 % de tous

les malades bientôt après l'accident et qui appartient au tableau clinique de la maladie de brûlure.

Dans la partie suivante, on discute l'étiopathogenèse de la lésion secondaire du foie et on fait entrevoir les principes du traitement de celle-ci dans le cadre de la thérapie complexe de la maladie de brûlure.

ZUSAMMENFASSUNG

Das klinische Bild der Leberschädigung bei der Verbrennungskrankheit

Chlumský J., Vrabec R., Hynčík V., Mareček B., Chlumská A.

Die Autoren beschrieben das klinische Bild der Leberschädigung, die einen regelmässigen Teil jeder schwereren und umfongreicheren Verbrennungskrankheit darstellt. Weniger häufig manifestiert es sich in ihrem Schockstadium, regelmässig dagegen im Stadium der Toxämie oder Sepsis. Verhältnismässig häufig ist auch die Gelbsucht, die bei den Kranken frühzeitig nach dem Unfall bis in 80 % aller Fälle erscheint und zum klinischen Bild der Verbrennungskrankheit gehört.

Im weiteren wurde die Äthiopathogenese der sekundären Leberstörung diskutiert und die Grundsätze ihrer Behandlung im Rahmen der Komplextherapie der Verbrennungskrankheit angedeutet.

RESUMEN

Cuadro clínico de una lesión hepática en la enfermedad por quemadura

Chlumský J., Vrabec R., Hynčík V., Mareček B., Chlumská A.

Fue descrito un cuadro clínico de la lesión hepática que es una parte normal de cada enfermedad por quemadura más grave y más extensa. Se manifiesta menos a menudo en su estadio de choque, sin embargo aparece en el estadio toxémico o séptico con regularidad. Relativamente frecuente suele ser también la ictericia que aparece hasta en los 80 % de todos los enfermos pronto después del accidente y pertenece al cuadro clínico de la enfermedad por quemadura.

Más adelante está discutida la etiopatogenesis de la lesión hepática secundaria e indicados los principios del tratamiento de la misma en el marco de la terapia compleja de la enfermedad por quemadura.

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STANDARD NON-CONTACT BURN
Subcutaneous Temperature Dynamics During a Thermal Injury

J. MOSEROVÁ, Z. PROUZA

Temperature measurements of affected tissues carried out during an affliction of an experimental burn represent an important factor in the standardisation of a burn.

Besides describing basic data characterising the heat source, several experimental studies deal also with thermal conditions of the surface of the object exposed to heat, or thermal conditions within the tissues. Entin and Baxter (1950), for instance, carried out temperature measurements between a contact source and tissues exposed to the source in human volunteers. These authors expressed graphically the relation between the degree of heat, exposure time and the resulting thermal injury (erythema, blistering, thermal coagulation of the skin). The authors worked with source temperatures between 40—110 ^{0}C and they used exposures of 0,5—3 minutes (Fig. 1).

Comparing (Tab. 1) the results of Entin and Baxter (1950) who measured temperatures on the surface of the traumatised skin and the results of Mendelsohn and Rositter (1943—4) and Henriques and Moritz (1947), who measured subcutaneous temperatures in experimental animals, we can see that the temperature values obtained on the surface of the skin are approximately 6—18 ^{0}C higher than values obtained subcutaneously (with higher temperatures this difference is more pronounced).

Besides studying the dependence of tissue destruction on source temperature and exposure time, some authors followed also tissue temperature changes during exposure to the heat source. In the majority of such studies, the length of exposure exceeded 30 sec.; some authors even used exposures lasting several hours.

In this study, the authors concentrated on subcutaneous temperature measurements obtained under the skin exposed to the heat source using very short time exposures to a high density thermal flux. With extremely short exposure times temperature changes during the burning appeared less significant than tissue temperature changes occurring immediately after ter-

Tab. 1

Surface temperature (°C)	Subcutaneous temperature (°C)	
(Entin, Baxter)	(Mendl., Rossiter)	(Henriques, Moritz)
45	38,4	39,5
50	41,4	—
55	44,8	47,0
60	48,4	—
65	—	53,0
70	54,4	—
80	—	71,0
90	—	74,0

(reprinted from Entin & Baxter, 1950)

mination of exposure to the heat source. The authors also aimed to find, whether temperatures, detrimental to the living cell, critical temperatures, may persist in the tissues for any length of time after the termination of the tissue exposure to the thermal source.

METHOD

A plasma burner was used as a heat source. The plasma beam of (29000 ± 1000) ^{0}C is directed on a graphite electrode the front of which can reach temperatures as high as 3000 ^{0}C. On the apparatus used, thermal energy transfer can be achieved in two ways:

— direct thermal energy transfer from source to object; in this case, an area of 50 cm^2 is exposed to the thermal flux, the density of which is (9,8 ± 0,91) cal. cm.$^{-2}$sec.$^{-1}$,

— indirect thermal energy transfer using an optical system of two parabolic mirrors (∅ 2000 mm, f ± 848 mm); in this case, the highest intensity of energy may be found between the focal points of both mirrors, where an area of 20 cm^2 may be exposed to a thermal flux of (22,5 ± 0,85) cal.cm.$^{-2}$sec.$^{-1}$ mean density (Prouza et al., 1972).

Male Wistar (Mezno) rats, weighing 200—250 g were used as as experimental object. After two weeks of quarantine, the animals were placed in separate cages and were kept at standard room temperature of (20—22 ^{0}C). The animals were fed a standard diet and received water ad lib.

The depilation of animals, fixation and burning technique were described in a previous study (Prouza et al., 1972).

Temperature changes were being measured continuously by iron-constantan thermocouples, placed in the tips of ordinary intramuscular injection needles. The thermocouples were introduced under the skin muscle in the dorsal area designated to burning approximately 5 min., before exposure to the thermal flux, and were left in place for 5 to 10 minutes after termination of the exposure. Temperatures were recorded continuously by a linear register

EZ 3, starting one minute before burning until 5 min. after termination of burning.

The calibration of thermocouples as well as the evaluation of temperature values thus obtained and statistical evaluations were described in a previous study.

Results obtained in 116 animals will be presented.

RESULTS

In Tables 2 and 3 and in Fig. 2 and 3 absolute subcutaneous temperature values (in ^0C) in different concentric zones of the exposed area in relation to time are listed. These values were obtained using the indirect heat transfer.

The grey area on the graphs represents control values of normal, initial temperatures, obtained 1 min. before burning.

In Tab. 4 and on Fig. 4 and 5, subcutaneous temperatures obtained under the centre of the exposed area with the use of direct heat transfer are listed. The values are represented in ^0C. (Graph 4 and 5 represent results for 8 sec. and 12 sec. exposure to the heat source.)

DISCUSSION • EVALUATION OF RESULTS

This particular study is the second part of a series of studies which will be published in this journal and which aim to characterise a standard, non-contact experimental burn. In all of them, the authors aimed to characterise

Tab. 2

1 sec exposure

Temp. (°C) / time after exp. (min)	Centre		Paracentre		Periphery	
	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
−1	34,8	2,21	34,8	2,21	34,8	2,21
0	94,5	7,91	78,3	9,02	60,1	11,30
1/4	55,1	3,38	51,2	5,20	44,3	4,00
1/2	53,1	2,95	49,6	4,82	43,8	3,88
1	49,5	2,82	47,1	3,88	42,6	2,35
2	45,0	2,89	43,4	3,14	40,8	2,44
3	42,3	2,59	41,4	2,74	39,8	2,07
4	40,1	3,15	39,8	3,75	38,6	3,32
5	39,0	3,16	38,6	3,00	37,7	2,32
6	31,0	2,95	37,4	1,68	37,1	3,16
7	37,2	3,58	36,6	3,10*)	36,8	2,38
8	36,7	2,78	36,9	2,55*)	36,5	2,22
9	36,1	3,62*)	36,6	1,96*)	36,2	2,97*)
10	35,7	2,46*)	—	—	36,1	2,25*)
11	35,2	2,65*)	—	—	—	—

*) statistically non-significant values at 5% significance level

a standard burn which could be used for further studies of local changes and the therapy of local changes. Of all exposures used, the burn resulting from the 1 sec. exposure, using indirect heat transfer, proved to be most convenient for this purpose. Therefore, this type of burn was studied most thoroughly. Nevertheless, measurements obtained in all other types of burns, using different time exposures are also listed, as there are some points of interest in their interpretation.

