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COMBINED METHOD OF PLASTIC OPERATIONS ON CENTRAL FACIAL REGION FOLLOWING EXTENSIVE RADICAL SURGERY

S. D. Sidorov

Extensive forms of malignant neoplasms arising from the maxillary and palatine mucosa with pterygoid muscle infiltration often call for an extended combined resection of the organs and tissues involved, thus giving rise to combined defects of the central region of the face resulting in the absence of the orbital base, palate, Highmore's antrum wall, the sphenoid bone pterygoid processes, pterygoid muscles, the lateral wall of the pharynx and mandibular ramus [F. M. Khitrov, 1954; A. I. Paches, 1971; P. V. Naumov, 1973; V. A. Dunayevski, 1976; B. D. Kabakov et al., 1978; L. Bařinka, 1977, etc.].

MATERIAL AND METHODS

Replacements for the above listed defects were performed using wedge-shaped faciocervical and tube flaps according to Filatov in 7 patients aged 37 to 60 years. Radical surgery was followed first by primary plastic operation of the orbital face to cover the defect. Alongside with the surgical approach to the organs of the neck and facial bones, an incision from the nasal canthus down the lateral edge of the nose and the nasolabial sulcus via the corner of the mouth, the face and the mandibular body towards the centre of the clavicular bone and from the mastoid process towards the middle of the freshly performed cervical incision was used for the creation of a wedge-shaped cervicofacial skin flap (Fig. 1 and 2), for building the base of the orbit and for covering the maxillary and facial defects. For that purpose the flap was rotated 170—180° inside the defect and its end pulled towards and beyond the lower orbital canal in order to reconstruct the orbital base and the skin on the face (Fig. 1, 3).

After a period of 2 to 3 weeks, using the typical Filatov's method, we prepared a tube flap of the required size using the anteriolateral surface of

the abdomen and transferring it in the form of a forearm jump flap to the facial edge of the defect with a view to reconstructing the palate, the lateral wall of the pharynx, and the face. At the same time, the previously constructed rotated cervicofacial flap was cut off from the recreated bottom of the orbit

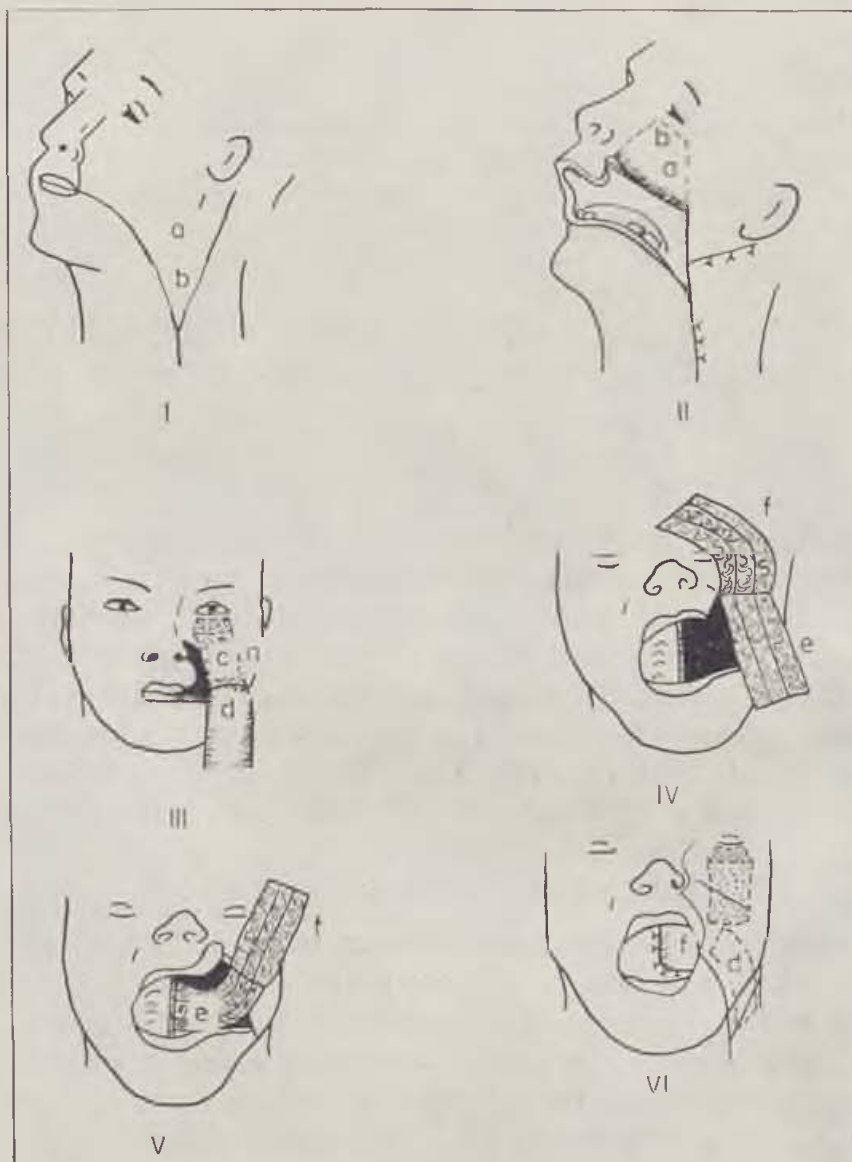


Fig. 1. Diagram of combined plastic operation using cervicofacial flap and Filatov's tube flap for postoperative complicated defect of the central facial zone. I. Outline of wedge-shaped cervicofacial flap (a — pedicle, b — flap cusp). II. Orbit base construction (b), facial wound closure (a). III. Flap transplanted to defect edge: part of cervico-facial flap detached from the face, rotated downward and sutured to rope flap. Facial wound thus created is covered with skin-connective tissue edge (c) from the rope flap end (d). IV. Skin-fibrous stripes constructed of rope flap: e — internal, f — external. V. Flap duplication and palate reconstruction. VI. Reconstruction of lateral pharyngeal wall (d)



Fig. 2. Patient K.: Extensive form of palatine mucosa cancer. On operating table: lines of incisions, top: outlines of wedge-shaped cervico-facial flap. Tracheostomy and intubation (Fig. 3—8 — same observation)

at the level of its lower edge, separated from the inner surface of the face back to the initial position, and sutured to the rope flap wound. The newly created facial wound was covered using the skin-fibrous portion of the rope



Fig. 3. Postoperative defect. Cervico-facial flap rotated 170—180° inside the defect to cover the wounds of the face, eye, sphenoid bone; flap cups forms orbit bottom



Fig. 4. Tubular flap healed to defect edge. Cervico-facial flap buccal part separated from wedge-shaped cusp, detached from inner side of cheek, rotated downward, and sutured to rope flap

flap, thus reconstructing the tissues from the flap, skin, and connective tissue of the face (Fig. 1 and 4).

After a period of 3 to 4 weeks, the rope flap was separated from the fore-



Fig. 5. Rope flap cut off from forearm and divided by longitudinal incisions into inner [bottom] and upper [top] full-thickness bands

arm. To reconstruct the palate, we made two longitudinal incisions to create two full-thickness bands — internal and external (Fig. 5) — used for the cutting out of flaps shaped and sized so as to cover the palatine defect. The



Fig. 6. Palate reconstruction. Inner band with skin turned to "Highmore's antrum" and partially sutured to nasal cavity mucosa. Outer band lying sideways

flaps were rotated into the oral cavity and sutured layer by layer: the inner flap (with the skin turned into the antrum of Highmore) to the nasal cavity mucosa, the outer one (with the skin turn into the oral cavity) to the mucosae of the palate and the upper lip (Fig. 1 and 6). This duplicate of full-skin flaps



Fig. 7. Skin reconstruction of the palate. Outer band turned into oral cavity

resulting from this served as a replacement of the palate with the epithelial cover of the "Highmore's antrum" bottom complete from the oral cavity side. Highmore's antrum was filled with iodoform-soaked tampons, the palate was covered with a protective platelet.



Fig. 8. Post-treatment appearance of reconstructed palate

The final stage of the plastic reconstruction took place after a period of 4 to 5 weeks: the flap pedicle was cut off from the face; the flap was cut longitudinally to form a full-thickness band which in turn — following lateral wall scar resection — was sutured to the wound, thus reconstructing the lateral wall of the pharynx from the newly created palated down to the epiglottis (Fig. 1, 7, and 8). The mandibular ramus was reconstructed afterwards.

The basic plastic operations were performed using endotracheal narcosis (sometimes with tracheostomy).

RESULTS AND DISCUSSION

During the basic phases of healing primary union was achieved in most of the patients operated on. The anatomical and functional results were satisfactory.

The primary plastic operation using a cervico-facial flap permitted the reconstruction of the base of the orbit, closing the eye wound, the sphenoid

bone and the face, thus preventing complications involving sight and cicatrization deformation of the circaorbital and facial regions. The internal buccal surface lining subsequently reconstructed from the rope flap skin developed into a sort of epithelial cover of the anterior wall of the reconstructed antrum of Highmore.



Fig. 9. Post-operative defect. Maxilla together with mandibular ramus, lateral wall of pharynx, pterygoid processes and muscles complete with lymphatic apparatus — all removed. Upper and lower lips held apart with forceps. Top: Inverted wedge-shaped cervico-facial flap

Plastic operations using Filatov's tube flap for the reconstruction of the base of the orbit, Highmore's antrum, palate and pharyngeal lateral wall created the necessary conditions for the normalization of speech and swallowing, as well as for the construction of suitable prostheses.

Two of the patients had mandibular autoplasty performed using costal cartilage. The rest of the patients refused to undergo this type of operation.

To give an illustration here goes with one observation: Patient K., aged 37, operated on for cancer of the palatine mucosa infiltrating Highmore's antrum, the orbit and pterygoid muscles, had — on Oct. 2, 1968 — a cervico-facial flap created under endotracheal narcosis and with the aid of tracheostomy (Fig. 2), and a combined extended block operation performed. The resulting defects of the palate, face, orbit, pterygoid processes of the sphenoid bone, lateral wall of the pharynx, and mandibular ramus (Fig. 9) were covered using primary plastic surgery. On Oct. 25, 1968, a Filatov's jump rope flap was taken from the anteriolateral surface of the abdomen, and, using a forearm jump, attached to the edge of the facial defect (Fig. 3). On Jan. 21, 1969, the rope flap was cut into full-thickness bands for the reconstruction of the palate and the bottom of "Highmore's antrum" (Figs. 4—7). On Feb. 26, 1969, a resection was performed of the disfiguring scar on the lateral wall of the

pharynx. The flap pedicle was cut off the face and turned into a skin flap for the closing of the wound on the lateral wall of the pharynx. The graft extended right from the newly reconstructed palate down to the epiglottis. The defect in the cheeks was covered by mobilizing its edges and by suturing layer. The postoperative period remained without complications.



Fig. 10. Patient after termination of treatment

In this way, the combined plastic operation of complicated defects involving the central facial region, using cervico-facial and Filatov's rope flaps, permits the reconstruction of the orbital base, Highmore's antrum, and lateral pharyngeal wall, and proves satisfactory from the functional and cosmetic points of view (Fig. 8 and 9). Fig. 10 shows the patient's appearance after the termination of the course of treatment.

The positive results obtained in plastic operations on the central region of the face can provide the basis for extending the range of operations as part of the radical treatment of patients with extensive malignant tumours of the face and oral cavity.

J. H.

SUMMARY

To replace complicated post-operative defects of the central facial zone, a combination of wedge-shaped facio-cervical and Filatov's tube flaps was

used for the reconstruction of the orbital base, palate, Highmore's antrum and lateral pharyngeal wall. The positive results thus obtained can serve as a basis for extending the volume of surgery in radical operations on patients with extensive malignant tumours of the face and oral cavity.

RESUME

Méthode de la plastie combinée de la zone centrale de la face après les opérations radicales

Sidorov S. D.

Pour la réparation des déformations compliquées après les opérations de la zone centrale de la face on utilise la combinaison de la plastie du lambeau en chéville de la face et du cou et du lambeau tubulé d'après Filatov. Les résultats positifs obtenus peuvent devenir la base pour l'augmentation du nombre des opérations en cas du traitement radical des malades avec les tumeurs malignes de la face et de la cavité de la bouche.

ZUSAMMENFASSUNG

Kombinierte Methode der Plastik der mittleren Gesichtszone bei erweiterten radikalen Operationen

Sidorow S. D.

Bei dem Ersatz kombinierter postoperativer Defekte der mittleren Gesichtszone benutzte man eine Kombination der Plastik der keilförmigen Wangen-Halslappens und des Rundstilllappens nach Filatow zur Wiederherstellung des orbitalen Bodens, des Gaumens, der Highmoreschen Höhe und der Seitenwand des Rachens. Die gewonnenen positiven Ergebnisse können als Grundlage dienen zur Erweiterung des Operationsumfanges bei radikaler Behandlung von Kranken mit umfangreichen bösartigen Tumoren des Gesichtes und der Mundhöhle.

RESUMEN

Método combinado de plástica de la zona central de la cara después de ampliadas operaciones radicales

Sidorov S. D.

Al rehacer los complicados defectos postoperatorios de la zona central de la cara se ha utilizado una plástica combinada con lóbulo cuneiforme cervico-facial y el tubular, según Filatov, para la reconstrucción del fondo orbital, del paladar, la cavidad Highmor y la pared lateral del fátige. Los obtenidos resultados positivos pueden servir como base para ampliar el volumen de las operaciones en el tratamiento radical de los enfermos con extensos tumores malignos de la cara y la cavidad bucal.

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NOTE

A joint annual meeting of the Austrian Society for Plastic Surgery, the Union of German Plastic Surgeons, the Swiss Society for Plastic and Reconstructive Surgery, and the German speaking Working Society for Peripheral Nerve and Vessel Microsurgery will be held in Innsbruck from **Sept. 23 to 26, 1981.**

University of Copenhagen, Hvidovre Hospital, Copenhagen (Denmark)
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ACUTE EXCISION OR EXPOSURE TREATMENT?

B. Sørensen, E. K. Pedersen, N. P. Fisker, J. P. Steensen

INTRODUCTION

One of the main themes of the 5th International Congress on Burn Injuries in June 1978 was Acute Excision, and at NIH's Consensus Development Conference in Supportive Therapy in Burn Care in November 1978, the debate on acute excision was concluded in the following manner: "In summary, excisional therapy not only has a place in the treatment of the thermally injured patient, but in theory can also promptly terminate the burn illness. Under the circumstances of adequate facilities, personnel, control and expertise, clinical trials in the efficacy of burn therapy can be recommended."

Several preliminary results of the thus suggested randomized controlled clinical trials will be presented in the following.

The Burns Unit has previously published accounts dealing with the philosophy supporting acute excision (2, 3, 4, 5, 6). To recapitulate, group A (acute excision) is evaluated, this concerns patients treated with surgical excision at the earliest possible moment, and never later than 24 hours post burn, when all burned tissue to viable tissue is excised and followed by immediate grafting of all excised areas. This group A is evaluated versus group E (exposure treatment) which is comprised of patients treated with exposure under 14 days, followed by excision of non-healed areas and immediate grafting.

The trial ran for three years, it started three months after the Burns Unit moved from Kommunehospital to Hvidovre Hospital.

STRATIFICATION

All patients admitted suffering from acute burn injuries over the three years in question, were placed in one of three groups:

Group U: Unsited for trial, comprised of different categories of patients that for various reasons were not suited to take part in any trial if the results were to be comparable (Table 1).

The remaining patients took part in the trial and were treated with either acute excision or exposure method and were stratified according to the criteria:

Table 1

	No. of pts.	B & F*)	Died
>6 years old	170	3.4	2
Face and hands	75	5.2	4
Electricity	16	0.5	0
Corrosions/congelations	33	0.6	0
Non treated	22	21.8	22
Admitted later than 24 h. post burn	61	5.3	4
Vacation and errors	44	7.4	4
Total	421	44.2	36

*) Expected mortality according to Bull & Fischer.

3-years material (1976—1979).

Group U = unsuited for trial.

ethiology: scalds or other burn injuries, age: over or under 60 years, extent of burn: small burns up to 15 %, medium: between 15 and 40 %, extensive: more than 40 % E. A. B.

After stratification and subsequent randomized distribution every even patient went into group A, every odd to group E.

COMPARABILITY

The purpose of stratifying the material is in fact to achieve reasonable comparability between group A and E. In doing so, however, only a few — although important — conditions are taken into consideration (Fig. 1).

One method of evaluating the stratification on one hand and comparability on the other is by analyzing the two materials by means of Bull & Fi-

Table 2

E. A. B.	No. of pts.		B & F*)		Died	
	A	E	A	E	A	E
0—15 %	33	36	0.6	0.8	1	1
15—40 %	19	17	4.5	3.5	4	2
≥ 40 %	8	9	5.6	7.0	3	5
≥ 60 years	13	14	9.4	9.2	8	8
Total	73	76	20.1	20.5	16	16

*) Expected mortality according to Bull & Fischer.

3-years material (1976—1979).

Group A (Acute Excision) and E (Exposure).

Mortality.

scher's formula (1). Bull & Fischer's formula indicates the risk a patient has of dying from burn injury in terms of a decimal fraction, this risk being decided by the age of the patient and the extent of the burn. If the material is divided into four groups as indicated by the figure:

1. Patients with a stipulated mortality of 0 % according to Bull & Fischer's formula.

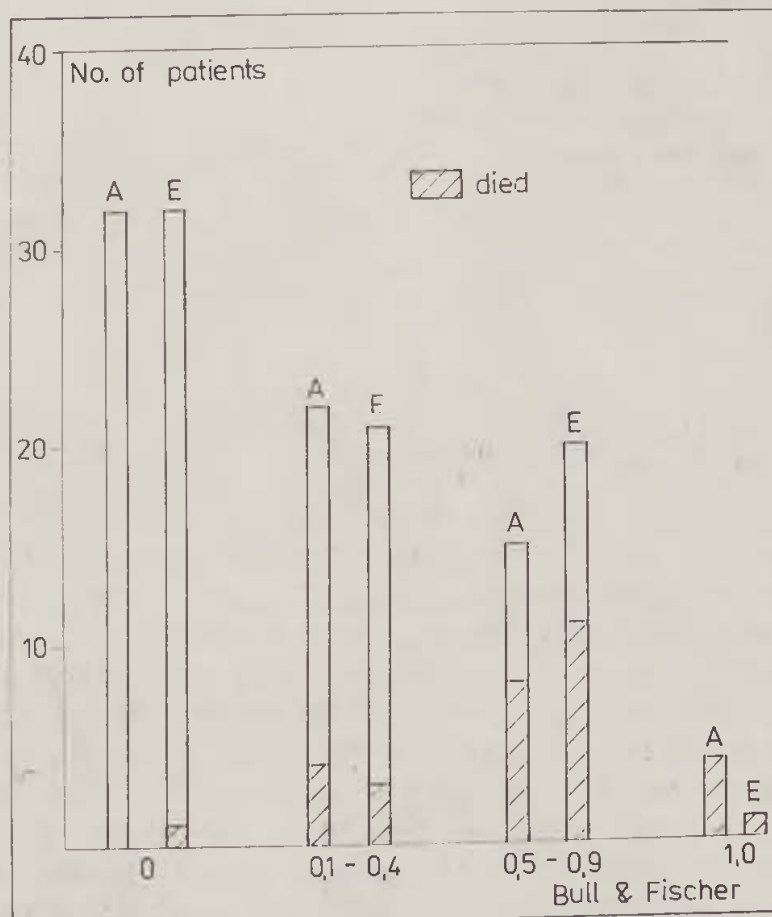


Fig. 1. 3-years material (1976—1979). Group A (Acute Excision) and E (Exposure). The material is divided into groups of severity of burns according to Bull & Fischer's formula:

Group I: Patients with an expected mortality of less than 0,1

Group II. Patients with an expected mortality from 0.1—0.4

Group III. Patients with an expected mortality from 0.5—0.9

Group IV: Patients with an expected mortality over 1.0

2. Those with a stipulated mortality of 0.1—0.4, in other words that have a 10—40 % chance of dying.

3. Patients with an expected mortality from 0,5—0,9.

4. Those with a stipulated mortality of more than 1.0, or a 100 % chance of dying.

If the two materials are subdivided thus, Figure 1 demonstrates practically identical figures for mortality for each group.