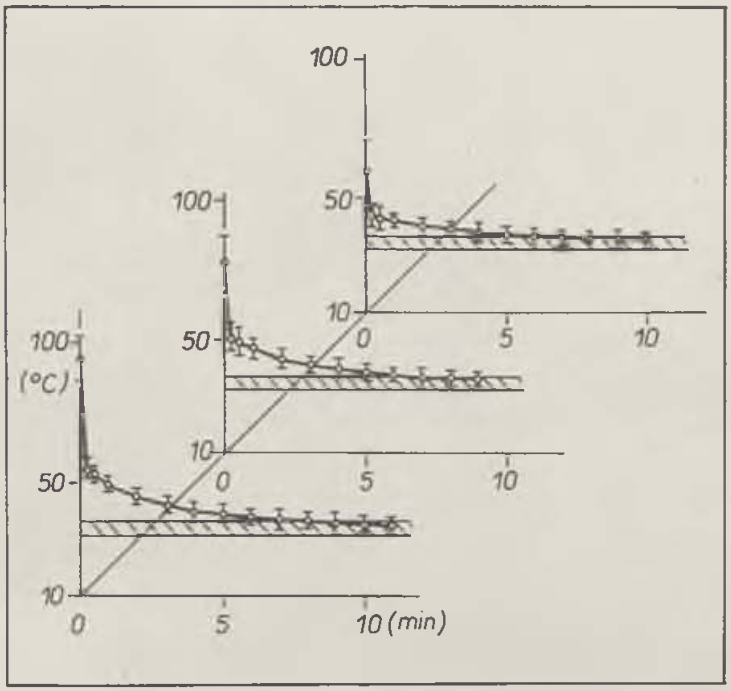

Fig. 1. Subcutaneous temperatures [^{0}C] in relation to exposure time [min.] 1 sec. exposure — indirect heat transfer. From lower left to upper right: central zone, paracentral zone, periphery

Temperature dynamics with the use of 1 sec. exposure and indirect heat transfer are presented on Graph 2. It will be seen that the curves obtained in the three concentric zones of the exposed area are quite characteristic. At the onset of the exposure, in all zones the temperature reaches abruptly its maximum and within 15 sec. after the termination of the exposure a relatively steep temperature fall may be observed [in the central zone 66 %, in paracentral zone and peripheral zone 68 % of the difference of the initial and maximum temperatures]. After this period, the temperature fall is slower. Temperatures, which do not significantly differ from initial values are found 7 to 9 min. after termination of exposure.

In 4 sec. exposure [Graph 3] the temperature fall which may be observed immediately after termination of exposure, is less steep. Within 15 sec., the temperature fall in all three concentric zones is only 34—35 % of the initial and maximum temperature difference. Also, in this case, the subsequent slower rate of temperature fall is less pronounced than in the case of 1 sec. exposure.

Tab. 3

4 sec exposure

Temp. (°C) / time after exp. (min)	Centre		Paracentre		Periphery	
	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
−1	34,8	2,21	34,8	2,21	34,8	2,21
0	100,3	17,70	100,2	13,60	77,5	16,29
1/4	77,6	7,99	76,4	7,11	61,9	10,89
1/2	71,0	7,10	71,2	5,57	59,2	8,44
1	62,3	4,45	64,0	5,05	54,3	6,10
2	53,3	3,89	54,2	4,80	47,4	5,60
3	45,9	3,54	48,5	5,67	43,2	5,62
4	40,6	2,38	44,8	5,49	41,3	5,12
5	36,3	2,76*)	41,5	3,36	38,8	5,57
6	33,5	2,60*)	40,0	5,95	37,1	5,35*)
7	32,2	1,87*)	39,4	4,85	—	—

*) statistically non-significant values at 5% significance level

The temperature values approach initial values already 5 min. after exposure termination; in central zones temperature even falls under the initial values. The character of the curves for all three zones is analogous.

The differences in the character of the subcutaneous temperature curves which may be observed immediately after exposure termination, are based on fundamental laws of heat transfer in tissues. Using the 1 sec. and 4 sec. exposure, the tissues were exposed to a thermal flux of the same density but for a different time period. It can be presumed that with a longer exposure the heat will invade deeper into the tissues. Nevertheless, the thermo-

Tab. 4

Temp. (°C) / time after exposure (sec)	Exposure							
	6 sec		8 sec		10 sec		12 sec	
	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE	$\bar{x}$	SE
−60	39.00	3.12	30.75	4.35	30.00	3.80	32.27	4.51
4	66.49	7.61	60.25	4.19	65.73	13.27	74.60	14.71
8	70.66	7.60	61.25	4.57	65.93	12.36	74.40	13.98
12	70.66	6.12	61.25	5.25	65.33	11.47	73.13	12.74
16	70.10	5.54	59.75	4.92	64.80	11.21	71.53	11.08
20	69.11	4.95	58.50	5.07	63,73	9.37	70.60	10.23
30	66.51	6.12	55.50	3.37	61.33	8.01	66.47	8.68
60	60.55	4.48	49.25	4.79	55.53	6.10	63.60	5.67
120	55.93	1.79	44.50	4.65	49.00	5.54	51.20	6.38
180	52.57	1.42	41.50	5.80	45.80	5.16	46.60	5.72
240	—	—	40.00	6.53	43.27	5.40	43.27	5.40
300	—	—	—	—	41.18	5.85	41.33	5.29
mid-exp.	57.45	10.26	45.50	3.32	47.53	10.90	60.07	11.91
end of exp.	66.31	11.42	59.75	2.36	67.13	15.04	75.27	16.72

couple was placed in both cases in the same depth, i.e. immediately under the skin muscle. As was ascertained by histological examination with 1 sec. exposure, the thermocouple was located on the border zone of thermal damage (vertically). Consequently, it can be presumed that in this particular location there was a marked temperature gradient immediately after termination of exposure; the temperature in the tissues above the thermocouple was markedly higher than the temperature of the underlying tissues. Thus, a relatively rapid heat balance was achieved by active and passive heat transfer into the

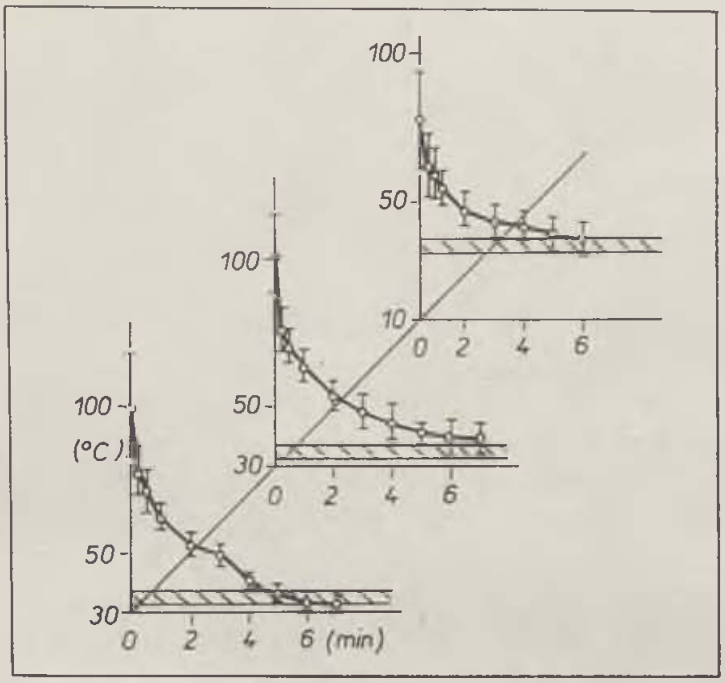

Fig. 2. Subcutaneous temperature (°C) in relation to exposure time (min.) 4 sec. exposure — indirect heat transfer. From lower left to upper right: central zone, paracentral zone, periphery

surrounding tissues. By passive transport direct heat conduction to surrounding tissues is understood; active transport is represented by heat transfer by intravascular fluid through undamaged vessels (Sevitt, 1957). It follows that by gradual heat transfer from the zone in which the thermocouple is placed, the temperature gradient decreases and consequently the rate of temperature fall, as seen on the curves, decreases as well.

In the case of the 4 sec. exposure, the heat pervaded during the exposure beyond the skin into the underlying tissues in the centre of the burned area even as far as into the skeletal muscles. During this exposure the heat capacity of the skin was evidently reached and heat transfer occurred both vertically and horizontally (centrifugally). (Henriques and Moritz, 1947) (Sevitt, 1957; Prouza et al. 1972.) At the termination of exposure the tissues under the thermocouple were also at higher temperatures and therefore the heat from the vicinity of the thermocouple could not be speedily transferred to surrounding tissues. Histological examination showed that both in the central and

paracentral zone the vessels, which otherwise would enable active heat transfer, were damaged in the underlying tissues. This fact explains the slow temperature fall observed immediately after termination of burning at the 4 sec. exposure. The temperature fall to lower values than the initial values, which occurs at the terminal phase of the 4 sec. exposure curves, points also to a severe active heat transport disorder. The tissue temperature is levelling up with the temperature of the outer environment.

In the case of direct heat transfer, analogies were found in 6 and 8 sec. exposure curves and in 10 and 12 sec. exposure curves. A graphical expression of the results is presented for the 8 sec. and 12 sec. exposure (Graph 4 and 5).

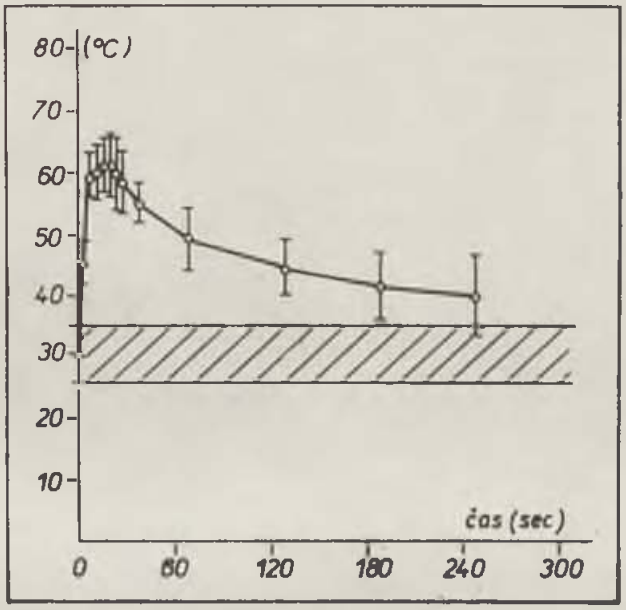

Fig. 3. Subcutaneous tempratures (°C) in relation to exposure time (sec.) 8 sec. exposure — direct heat transfer

All values that were measured in different exposures using direct heat transfer are listed in Tab. 4.

With the 6 sec. and 8 sec. exposure a temperature rise may be observed even after the termination of the exposure, while with the 10 sec. and 12 sec. exposure there is an immediate temperature fall. It appears that with shorter exposures to the thermal flux (6 sec. and 8 sec.) the heat capacity of skin is not attained, while with longer exposures, namely with 12 sec. exposure, the temperature of the measured tissues is approaching its heat saturation (the maximum temperature difference in 10 sec., 12 sec. exposures is statistically not significant), though the maximum temperatures in the consecutive exposures are gradually rising. In the 10 sec. and 12 sec. exposure it is important to remember that with these two types of burning a higher capillary permeability in the underlying tissues may occur during the burning or immediately after its termination. The oedema absorbs part of the heat; this may explain the relatively rapid temperature fall during the first 20 sec.

after the termination of these exposures. In the next phase the temperature fall in the measured layer is slow, and the curves obtained with different exposures do not significantly differ.

In the case of direct heat transfer the tissues were exposed to a heat flow of lower density than that when using indirect heat transfer. Nevertheless, in this case the tissues were exposed to the heat for a longer time period and therefore it is to be expected that the critical temperature-raise could penetrate to a greater depth notwithstanding the fact that maximum temperatures obtained by measurements were lower than in the case of indirect heat transfer. The temperature levelling-out within the layer into

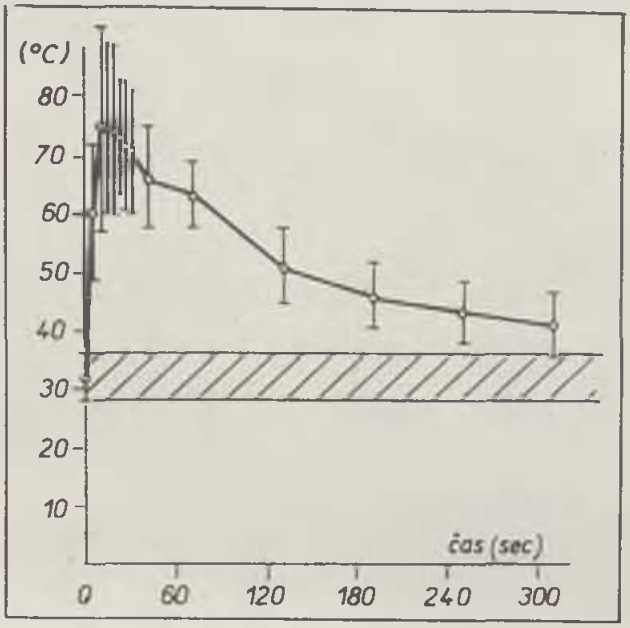

Fig. 4. Subcutaneous temperatures [^{0}C] in relation to exposure time [sec.] 12 sec. exposure — direct heat transfer

which the thermocouple was introduced is slowed down. This supposition is confirmed by the overall shape of the curves obtained in all exposures with direct heat transfer.

The most important finding of this study lies in the fact that critical temperatures persist in the tissues surprisingly long after the termination of the tissue exposure to the thermal flux.

The term "critical temperatures", i.e. temperatures that may cause both reversible and irreversible changes in living tissues, is not well defined. Criteria, according to which critical temperatures may be ascertained, differ. Sevitt [1957] defined a temperature threshold for skin erythema a threshold for increased capillary permeability and a treshold for the development of stasis. He states. for instance, that erythema develops with a contact heat source of 51 ^{0}C applied to tissues for 60 sec. If a 52 ^{0}C contact heat source is applied for 60 sec., increased capillary permeability is induced. Capillary statis occurs after the application of 58 ^{0}C contact heat source for 60 sec.

With heat sources of lower temperatures these changes, of course, develop only after a prolonged contact with the tissues. (For instance, with 50 ^{0}C temperature increased permeability occurs after three minute period.) According to Sevitt (1957) the threshold dermal temperature for the development of oedema and stasis in guinea pigs is 41—48 ^{0}C.

After comparing the values in Table 1, where surface skin temperatures obtained by measurements are listed (Entin and Baxter, 1950) as well as the corresponding subcutaneous temperatures (Mendelsohn and Rositter, 1943—44; Henriquez and Moritz, 1947), it may be stated that with surface skin temperatures within the range of 50—60 ^{0}C the subcutaneous temperature is approximately 10 ^{0}C lower. (This, of course, is rather a gross approximation, as, according to the above mentioned findings, the temperature gradient between the surface of the skin and the subcutaneous tissues depends on the intensity of the thermal flux and the exposure time the heat source.)

Considering the results of the studies of Sevitt (1957), Henriques and Moritz (1947), Leach and al. (1943), Mendelsohn and Rositter (1943—44) and Entin and Baxter (1950), which dealt with the problem of critical temperatures, the authors of this study were able to reach and approximation of the time period, for which critical temperatures are persisting in the subcutaneous tissues after different time exposures using both the direct and indirect heat transfer.

The temperature of 41 ^{0}C is considered critical by the authors if it persists subcutaneously for at least 3 minutes, and that of 51 ^{0}C if persisting at least 1 minute.

In the case of 1 sec. exposure to the heat flow with indirect heat transfer critical temperatures are persisting within the tissues in the central and para-central zone for 4 minutes after the exposure. After the 4 sec. exposure, critical temperatures are persisting in all three concentric zones up to 5 minutes.