RESULTS

The mortality found in group A and group E is astonishingly alike, as seen in table 2. The Bull & Fischer figures appearing in the table are the result of addition of decimal fractions. For example: of the 73 patients in group A,

Table 3

	A	E
I Pulmonary burns	7	2
II Pre-existing illness	4	5
III Complications to burns (— infection)	1	3
IV Infections	4	6
Total	16	16

Causes of death.

3-years material (1976—1979).

Group A (Acute Excision) and E (Exposure)

20.1 patient (not per cent, but patients in absolute numbers) were stipulated to die, that is to say if treatment results were as those found in Bull & Fischer's material. The actual number of deaths observed was 16. The same applies to group E: the total number of patients was 76, the calculated mortality was 20.5 patient, and the observed number was 16. In other words: quite equivalent numbers; furthermore if the results are divided up according to stratification groups, the numbers will also be found to be astonishingly equivalent.

The obvious — and correct — conclusion must be that whether acute excision or exposure treatment is the method of choice, when applied to an unsorted material, mortality will be found to be absolutely the same in both groups.

Profitable knowledge will, however, be gained if the causes of death also are studied (Table 3).

The causes of death are not found to be the same in the two groups. In group A lung burns predominate, whereas fewer lung burns appear in group E, where infections and complications are the predominant causes of death. This latter group is also characterized by generally dying after a long interim.

From a coarse measure: mortality, we shall now proceed to a finer: morbidity (table 4).

As far as morbidity is concerned table 4 demonstrates that it is found to be less in group A than group E. It may especially be noted that the number of cases of thrombosis is found to be greatest in group E, and that the number of survivors with proved sepsis is smallest.

The amount of resources (shown in table 5) needed for a particular method of treatment may be calculated by means of many criteria and by using various parameters.

An indication of the resource-consumption may be found by employing the percentage of full skin thickness burned as a criterium, and as parameters: the duration of the operation, the number of blood transfusions and the time spent in hospital.

Table 4

	A		E	
	All (73 pts)	Survivors only (57 pts)	All (76 pts)	Survivors only (60 pts)
Infections	26	23	44	40
Infections + possibly sepsis	7	4	9	6
Sepsis	5	3	7	1
Pneumonia	8	1	4	1
Gastro-intestinal ulcers	8	1	11	4
Burn psychosis	2	2	7	6
Emboli	1	0	5	2
Complications total	57	34	87	60

3-years material (1976—1979).

Group A (Acute Excision) and E (Exposure).

Morbidity.

It is quite logical that surgery takes longer in group A as opposed to group E, — in fact 26.5 minutes for the former, 19.6 minutes for the latter — still calculated in terms of per cent full skin thickness burned.

Table 5

Per 1 % full thickness burn:

Per 1 % full thickness burn	A	E
— Surgery time (minutes)	26.5	19.6
— Blood transfusions (number)	1.2	1.2
— Days in hospital	3.2	3.6

3-years material (1976—1979).

Group A (Acute Excision) and E (Exposure).

Resources.

The necessary number of blood transfusions is astonishingly alike in both groups, being 1.2 portions which is equivalent to 540 ml of blood.

The time spent in hospital is a little longer in group E.

If the resource-consumption is calculated in this manner, the two methods of treatment are found to consume almost the same.

SUMMARY

In a randomized controlled clinical trial two methods of treatment are tried:

Acute Excision, group A, excision and grafting in the shock-phase.

Exposure, group E, exposure for about 14 days, followed by excision and grafting.

Randomization is successful, and the two groups are found to be comparable.

On the two unsorted materials it is shown that

- 1: the crude mortality is the same,
- 2: the causes of death appear to differ,
- 3: the morbidity is least in group A,
- 4: both groups consume almost the same amount of resources,
- 5: a follow-up examination has as yet, not taken place.

The final conclusion will have to bide point 5, but will probably be that certain categories of patients should be treated with exposure, others with acute excision.

RESUME

Traitement par l'excision urgente ou la méthode expectative?

Sørensen B., Pedersen E. K., Fisquer N. P., Steensen J. P.

L'expérience clinique de contrôle avec les groupes des clients brûlés a vérifié deux méthodes: l'excision urgente avec la transplantation dans la phase de choc (le groupe A) et la méthode expectative (le groupe E) avec l'excision et la transplantation après 14 jours. Le groupement des clients sans choix en du succès parce qu'on pouvait faire la comparaison. Notre expérience a montré, que: 1. la mortalité était presque la même dans les deux groupes; 2. les causes de la mort étaient différentes; 3. la morbidité était plus petite dans le groupe A; 4. le traitement des deux groupes exige presque les mêmes moyens; 5. les observations de longue durée n'étaient pas encore faites. Pour prononcer une conclusion définitive, il faut d'abord obtenir les résultats quant au point 5, mais on peut dire, que dans certains cas il faut mieux choisir la méthode expectative et dans les autres cas il faut préférer l'excision urgente.

ZUSAMMENFASSUNG

Behandlung durch akute Exzision oder Methode des Abwartens?

Sørensen B., Pedersen E. K., Fisker N. P., Steensen J. P.

Im kontrollierten klinischen Versuch mit Gruppen ohne Auswahl zusammengestellter Kranken wurden zwei Methoden überprüft: akute Exzision mit Transplantation bereits in der Schockphase (Gruppe A) und Methode des Abwartens (Gruppe E) mit Exzision und Transplantation etwa nach 14 Tagen. Die zufällige Zusammenstellung von Patienten war erfolgreich, man konnte sie vergleichen. Unser nichteingeteiltes Material hat gezeigt: 1. die Mortalität war in beiden Gruppen ungefähr gleich; 2. die Todesursachen waren verschieden; 3. die Morbidität war in der Gruppe A geringer; 4. die Behandlung beider Gruppen erfordert fast gleiche Menge von Mitteln; 5. langzeitige

Überwachung wurde nicht unternommen. Für definitive Schlussfolgerungen müssen die Ergebnisse des Punktes 5 abgewartet werden, diese werden aber wahrscheinlich so sein, daß es bei einigen Patienten besser ist abzuwarten, während bei anderen eine akute Exzision notwendig ist.

RESUMEN

Tratamiento a través de excisión aguda o el método de expectación?

Sørensen B., Pedersen E. K., Fisquer N. P., Steensen J. P.

Por experimento clínico de control con grupos de quemados, compuestos sin selección, se estuvieron chequeando dos métodos: el de excisión aguda con transplantación ya en la fase de conmoción (grupo A) y el de expectación (grupo E) con excisión y trasplante al cabo de unos 14 días. La agrupación casual de pacientes resultó exitosa, dando posibilidad de comparaciones. Nuestro material no seleccionado demostró como sigue: 1. la mortalidad fue aproximadamente la misma en ambos grupos; 2. las causas de la muerte fueron distintas; 3. la morbilidad era menor en el grupo A; 4. el tratamiento de ambos grupos requiere de casi igual cantidad de recursos; 5. no se ha hecho observaciones a largo plazo todavía. Para hacer una conclusión definitiva hay que esperar los resultados del punto 5, pero es de suponer que la definitiva sería la de que en algunos pacientes resulta mejor esperar mientras que en otros se hace necesaria la excisión aguda.

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BURN-INDUCED IMMUNOLOGICAL MODIFICATIONS. PRELIMINARY RESULTS WITH LEVAMISOLE

F. Mazzoleni, A. Chiarelli, A. Corsano, O. Dal Lago, U. Fagiolo

Many clinical and experimental studies indicate that significant changes occur in the immune system following thermal injury (1, 3, 4, 5, 6, 7, 11). The reduction in immunocompetence seems to consist of an impairment in cellular immunity, which appears responsible for the particular susceptibility to infective agents and high incidence of septicemia among the causes of death observed in burn patients.

Levamisole has been widely used as in anthelmintic therapy. In 1971, it was reported that Levamisole enhanced the efficacy of antibrucellosis vaccine (8) since its administration 2 days after vaccination conferred higher protection without inducing variations in specific antibody titers. Subsequent studies showed that this protective effect was due to significant capacities of "immunomodulation" at the cellular immunity level.

In the view of these results, Levamisole is currently being tested in many diseases where immune system disorders have been recognized as the underlying cause. We have studied the therapeutic effects of Levamisole in burn patients. To this end, we have evaluated its effects on some immunological parameters and on the evolution of the disease, with particular reference to the incidence and course of septicemias.

MATERIAL AND METHODS

Patients

This study concerns 234 patients of both sexes, ranging in age from 1 to 82 years with burn injury evaluated at 15–90 % BSA, admitted during 1977, 1978, and 1979.

In 1979 all patients (70 ranging in age from 1 to 79 years, thermal injury: 15–90 % BSA) were treated with Levamisole (2.5 mg/Kg BW per week for 3–5 weeks in 15 patients, and 7.5 mg/Kg BW per week in 55 patients). Treatment was initiated at 3rd–4th day after injury. Wound sepsis and septicemia were checked according to a standard protocol at least twice a week in all patients, and more frequently in severe or clinically suspect cases.

Patients hospitalized during 1977 and 1978 were employed as controls. Standard therapy was essentially similar in both groups.

Immunological follow up

This study concerns 17 patients: 12 were treated with Levamisole (age: 2—79, thermal injury: 20—90 % BSA) and 5 were treated with standard therapy (age: 12—61, thermal injury: 20—35 % BSA). In these patients the following parameters were evaluated weekly for 4 weeks or until death:

1. Peripheral blood monocytes, Ig_s^+ cells (Ig_s^+), E-active rosette forming cells (E-ARFC), E-rosette forming cells (E-RFC), T lymphocytes with Fc receptors for IgG (T_G). In addition, T lymphocytes with Fc receptor for IgM (T_M) were evaluated in 3 patients.

2. Skin reactivity to PPD, varidase and DNCB at the first and fourth week after injury;

3. Antibody response to HL-A antigens infused with plasma and blood transfusion.

The methods employed for the isolation of peripheral blood lymphocytes and for the identification of monocytes, Ig_s^+ , E-ARCF, E-RFC and lymphocytes with Fc receptors for IgG and IgM have been described in detail previously (1). Skin test were performed as suggested by Friedman and Gold (2). Anti HL-A antibodies were detected by testing sera against a panel of HL-A ABC typed peripheral lymphocytes from healthy donors in complement depended microtoxicity (10). Sera were collected at regular intervals after thermal injury and stored at $-20^{\circ}C$ until use.

RESULTS

Clinical results are reported in Table 1 which indicates the number of cases and incidence of septicemia (ascertained with blood culture) with

Tab. 1. Levamisone Treatment: Incidence and Outcome of Septicemia* and Pseudomonas Induced Septicemia* in Relation to Preceding Years

Year	N° of hospitalized patients	BSA burnt (% average)	Age Average	Total septicemia			Pseudomonas induced septicemia		
				N°	% of hospitalized	Survival	N°	PS. Sept. Total $\times 100$	Survival
1977	90	35 %	26	14	15.5 %	6 (42.8 %)	12	87.7 %	4 (33.3 %)
1978	74	32 %	37	12	16.2 %	6 (50.0 %)	9	75.0 %	3 (33.3 %)
1979 (Levamisole)	70	33 %	26	10	14.2 %	7 (70.0 %)	6	60.0 %	3 (50.0 %)

*Verified by blood culture

Tab. 2. ANTI HLA Antibodies in Burn Patients

	N° patients	Plasma Transfusions (mean)	Blood Transfusions (mean)	With ANTI HLA antibodies	Without ANTI HLA antibodies
Treated with levamisole	12	21	4	5 (41.6 %)	7 (58.4 %)
Untreated with levamisole	5	8	3	0	5 (100 %)

regard to the number of patients in each single year, as well as the number of cases and incidence of septicemia due to *Pseudomonas* with regard to the total number of septicemia cases, and relative survival. While septicemia incidence does not vary in the three years considered, the percentage of patients

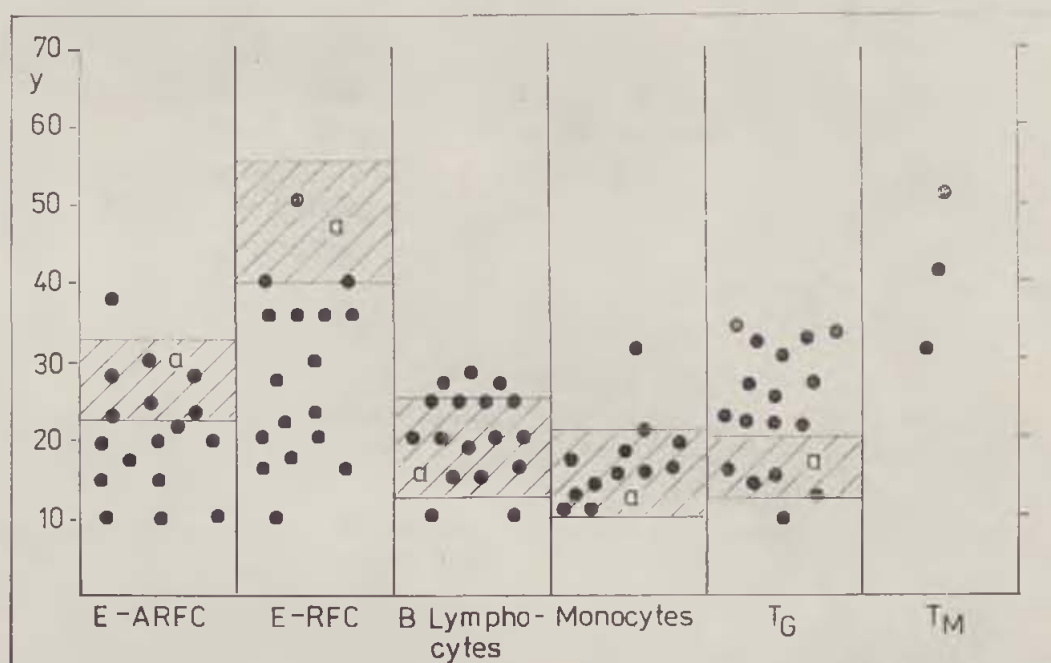


Fig. 1. Mononuclear cell (%) in burn patients. y — cell percentage, a — values in normal subjects

surviving septicemia is higher in 1979 compared to the previous years (42.8 % in 1977, 50 % in 1978, and 70 % in 1979). This variation depends on both a lower incidence of septicemias from *Pseudomonas* as well as greater survival in these patients. This finding is in agreement with observation made during therapy, since onset and course of septicemia were not acute. In one high burn patient (90 % predicted mortality according to McCoy et al.) the septicemia episode had a long duration and blood cultures were positive for *Pseudomonas* from the 8th to the 22nd day of disease.

Immunological results are illustrated in Figures 1, 2, 3, and 4. During the first week of treatment, no differences were observed between the 2 patients groups. Fig. 1 shows the percentage values of the different subpopulations of mononucleated cells in treated and non-treated burn patients

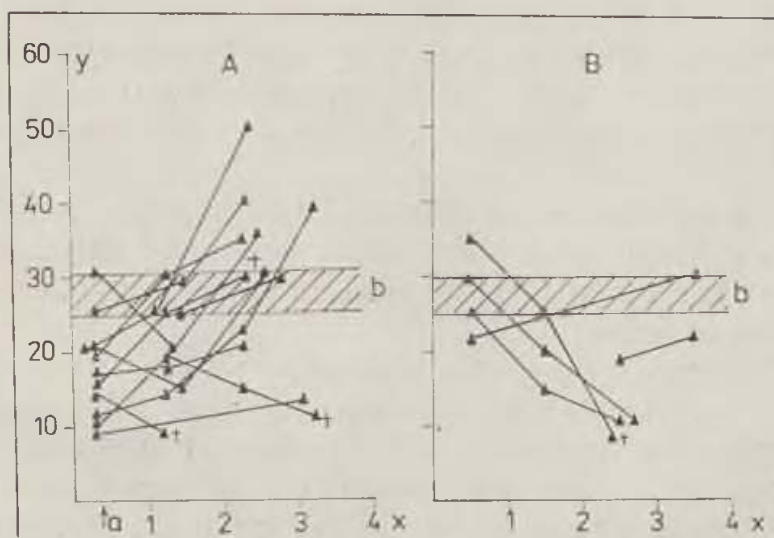


Fig. 2. E — active rosette forming cells (%) in burn patients. A — Treated with Levamisole, B — Untreated with Levamisole, x — weeks, y — E-active rosette forming cells (%), a — start of treatment, + — dead, b — values in normal subjects

compared to normal values. E active rosettes are decreased in 10 out of 17 cases, and E standard rosettes in 14 out of 17. B lymphocyte and monocyte percentages were within normal limits. Tc instead were increased in 12 patients. Anti-HL-A antibodies were absent. Cutaneous reactivity was negative

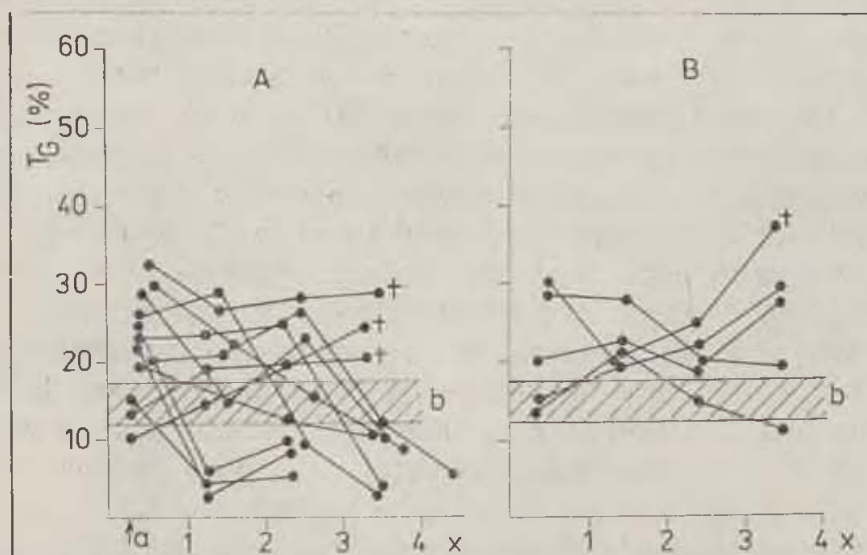


Fig. 3. T_G (%) in burn patients. A — Treated with Levamisole, B — Untreated with Levamisole, x — weeks, a — start of treatment, + — dead, b — values in normal subjects

for all antigens in 7 cases, and positive for one or two antigens in the other 10.