In the case of direct heat transfer, after 6 sec. and 8 sec. exposures, critical temperatures persist 3 minutes.

It may be stated that in all exposures critical temperatures persist for a time period several times longer than the time period of the exposure to the termal flux itself. Š. H.

SUMMARY

By analyzing the results of this study, the time period, for which temperatures detrimental to the living cell can persist in thermally injured tissues, was ascertained. It was found that in all exposures to the thermal flux critical temperatures persist in the tissues for a time period several times longer than the time period of the exposure to heat. These findings may prove to be of importance for first-aid in cases of burns; it appears that by insuring swift heat conduction from the affected tissues immediately after the thermal injury, changes which develop with the persisting critical temperatures within the tissues could be slowed down or stopped.

RÉSUMÉ

Brûlure normale, sans contact. Dynamique des températures subcutanées au cours du traumatisme thermique

Moserová J., Prouza Z.

En conséquence de résultats mentionnés a été constaté le temps pendant lequel les températures traumatisantes nonphysiologiques peuvent survivre dans le tissu. On a constaté sur toutes les expositions dans tous les cas que ces températures critiques survivent pendant un temps beaucoup plus long que celui de la durée de l'action de la source. Ce fait est d'une importance incontestable pour la pratique clinique. Il démontre qu'une intervention faite immédiatement après l'action thermale nocive permettant l'élumination plus vite de la chaleur du tissu atteint pourrait arrêter ou ralentir les modifications successives qui se produisent par une action prolongée des températures nonphysiologiques.

ZUSAMMENFASSUNG

Kontaktfreie Standardverbrennung. Die Dynamik der Unterhauttemperaturen während des Wärmetraumas

Moserová J., Prouza Z.

An Hand der angeführten Ergebnisse wurde ermittelt, wie lange in dem Gewebe die traumatisierenden nichtphysiologischen Temperaturen erhalten bleiben können. Bei allen Expositionen wurde festgestellt, dass diese kritischen Temperaturen vielfach länger erhalten bleiben, als die eigentliche Einwirkung der Quelle dauerte. Diese Feststellung ist von unbestreitbarer Bedeutung für die klinische Praxis, da aus ihr hervorgeht, dass ein rechtzeitiger Eingriff unmittelbar nach der Wirkung der thermischen Noxe, der ein schnelleres Wärmeableiten aus dem betroffenen Gewebe ermöglichen würde, die forschreitenden, durch protrahierte Wirkung der nichtphysiologischen Temperaturen eintretenden Veränderungen entweder zum Stillstand bringen oder verlangsamen könnte.

RESUMEN

Quemadura corriente sin contacto. Dinámica de las temperaturas subcutáneas durante el traume térmico

Moserová J., Prouza Z.

A base de los resultados mencionados fue constatado durante cuanto tiempo perduraban en el tejido las temperaturas no fisiológicas que causan un trauma. En todas las exposiciones fue constatado que estas temperaturas críticas perduraban por un tiempo mucho más larga de lo que había sido la misma acción de la fuente. Esta averiguación tiene importancia indiscutible para la práctica clínica, porque de ello resulta que una intervención, hecha a tiempo inmediatamente después de la acción de la noxa térmica, que haría posible que se llevase el calor del tejido afectado más rápidamente, pararía o retardaría los cambios que proceden de la acción prolongada de las temperatures no fisiológicas.

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AN EPIDEMIOLOGICAL STUDY OF CLEFT LIP AND PALATE IN BOHEMIA

O. KLÁSKOVÁ-BURIANOVÁ

Academician Burian: "The aim of congenital defects research is one of prevention. The essential thing here is to have as reliable a registration of congenital defects as possible. This will help to find out about the density of their incidence in certain regions under certain epidemiological conditions."

*

The Laboratory of Plastic Surgery of the Czechoslovak Academy of Sciences keeps a file on all surgically treated cases of cleft lip and/or palate since the very beginning of plastic surgery in Czechoslovakia after the first world war. By the end of 1971, this clinical registration contained case histories of a total of 6,016 individuals suffering from cleft lip and/or palate. This number included 4,151 cases of cleft lip (CL) and cleft lip and palate (CLP) — 67 %, and 1,865 cases of isolated cleft palate (CP) — 31 %.

The first surgical operations were mostly of a palliative nature. But as plastic surgery was gradually gaining ground thanks to its therapeutical successes, more and more children with congenital clefts came to be treated at the Institute of Plastic Surgery, at that time the only one in the country. After the second world war, departments of plastic surgery were established in more Czechoslovak towns and cities thus giving rise to the establishment of areas of treatment. With a few exceptions, all children born in Bohemia receive treatment at the Department of Plastic Surgery of the Medical Faculty of Hygiene, Charles University, Prague. Our own laboratory works in close co-operation with the Department.

Registration of apparent congenital defects was introduced throughout Czechoslovakia in 1964 — mainly thanks to the efforts of Academician Burian who was the first to propose compulsory registration after the second world war.

New born infants afflicted with congenital deformities are reported on special punch-cards within seven days by the maternity pediatrician to the Ministry of Health. Child's name and date of birth, congenital defect, the

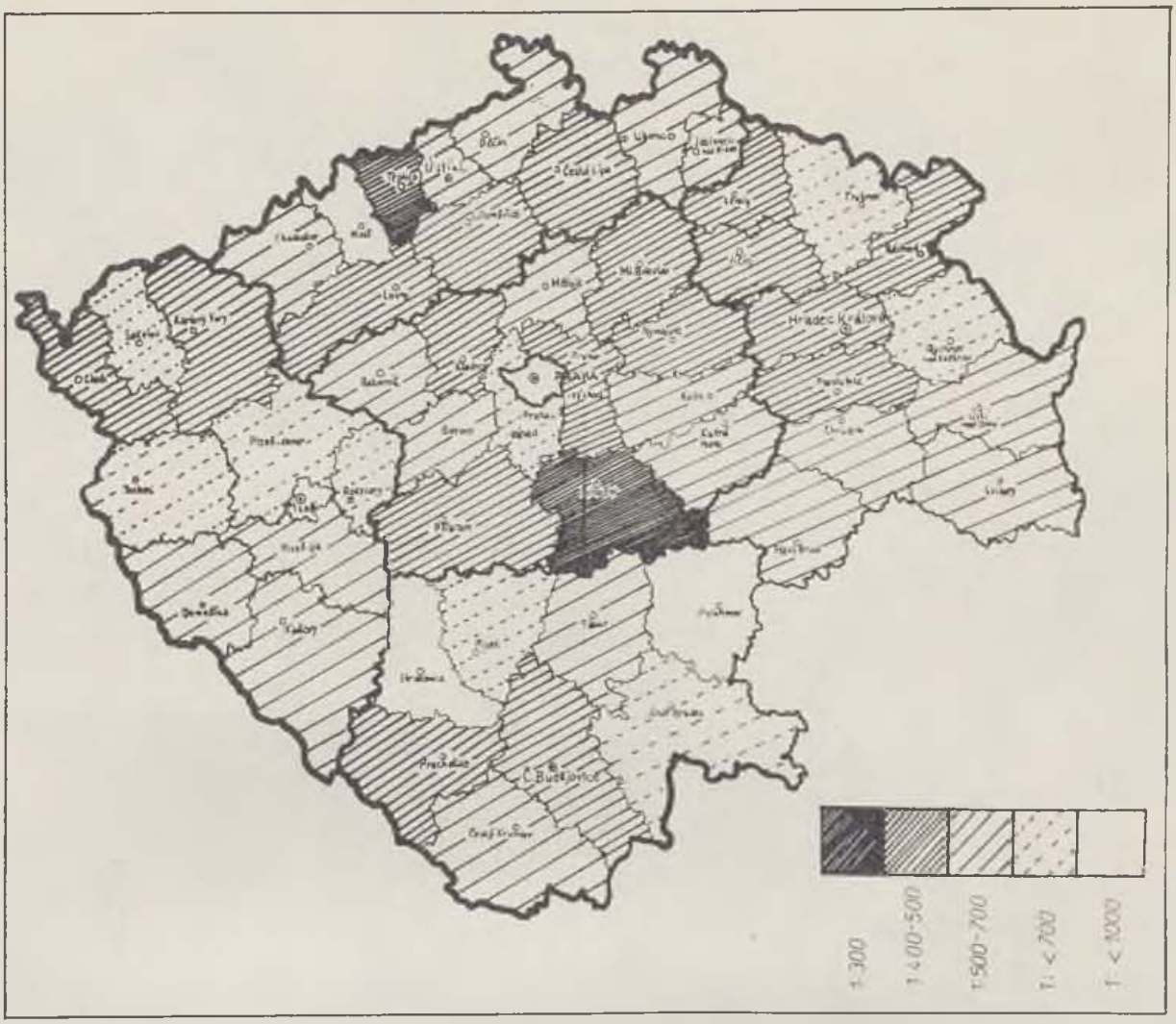

Fig. 1. Cleft rate on the territory of Bohemia over the years 1964—1970. — One cleft per number of live born children. — The frequency on the chart is given by the ratio of one cleft per number of live born children.

parents' dates of birth, the infant's weigh at birth, duration of pregnancy, rate are reported. The data are computerized on an annual basis and published.

Children with cleft lip and/or palate, born on the territory of Bohemia, are gradually registered at our laboratory. At the age of 2 months, they are examined by a plastic surgeon and their mothers are briefed on the therapeutical procedure. The laboratory carries out the basic investigation. We fill in the epidemiological form and set up a pedigree on a coded genealogical form. The parents are examined for possible microforms. The family history is added to during further calls. In case of young mothers and perticularly primaparae, we explain the possibilities of family planning and provide mothers with information about our genetic counselling. The counselling system is based on empirical risk-figures calculated from our material and gradually published for different genetic situations.

Six months after the termination of the calendar year we compare our registration notes with the state register. Each year, some 20 to 30 children with clefts (11—17 %) who are registered in our laboratory fail to be reported in the state registration. These are mostly mild cases which escaped notice: incomplete harelips, clefts of the velum and submucous cleft. However, it does happen each year that even total clefts are reported at the Department but fail to be reported for the state registration.

We make written inquiries at district pediatricians about the progress of children registered at the Ministry of Health but missing in our own register. A small proportion of these children from the Eastern part of Bohemia are treated at the Department of Plastic Surgery in Brno. Another part, some 5

Table

	Live born	Absolute number of clefts	Number per 1,000	1 cleft per number of live born
1964	91,527	154	1.7	594
1965	86,775	176	2.0	493
1966	82,985	166	2.0	500
1967	82,070	162	2.0	507
1968	81,657	156	1.9	523
1969	84,875	173	2.0	491
1970	88,895	185	2.1	479
1971	92,828	174	1.9	533
Total	689,967	1346	1.9	513

to 12 children a year, die before operation. These are invariably immature children suffering from additional severe congenital malformations. On the basis of long years of experience, Academician Burian had arrived even before 1960 to the estimated frequency of one cleft per 500 live-born children. This estimate is corroborated by the eight year registration described elsewhere in this paper. During the period of 1964—1971, 1,346 out of a total of 689,967 live-born children were registered for congenital clefts, i.e. one cleft per 513 live born children.

The frequency for the single years does not show much difference:

We failed to find out more details about 68 children (5,8 %) reported for state registration. 15 of them are in children's homes (for their parents' lack of interest), 16 others are treated elsewhere. The remaining 37 may be presumed to have died prior to surgical treatment, judging by the punch-card data.

29 children (2.1 %) have cleft palate or total cleft as part of syndromes: 11 syndrome of lower lip fistulae, 10 Pierre Robin's syndrome (7 autopsy finding), 2 Apert's syndrome, 1 Klippel Feil syndrome, 1 Treacher Collins' syndrome (autopsy finding), 1 Turner's syndrome, 1 Marfan's syndrome.

These children apart, there remain 1,318 individuals (a rate of 1.9 per 1,000 live-born, one cleft per 523 live-born) out of whom 885, i.e. 67 % have group I. clefts — CL and CLP with a rate of 1.3 per 1,000 or one cleft per 780 live-born. 433 children have group II. clefts — CP — i.e. isolated cleft palate or submucous cleft. In this group the rate is 0.6 per 1,000 or one cleft per 1,593 live-born infants.

The sex ratio is 62 % : 38 % in group I. (CL plus CLP) and 44 % : 56 % in group II. (CP).

89 children (6.9 %) had died prior to operation, most of them by the end of the first month of life. 30 % of these (27) had suffered from congenital heart defects and other serious associated defects or syndromes. Most of these children were immature with bronchopneumonia as the immediate cause of death. In only 25 children no congenital deformity other than cleft was found. The causes of death included an early asphyctic syndrome and again bronchopneumonia and immaturity.

Of the living children with clefts, a congenital heart defect was diagnosed in fewer than 1 % (8 children).

The sex ratio among the deceased of group I was 32 : 22, in group II — 16 : 19; i.e. similar to that of the whole material. Among the 54 children who have died in the course of the first month of life, the male sex predominated not only in group I., 22 : 12, but also in group II., in cases of isolated cleft palate — 13 : 7.

There are 21 pairs of twins in our eight years collection. Two pairs are concordant in respect to cleft, one of them being of the same sex. 19 pairs are discordant, eleven of these are of the same sex. In the only case of triplets, one boy has cleft palate while his brother and sister are unaffected.

Each individual case of cleft is recorded on a cartogram. Major density is discernible in the industrial border regions of northern Bohemia. The Teplice district, e.g., is marked by one cleft per 344 live-born. The second area with a major accumulation is in Central Bohemia, south-east of Prague. Here is a district with the second largest frequency, 1 cleft per 322 live-born. Unlike these, southern Bohemia has the lowest incidence rate, 1 : 585 for the whole region, one of its districts showing the lowest frequency of 1 : 1.93. In some of the neighbouring districts there are great differences in rate. The above described system of registration at our laboratory makes it clear that the differences cannot be explained by incomplete registration.

We co-operate with geological research. Regardless of the administrative territorial division, two areas in Bohemia are to be examined for hydrogeo-factors and other possible environmental factors: the industrial region of northern Bohemia with its high rate of congenital clefts and migration and southern Bohemia with a low rate and with its mostly oldsettled population. The results will be published.

In our opinion, only a systematic confrontation of clinical and national registration and co-operation of plastic surgeons and pediatricians can yield precise data to ascertain the frequency of congenital defects.

It follows from our own as well as foreign studies so far that the state of epidemiological factors and frequency of clefts can only be approached by examining large territorial units over long periods of time. Therefore, we hope for collaborators not only from Czechoslovakia but also from the neighbouring countries.

jh:

SUMMARY

The subject is that of evaluating the eight year registration of cleft lip and palate on the basis of clinical and state registration.

RÉSUMÉ

Etude épidémiologique sur la fente de la lèvre et du palais en Bohême

Klásková-Burianová O.

On a évalué l'enregistrement dans la période de 8 ans des fentes de la lèvre et du palais réalisé sur la base de l'enregistrement clinique et d'état.

ZUSAMMENFASSUNG

Epidemiologische Studie der Lippen- und Gaumenspalte in Böhmen

Klásková-Burianová O.

Es wurde die auf der klinischen und staatlichen Registrierung beruhende achtjährige Lippen-und Gaumenspaltenregistrierung ausgewertet

RESUMEN

Estudio epidemiológico de la fisura del labio y del paladar en Bohemia

Klásková-Burianová O.

Registración de la fisura del labio y del paladar durante el período de 8 años está evaluada en base de la registración clínica y estatal.

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SEPTIC SHOCK IN THE BURN ILLNESS

V. MATĚJÍČEK, Z. KONÍČKOVÁ, R. VRABEC, J. ŠTEFAN

The growing frequency of gram-negative infections encountered in clinical practice and the shift of the critical period of the burn illness to the stage of fully developed bacterial infection, draws attention to the importance of septic conditions in the course of the burn illness and thus also to the increased danger of bacterial shock.