Appreciable differences in the two patient groups were observed in the successive phases of the disease (Figs. 2, 3, 4). In treated patients, E active rosettes tended to increase by the end of the second week (Fig. 2), and in 8 cases normal reached or higher than normal values within the 3rd or 4th weeks. T_c also displayed a net tendency towards reduction, and at the 3rd week T_c were normal in 9 cases. In parallel with these findings, skin reactions became more positive. In 5 cases out of 12, anti-HL-A antibodies were detected (Fig. 4).

In the group of non-treated patients, after 3 weeks T_c values (Fig. 3) were higher than normal in 4 out of 5 cases, with active and normal E rosettes (Fig. 2) reduced in 4 out of 5 cases. Skin tests were not significant and anti-HL-A antibodies were absent (Fig. 4).

Figure 3 illustrates a significant correlation between T_c decrease and survival in the treated patients. The 9 patients in whom a progressive reduction in this lymphocyte sub-population occurred are all surviving. On the other hand, 3 burn patients at high death risk (80 %, 96 % and 100 % respectively, according to McCoy et al.) who showed increasing T_c percentage values during the course of their disease died following septicemia. In all three patients, several blood cultures were positive for *Pseudomonas* and other agents. In the group of non-treated patients, one died following an episode of bronchial pneumonia. In this case also, T_c percentage was high (39 %) at the moment of death.

CONCLUSION

In the light of these observations, it is clear that burn injury brings about appreciable changes in some immunologic parameters, and these effects are more detectable at the cellular immunity than at humoral immunity level. Some subpopulations of the T lymphocytes are mostly affected. In many patients, the percentages of T cells with Fc receptors for type G immunoglobulins are significantly increased. Miller and Baker (6) have shown that the T cells of burn patients with reduced PHA response exert a suppressive effect on mixed lymphocyte reactions between high responsive normal subjects. In another study, Miller and Claudy (7) observed that the T cells obtained from the spleens of mice with burn injury are able to suppress in vitro the primary antibody response to sheep red blood cells by normal syngeneic cell. It is therefore likely that T_c represent a T lymphocyte subpopulation that acts negatively on the regulation of immune response, and thus may be responsible for the remarkable susceptibility to infective agents observed in extensively burned patients. The correlation observed in our study between T_c variation on one hand, and incidence and course of septicemia on the other supports this interpretation. Levamisole appears to influence the immunological modifications induced by burn injury by favoring a re-equilibrium between various populations of mononucleated cells. In a good number of cases Levamisole brings T_c and E-ARFC values back to normal limits. This action corresponds

clinically with a lower incidence of septicemia from *Pseudomonas* and a more favorable course of the septicemias in general.

SUMMARY

Clinical and immunological observations suggest that fatal septicemia in severe burns is related to an impairment of the immune system. In 1979 all patients admitted to our Burn Center were treated with Levamisole, an immunomodulating substance (2.5—7.5 mg/kg BW weekly for 3—5 weeks starting on the 4th—5th day of hospitalization). We observed a reduction in septicemia from *Pseudomonas* and a higher survival rate compared to previous years (1977—1978).

17 burn patient, of whom 12 were treated with Levamisole and 5 untreated, underwent immunological study. We examined: 1. changes in mononuclear peripheral blood cells: monocytes, E active rosette forming cells (E'ARFC), E rosette forming cells (E'RFC), Ig_s+ cells, and T lymphocytes with Fc receptors for IgG (T_G); 2. skin reactivity to PPD, varidase, and DNCB; 3. antibody response to HL-A antigens infused with plasma and blood transfusions. In the first week, low levels of E'ARFC and E'RFC were observed in 14 of 17 patients, and an increase in T_G in II. Skin reactivity was negative in 7 patients and positive for only one or two antigens in the other ten. Monocytes and Ig_s+ cells were in the normal range. Anti HL-A antibodies were absent. In the Levamisole treated group (age: 2—79, thermal injury: 20—90 % BSA), after three weeks, 9 patients had normal values for % T_G, E'ARFC and E'RFC, positive skin tests and a favorable clinical outcome. In contrast, in 3 patients dead with septicemia the number of T_G remained high. In the control group (age: 12—61 years, thermal injury: 20—35 % BSA), after three weeks the immunological changes observed during the first week remained generally unmodified. Anti HL-A antibodies were detected only in five Levamisole treated subjects.

RÉSUMÉ

Modifications immunologiques dues aux brûlures. Résultats préalables avec Levamisol
Mazzoleni F., Chiarelli A., Corsano A., Dal Lago O., Fagiolo U.

Les observations cliniques et immunologiques indiquent que la septicémie fatale chez les clients gravement brûlés est en connexion avec les déformations du système d'immunité. En 1979 on a traité tous les clients dans notre Centre pour le traitement des brûlures par Levamisol. Ce médicament fut administré (2,5—2,7 mg/kg du poids du corps par semaine) pendant 3—5 semaines. L'administration de ce médicament commença le quatrième ou le cinquième jour de l'hospitalisation. On a remarqué la réduction de la septicémie des *Pseudomonas* et le nombre des survivants a été plus grand en comparaison avec les années 1977—1978. On a observé 17 clients dont 12 ont été traités par l'administration de Levamisol et dont les autres se sont soumis à la thérapeutique courante. On a remarqué: 1. les transformations dans les cellules mononucléaires du sang périphérique; 2. la réactivité cutanée pour PPD, DNCB et pour la varidase; 3. la réaction des anticorps sur l'antigène HL-A et la transformation du sang. Chez 14 des 17 clients brûlés on a trouvé le volume bas de E'ARFC et E'RFC dans la première semaine et l'augmentation de T_G dans la deuxième semaine. La réactivité cutanée fut négative chez 7 clients et positive avec un seul des deux antigènes dans les autres cas. Les monocytes et les cellules Ig_s+ étaient dans la norme. Les

anticorps Anti-HL-A n'étaient pas présents. Dans la groupe des clients traités par l'administration de Levamisol (2—29 ans, 20—90 % de la surface du corps brûlé) 9 clients avaient au bout de trois semaines des valeurs normales de % T_G , E'ARFC, E'RFC et des résultats positifs des réactions cutanées et l'état clinique favorable. Chez trois clients qui sont morts à cause de la septicémie, le nombre de T_G restait au contraire élevé. Dans le groupe de contrôle (12—61 ans, 20—35 % de la surface du corps brûlé) les transformations immunologiques observées au cours de la première semaine n'ont presque pas changé après trois semaines. Les anticorps Anti-HL-A n'ont été remarqués que chez 5 clients qui ont été traités par l'administration de Levamisol.

ZUSAMMENFASSUNG

Durch Verbrennung ausgelöste immunologische Veränderungen. Vorergebnisse mit Levamisol

Mazzoleni F., Chiarelli A., Corsano A., Dal Lago O., Fagiolo U.

Klinische und immunologische Beobachtungen deuten daraufhin, daß die fatale Septikämie bei Schwerverbrannten mit einer Schädigung des Immunitätssystems verbunden ist. Im Jahre 1979 wurden alle in unserem Zentrum für die Behandlung von Verbrannten hospitalisierten Patienten mit Levamisol behandelt. Diese immunomodulierende Substanz wurde verabreicht (2,5—2,7 mg/kg Körpergewicht wöchentlich) 3—5 Wochen lang vom vierten bis fünften Tag der Hospitalisierung. Man beobachtete eine Verminderung der Pseudomonadenseptikämie und einen Anstieg in der Zahl der Überlebenden im Vergleich zu den vorhergehenden Jahren (1977—1978). Die Beobachtungen umfassten 17 Patienten, von denen 12 mit Levamisol und 5 mit standardmäßigem Therapieverfahren behandelt wurden. Es wurden beobachtet: 1. Veränderungen in den mononukleären Zellen des peripheren Blutes: Monozyten, aktive rosettenbildende E-Zellen (E'ARFC), rosettenbildende E-Zellen (E'RFC), Ig_{S+} und T-Lymphozyten mit Fc-Rezeptoren für IgG (T_G); 2. Hautreaktivität für PPD, Varidase und DNCB; 3. Antikörperantwort auf das mit dem Plasma eingeführte HL-A-Antigen und Bluttransformation. Bei 14 unter den 17 Verbrannten beobachtete man einen niedrigen Gehalt an E'ARFC in der I. Woche und einen Anstieg von T_G in der zweiten Woche. Die Hautreaktivität war bei sieben Kranken negativ, positiv war sie lediglich mit einem von zwei Antigenen bei den übrigen zehn Patienten. Monozyten und Ig_{S+} -Zellen waren in der Norm. Anti-HL-A-Antikörper fehlten. In der mit Levamisol behandelten Gruppe (Alter 2—79 Jahre, Verbrennung im Umfang von 20—90 % der Körperoberfläche) hatten 9 Patienten nach drei Wochen normale Werte für % T_G , E'ARFC und E'RFC, positive Hautteste und einen günstigen klinischen Verlauf. Bei drei Kranken dagegen, die infolge von Septikämie gestorben sind, blieb die T_G -Zahl hoch. In der Kontrollgruppe (Alter 21—61 Jahre, Verbrennung im Umfang von 20—35 % der Körperoberfläche) blieben die in der ersten Woche beobachteten immunologischen Veränderungen nach drei Wochen im allgemeinen unverändert. Anti-HL-A-Antikörper wurden lediglich bei fünf mit Levamisol behandelten Patienten beobachtet.

RESUMEN

Modificación inmunológica provocada por quemadura. Resultados preliminares con Levamisol

Mazzoleni F., Chiarelli A., Corsano A., Dal Lago O., Fagiolo U.

Los estudios clínicos e inmunológicos atestiguan de que la septicemia fatal en los gravemente quemados está acompañada del deterioro del sistema de inmunidad. En

año 1979 todos los pacientes ingresados en nuestro Centro de Tratamiento de Quemaduras fueron tratados a base de Levamisol. Esta materia inmunomodulatoria se administró [2,5—2,7 mgs./kg. del peso del cuerpo a la semana] durante 3 a 5 semanas, empezando por el cuarto a quinto día de ingresar el paciente. Se observó una reducción de la septicemia de pseudomónades y una mayor cantidad de sobrevivientes comparado con los años precedentes (1977—78). Se siguió a 17 pacientes de los que 12 fueron tratados con Levamisol y 5 por terapia estandarizada. Se pudo observar: 1. cambios en células mononucleares de la sangre periférica: monocitos, células E activas que crean rosetas (E'ARFC), células E que crean rosetas (E'RFC), Ig_s+ y linfócitos T con Fc receptores para IgG(T_G); 2. reactividad dérmica para PPD, varidasis y DNCB; 3. respuesta de factores de protección a la introducción, junto con el plasma, del antígeno HL-A y una transformación de la sangre. En 14 de los 17 quemados se observó un bajo contenido de E'ARFC y E'RFC en la primera semana y un aumento de T_G en la segunda. La reactividad dérmica fue negativa en 7 pacientes siendo positiva, con sólo uno de dos antígenos, en los demás diez. Los monócitos y células Ig_s+ se encontraban en conformidad con la norma. Faltaron las antimaterias anti-HL-A. En el grupo tratado por Levamisol (de 2 a 79 años de edad, superficie quemada del cuerpo 20 al 90 %) después de tres semanas presentaban 9 pacientes cantidades normales de tanto por ciento de T_G, E'ARFC y E'RFC, pruebas dérmicas positivas y un favorable desarrollo clínico. En cambio, en tres enfermos, que murieron de septicemia, la cantidad de T_G se quedaba elevada. En el grupo de control (de 12 a 61 años de edad, superficie quemada del cuerpo 20 al 35 %) los cambios inmunológicos observados en la primera semana quedaban incluso después de tres semanas sin alteración. Las antimaterias Anti-HL-A se observaron sólo en 5 pacientes tratados con Levamisol.

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PREVENTION OF BURNS IN CHILDREN

J. Babík

The growing number of burns and scalds affecting a rising proportion of children makes us think not only of the predicament of the victims but also of the causes of these emergencies and of what we ourselves have done in the way of burns prevention. A severe and painful thermal injury represents a physical and mental strain on the victim, on members of his or her family, as well as on the medical and nursing staff. In addition, the treatment is costly, too, coming to an average of about 80,000 Czechoslovak crowns for a single severely burned patient. As follows from statistics, roughly 60 people in Slovakia die annually as a result of burns sustained. The total number of burns amounts to 50,000 every year including 2,000 hospitalized cases (in accordance with methodological instructions issued by the Ministry of Health of the Slovak Socialist Republic) while the rest receive outpatient or lay treatment. The striking feature is that children up to the age of 15 represent 48% of the total number of burns, and that the most numerous group (33%) are children aged 2 to 5 years. The cost of material damage and medical and surgical treatment run into millions of crowns. This brief statistical survey leaves us little to boast of as it makes us guilty of inconsistency in the prevention of children's accidents in particular. The cost of prevention is difficult to estimate but it cannot be more than negligible compared with the cost of burns treatment.

Precise statistical processing of injuries and their causes is an essential condition for consistent prevention. Until now there have been no specific statistics on burn injuries in this country; consequently no effective counter-measures have been taken. How appropriate such measures are could be seen from the consistent processing of statistical data on injuries accomplished at some of the large enterprises such as the VSŽ East Slovak Iron and Steel Works and the ZŤS Heavy Engineering Works whose departments of safety at work took them as a basis for the adoption of such measures as would reduce substantially the rate of severe occupational accidents. Also, how substantiated such solid prevention of thermal injuries is can be seen from statistics avail-

able in those countries where prevention has already reached the necessary standard.

Proceeding from our own experience of the treatment of more than 5,000 burned patients at the specialized department for the treatment of burns in Košice-Šaca with the whole of Slovakia as its catchment area we were able to follow-up the causes of such accidents and to determine which way their prevention ought to be directed. The simplest method of accident prevention is the creation of the sort of environment which in itself would prevent accident from happening, though it is often equally necessary to try and change people's behaviour and habits.

Prevention requires the active interest of not just individuals but whole production organizations as well, indeed, the whole of our society as it is often changes in industrial production but also changes in attitudes and technological procedures that are called for. A large proportion of burns, particularly in children, occur as a result of the ignition and burning of garments, clothes, underwear, pyjamas, etc. As the results of our own research show, most of such products available on the Czechoslovak market contain 60 to 80% polyester but also other types of synthetic, easily inflammable constituents which together with expanding electrification and gas supply in households have been responsible for hundreds of severe injuries. Their inflammability and fast burning are even aggravated in that at higher temperatures they get stuck to the body thus preventing their removal as part of first aid. As a result there are secondary burns caused by burning garments. Such secondary burns tend to be very severe, indeed; no wonder then more than half the victims who succumbed to their burn wounds showed signs of secondary burns. Similar properties can be found in clothes made of other artificial fibres such as silon, chemlon, etc., the burning of which releases toxic substances with toxic effects on the respiratory tract and indeed the whole body. In the United States the proportion of burns in children resulting from the ignition and burning of garments was successfully reduced by introducing legislation (1971) which permits the use of exclusively non-inflammable or poorly inflammable materials for the production of children's clothes and underwear. This made the rate of such injuries drop considerably. It would, of course, be an exaggeration to demand the complete exclusion of synthetic fibres. It seems more reasonable to propose the use of synthetic fibres with flameproof finish, or at least to tag cloths, garments, underwear, upholstering and decorative fabrics with warnings of the presence of potentially dangerous inflammable substances.

As for electric contacts and injuries in children resulting from immediate contact in the socket or at the other end of plugged in cords, the number of children aged 2 to 5 who suffer such injuries is very high indeed. Older children are likely to get hurt at play near high-voltage electric lines, electric railway traction lines, unprotected transformer stations, etc. It was certainly no fortunate idea to place electric sockets a mere 40 cm above the floor in our new blocks of flats as this makes them within easy reach of the youngest and the most inquisitive children. Not even protective covers of synthetic materials have proved to be adequate protection. Obviously, if the sockets were installed

high enough to be out of children's reach a number of severe accidents could be prevented. Most children playing in the open air know nothing of the fact that an electric arc develops already at a certain distance from high-voltage electric lines. Balls thrown or kicked into the open windows or doors of transformer stations, too, have already caused serious accidents. The organizations responsible ought to be more consistent in protecting and labelling such installations as children are likely to ignore or not to recognize the conventional lightning symbols indicating dangerous high voltage. In wintertime and during flu epidemics we are always called to treat dozens of children suffering from scalds caused by hot steam administered by their parents. Bronchitis is thus complicated by scalds in the face, limbs, indeed, the whole body. Our ancestors' type of treatment is thus likely to do more harm than good. A much simpler and far less dangerous way is the hanging up of wet bed sheets in order to moisten the air in rooms. It was a striking revelation to learn that advice on the use of boiling camomille decoction often comes from doctors and other health personnel. Children are often intrigued by automatic washing machines; these heat water up to 90 °C, and this is where scalds are often likely to occur, too, in the process of opening the side insertion hole. Severe scalds might easily be prevented by placing a safety catch out of children's reach. In Denmark where such safety catches were introduced in 1971 there have been no such children's injuries since. Scalds are also likely to occur in the showering or bathing of children when for unknown reasons cold water supply is suddenly stopped — whether by mistake or due to some technical fault. The point to be borne in mind here is that 70 °C hot water will cause 3rd degree scalds in a matter of half a second. This particular problem could easily be coped with by bringing in more reliability in the heating of water, the temperature of which should never exceed 60 °C (not to mention the need for energy saving).