This clinical syndrome, first described by Waisbren, was originally considered to be rather rare. In 1958, Weil and Spink (10) reported 278 cases of bacteraemia, in 15 % of which a fully developed bacterial shock was encountered; the shock was lethal in 10 % of the total number of cases. Later reports by other authors (1 to 9) confirmed this surprisingly high incidence of bacterial shock and stressed that it is usually overlooked and frequently lethal. It can be stated that the possible incidence of this acute state which threatens the life of the patient, must be considered in every medical branch, especially at the time, when the natural endotoxin detoxicating mechanisms are weakened.

From this aspect, 1496 case histories of patients, hospitalized at our Department in the years 1967 to 1969 for burns of various extent, localisation and depth, have been evaluated. It is necessary to state though that some of the case histories did not include some of the necessary laboratory results and other data. We were guided chiefly by the following criteria: sudden rise in temperature to 39 °C and more, especially in relation to surgery, possibly accompanied by psychic disorders and by the bacteriology of the wounds. Of the total number of 1496 patients, 53 i.e. 3,5 % died. In these 53 deceased, the chronic septic condition (caused by gram-negative microorganisms), occured in 28 cases, i.e. in 52 %. We were able to prove furthermore that 147 out of 1496 patients, i.e. practically 10 %, were threatened by bacterial shock. In 5 patients, i.e. in 0,3 % there was definite suspicion of a developed bacterial shock. We may characterize the typical manifestations of the initial phase of acute endotoxin shock by a sudden rise of the temperature with chill, tachy-

pnoea, nausea and sudden psychic manifestations of unrest and desorientation. In the course of the shock, there is a decrease in blood pressure and diuresis, the skin of the patient is cold and moist and a general psychic depression may be observed. Predominant symptoms of the terminal stage are: unconsciousness, acidotic breathing, anuria, weak pulse, a marked blood pressure fall, circulation collapse and subsequent cardiac failure.

These clinical manifestations correspond to the laboratory findings of sudden increase of catecholamines and histamine, decrease of leucocytes, respiratory alkalosis and to the differences in hematocrit values from centrally cathetrized venous blood and from peripheral blood; these manifestations also correspond to changed haemodynamic values obtained by central cathetrization. The frequent positive findings of haemoculture represent an important parameter. Subsequent progression leads to metabolic acidosis, leucocytosis, thrombocytopenia, decrease of fibrinogen values, increased lactate, pyruvate, blood sugar, neutral fats and transaminase values and a rise of oxygen debt.

Clinical and laboratory findings correspond to the pathophysiological conception based mostly on experimental studies. The basic relations of neurohumoral response to microcirculation and to RES activation (possibly RES blocking) as sequel to inundation of the organism by endotoxin, step into the foreground. Disturbance of the integrity of the capillary wall, disorder in the intravascular stability of blood with a picture of disseminated intracapillary thrombosis, permanent hypoxia, tissue acidosis with subsequent metabolic- and energy break-down, corresponds to prolonged defficiency flow, leads to cellular destruction in the shock organs with subsequent release of the vasoactive substances and proteolytical ferments into circulation and ends in generalized paralytic vasodilatation with cardiac insufficiency and definite collapse of circulation.

As this course is dramatic in clinical- and laboratory findings of acute and typical progress of the shock condition (1—10), it leads nowadays already to therapeutic steps in the early phase of developing endotoxin shock, thus often stopping acute progress. There are, however, diagnostic difficulties due to the atypical course especially in old exhausted people and in protracted course of the basic disease, namely if the condition is infectious. This question and the question of repeated mobilisation of sublethal doses of endotoxin, is extremely important in the course of burn illness.

We should like to report on a case of septic shock in the course of burn illness, which had been diagnosed at our Clinic and was confirmed by the post mortem examination.

Patient L. P., Nr., of case history 226/72, age 3[illegible] years, weight 80 kg, was burned in a petrol explosion to an extent of 54 % of the body surface II—III^0. Practically all of the upper part of the body, the medial part of both thighs and shins, were affected. Special medical care was afforded at the surgical department of the respective hospital immediately after the accident and the patient was transported to the shock department at our Institute within one hour after the accident. At admission he was quiet and in comparatively good condition, with infusion by Portex-cannula. It is important in the case history

that he went through diphtheria in childhood, through infective hepatitis in 1950 and that in the last two years he had digestive troubles with pain in the stomach region during the spring- and autumn season. Primary definite treatment was afforded in anaesthesia at the operational theatre on the second day after the accident and Sulfamylon crème was applied. Incisions releasing tension, were carried out because of the deep circulary affliction of the right upper extremity and progressing oedema. The Portex cannula introduced into the vessel above the right ankle, was removed on the same day. Achromobacterium sensitive to Gentamycin and Polymyxin, was bacteriologically cultivated from its part in the vein.

We may characterize the first 6 days after the accident as the therapeutically compensated course of the burn illness. On day 6 after the accident, clinical symptoms of bronchopneumonia on the left, supervened. The condition suddenly worsened in the patient on day 8. The temperature curve which had been originally continuously within the range of 38—39 ^{0}C, is approaching sepsis. At first the patient complains of nothing, being mostly fully conscious, occasionally he becomes restless, desorientated, hallucinates and shivers all over the body at irregular intervals. The pulse is regular and full, blood pressure 120/70, with pH decreasing to 7,290. The internal specialist diagnose sharply developing bronchopneumonia. The following bacteria were cultivated from smears of the wound area: E. coli sensitive to streptomycin, Chloramphenikol, Kanamycin, Gentamycin, Septrin, Pyopen, Achromobacterium sensitive to Gentamycin and Polymyxin, Pseudomonas aeruginosa sensitive to Streptomycin, Gentamycin, Pyopen, Septrin, Polymyxin, Staphylococcus pyogenes aureus sensitive to Oxacilin, Gentamycin, Kanamycin, Vancomycin, Bacitracin, viridating streptococcus sensitive to Penicillin, Chloramfenikol, Gentamycin, Septrin, Vancomycin, Proteus morgani sensitive to Streptomycin, Chloramfenikol, Kanamycin, Gentamycin. From the canulla there was again cultivated Achromobacterium sensitive to Gentamycin and Polymyxin. Besides the daily therapeutic prescription there was prescribed Hydrocortison 100 mg every 12 hours, of antibiotics megadoses of Gentamycin 240 mg every 8 hours in combination with Pyopen 2 g every hour. Establishment of contact with the patient in the further days is rather difficult, he has permanent tachycardia and tachypnoea, there appears systolic murmur on the apex of the heart, occasional cyanosis and meningeal symptoms. Laboratory results: N-urea 32 mg%, TZR 3,3 units, SGPT 18 mU/ml, SGOT 29 mU/ml. bilirubin 2,1 mg%, P anorg. 2,9 mg%, Ca 9,9 mg%, relation Na : K : Cl in the serum = 158 : 3,5 : 145 mekv/l in the urine 212 : 111 : 240 mekv/l, creatinine 1,7 mg% in the serum, at normal diuresis in consideration of the liquid intake, Hb 58 %, Ery 2,960.000 Ht 28, FW 140/157. Later there appears subicterus of sclera, on the trunk rose coloured — and on both lower extremities map-like — cyanosis. On palpation the abdomen is soft, the hypogastrium is bilaterally sensitive, Astrup values normalized to pH 7,410, pO_2 63, pCO_2 42. Decubitus appears on the right calf and blisters on both soles. The Ekg. demonstrates sinus tachycardia with signs of hypoxia of the myocard. Inderal is prescribed and Rheodextran is added to the infusion therapy, the daily dose of Hydrocortison is increased to 2 g.

If we sum up these findings, we arrive at the following conclusion. Since day 9 after the accident, the patient is in grave sepsis with chill, hyperpyrexia, tachycardia, tachypnoea with deteriorating psychic condition, with progressing bronchopneumonia and map-like cyanosis appears on both lower extremities. The condition of the patient is getting worse in spite of all therapy. On day 13, failure of circulation starts, the patient only reacts to painful stimuli, the blood pressure can not be measured, the pulse can not be felt, the heart response is dull and can not be evaluated, arrhythmia, wandering motions of the eyeballs, the pupils isocoric, meningism, superificial breathing with numerous rattling, the temperature decrease suddenly to 35 ^{0}C and anuria appears. After brief clearing of the sensorium, exitus letalis sets-in under symptoms of circulation failure at septic shock on day 14 after the accident.