The parents, brother or sisters, grandparents or educators are always directly or indirectly responsible for each new case of burns in children. This responsibility ought to be emphasized and enforced, if necessary, too. Children often sustain thermal injuries when they are being looked after by their elder brothers or sisters (often only a little older themselves) or by their grandparents, whose technical knowledge is limited and attention inadequate.

Safety precautions are the most frequently violated in people's homes. The mishandling of gas and electric appliances inadequately stored and unprotected inflammable substances such as industrial petrol, solvents, volatile substances, or even matches all too often cause irreparable damage and injury not only to adults but particularly to children who often suffer for their own parents' negligence or lack of caution.

Keeping the public informed is an important method of prevention as this is often the only way to achieve the necessary changes in the environment, in people's behaviour, attitudes and knowledge, as well as in the provision of first aid. There are two important factors likely to assist effectively in the prevention of accidents in general and burns in particular. First, the mass

media, especially the television and the press, second, our schools were the principles of prevention ought to be promoted all the time alongside with instructions on first aid and ways and means of providing it. This in itself would be a valuable contribution to society and to those affected. Warnings should be made particularly as regards inflammable liquids, petrol, acetone, etc., but also as regards methods of first aid in cases of burning garments, fires in rooms and building, plans of fire protection, the correct procedures in bathing babies, the dangers of playing with matches, electrical appliances, etc. Parents should be warned not to cook or consume hot liquids in the presence of very young children. Unfortunately, instructions on first aid do not contain adequate information on how to deal with thermal injuries. Thus we keep coming across cases where the burned were given first aid in the form of milk, eggs, flour, Diesel oil, etc. Only very few burned patients had their burn wounds cooled as they should.

Compared with cardiovascular or neoplastic diseases the rate of incidence in burns is lower but the cost of treatment is substantially higher, especially in severe burns. Those affected suffer for the rest of their lives due to burn-induced disorders, scars or deformities and disfiguration, and so do their parents and other relatives. As burn injuries affect mainly children aged 1 to 5 years, effective ways and means of prevention ought to be introduced as fast as possible. The precautions recommended and well-developed prevention ought to reduce the proportion of burn injuries especially in the presence of stepped up co-operation involving the health services, fire brigades, television, press, schools, etc. Also involved in the prevention efforts should be cured patients who are best called to describe their often shattering experiences. Investments ought to be made into preventive measures and programmes if only in order to reduce the cost involved in the treatment of the burned, and thus to prevent more suffering by innocent children.

The idea of the present paper was to analyze at least some of the many causes and effects, and to give a few facts and figures as seen in terms of effective prevention.

J. H.

SUMMARY

The growing number of accidents involving children up to the age of 5 is discussed. The causes of some of the injuries resulting from this are analyzed, and methods of preventing burn injuries are proposed.

RESUME

Prévention des brûlures chez les enfants

Babík J.

Dans cet article l'auteur s'occupe de l'augmentation du nombre des blessures chez les enfants de 0 jusqu'à 5 ans. L'auteur fait l'analyse des causes de certaines blessures et il propose en même temps les mesures de prévention.

ZUSAMMENFASSUNG

Vorbeugung der Verbrennungen bei Kindern

Babík J.

In seiner Arbeit befaßt sich der Autor mit der wachsenden Zahl von Unfällen, die Kinder im Alter von 0 bis 5 Jahren betreffen. Er analysiert die Ursachen der Entstehung einiger Unfälle und legt zugleich Vorschläge für die Prophylaxe und Vorbeugung der Entstehung von Verbrennungsunfällen vor.

RESUMEN

Frecuencia de quemaduras en los niños

Babík J.

El autor llama la atención a la creciente tendencia al producirse accidentes que afectan a los niños entre 0 y 5 años de edad. Analiza las causas de algunos de los accidentes proponiendo al mismo tiempo la prevención de estos accidentes por quemadura.

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EXPERIENCE IN BURNS TREATMENT IN PATIENTS OVER 60

J. Bláha

Thanks to the widening range of therapeutical opportunities as well as to the rising standards of living and hygiene the average life expectancy has been rising accordingly. This is in connection with the constantly growing proportion of hospitalized patients over 60. While it is difficult to define the exact age limit for old age, the lower limit of senescence is generally put at 60 years of age. Patients over 60 years of age are reported to have as much as 75% death rate in deep burns affecting 25—30% of the body surface.

Thermal injuries rank among the severest affections, and their management in elderly patients belongs among the most intricate problems of burns treatment. While survival depends predominantly on the extent and depth of burns it is also substantially influenced by age, previous illnesses, and concomitant injuries. Burns provoke profound pathophysiological changes in the organism, the management of which places enormous demands particularly on the cardiovascular and respiratory systems. The most frequent concomitant diseases likely to increase the risk of major damage in elderly patients are: arteriosclerosis, diabetes mellitus, advanced coronary sclerosis, hypertension, obesity, chronic pulmonary emphysema, bronchitis, chronic hepatic and renal insufficiency. Advancing age is associated with increasing multimorbidity involving mainly chronic and degenerative illnesses. An ageing man is no longer able to adapt to changing conditions; hence the need for adapting environmental changes to his or her capabilities. Nearly half the old population of Czechoslovakia die of cardiac and circulatory diseases.

The causes of increased risk may vary and are sometimes difficult to define, though a history of cardiac, pulmonary, renal and hepatic diseases is likely to feature prominently. Primary strain on the organs involved comes in the phase of shock, in the early post-accident period, and at the time of incipient hypovolaemia and resuscitation. In elderly patients only minor differences in fluid replacement dosage are permitted, hence the need for well-planned and conducted resuscitation. The formulas in use are much the same as in other age groups. The amount of fluids supplied ought to be large enough to maintain

urine output at 30 to 50 ml per hour. In older patients the likelihood of renal damage — oliguria or even anuria — is five times that in patients under 40.

Also, in older patients, impaired movement is likely to affect the extent and depth of burns. The injury may also be aggravated by diminished skin thickness and loss of elasticity. Similarly increased susceptibility can be observed in paraplegics. Then there is retarded temperature perception which slows down reaction time, thus prolonging exposure (neuropathies in diabetics).

Elderly and old patients often suffer from indigestion which, too, contributes to defence mechanism impairment. An elderly patient no longer has the necessary metabolic reserve to help him adequately cope with the injury and with the stresses of the subsequent illness. As shown by Wilmore maximum metabolic response is provoked in burns involving 40% of the body surface. Elderly patients will require ambient temperature of about 30 to 33 °C, their wounds take longer to heal and to remove necrotic tissues spontaneously, thus prolonging the periods of hospitalization and immobilization and creating the conditions for the development of all manner of complications.

The most serious of these are cardiopulmonary diseases. According to Pruitt, 80% of patients in the higher age groups die of pulmonary or cardiac involvement, the ratio being 1:2. Most of those patients are likely to have suffered from different forms of the disease prior to the injury, though quite possibly mishandled resuscitation may have contributed to a lethal outcome as well. The severity of such aftereffects may be alleviated by rational fluid replacement, early mobilization of patients, and by strict respiratory tract toilet.

Infection represents a great potential risk for all patients of all age groups, mainly, however, for the elderly noted for their considerably reduced resistance to infection. Malnutrition, too, carries the risk of impaired immunocompetence. For that reason, it is important to check the wound for infection. Special attention should be devoted to intravenous catheters making sure that the catheter is introduced under aseptic conditions, cleansed daily completely with the point of entry through the skin, and exchanged every third day as the incidence of infection and thrombosis starts rising steeply after three days. Specific antibiotic treatment is essential.

In the treatment of burn wounds in patients of all age groups it is imperative to cover the defect as soon as possible, to cut to a minimum the interval of injury by providing a definitive cover, particularly where elderly and old patients are involved. The sooner the patient is covered with autografts, the greater his hope of survival and the lesser the danger of post-operative complications. Excision of the burn area and its covering with autografts are the most effective approaches. For this the patient should be well balanced in terms of haemodynamics. Where for any reasons surgery is ruled out necrotic tissue removal should be sped up as all methods actively conducive to the removal of necrosis and to transplantation, provided they are employed appropriately and in good time, will reduce the risk of invasive infection, and are far more effective than just waiting for the spontaneous separation of necrotic tissue, a process which may take 6 to 8 weeks. While waiting for this to happen, the patient remains bed-ridden, unable to take care of himself, which together

with chronic stress cannot but increase the risk of complications and death. All our effort therefore must be geared to the earliest possible covering of the wound and to rehabilitation as a means of avoiding any of the above complications.

The conclusions to be drawn from this are that therapy in the elderly ought to be carefully planned, and that resuscitation involving fluid replacement should be conducted with a view to cutting down to a minimum the most likely complications, particularly cardiopulmonary and renal side effects. Undesirable complications should be prevented by early mobilization and care for the respiratory tract. The burned patient's nutrition should be borne in mind and, last not least, the patients' co-operation with the medical and nursing staffs ought to be solicited. Loss of interest in co-operation and in life together with depression may often prove to be the cause of lethal end. In other words, for the successful management of an injury as severe as a major burn it is necessary for the whole team of medical and paramedical workers to maintain close co-operation throughout.

A total of 4,027 patients were admitted in our burns department from the day it was opened in 1971 to the end of 1979. 138 of them were over the age of 60. A total of 239 patients died, an average death rate of 5.9%. The over-sixty group consisted of 51 women and 87 men. 55 of them died, thereof 19 women and 36 men, a death rate of 39%. The majority of the patients admitted were in the 60—70 group, a total of 78 patients, 31 of whom died. In the 70—80 age category 19 of the patients admitted for treatment died. In the group of over 80 years of age there were 9 patients admitted for treatment; 5 of those died, a death rate of 62.5%.

The predominant extent of burns in the surviving group of patients was up to 20% of the body surface with mostly 2nd to 3rd degree burns of the more superficial type. Those who failed to survive had suffered deep burns involving more than 30% of the body surface. Surgical operations were the predominant type of therapy in both groups with a total of 77 patients operated on. The maximum period of hospitalization was up to 30 days. Most of the deaths occurred within 10 days of the accident.

The primary immediate causes of death as established by postmortem examinations were heart failure, pulmonary damage, and shock with a smaller share going to sepsis, and even less so to alimentary tract haemorrhage and internal environment breakdown.

The above facts underscore the seriousness of thermal injury in the old-age group. Far from all the problems involved in the treatment of elderly patients can be dealt with in a single report. Nevertheless, the facts as such deserve a more detailed analysis to be made and conclusions to be drawn for an improvement in the prognosis of elderly people thus afflicted. Stress should, in our opinion, be laid on prevention. As an example we can refer to the cases of 5 people with impaired mobility who died in 1976 in the process of burning out grassfields. After the introduction of stiffer penalties for this offence there was not a single case of this type of injury at our department. Apparently, all available forms of prevention should be used.

The point is to improve the techniques of shock management, to provide appropriate first aid in good time, to take the victims to hospital, at the respective hospital departments and wards to improve shock management by supplying adequate amounts of quantitatively and qualitatively balanced fluids for intravenous replacement with full regard to possible consequences in the subsequent period of burn disease treatment, all available methods to be employed in order to speed up necrotic tissue removal and defect covering, increased attention to be paid to the pre-operative preparatory period as most of those over 600 suffer from different degrees of cardiovascular or respiratory system involvement.

J. H.

SUMMARY

The author presents a brief analysis of burns sustained by patients over 60 years of age in order to point to the problems and pitfalls of therapy, and to propose measures designed with a view to improving the prognosis of thermal injury in this particular age category.

RESUME

Expériences avec le traitement des brûlures chez les clients au-dessus de 60 ans

Bláha J.

L'auteur publie une courte analyse des brûlures chez les patients qui ont plus de 60 ans. Il fait remarquer les moments problématiques du traitement et propose les méthodes qui peuvent améliorer le pronostic des blessures thermiques dans cette catégorie d'âge.

ZUSAMMENFASSUNG

Erfahrungen mit der Behandlung der Verbrennungen bei Patienten in der Altersgruppe über 60 Jahre

Bláha J.

Der Autor legt vor eine kurze Analyse der Verbrennungen bei Kranken in der Altersgruppe über 60 Jahre. Er weist auf die Schwierigkeiten der Therapie hin und empfiehlt Massnahmen zur Besserung der Prognose der thermischen Verletzung in dieser Altersgruppe.

RESUMEN

Experiencias con el tratamiento de quemaduras en pacientes de más de 60 años de edad

Bláha J.

El autor presenta un breve análisis de casos de quemaduras en los enfermos mayores de 60 años. Señala los escollos del tratamiento y propone medidas a tomar para mejorar la prognosis de las lesiones térmicas en las personas de dicha edad.

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DEATHS IN FLAME BURNS

J. Koller, M. Sabo

As to the seriousness of injury flame burns come immediately next to high-voltage electrical burns. Although considerable progress has been made in the treatment of burns over the past twenty years or so, the rate of deaths caused by flame burns remains relatively high, especially in the presence of concomitant inhalation injury. A number of authors have already produced through-going analyses of the causes of death in burn victims (1, 2, 4, 5, 6). Our aim was to identify the causes of death in flame-burned patients treated at our department.

To do this, we chose to process data on 104 patients who died in the years 1974—1979 as a result of flame burns, and were examined post mortem at the Institute of Forensic Medicine, P.J. Šafárik University Medical Faculty, Košice. The deaths were divided into three categories: *i m m e d i a t e* deaths, i.e. deaths on the spot or while being taken to hospital; *e a r l y* deaths, i.e. those which occurred within four days of hospitalization; *l a t e* deaths, i.e. those which occurred at any time from the 5th post-injury day on, (see Tab. 1).

Findings of Pathological Anatomy

I m m e d i a t e deaths; a total of 22 postmortem examinations were evaluated in this particular group. The *i m m e d i a t e causes of death* (Tab. 2) were identified as follows: shock — 7 cases, shock in combination with carbon monoxide poisoning — 7 cases, carbon monoxide poisoning alone — 6 cases. There was one case of ruptured aorta (vehicle in flames after car accident), and one case of suffocation with vomitus in a 2-year old child with burns covering 17% of the body surface and 32% COHb blood level.

COHb blood levels were examined in all cases of this group; elevated levels (over 5%) were found present in 19 cases. The highest COHb blood level was identified in a 25-year old man who, in alcohol intoxication, fell asleep in a van with a burning cigarette and was burned to death after the van had caught fire. At the time of death he had 81% COHb in the blood.

Other findings (Tab. 3) included 12 cases of soot deposits in the respiratory tract, 2 cases of thermal changes in the respiratory tract mucosa, 13 cases of acute focal emphysema, 6 cases of grey-black deposits in the lumen of the lower respiratory tract, 3 cases of fat embolism (thereof two in polytraumatized subjects), and 1 case of focal pulmonary haemorrhage.

Table 1. Deaths 1974–1979. Burns Unit EINH Košice-Šaca

Immediate	Early	Late	Total
22	17	65	104

Early deaths; this particular group consisted of 17 subjects. The *immediate causes of death* (Tab. 4) were identified as follows: 12 cases of irreversible burn shock, 4 cases of major pulmonary changes, and 1 case of shock with carbon monoxide poisoning. *Other findings*: 8 cases of suppurative

Table 2. Immediate death (immediate causes of death)

Shock	7
Shock and CO poisoning	7
CO poisoning	6
Ruptured aorta	1
Inhalation of vomitus	1

bronchopneumonia (Tab. 5), 3 cases of hypostatic pneumonia, 4 cases of pulmonary vascular bed thrombosis, and 1 case of fat embolism of the lungs. Parenchymatous dystrophy of the lungs was found in 12 cases, of the kidneys in 11 cases, of the myocardium in 11 cases, and of the suprarenals in 9 cases.

Table 3. Immediate death (other findings)

Acute focal pulmonary emphysema	13
Soot in upper respiratory tract	12
Soot in lower respiratory tract	6
Fat embolism of the lungs	3
Respiratory tract burns	2
Inhalation of gastric contents	1

In the upper respiratory tract there were four cases of immediate thermal injury and three cases of soot deposits in the lumen. Gastric mucosa ulceration was found in 5 cases, duodenal mucosa ulceration in 1 case.

Late deaths; these were the most numerous group — 65 cases in all, all of them attributable mainly to infection found to have been responsible for a breakdown of the internal environment and for the terminal failure of one or more organs.

Table 4. Early death (immediate causes of death)

Burns shock	12
Shock lungs	4
Shock and carbon monoxide poisoning	1

The most frequent *immediate causes of death*: bronchopneumonia with progressive pulmonary insufficiency (22 cases), sepsis (15 cases); next came internal environment breakdown (11 cases), cardiac failure (6 cases), renal

Table 5. Early death (other findings)

Parenchymatous dystrophy of the liver	12
Parenchymatous dystrophy of the myocardium	11
Parenchymatous dystrophy of the kidneys	11
Parenchymatous dystrophy of the suprarenals	9
Suppurative bronchopneumonia	8
Hypostatic pneumonia	3
Catarrhal pneumonia	1
Thrombi in pulmonary vascular bed	4
Acute gastric mucosa ulceration	5
Acute duodenal mucosa ulceration	1

failure (4 cases), liver function breakdown (2 cases), fatal bleeding from gastrointestinal tract ulceration (4 cases), infarction of the myocardium (1 case). For survey see Tab. 6.

Table 6. Late death (immediate causes of death)

Bronchopneumonia	22
Sepsis	15
Internal environment breakdown	11
Cardiac failure	6
Renal failure	4
Hepatic failure	2
Fatal loss of blood from GIT ulcerations	4
Infarction of the myocardium	1

In view of the longer survival time in this group of patients, the *other findings* were far more varied than in the previous two groups (Tab. 7). Findings in the respiratory tract were made up of 26 cases of suppurative bronchopneumonia, 23 cases of hypostatic pneumonia, and 3 cases of catarrhal

pneumonia. Thrombi and emboli in the pulmonary vascular bed were present in 11 cases, emphysema in 9 cases, pleuritis in 4 cases, pulmonary abscesses in 2 cases, and fat embolism in 2 cases. The most frequently found changes in

Table 7. Late death (other findings)

Suppurative bronchopneumonia	26
Hypostatic pneumonia	23
Catarrhal pneumonia	3
Thrombi and emboli in pulm. vascular bed	11
Emphysema	9
Parenchymatous dystrophy of the liver	59
Parenchymatous dystrophy of the kidneys	50
Parenchymatous dystrophy of the myocardium	41
Suprarenal exhaustion	47
Shock changes in the kidneys	19
Gastric (duodenal) mucosa ulceration	25 (11)

the other vital organs came under the heading of severe parenchymatous dystrophy involving the liver in 59 cases, the kidneys in 50 cases, the myocardium in 41 cases. Suprarenal exhaustion was found in 47 cases, and evidence of shock changes in the kidneys in 19 cases. Acute ulceration in the gastric mucosa was present in 25 cases, in the duodenal mucosa in 11 cases. There was one case of acute gastric ulcer perforation. Septic tumours of the spleen were found in 8 victims. Hyperkalaemic necrosis of the myocardium (2 cases), nephrosis of the lower nephron (3 cases), suppurative foci in the kidneys (4 cases), in the brain (1 case) and in the liver (2 cases) were among the rarer findings. There was 1 case of oesophageal ulceration, 1 case of adrenal cortex infarction, and 1 case of red malacia of the left temporal lobe of the brain.

DISCUSSION

As for the group of immediate deaths our findings were similar to those of other authors (1, 4, 5). Shock and carbon monoxide poisoning were the predominant causes of death. Death invariably occurred so early that there was not enough time for any major pathological organ changes to develop. Acute focal emphysema proved to be the characteristic finding in the lungs, a phenomenon obviously connected with suffocation resulting from reflex spasms of the glottis in the inhalation of hot and irritating gases. In other words, any hope for survival in this group of victims would depend on their early removal from the site of the accident and on-the-spot resuscitation with the therapy geared to carbon monoxide poisoning management, as this was present in most of the cases. In addition, it would seem necessary to improve the material and personnel standard of ambulance cars to cope with such emergencies.

Transport appeared to be an essential factor in the assessment of the group of early deaths. 13 out of 19 patients taken to our burns unit from some of the more remote places of work on the day of the accident died, i.e. 68.4%. The death rate in patients transported to our unit after prior treatment in other

health care institutions during the first four post-accident days proved to be a mere 25%. This fact is brought up also by postmortem findings in the group of early deaths where shock was the main cause of death. Concomitant inhalation injuries were the cause of death in 23.5% (4 cases), and contributed to a fatal outcome in 5 more cases. We noted a relatively frequent occurrence of pulmonary vascular bed thrombosis (23.5%), a fact which supports the hypothesis of disseminated intravascular coagulation (8) in cases of burn shock and, in particular, in inhalation injuries. The liver appeared to be the most frequently affected parenchymatous organ. Pardy (7) says that the liver protects the lungs so that its cells as part of the reticulo-endothelial system takes up phagocytatable material from circulation, thus reducing its supply to the lungs. If the liver function is impaired the lungs, already damaged by shock, receive an increased supply of phagocytatable particles which they are unable to process adequately, thus precipitating a whole range of other changes (disseminated intravascular coagulation, infection, progressive pulmonary insufficiency).

As already mentioned, infection featured prominently in the late death group. Pneumonia was responsible for 31.6%, sepsis for 22.8%, and metabolic breakdown for 17.5% deaths. Major pathological changes were also found in parenchymatous organs, in the suprarenals, and on the alimentary tract mucosa. All of those changes, however, are only secondary after-effects in cases of failure to remove in time burn necroses and to provide a temporary or else definitive cover for the burn wound.

J. H.

SUMMARY

Although mortality and morbidity among burned patients has been markedly reduced in the past few years there are many open problems to be coped with yet. Some of them are pointed out in the authors' analysis of the causes of death of 104 patients. Shock and CO poisoning predominated in the category of immediate deaths (22 patients). While burn shock has ceased to be the main cause of worry in the early post-burn period it continues to be a major risk in those patients where substitution therapy was started late or where transport to a remote burn center was inadequately indicated at the time of the accident. This is corroborated by the authors' findings in the early death category. Infection spreading from the burn wound remains problem No. 1 in the category of late deaths. Early removal of necrotic tissues and provision of temporary and or preferably definitive cover are now the most effective prevention of infection.

RESUME

Mortalité en conséquence des brûlures par le feu

Koller J., Sabo M.

Dans les dernières années on a réussi à diminuer la mortalité et la morbidité des clients brûlés, mais il y a encore beaucoup problèmes qu'il faut résoudre. Notre analyse

des causes qui menaient jusqu'à la mort chez 104 clients fait remarquer quelques - uns de ces problèmes. Dans les cas de la mort immédiate [22 clients] dominait le choc et l'intoxication par CO. Tandis que le choc après les brûlures ne présente plus un grand danger dans les premières phases après la brûlure, il reste toujours très dangereux pour les blessés chez lesquels on n'a pas commencé à temps avec la thérapeutique substituée ou bien pour ceux, qui ont été transportés le jour de l'accident dans les endroits éloignés. Ce sont les résultats de notre analyse des morts immédiates qui le prouvent. Le problème principal dans les cas de la mort postérieure reste toujours l'infection qui se répand du point brûlé. La prévention la plus efficace de cette infection est l'écartement opportun des necroses et la protection de la blessure par la couverture provisoire et surtout définitive.

ZUSAMMENFASSUNG

Todesfälle bei Flammenverbrennungen

Koller J., Sabo M.

Auch wenn in den letzten Jahren die Mortalität und Morbidität der verbrannten Patienten weitgehend herabgesetzt werden konnte, bleiben noch viele Probleme auf diesem Gebiet ungelöst. Auf einige von ihnen hat unsere Analyse der Todesursache von 104 Patienten hingewiesen. In der Gruppe der unmittelbaren Todesfälle (22 Patienten) dominierte der Schock und die CO-Vergiftung. Obwohl der Verbrennungsschock nicht mehr ein Gespenst des Frühstadiums der Verbernnung ist, stellt er eine ernste Gefahr bei Patienten dar, bei denen mit der Substitutionstherapie spät begonnen und wo der Transport zu einer mehr entfernten Arbeitsstätte am Unfalltag ungeeignet indiziert wurde. Dies bestätigen auch unsere Befunde in der Gruppe der frühzeitigen Todesfälle. Das Problem Nummer 1 bei späten Todesfällen bleibt die Infektion, die ihren Ursprung in der Verbrennungswunde hat. Ihre wirksamste Vorbeugung ist derzeit die frühzeitige Beseitigung der Nekrosen und Schliessung der Wunde mittels einer vorübergehenden und vor allem definitiven Deckung.

RESUMEN

Fallecimientos a causa de quemaduras por llama

Koller J., Sabo M.

Aunque en los últimos años se ha logrado reducir considerablemente la mortalidad y la morbilidad de los pacientes quemados quedan muchos problemas pendientes sobre este campo. Algunos de ellos fueron señalados por nuestro análisis de causas de muerte de los 104 pacientes. En el grupo de fallecimientos inmediatos (22 pacientes) prevaleció como causa la conmoción y el envenenamiento por CO. Bien que la conmoción producto de quemaduras haya dejado de ser el espantajo en los estadios tempranos de las quemaduras, sigue constituyendo un grave peligro en los pacientes que no se han sometido a tiempo a la terapia de substitución o los que indebidamente se han transportado a lugares de trabajo lejanos el día del accidente. Lo confirman nuestras diagnosis en el grupo de fallecimientos tempranos. El problema principa en caso de fallecimientos tardíos lo sigue constituyendo la infección originada en la herida por quemadura. Su prevención más eficaz actualmente es la eliminación de las necrosis y el cerrar la herida con una cobertura temporal y, sobre todo, definitiva.

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BRAIN EDEMA AND CARBOHYDRATE METABOLISM IN THE EARLY STAGES OF THERMAL INJURY AND BURN SHOCK

B. Domres, W. Heller, W. v. Kothen

INTRODUCTION

One of the most salient characteristics of the pathogenesis of acute burn shock is the generalized increase in blood vessel permeability. This causes marked edema not only in the area of the dermis directly damaged by thermal energy, but in the organism as a whole.

This generalized disturbance of vessel permeability cannot be explained alone by the immediate effect of thermal energy on the vessels. The permeability disturbance is due more to chemical substances such as histamine, prostaglandine, polypeptides, catecholamines and toxic burn products which are liberated partially from the organism and cause neurogenic and chemical irritation.

A secondary effect is that all organ functions are handicapped throughout the course of the burn disorder.

Clinical and experimental interest has up until now centered on the functional and pathological changes in the kidneys, lungs, liver, gastrointestinal system, endocrine glands as well as in the reticuloendothelial system. Disturbances of the central nervous system have received hardly any attention, although seriously injured patients exhibit regularly neurological and psychiatric symptoms.

Feller and Archambeault [1] found, that of 1974 deaths, 58 were caused by brain damage. In 24 of 30 burn cases who expired in the Chirurgische Universitätsklinik in Tübingen between 1966—1975, autopsy showed cerebral edema (Fig. 1). Of 12 patients who died in the first five days following a burn injury, 11 showed brain edema.

SUBJECT OF INQUIRY

The following questions are of importance in this connection: Does cerebral edema occur regularly following a standardized thermal trauma?

What happens to concentrate ions such as sodium and potassium in the brain? Do disturbances in glucose or energy metabolism or lessening of the supply of oxygen play any decisive part in the pathogenesis of cerebral edema?

METHODS AND MATERIALS

Using a standardized burn model, 467 male SPF-Sprague Dawley rats received third degree burns over 20 % of their body surface with an electrically heated copper stamp at a temperature of 250 °C for 30 seconds.

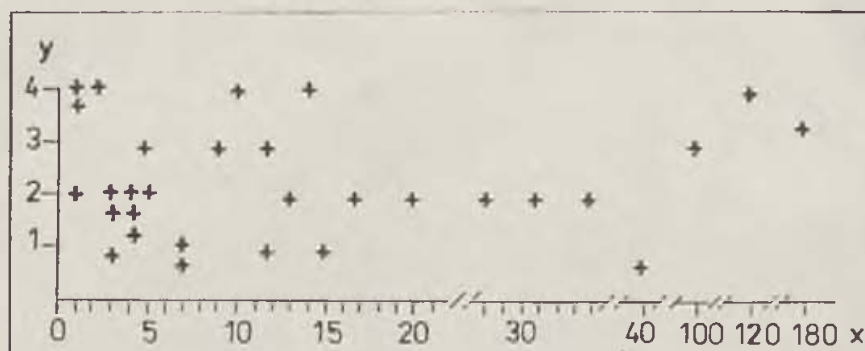


Fig. 1. Extent of cerebral edema as shown by autopsy of 30 mortalities. y: 1 — none, 2 — light, 3 — medium, 4 — severe, x: in days

At 12 and 48 hours, as well as at 14 days, the brains were removed by means of the freeze stop technique and the concentration of the following parameters was measured: sodium and potassium, glucose, glucose-6-phosphate, fructose-1, 6-disphosphate, dihydroxyacetone phosphate, lactate, pyruvate and adenosinetriphosphate. In addition, cerebral fluid content at 72 was

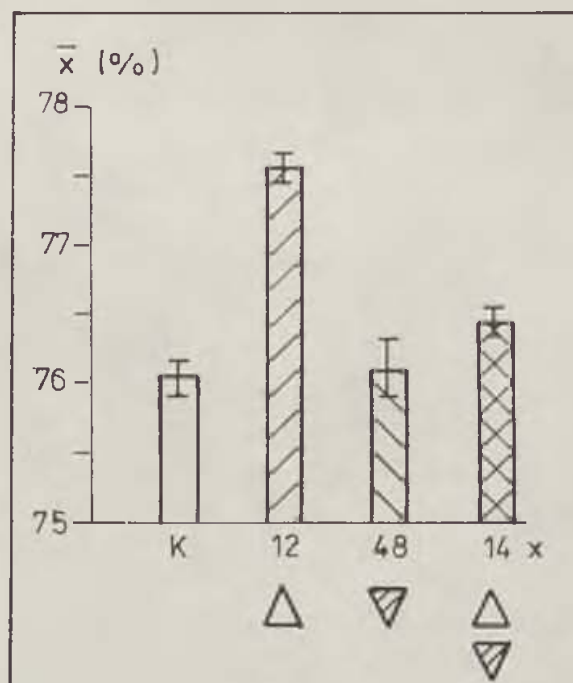


Fig. 2: Fluid content of brain following experimental trauma. x: control — 12 hours, 48 hours, 14 days after burn

determined by weighing subsequent to removal and lyophilisation with liquid nitrogen.

For statistical analysis Fischer's F-test, the t-test according to Gosset and the Aspin test and U-test according to Wilcoxon, Mann and Whitney were used.

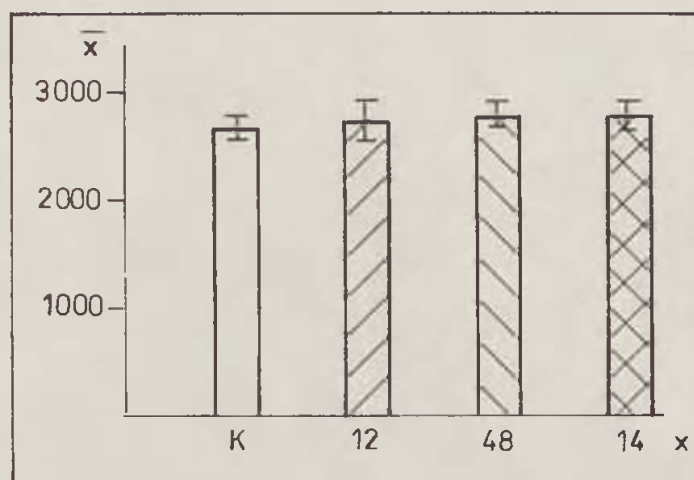


Fig. 3. Sodium concentration in the brain following experimental burn trauma. \bar{x} — mval/kg dry weight, x — control — 12 hours, 48 hours, 14 days after burn

RESULTS

1. Mortality due to trauma lay at 22.7 % in the first 48 hours and climbed to 61.75 % at the end of the experiment in 14 days.

2. Fluid content of the brain (Fig. 2) climbed 1.5 % at 12 hours and another 0.5 % at 14 days over control values. The 1.5 % increase in fluid content caused

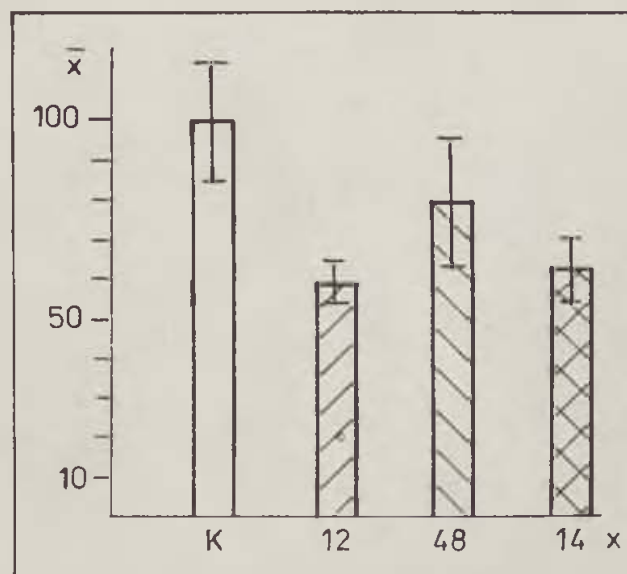


Fig. 4. Potassium concentration in the brain following experimental burn trauma. \bar{x} — mval/kg dry weight, x — control — 12 hours, 48 hours, 14 days after burn

(3) an increase of 6.3 % in volume. While the burn patients in the clinic showed an increase in weight because of a tendency to edematize with a calculated fluid administration in the first days, the animals in the experiment, who re-

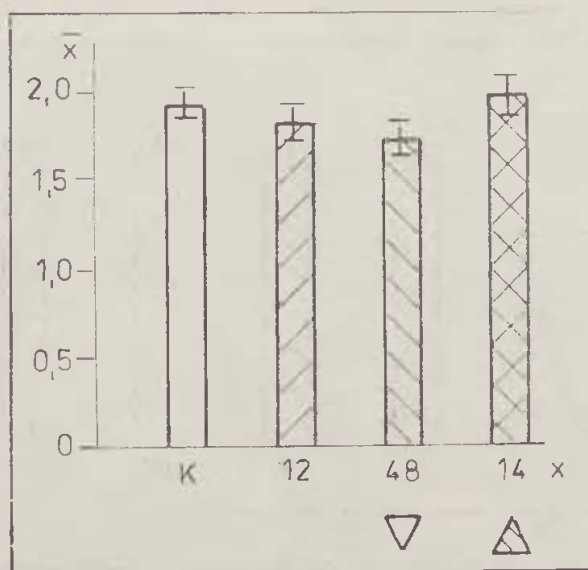


Fig. 5. Glucose concentration in the brain following experimental burn trauma. \bar{x} — $\mu\text{mol/g}$ wet weight, x — control 12 hours, 48 hours, 14 days after burn

ceived no fluid substitution, showed a marked decrease in weight. A generous fluid administration would have probably increased cerebral fluid content over that found here.

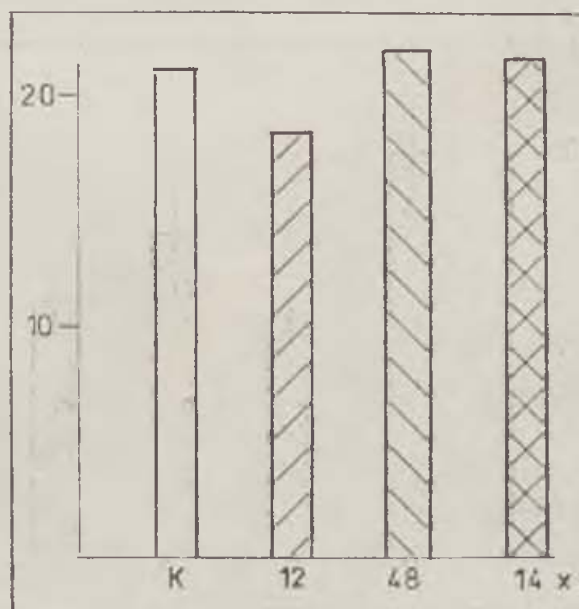


Fig. 6. Lactate/Pyruvate quotient in the brain following experimental trauma. x — control — 12 hours, 48 hours, 14 days after burn

3. The concentrations of sodium and potassium in cerebral tissue (Figs. 3, 4) behave in opposite ways. The Na^+ concentration increases slightly, while the K^+ concentration decreases significantly.