The results of the post mortem examination with bacteriologic proof of Pseudomonas aeruginosa from the spleen, also tended to prove septic shock. Blood stasis in the lungs and symptoms of dextrolateral heart failure were ascertained, chiefly considerable broadening of the right heart and hyperaemia in the splanchnic region. In the histologic examination, the most important for diagnosis of the septic shock was the finding of dispersed microthrombs rich on fibrine in the final parts of the blood bed in the lungs, heart, in the soft meninges, in the brain and in the liver sinuses. Besides the mentioned changes confirming the septic shock, there was ascertained bilateral bronchopneumonia, with infection of the bronchi and trachea (Staphylococcus pyogenes aureus was found in the respiratory ways) and toxic damage of liver and kidney, due to sepsis.

We should like to point out in conclusion that the more detailed analysis of the role of endotoxin in the course of the burn illness, requires wide team cooperation and co-ordination in diagnostics, therapy and research, with precise documentation and with a broad laboratory base working according to the demands and principles of intensive care. There remains last not least the rather very important and basic question of direct, rapid and specific laboratory proof of endotoxin in the blood of patients. H. S.

SUMMARY

The report compares the literary data on the incidence of septic shock with a group of 1496 burned patients hospitalized at the Unit of Burn, Clinic of Plastic Surgery in Prague. A case of clinically diagnosed and by section confirmed septic shock in the course of burn illness, is described. The special team cooperation according to the demands and principles of intensive care, is stressed in conclusion.

RÉSUMÉ

Choc septique au cours de la maladie causée par la brûlure

Matějíček V., Koníčková Z., Vrabec R., Štefan J.

Dans l'étude, on compare les indications littéraires sur l'existence du choc septique avec un ensemble de 1496 personnes brûlées et hospitalisées dans le service de brûlures de la chirurgie plastique à Prague. On décrit le cas d'un choc septique

au cours de la maladie causée par la brûlure qui a été diagnostiqué et confirmé par la section. En conclusion, on accentue la collaboration spécialisée de l'équipe selon les exigences et principes des soins intensifs.

ZUSAMMENFASSUNG

Septischer Schock im Verlauf der Verbrennungskrankheit

Matějíček V., Koníčková Z., Vrabec R., Štefan J.

In der Arbeit werden die Schrifttumangaben über das Vorkommen des septischen Schocks mit einer Aufstellung von 1496 Verbrannten verglichen, die an der Abteilung für Verbrennungen der Klinik der plastischen Chirurgie in Prag hospitalisiert wurden. Beschrieben wird der Fall eines klinisch diagnostizierten und durch Sektion bestatigten septischen Schocks im Verlauf der Verbrennungskrankheit. Abschliessend wird die Fach- und Teamarbeit nach den Forderungen und Grundsätzen der intensiven Pflege hervorgehoben.

RESUMEN

Un shock septicémico a consecuencia de quemaduras

Matějíček V., Koníčková Z., Vrabec R., Štefan J.

En el trabajo se comparan los datos obtenidos de la literatura sobre la frecuencia del schock septicémico con una serie de 1496 quemados hospitalizados en el departamento de quemaduras de la cirujía plástica de Praga. Se describe un caso de shick septicémico que se presentó durante un caso de quemadura diagnosticado clínicamente y probado por disección. En conclusion se subraye la colaboración colectiva de especialistas según las exigencias y principios de la terapia intensiva.

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NEWS

Report about Symposium on Treatment of Burns held in Prague

The Czech Society of Plastic Surgery, branch of J. E. Purkyně Medical Society, held a Symposium on the Treatment of Burns on Sept. 13 to 15, 1973, on the occasion of the XXth aniversary of the foundation of the Prague Burn Unit.

By its content and course, the Symposium became a meeting of Czechoslovak and foreign authors, according to the tradition of the Third Congress held in Prague in 1970.

The program, this time condensed to four problems, i.e. surgery, resuscitation and anaesthesiology, infection and complications in burns, made it possible to demonstrate the contribution made by the Prague Unit which has found general acknowledgement. The Symposium brought a total of 110 Czech authors and foreigners from 25 countries, both capitalist and socialist, together, who participated in the lectures in a respective proportion. There where experts from the USSR, Poland, Bulgaria, Yugoslavia, Roumania, Hungary and the GDR, apart from those who had come from France, England, the USA and, suprisingly, also from South America, Mexico and even Australia and Pakistan.

The main topic of the first part was the problem of early excision of the burned area. The surgeon-in-chief of the Prague Unit, Doc. Vrabec, attempt to find a synthesis between the methods used hitherto for this risky operation, which has proved a great contribution to saving the life of patients whose injuries had previously been considered fatal. Evidence has been furnished that the operation shortens the duration of treatment and that it also leads to a decrease in the costs of treatment. The method has centred the interest of all burns units and a number of new improvements have been introduced. MacMillan of Cincinnati spoke about the immediate application of a net autotransplant. Xenotransplants taken from the skin of pig were used by Doc. Vrabec, Moserová et al. (Praha), Bohmert (GDR) and others. When estimating the depth of excision, Millesi (Vienna) recommends the microsurgical technique and preoprative biopsy. Burke et Koumas (Boston and Rotterdam) have used immunosuppression in order to extend the survival time of homologous transplants laid on after primary excision. In an analysis of postoperative conditions, Janžeković of Maribor (Yugoslavia), recognized all over the world as the author of the method of primary excision followed by immediate coverage of the defect, pointed to the part played by early excision in the development of burns shock.

Many units in the world try to overcome the obstacle to such operations, i.e. the lack of donor sites for transplants, by seeking suitable alloplastic coverages. A number of lecturers at the Symposium evaluated the new dressing Epigard (Bruck, New York), Winter (Stanmore) and Sutherland (Bangour). The Brno authors, Samohýl et al., reported on the Czechoslovak material for coverage, Synkryt (Syncover) which is of similar composition. Dr Ježek, surgeon-in-chief of the Ostrava Burns Unit, and

collaborators demonstrated the good results achieved with collagen foam hardened with a plastic substance. This goes to show that they have not stayed behind the general trend of development in the world. However in Czechoslovakia the question of easy acquisition of homologous transplants from cadavers has still remained unsolved. This is the reason why a substitute for biological material is always sought in lyophilized chorion or amnion (Simko, Kosice; Klen, Hradec Králové).

Hermans (Beverwijk) and Pashev et al. (Varna) believe the solution to lie in the employment of micrografts and net grafts which these authors have used in the method of Mowglen-Jackson.

Another topic was anaesthesiology, dealt with on a widened scale including resuscitation and intensive care. It has been shown that the experience of anaesthesiologists has essentially contributed to the improvement of therapeutic results, to the decrease in death rate and the perfection of organization in burns units. The topic of analgesia was dealt with by a number of authors such as Počta (Praha), Spijker (Beverwijk), Vojnov (Maribor) and Skácel (Ostrava). Sørensen (Hellerup) and Berger et Bruke (Boston) spoke about the administration of electrolytes per os and infusion.

The field of therapy was entered by hyperbaric oxigenation (Šimko, Kosice; Hartwig, Freiburg). Suggestions as to nutrition by catheter were given by Ranev (Sofia) and Slánská (Praha) recommended a nutritional combination which need not be interrupted during anaesthesia. The Symposium furnished evidence that the question of the first severe stages of burns sickness are still under discussion and constantly influenced by the demands made by primary and early excision (Janžeković, Maribor). Arturson (Uppsala) spoke about the loss of water in early application of homologous transplants.

The second day of the Symposium was assigned to the problems of contamination of burns at the burn unit, infection and the sources of microbes, the dynamics of their development and their reactivity to new antibiotics, because they are still dealt with by the people engaged in the treatment of burns. The topic was spoken about by clinicians and bacteriologists, such as the Czechoslovak authors Pávková, Pelar et Vacek and MacMillan (Cincinnati) and Novak (Budapest) as representatives of the gustes. The possibility of achieving aseptic environments was regarded with scepsis. However, the immunological response to carried-in infection is being investigated. Immunology seems to become one of the branches indispensable for the research in burns. Alexander (Cincinnati) and Day (Minesota), Lemperle (Frankfurt) and Leguit (Amsterdam) spoke about it.

It was shown that the endotoxin plays a role in the development of infection, which has not been recognized for a long time. Koníčková spoke about its detection and the first experience with the haemolymph Limula.

The disturbances of the milieu interne were summarized in the classical paper of M. D. Jackson (Birmingham) in his description of the syndrome of the "diseased cell". The pulmonary complications were spoken about by Wolf (Giessen) and Howie (Bangour). Petrásek (Praha) dealt with the damage to kidneys and Doleček (Ostrava) with secretion of insulin, somatotropin, renin and angiotensin. Disorders in the liver were referred to by Chlumský et al. (Praha).

In conclusion, a number of individual suggestions, such as the treatment with hot air (Ugland, Oslo), cooling (Šimko, Košice) and the treatment of sequelae (Kufner, Praha), were discussed. Tošovský et al. (Praha) spoke about the specific treatment of children and their experience with Fox's procedure.

Every participant of the Symposium received the Abstracts, and each lecture was translated into Russian, English or Czech. A collection of these papers is in print.

The participation of so many experts of different branches again pointed to the complexity of the problems of burns and the necessity of complex treatment. It has been shown that the conception of treatment as outlined by Burian is correct, and the team of the Prague Burn Unit deserves full acknowledgement and support.

The Symposium which was originally planned as a domestic affair, grew into a small international congress thanks to the great interest and the participation of authors from abroad. The Symposium was, therefore, classified among the meetings which are held under the auspices of the International Society of Burns Treatment (ISBI) which is to culminate in a world congress within five years. The meeting in Prague has given many an organizational and topical stimulus to the organizer of the coming World Congress, Dr. Benain of Buenos Aires, who conveyed his hearty invitations to Argentina for the coming year. The Czechoslovak authors who had organized the previous World Congress and this Symposium should not be absent there.

Prof. V. Karfík, M.D., DrSc.

International Symposium on Wound Healing 8—12 April, 1974, Rotterdam, the Netherlands

At the new premises of the Medical Faculty of the Erasmus University, Rotterdam, an International Symposium on Wound Healing will be held from 8—12 April, 1974.

Primary purpose of this Symposium is to create a forum for surgeons and other medical specialists to exchange ideas on wound healing. Ultimate goal is the integration of the latest knowledge of experimental and clinical origin in order to provide a rational basis for surgical procedures and for pre- and postoperative management of patients.

Biological, biomechanical, physiological, pharmacological, pathological and clinical aspects of the process of healing in different tissues will be discussed. These topics will be dealt with by authorities of international repute.

Chairman of the Scientific Programme Committee is Dr. J. C. van der Meulen, M.D., Rotterdam, the Netherlands.

Other members of the Committee are: Professor A. Algöwer, M.D., Basel, Switzerland. Mr. T. Gibson, M.D., Glasgow, Great Britain. Professor Ch. M. Lapière, M.D., Liège, Belgium. Professor W. van Winkle, M. D., Tucson, Arizona, U.S.A. Professor B. Zederfeldt, M. D., Malmö, Sweden.

Experts from all over the world are invited to attend the Symposium, to which a book exhibition, a scientific exhibition as well as a technical exhibition of medical equipment and pharmaceutical products will be attached. Those, who intend to present a paper should submit an abstract of maximum 300 words before 15 December, 1973 to the Secretariat.

For further information please apply to the Secretariat, c/o Holland Organizing Centre, 16 Lange Voorhout, The Hague, the Netherlands.

Argentina Society of Plastic Surgery, Santa Fe 1171, Buenos Aires

At the Assembly of December 1972, that this society brought about, the Directing Committee was renovated for the period of April 1973 — April 1974 and was constituted in the following way: President Dr. Francisco Arespacochaga, Vice-president Dr. Eduardo Marino, Secretary-general Dr. Francisco S. Urbistondo, Secretary of the agenda Dr. Victor Nacif Cabrera, Treasurer Dr. Pedro Mugaburu, Director for publication Dr. Eduardo Rizzo, members, Dr. Anibal Oris de Roa, Dr. Mratin J. Saubidet.

A new journal "Thermal Injury" will be issued by John Wright et Sons, Ltd., Bristol, England. It is expected to come out first in Oct., 1973, and all contributions should be sent to Dr. J. E. Laing.

Editorial Board: Prof. G. Arturson, Sweden, Dr. A. M. Clarke, Australia, Dr. F. E. de Salamanca, Spain, Dr. S. Ohmori, Japan, Dr. B. C. MacMillan, USA.

Acting editor: Dr. J. E. Laing, Odstock Hospital, Salisbruy, England.

Announcing

The Thirteenth Congress of the Pan-Pacific Surgical Association — February 15—21, 1975. — Place Hilton Hawaiian Village Hotel Honolulu, Hawaii.

Concurrent meetings will be held in Anesthesiology, Colon & Anorectal Surgery, General Surgery, Neurosurgery, Obstetrics & Gynecology, Ophthalmology, Orthopedic Surgery, Otolaryngology, Plastic Surgery, Thoracic-Cardiovascular Surgery, Urology.

For details, write Cesar B. DeJesus, M.D. Pan-Pacific Surgical Association 236 Alexander Young Building Honolulu, Hawaii 96 813.

INSTRUCTIONS TO AUTHORS

Acta Chirurgiae Plasticae, the international journal of plastic surgery, is issued in two versions four times a year. One version is in English (or, as requested by the author, in French or German) and the other in Russian. The aim of the Journal is to make specialists acquainted with the work of authors of the socialist countries, but studies from other countries are also published and welcomed.

Articles are accepted for publication which deal with the problems of plastic surgery and allied branches (clinical, laboratory, experimental studies); they must be original and not yet been published elsewhere. Articles written by authors of the countries which are represented in the editorial board of the Journal, must be given their imprimatur by the respective members.

Kindly send your manuscripts to the following address: Acta Chirurgiae Plasticae, c/o R. Vrabec, M. D., the secretary, Legerova 63, Praha 2, Czechoslovakia.

The manuscript must be typewritten in two copies (1 original plus 1 carbon-copy), one page per sheet, with doublespacing between the lines, 60 types per line and no more than 30 lines per page. There must not be more than five corrections by handwriting per manuscript. The manuscript should not exceed eight pages and contain no more than 10—12 illustrations. The institute the author works at, its director, the title of the article and the full name of the author (or authors), must be stated on the first page. All other pages should be numbered consecutively. Every paper must have a summary which is then translated into French, German and Spanish. The summary, the references and the captions to the figures are to be written each on a separate page and added to both copies of the manuscript. The address of the main author should be given at the bottom of the references. The place where the tables are to be inserted, must be marked in ink on the margin of the text. Figures are to be separate and not affixed in the text. On the back of each figure, the author is requested to write his name, the short title of the paper and the consecutive number of the illustration which must tally with the number marked on the margin of the text. An arrow indicates the way the figure should be set. Photographs must be clear, with good contrast and of the same size (best 6×9 cm.). The tables and graphs should be lined with Indian ink on white paper so as to make them well readable.

References should be limited, quoted from internationally accessible sources and not older than five years. If the number of references exceed ten, the editors are entitled to pick their choice.

Quotations should be adjusted according to Czechoslovak norm as follows: Articles in journals — author's surname and initials, title of the article (may be left out), international abbreviation of the journal, volume, number, page and year of issue. For instance: Frazer F. C., Warburton D.: Plast. reconstr. Surg., 33, 4 · 395, 1964.
Books and monographs — name of author, title of publication, place of issue, publisher, year of issue and — maybe — also page from which quotations has been taken. For instance: Burian F.: Surgery of Cleft, Praha, SZdN 1954.

Manuscripts which do not comply with these requirements, cannot be published.

The editorial board reserves the right to suggest to the author publication of his article in the form of an annotation, shorten the original manuscript, make corrections or, on account of comments made by the reviewers, return the manuscript to the author for redrafting. The papers must be sent to the editor in their final formulation. The galley proofs are done by the author, but no essential changes are permitted. The authors of original papers receive 50 reprints free of charge and without special order.

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