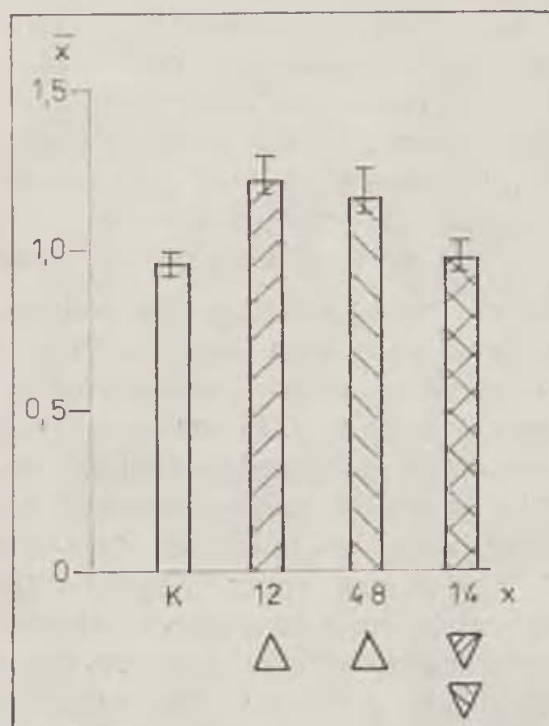


Fig. 7. Adenosintriphosphate concentration in the brain following experimental burn trauma. \bar{x} — $\mu\text{mol/g}$ wet weight, x — control — 12 hours, 48 hours, 12 days after burn

4. Glucose concentration (Fig. 5) is decreased at 12 hours and declines still more following 48 hours-Glucose-6-phosphate, on the other hand, is significantly increased. At 12 hours, the animals had a diabetic metabolism with hyperglycemia, followed by a hypoglycemic phase. Of the other intermediate products of glycolytic glucose metabolism, the concentration of fructose-6-phosphate and fructose-1/6-disphosphate are lowered. The concentration of dihydroxyacetone phosphate is increased.

5. The lactate/pyruvate quotient (Fig. 6) shows a significant decline, since the concentration of pyruvate in the brain increases and the lactate decreases.

6. The concentration of adenosinphosphate (Fig. 7), the universally usable form of free energy, is significantly increased.

DISCUSSION

The analyses of cerebral glycolytic glucose metabolism would seem to indicate that glucose, being the chief substrate for energy procurement in diabetic metabolism, can become a limiting factor in metabolic function. The concentrations of glycolytic intermediate products indicate an inhibition in enzyme activity of phosphofructokinase in the brain.

The decreased lactate-pyruvate quotient and the increased ATP concentration exclude as cause of the edema a diminished cerebral arterial circulation. This is in accord with measurements of organic circulation [4] with Xe^{133} in burn patients. In these patients there was decreased circulation in the dermis, musculature and liver, but cerebral circulation was increased. When a critical amount of cerebral edema is at hand, however, cerebral circulation will be secondarily hampered. In this case ATP-concentration decreases and the lactate/ pyruvate quotient increases. Cerebral edema is pathologically and anatomically a constant finding in severely burned patients who die in the earliest stages. Cerebral edema is probably more frequently the direct cause of death in the acute shock phase than has previously been assumed.

Children are especially endangered since the clinical symptoms of cerebral edema are more pronounced with them than in adults. Unbearable headache, loss of consciousness to coma and convulsions are the precursors of imminent danger. An electroencephalogram will in the majority of cases indicate the cerebral lesion. Very frequently prolonged rhythm or even convulsive potential can be found. The changes are generally reversible for those who survive, however, permanent damage such as lowering of the intelligence quotient, hysterical blindness and plegias can occur. Koepke [2] found in follow-up studies of 128 burn patients 31 cases of electrodiagnostic denervation or electromyographic delay in potential in 2 or more peripheral nerves. Avoidance of an over-balanced transfusion treatment is of the greatest importance in the prophylaxis of cerebral edema.

For therapy of cerebral edema as a result of burning, the administration of 40 % Sorbit in a dose of 1 mg/kg body weight 3X daily is recommended.

Dexamethason in an initial dosage of 100 mg followed by 8 mg 3X daily likewise produces good results. The dosis is reduced daily. One disadvantage of Dexamethason treatment is that it still further reduces the already lowered infection resistance of the burned patient, so that coverage with antibiotics is necessary. Other than this no routine antibiotic treatment is undertaken with burn patients. Furosemit, albumin and hyperosmolar transfusions can also be recommended for supplemental treatment of the cerebral edema.

S U M M A R Y

Cerebral edema is experimentally producible in severe burn traumas. It occurred clinically and experimentally predominantly in the initial stages. Children are especially endangered.

In experiments on animals the fluid content in the brain rose 1.5 % at 12 hours, corresponding to increase in volume of 6.3 %. While the sodium content increases slightly, the potassium content decreases significantly.

In spite of hyperglycemia the glucose concentration in the brain decreases rapidly. Concentrations of glucose intermediate products indicate an inhibition in enzyme activity of phosphofructokinase.

The increased concentration of ATP and the lowered lactate/pyruvate quo-

tient negates the assumption of lowered arterial circulation as cause of the cerebral edema.

Pathological anatomical finding of cerebral edema is constant in the early stages of burn mortalities and can be presumed to be much more frequently the cause of death than has previously been assumed.

Prophylactically, an over-balanced fluid supplementation is to be avoided in the early stages above all. In surviving patients the cerebral edema can lead to permanent damage with psychiatric and neurological symptoms.

RESUME

Fluxion du cerveau et le métabolisme des hydrates de carbon dans les premières phases du choc après les brûlures

Domres B., Heller W., Kothen W. v.

À l'aide de graves brûlures on peut provoquer une fluxion expérimentale du cerveau. Cette fluxion apparaît surtout dans les premières phases de la maladie des brûlures. Ce sont notamment les enfants qui sont menacés. Les expériences avec les animaux ont prouvé que la quantité des liquides monte dans le cerveau de 1,5 % dans 12 heures. Le volume s'accroît de 6,3 %. Tandis que la quantité du sodium monte, la quantité du potassium s'abaisse. Malgré l'hyperglycémie la concentration du glucose dans le cerveau est diminuée. La concentration des produits du glucose indique l'inhibition de l'activité enzymatique de la phosphofructokinase. La raison de la fluxion du cerveau ne se trouve pas dans la circulation artérielle réduite. Le diagnostic pathologique et anatomique de la fluxion de cerveau est un phénomène constant dans les phases précoces des brûlures mortelles. On peut supposer, que cette fluxion est une cause de la mort plus souvent que l'on pensait avant. Il faut surtout éviter la compensation déséquilibrée des liquides dans les premières phases après les brûlures. Chez les clients survivants les fluxions peuvent mener aux défauts persistants avec des symptômes psychiatriques et neurologiques.

ZUSAMMENFASSUNG

Hirnödeme und Kohlenhydratstoffwechsel in den Frühstadien des Verbrennungsschocks

Domres B., Heller W., Kothen W. v.

Mittels schwerer Verbrennung kann ein experimentelles Hirnödem ausgelöst werden. In der Klinik und im Versuch erscheint dieses Ödem überwiegend in den Initialstadien der Verbrennungskrankheit. Besonders gefährdet sind die Kinder. In Tierversuchen steigt der Flüssigkeitsgehalt im Gehirn binnen 12 Stunden um 1,5 %. Dem entspricht ein Volumenanstieg um 6,3 %. Während der Natriumgehalt ein wenig steigt, kommt es zu einem wesentlichen Absinken des Kaliumgehaltes. Trotz der Hyperglykämie vermindert sich schnell die Glukosekonzentration im Gehirn. Die Konzentration der Glukosezwischenprodukte deutet auf eine Hemmung der enzymatischen Aktivität der Phosphofructokinase. Die erhöhte ATP-Konzentration und das niedrigere Verhältnis zwischen dem Laktat und Pyruvat zeugen gegen die Voraussetzung, daß die Ursache des Hirnödems in der Abnahme der arteriellen Zirkulation liegt. Der pathologisch-anatomische Befund des Hirnödems ist eine konstante Erscheinung in den Frühstadien der tödlichen Verbrennungen. Es ist anzunehmen, daß es die Todesursache viel häufiger

bildet, als früher vorausgesetzt wurde. Prophylaktisch ist vor allem ein unausgeglichener Flüssigkeitsersatz in den Frühstadien nach der Verbrennung zu meiden. Bei überlebenden Patienten können die Hirnödeme zu einer dauernden Schädigung mit psychiatrischen und neurologischen Symptomen führen.

RESUMEN

Hinchazón del cerebro y el metabolismo de hidratos de carbono en estadios temprano de cenmoción por quemadura

Domres B., Heller W., Kothen W. v.

Por medio de graves quemaduras se puede provocar la hinchazón experimental del cerebro. En la práctica clínica así como en el experimento esta hinchazón aparece mayormente en estadios iniciales de la enfermedad por quemadura. A particulares peligros se ven expuestos los niños. En experimentos con los animales el contenido de líquidos en el cerebro aumenta dentro de 12 horas hasta en un 1.5 % a lo que corresponde un aumento de volumen de un 6.3 %. Mientras que el contenido de natrio se eleva algo, el de potasio baja considerablemente. A pesar de hiperglicemia rápidamente va disminuyendo la concentración de glucosis en el cerebro. La concentración de la glucosis señala la inhibición de la actividad enzimática de la fosfoructokinas. La concentración elevada de ATP y una proporción menos grande entre el lactato y piruvato va en contra de la suposición que es causa de la hinchazón del cerebro la disminución de la circulación arterial. El diagnóstico patológico-anatómico de la hinchazón cerebral es fenómeno constante en estadios iniciales de las quemaduras letales. Es de suponer que es causa de la muerte con mucha más frecuencia de lo que antes se creía. A título de prevención debemos lógicamente evitar una sustitución desequilibrada de líquidos durante los iniciales estadios después de producirse la quemadura. En los pacientes que sobreviven, las hinchazones cerebrales pueden provocar deterioros insanables con síntomas siquiátricos y neurológicos.

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MONITORING OF FLUID REPLACEMENT IN EXTENSIVELY BURNED PATIENTS WITH OLIGURIC RENAL INSUFFICIENCY

M. Dobke, B. Wyrzykowski, W. Kondrat, J. Jaromczyk-Slisz, A. Dominiczak

During initial burn shock treatment early prophylaxis and diagnosis of renal failure is important. During first hours post severe thermal injury it is not easy to differentiate between "naturally" appearing oliguria (despite proper early fluid resuscitation) and oliguric acute renal failure (OARF). Oliguric and anuric states in this burn shock phase are routinely treated with additional fluid with the expectation that the blood pressure (RR) and urinary output (UO) will be re-established. This common procedure and massive intravascular flux of previously sequestered fluid, which is not excreted by the kidneys, after the first phase of burn shock, increases the danger of fluid overload.

In burned patients (PTS) with OARF the management and monitoring of fluid and electrolyte balance becomes considerably more complex than in "non-complicated" burn shock cases. UO, a sensitive guide of fluid resuscitation, can not be used to monitor fluid therapy. Cardiopulmonary and renal functions should be estimated by the measurements of a number of variables but many of them are of limited value. Plasma osmolality in "non-complicated" burn shock may be a sensitive index of dehydration, while in burned PTS with OARF usually hyperosmolality was observed (sometime hyperosmolality coincided with hypervolemia). Central venous pressure (CVP) is useful as a guide to avoid fluid overloading better than for accurate monitoring of hypovolemia. But there may be a circulatory volume deficit when the CVP has been restored to (or increased above) normal values.

MATERIAL AND METHODS

Six severely burned PTS with OARF were studied (Tab. I). Hemodynamic measurements were performed after admitting the patient to the Dept. of Plastic Surgery Gdansk (cases 001,002,005 and 006 were admitted from other hospitals). Observation of physical status and routine hemodynamic measurements (as RR, heart rate RH, hematocrit HCT, CVP, plasma and urine osmolality, serum and urine electrolytes, and body weight changes) were performed

Table 1. Presentation of 6 burned patients with oliguric acute renal failure

Case N°	Age Sex	Type and percentage of injury	Initial fluid resuscitation (formula)	Clinical course
001	18/M	Flame III° 90 %	Brooke	Severe shock and anuric state on admission. Plasma osmolality 320–340 mOsm/kg in the course of therapy. Hemodialysis on the 2nd post burn day. Hypervolemia on the 3rd day: PV 3389 ml/m ² , CVP 25 cm H ₂ O, HCT 35%, plasma Na 134 mEq/l. Fluid restriction to 2 l of 5% glucose. On the 4th day: UO 15–30 ml/hr, CVP 10–15 cm H ₂ O, PV 2178, EV 794 ml/m ² , plasma Na 140 mEq/l. Subsequently fluid administration was calculated according to the formula given in the text, maintaining CVP from 8 to 13 cm H ₂ O and plasma Na at an acceptable level. Body weight changes reflected calculated and measured by using isotopes blood volume changes. The patient has died on the 9th day with the signs of progressive respiratory insufficiency.
002	24/M	Flame II/III° 60 %	probably Evans	Admitted on the 2nd post burn day without hemodynamic monitoring chart. Anuric state since the injury. Since the 3rd post burn day a tendency to hypervolemia and increasing biochemical signs of OARF. Hemodialysis with negative fluid balance 1.5 l (respective body weight change, no response in CVP value). On the 5th day PV 3100, EV 820 ml/m ² . Hemodialysis. On the 6th day PV 2716, EV 759 ml/m ² . On the 7th day the patient has died with the signs of renal and respiratory failure. In the course of therapy hyponatremia and CVP 4–7 cm H ₂ O. Plasma osmolality not less than 335 mOsm/kg.
003	26/M	Flame II/III° 70 %	Evans	BV, PV, CVP, body weight changes are presented on the figure 1. Plasma Na 134–148 mEq/l in the course of therapy. HCT 20 to 30 % (after initial treatment). Shock lung, respiratory failure.
004	28/M	Flame II/III° 56 %	Evans	Burn injury during epileptic state. Delay in shock resuscitation. Anuria since the admission. CVP 1–2.5 cm H ₂ O during therapy. On the 3rd day signs of hypervolemia. Hiponatremia, despite sodium intake. Sepsis. Death from renal, respiratory and circulatory failure.
005	31/F	Flame II/III° 60 %	not known	Admitted on the 9th day after injury with signs of deep hypovolemia (PV 681, EV 366 ml/m ² , HCT 42%, RR 70/30 to 100/70 mmHg). Anuria, respiratory failure. No response to fluid infusion.
006	28/M	Electric injury III° 15 %	Evans	BV, PV, CVP, body weight changes are shown on the figure 2. A tendency to hyponatremia. Hyperosmolar state in the course of therapy: plasma osmolality from 315 to 330 mOsm/kg, urine (15–20 day) 385 mOsm/kg.

serially. Blood volume [BV], plasma volume [PV], red cell mass volume [EV] estimations using radioiodinated human serum albumin (^{131}I) have been made using Volemetron type apparatus (IMOK) and medical system of scintillating counters (ZM 1) was used for simultaneous determinations of cardiac index

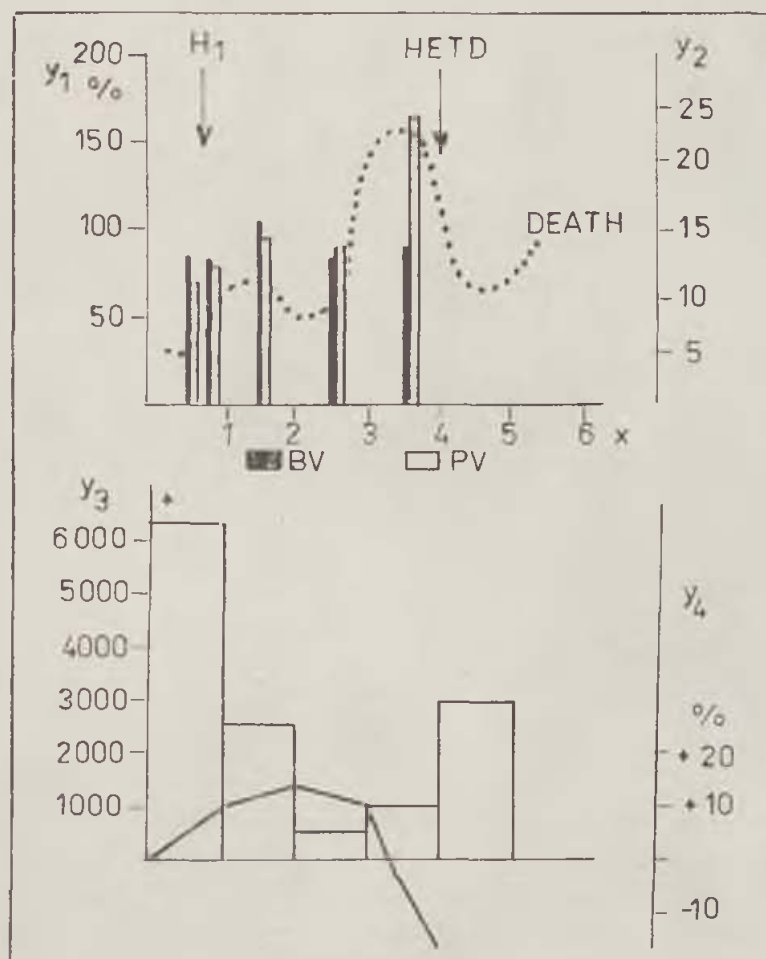


Fig. 1. Non-survived burned patient. Case No 003. x — DAYS, y_1 — FRACTION OF PREDICTED NORMAL, y_2 — CENTRAL VENOUS PRESSURE (cm H₂O), y_3 — FLUID BALANCE (ml), y_4 — BODY WEIGHT CHANGES
H — HAEMODYALYSIS, HETD — GASTROINTESTINAL BLEEDING, A — AMPUTATIONS OF LOWER EXTREMITIES, — CVP, ——— — BODY WEIGHT CHANGES

[CI], stroke volume index [SVI] and total peripheral vascular resistance index [TPRI]. PV and EV determinations was performed as soon as it was possible after patient's admission. In burned PTS with OARF daily fluid input was restricted to less than $1\text{ l} + \text{calculated evaporative water loss volume [EWL} = (25 + \% \text{ burned body surface area}) \times \text{total body surface area in m}^2/\text{ml per hour}]$. Clinically accepted BV in burned PTS with renal failure was $\text{BV} + 80$ to 105% of predicted normal value [6]. Fluids were administered under CVP control [maintaining a CVP at an acceptable level 6 to 13 cm H₂O]. If BV/PV exceeded accepted values, despite restrictions, the fluid input was limited

respectively to determined value of PV excess per day. In cases 002, 003, 006 PV/EV detreminations were performed before and after hemodialysis. If fluid overloading was detected before hemodialysis, the procedure was performed with negative fluid balance. Body weight was controlled once a day and before, and after hemodialysis.

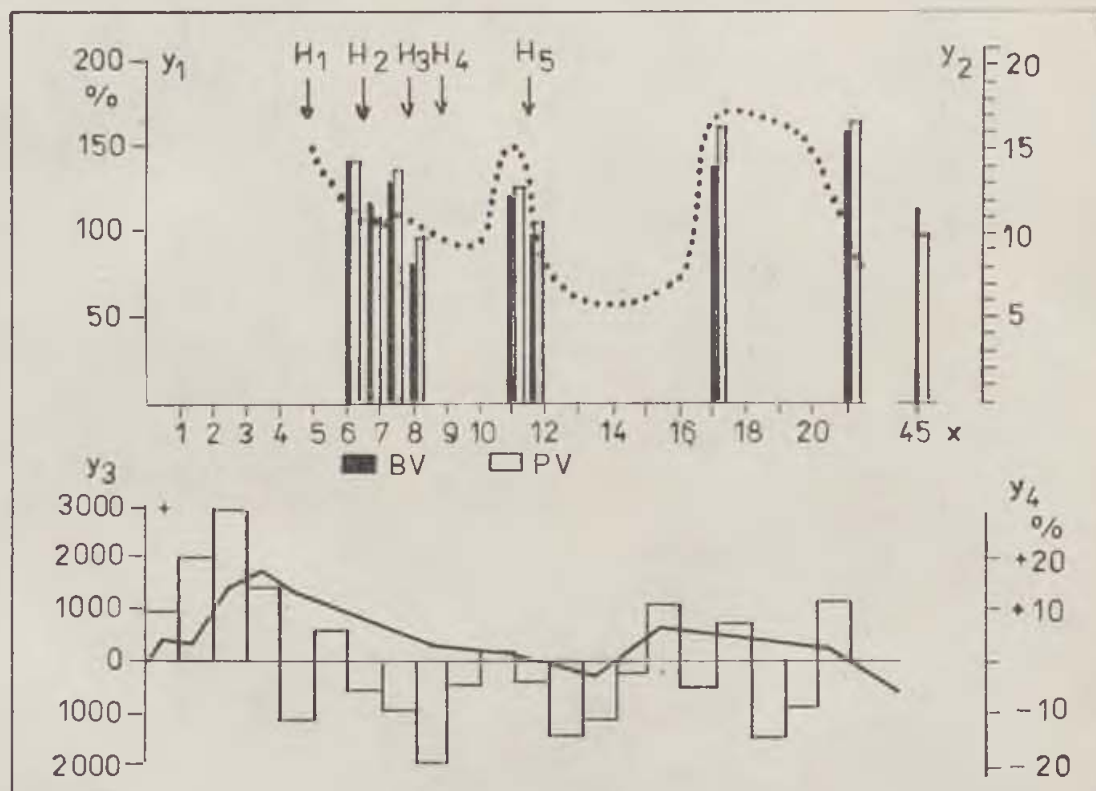


Fig. 2. Burned patient with acute renal failure. Case No 006. x — DAYS, y_1 — FRACTION OF PREDICTED NORMAL, y_2 — CENTRAL VENOUS PRESSURE [cm H₂O], y_3 — FLUID BALANCE [ml], y_4 — BODY WEIGHT CHANGES, H — HAEMODYALYSIS, — CVP, — — — — BODY WEIGHT CHANGES

RESULTS

Clinical and hemodynamic course of studied patients are presented in the table I, figure 1 and 2. A tendency to hypervolemia was observed, despite fluid restrictions. Calculated BV from actual and predicted values of HCT were lower from directly measured BV values by using isotopes. The PTS had elevated CI and SVI in the course of renal failure (case 003, 006) (Tab. II) although most of them have died with the signs of cardio-respiratory failure.

DISCUSSION

Every single hemodynamic measurement is not valuable in monitoring of severely burned PTS because of several limitations. In burn shock complicated by renal failure fluid administration and monitoring by using CVP and body weight changes determinations, serially controlled by direct and

objective BV/PV/EV measurements should aim to keep BV in the lower normal range, especially during restoration phase and no expansion of BV beyond upper normal or acceptable range during the post-hypovolemic phase of severe burn shock. Knowledge of the PTS weight prior to injury is important.

Table 2. Burned patients (Cases 003 and 006) with acute renal insufficiency. Cardiac index (CI), stroke volume index (SVI), total peripheral vascular resistance index (TPRI) values

Case No	CI ml/min/m ²	SVI ml/m ²	TPRI dyn. s/cm ⁵	PV ml/m ²	EV ml/m ²	HCT %	Clinical considerations
003	6007	50	886	1040	1199	23	13 hours after injury.
003	6800	56	820	1394	913	38	Prior to hemodialysis. After hemodialysis.
006	7575	69	1321	1367	549	32	Measurements after 3rd hemodialysis with negative fluid balance.
006	8698	92	1130	2509	1112	34	21st day post burn. The wound was healed (covered by autografts). Biochemical signs of renal recovering to normal function.
006	5195	61	1830	1619	1168	46	45 days post injury.

Fluid measurements of the body compartments (PV, interstitial fluid) is valuable because total body water by itself does not prove to be a suitable measure for body composition in hemodialysed PTS, because of the severity of water and electrolyte balance disorders.

SUMMARY

PV/EV determination by using isotopes is helpful in the evaluation of the effectiveness of fluid therapy. This non-invasive method can evaluate CVP and body weight changes interpretation in severely burned PTS with OARF.

RESUME

L'enregistrement à l'aide des moniteurs de la compensation des liquides chez les clients gravement brûlés avec l'insuffisance oligurique des reins

Dobke M., Wyrzykowski B., Kondrat W., Jaromczyk-Slisz J.,
Dominiczak A.

L'évaluation de la proportion entre le volume du plasma et le volume de la masse érythrocytaire à l'aide des radioisotopes est très utile pour l'appréciation de l'efficacité de la thérapeutique par la compensation des liquides. Cette méthode permet d'évaluer la pression veineuse centrale et le poids du corps chez les clients gravement brûlés avec l'insuffisance oligurique urgente des reins.

ZUSAMMENFASSUNG

Überwachung des Flüssigkeitersatzes bei beträchtlich verbrannten Patienten mit oligurischer Niereninsuffizienz

Dobke M., Wyrzykowski B., Kondrat W., Jaromczyk-Slisz J., Dominiczak A.

Die Bestimmung des Verhältnisses zwischen dem Plasmavolumen und dem Volumen der Erythrozytenmasse mit Hilfe von Radioisotopen ist bei der Beurteilung der Effektivität der Therapie durch Flüssigkeitersatz sehr nützlich. Durch diese nicht-invasive Methode kann man den zentralen venösen Druck und das Körpergewicht bei schwer verbrannten Patienten mit akuter oligurischer Niereninsuffizienz auswerten.

RESUMEN

Monitoramiento de la sustitución de líquidos en los pacientes con quemaduras considerables que padecen de insuficiencia renal oligúrica

Dobke M., Wyrzykowski B., Kondrat W., Jaromczyk-Slisz J., Dominiczak A.

La determinación de la proporción entre el volumen de plasma y el de la masa de eritrocitos con ayuda de radioisótopos es un método muy fructuoso al evaluar la eficacia de la terapia por sustitución de líquidos. Por este método no invasivo se puede evaluar la presión venal central así como el peso del cuerpo en los pacientes con aguda insuficiencia oligúrica de los riñones.

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SOME CLINICAL-MORPHOLOGICAL SPECIFICITIES IN PINNA BURNS

(Clinical and experimental observations)

K. Troshev, D. Markov, I. Kozarov

Burn injuries of the pinna are fairly frequent since the ears are likely to be affected in nearly 90 % of all facial burns.

Large-scale statistical studies seldom pay adequate attention to the pinnae, which is why it is rather hard to pinpoint their position in the localization of burns.

Severe ear burns and their complicated course are likely to cause considerable difficulties in treatment. The consequences of burns require repeated and complex reconstruction, and yet the outcome is seldom satisfactory in terms of aesthetic appearance. The ears invariably receive less attention as part of severely burned patients care. Most of the medical effort tends to be channeled to severe shock management exactly at the time that the pinna requires special care with regard to the aggravation of the destructive processes involved as well as to the development of complications.

Some authors refer to pinna chondritis without giving any precise data on its frequency (1, 4, 7, 8, 9). Nor is it always made clear if the clinical treatment of progressing chondritis is the same as in deep burns, and if it is consistent with the development of destructive processes or if there is, indeed, a secondary complication going on there. Dowling et al. were able to note suppurative chondritis in 23.8 % of the cases concerned.

Different scientific reports and monographs refer to the degree of pinna burns without much specification, so the degrees are very much the same as in the rest of burns (1, 4, 7, 9, 11). Nor are morphological reports sufficiently convincing in this respect (2, 3, 6).

All this poses certain questions concerning the classification of pinna burns and their complications. A solution to such problems would be of importance for the choice of correct therapeutical tactics. The course and the outcome of pinna burns treatment definitely have some specific features which prompted us the idea to try and specify some of the aspects.

Table 1

Age (years)	Number of cases
0 — 1	6
1 — 3	7
4 — 7	7
7 — 14	8
15 — 20	12
21 — 30	60
31 — 40	39
41 — 50	18
51 — 60	8
61 — 70	2
71 — 80	1
Total	168

MATERIAL AND METHODS

Clinical observations. 168 patients with auricular burns were observed (77.38 % men, 22.62 % women). All had suffered facial burns classified as extensive in 36.3 % of them, and were taken to hospital in a state of shock;

Table 2

Cause	Number of cases
Water	43
Fire	111
Electricity	1
Chemical substances	9
Contact	4
Total	168

76.68 % of them were people in the productive age (Table 1) — 15 to 59 years old. The other age groups were represented less prominently.

Flame-induced burns were a predominant feature (Table 2). A total of 283 pinnae were affected, which indicates a predominance of bilateral burns

[Table 3]. As regards the intensity of the trauma sustained, the Vishnevski, Vilyavin and Schreiber. As shown by Table 4 based on this type of classification 96.82 % of the lesions were superficial burns where no surgical treatment

Table 3

Side	Number of cases
Right	41
Left	12
Right and left	115
Total	283

was necessary; only the remaining 3.18 % were deep burns. During the convalescence period, chondritis, cartilage necrosis and other complications developed in 21.55 % of the pinnae affected [Table 5].

Table 4

Degree of burn	Number of cases	%
1st	37	96.82
2nd	135	
3rd-A	102	
3rd-B	8	3.18
4th	1	
Total	283	

Experimental observations involved 21 rabbits ("white California" strain) weighing 2000—2800 grams. 1st-, 2nd-, 3rd-A, 3rd-B, and 4th-degree burns were produced according to Gubler [5]. For histological tests the rabbits' ears were amputated according to the following pattern: 1st degree on days 3 and 7, and 2, 3, 4 and 5 weeks after the burns; 2nd degree and 3rd-A degree on days 3 and 7, and 2, 3, 4, 5 and 6 weeks after the lesion; 3rd-B and 4th degrees — on days 3, 5, 7 and 14 following traumatization.

The amputated pinnae were fixed in 10 % neutral formaldehyde solution. 6 fragments were taken from identical sites in each pinna for paraffin processing.

The sections were stained with hematoxylin-eosin for collagen fibres ac-

according to van Gieson, with azan for connective tissue fibres according to Kruchay, for reticular fibres according to Gomori, and for elastic fibres according to Weigert, and also with toluidine blue.

Table 5

Complication	Number of cases	%
Chondritis	51	
Necrosis	3	
After effects	7	
Total	61	21.55

RESULTS

Clinical observations. 30 (17.86 %) out of 168 burned patients received surgical treatment for burns of different localization. In only one case (0.50 %) the patient's condition made it necessary to operate on the pinna — 20 days after the trauma — for central colliquation of the cartilage and for signs of chondritis after the wound had epithelialized. The patients subsequently refused to have their disfigured auricles reconstructed.

Experimental observations. 1st-degree burns. Horny layer desquamation and epidermal cell dystrophic changes were observed as from the 3rd day. Cells of the germinative layer of epidermis showed signs of polarity disorganization, and vesicular dystrophy could just be seen. Some of the epidermal segments showed signs of stratification, and accumulation of exudate with large quantities of proteins — as well as signs of intraepidermal "blister" formation. Dilated and hyperaemic vessels — interstitial oedema (Fig. 1) — could be seen in the derma. There were no discernible morphological changes in the skin adnexa or in the hyaline cartilage. A week after that, the changes were much less prominent, though still present. Two weeks after and later still there were no longer any signs of pathological changes.

2-nd and 3-rd-A-degree burns. Severe granular cell dystrophy could be seen involving all layers of the skin, with the boundaries between individual cells and layers difficult to define. The cell nuclei were darker, hyperchromatic, and occasionally lysed; isolated necrotized cells could be seen in a small proportion of the sections. The skin showed signs of collagen fibre homogenization, interstitial oedema, vascular dilatation and hyperaemia (Fig. 2). Petechiae could be noted round hair follicles, and dystrophic changes in the cartilage. On days 3 and 5, fibrinous-suppurative exudation was noted on the burn surface, and necrobiotic and necrotic changes in all skin layers.. Marked inflammatory reaction with large quantities of eosinophil leucocytes (Fig. 3). The necrotic part of the skin peeled off completely after 2 weeks, and regeneration — proliferation of basal layer cells from the preserved neighbouring

parts — was noted in some of the lesion-affected sites. The derma showed signs of total connective tissue destruction, pronounced inflammatory reaction, oedema, and haemorrhage (Fig. 4). The predominant feature of the inflammatory reaction round the necrotized tissue were lymphoid and eosinophil cells and, in the cartilage, necrobiotic changes without morphological signs of regeneration.

After a period of three weeks the surface of the wounds was completely free of all necrotic tissue; there was profuse inflammatory exudation, and a predominance of eosinophil leucocytes. The edges of the wounds were marked by a pronounced process of regeneration — proliferation of isogenous squamous epithelium without signs of differentiation. The skin adnexa were missing. The necrotic cartilage tissue showed no signs of regeneration. The regenerative process came to be better expressed 4 weeks after the trauma. Granulation tissue could be seen at the base of the burn wound (Fig. 5). 5 to 6 weeks later, all the skin tissues had become almost entirely regenerated complete with skin adnexa. The epithelial tissue was very thin. There were well visible elastic and collagen fibres in the derma. Dystrophic changes could only be found in the cartilage (Fig. 6).

3-rd-B- and 4th-degree burns. There was complete necrosis in all the layers of the skin (Fig. 7), involving even vascular walls underneath the epithelium. There were thrombi in the vascular lumen — both in the arteries and in the veins (Fig. 8). Only part of the follicular cells remained preserved. The hyaline cartilage was strongly basophilic, the chondrocytes in a state of necrobiosis (Fig. 9). Complete necrosis of the skin and subepidermic tissue could be noted on days 3 to 5. The outer two thirds of the auricular cartilage had been necrotized with only the central part remaining relatively preserved. There were no subsequent signs of inflammatory reaction or regeneration.

DISCUSSION

In the course of our experiments the burn degrees were determined according to the routinely used type of classification and also on the basis of clinical observations (7). As it proved impossible to arrange doses of thermal lesions so as to produce the exact copies of clinical forms, it was necessary to create three groups of burns. The clinical signs and strategy of burns treatment were much the same for each group. In this way, our experiments approached the system of three-degree classification as proposed by many authors. For that reason, we believe that the interpretation of results should pose little difficulty.

The results of observations made in the third experimental group are quite unambiguous. The clinical picture shows necrosis, elimination of damaged tissue, total deformation and defects of parts of the pinna or, indeed, its total destruction.

There were no such cases in our group of patients except, perhaps, in five of them who died during the first day of the shock period (3rd-B and 4th-degree burns involving 90 % of the body surface) and in whom such

damage could be anticipated. Cases of this type were not included in our analysis.

The morphological picture in the 2nd experimental group was consistent with the most frequent clinical picture: the pinna was swollen, straightened, the auriculotemporal angle enlarged. In the presence of infection, fever and fluctuation the condition was complicated by persistent and unmanageable pain. This group requires concentrated care and infection prevention. In case colligation develops conventional treatment should be supplemented by the surgical removal of the necrotic parts of the cartilage (infected or otherwise).

Group I poses little in the way of clinical problems; no special treatment is called for except preventing infection or deterioration of the processes of destruction.

Our observations seem to warrant the conclusion that the usual classification of auricular burns is hardly satisfactory since it has to differ from that involving burns in the rest of the body surface. In view of the characteristic features of the anatomical structure of the ear and in view of the fact that the thermal agent does not affect equally all the parts and tissues of the pinna, we believe a two-degree classification would be more appropriate:

1st degree — auricular burns not requiring any surgical treatment, and healing spontaneously and without adverse consequences; changes in all the tissues and all over the pinna surface are reversible; regeneration is complete with no traces left either on the surface or in the shape of the ear.

2nd degree — auricular burns requiring surgical correction during the acute period, and subsequent reconstruction. The processes of destruction in a certain well defined part of the pinna or all over its surface are irreversible. Regeneration, so long as it does take place, results in deformities, so long as it does not, in definitive loss of tissue. Surgical treatment is essential during the acute period in order to prevent infection, and speed up regeneration and healing. Reconstruction following healing is essentially a cosmetic affair.

In our opinion, chondritis as referred to by many authors is really involved only in the presence of a demonstrable infection which, in addition to local changes, has also a bearing on the overall clinical picture. All other changes come as a result of destructive processes triggered off by the thermal agent. The processes of destruction and regeneration may be in equilibrium and cause no new symptoms.

If the processes of destruction prevail, colligation and tissue elimination may develop even in the absence of infection. As shown by our experimental observations, the development of morphological changes in a burned pinna is a long-term one, it may last 6 weeks or more. Hence the reason for making distinction between two groups of auricular burns: without infection and with secondary infection. All this, however, calls for taking into account the species-specific and anatomical differences between the pinna in man and in animals (rabbit).

All the above listed changes, processes and reflections come into the picture also in cases of burns involving the cartilaginous parts of the nose.

J. H.

SUMMARY

163 patients with 283 auricular burns were followed up. Experimental pinna burns were produced in rabbits using Gubler's method. Morphological examinations were made on days 3, 5 and 7 after burn injury, and then after 2, 3, 4, 5 and 6 weeks. The results are discussed against the background of literary data. A two-degree system of classification is proposed in relation to the morphological-clinical picture, therapeutic tasks and possibilities. The term "chondritis" is used only for the description of cases of secondary infection. In the rest of the cases the changes are the result of the development of destructive and regenerative processes. In the authors' opinion, the same processes and changes can be assumed to take place in nasal cartilage burns.

RESUME

**Quelques notes à propos des particularités cliniques et morphologiques des brûlures
du pavillon d'oreille
(Observations cliniques et expérimentales)**

Trochev K., Markov D., Kozarov I.

On a observé 163 malades avec les brûlures de 283 pavillons d'oreille. Les brûlures des pavillons d'oreille chez les lapins étaient évoquées expérimentalement par la méthode de Gubler. L'examen morphologique était faite le 3^{ème}, le 5^{ème} et le 7^{ème} jour après la brûlure et puis après 2, 3, 4, 5 et 6 semaines. Les résultats sont discutés. On a élaboré une classification de deux degrés, qui correspond à l'image clinique et morphologique et aux buts et aux possibilités de la thérapeutique. Le terme «chondritis» n'est usé qu'en cas de l'infection secondaire. Les transformations dans les autres cas sont effectuées par les processus destructifs et régénératifs. Les auteurs supposent, que les mêmes transformations se produisent au cas des brûlures du cartilage nasal.

ZUSAMMENFASSUNG

**Einige klinisch-morphologische Besonderheiten der Verbrennungen der Ohrmuschel.
(Klinische und experimentelle Beobachtungen)**

Troschew K., Markow D., Kosarow I.

Es wurden 163 Patienten mit Verbrennungen von 283 Ohrmuscheln beobachtet. Im Versuch wurden Ohrmuschelverbrennungen nach der Methode von Gubler bei Kaninchen hervorgerufen. Morphologische Untersuchungen erfolgten am 3., 5. und 7. Tag nach der Verbrennungen und dann nach 2, 3, 4, 5 und 6 Wochen. Die Ergebnisse werden auf der Grundlage der Angaben in der Literatur diskutiert. Es wurde eine zweistufige Klassifikation vorgeschlagen, die dem klinisch-morphologischen Bild sowie den therapeutischen Aufgaben und Möglichkeiten entspricht. Der Begriff „Chondritis“ wird nur für Fälle einer sekundären Infektion benutzt. Bei übrigen Zuständen sind die Veränderungen das Ergebnis von Destruktions- und Regenerationsprozessen. Die Autoren nahmen an, daß man auch bei den Verbrennungen des Nasenknorpels gleiche Prozesse und Veränderungen voraussetzen kann.

RESUMEN

Algunas particularidades clínico-morfológicas en las quemaduras del pabellón de la oreja

(Observaciones clínicas y experimentales)

Trochev K., Markov D., Kozarov I.

Se ha observado a 163 pacientes con quemaduras de 283 pabellones. Como experimento las quemaduras de pabellones de la oreja se provocaron por el método Gubler en los conejos. Exámenes morfológicos se hicieron a los 3, 5, y 7 días de producirse la quemadura y, a continuación, a las 2, 3, 4, 5 y 6 semanas. Los resultados son debatidos a base de datos de la literatura. Está propuesta una clasificación de dos grados correspondiente al diagnóstico clínico-morfológico, las tareas terapéuticas y las posibilidades. La acepción "Condritis" se usa sólo en casos de infección secundaria. Los demás estados presentan cambios que son producto de procesos destructivos y regenerativos. Los autores opinan que en caso de quemaduras del cartílago nasal son de suponer los mismos procesos y cambios.

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SOME CLINICAL-MORPHOLOGICAL SPECIFICITIES
IN PINNA BURNS

[Clinical and experimental observations]

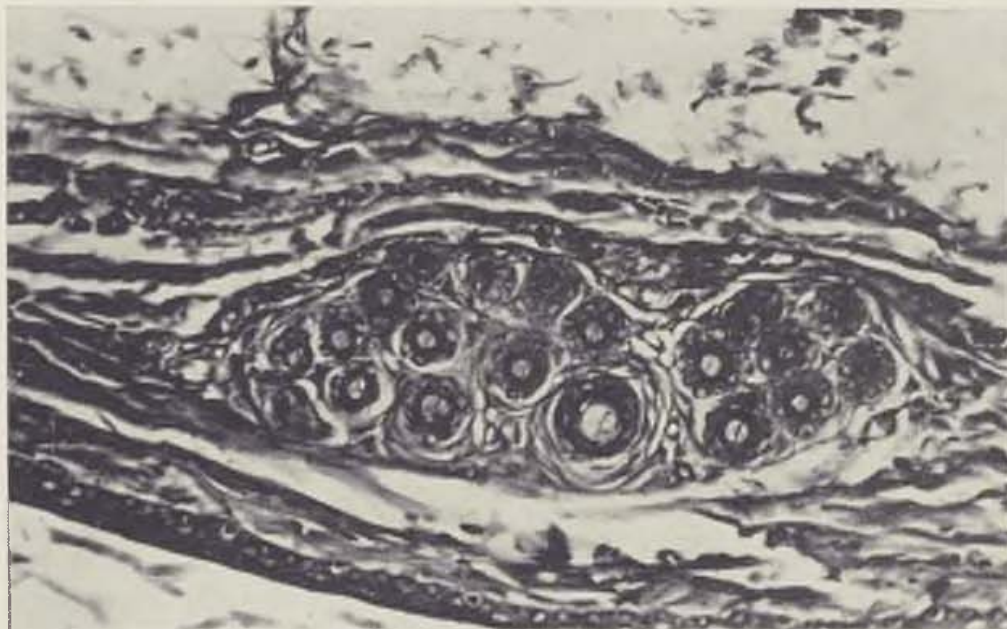


Fig. 1. Desquamation of epidermis, vesicular dystrophy of germinative layer cells, dermal layer oedema. HE — 10×10

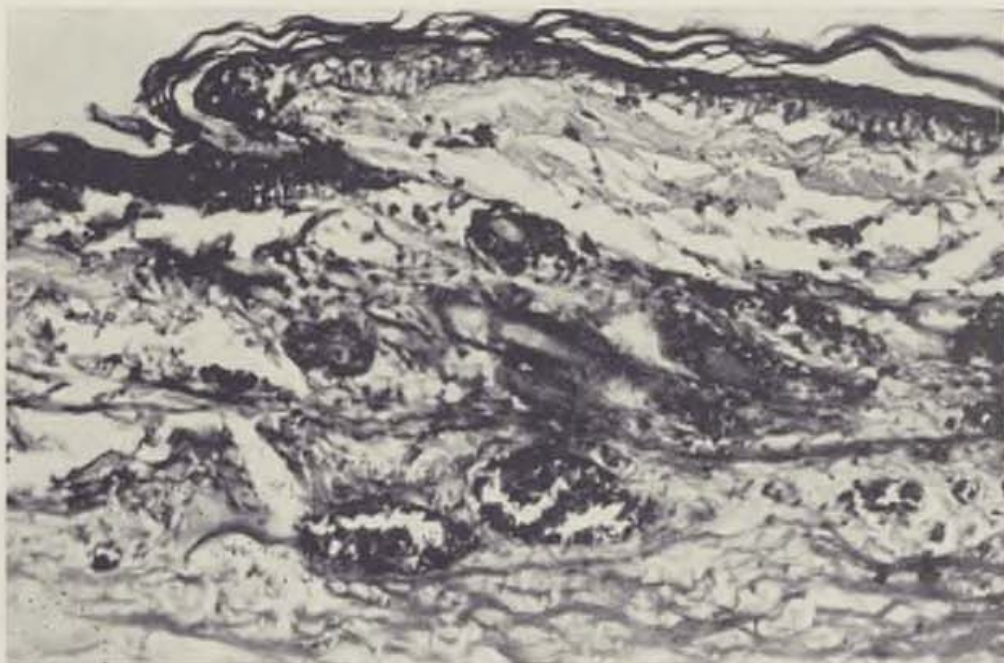


Fig. 2. Dystrophy and necrobiosis of epidermis, dermal layer fibres homogenization, interstitial oedema and haemolysis. HE — 10×10

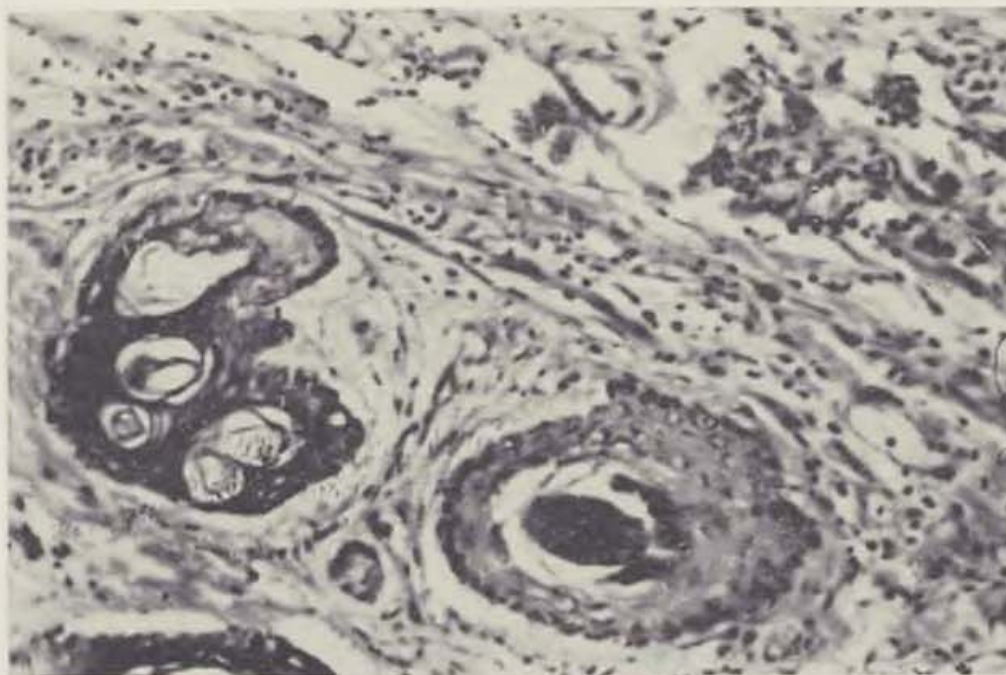


Fig. 3. Inflammatory oedema and mild inflammatory reaction in dermal layer. HE — 10×10

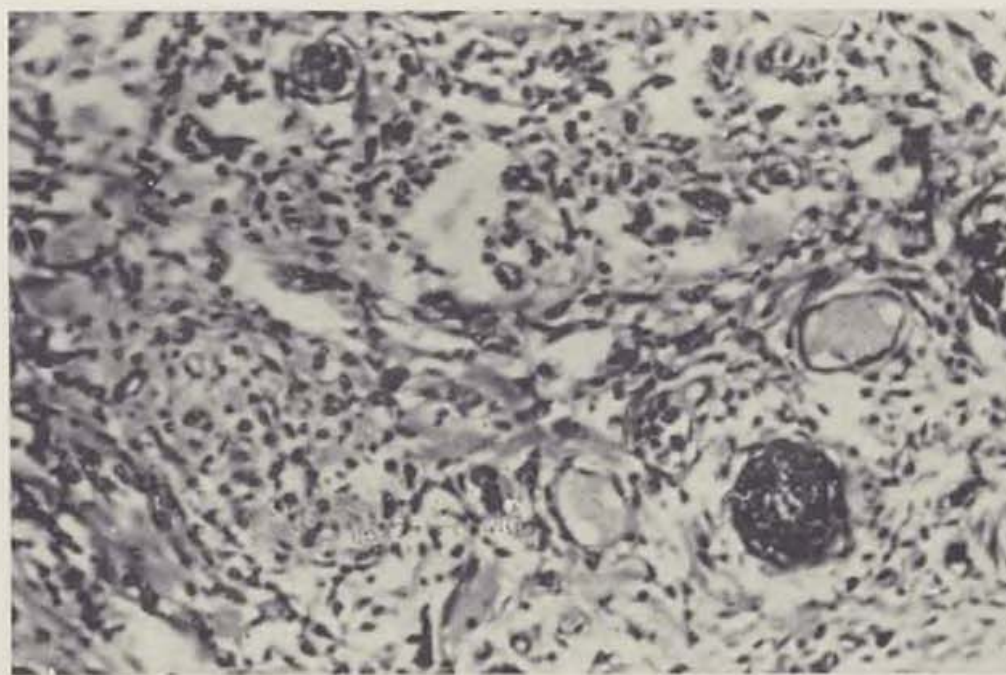


Fig. 4. Total disorganization of tissue in dermal layer with oedema and inflammatory infiltration. HE — 10×16

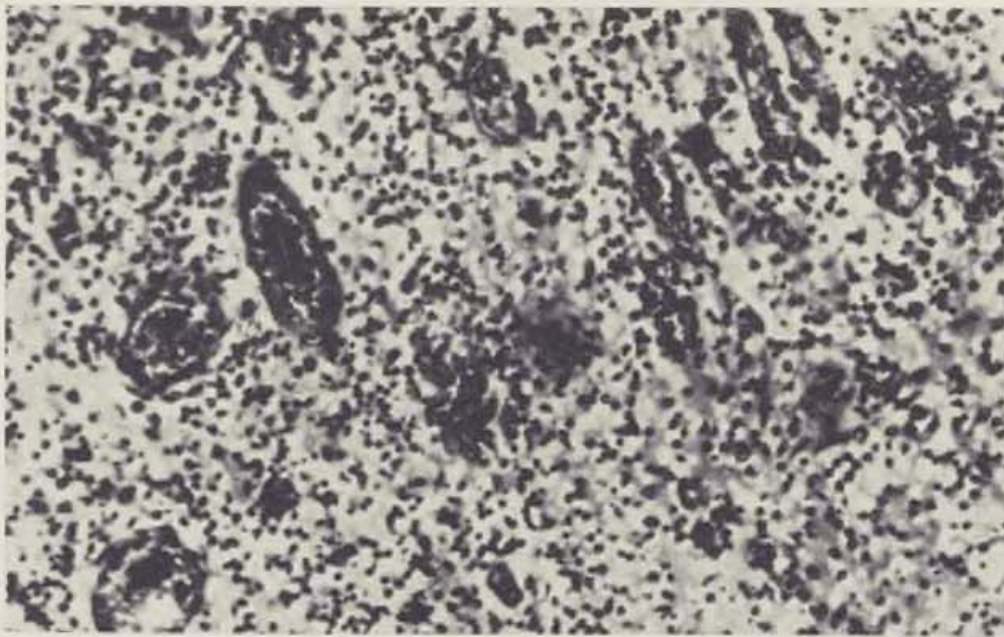


Fig. 5. Granulation tissue at burn wound base. HE — 10×16



Fig. 6. Epithelium and dermal layer regeneration. Chronic dermal layer inflammation. Dystrophy of cartilage. HE — 10×20

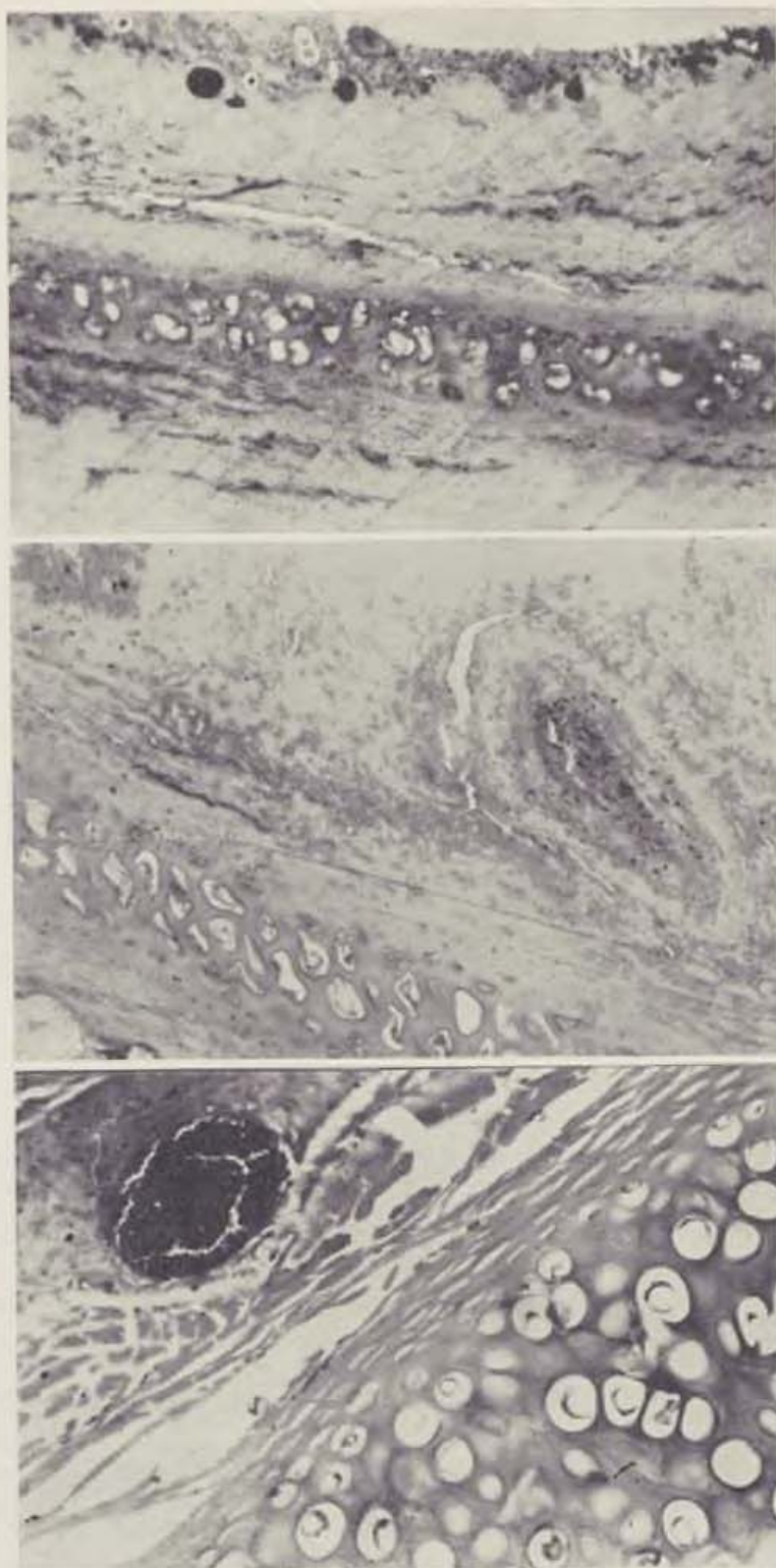


Fig. 7. Complete necrosis of all auricular layers and tissues. HE — 10×2.5 . — Fig. 8. Necrobiotic and necrotic changes in the pinna vascular wall and hyaline cartilage. HE — 10×10 . — Fig. 9. Major dystrophic necrobiotic and necrotic changes in auricular hyaline cartilage cells. HE — 10×20

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STOP FOR A MOMENT AND CONSIDER YOUR HEALTH



DAY AFTER DAY AND YEAR AFTER YEAR YOU ARE CONSTANTLY CHASING SOME AIM OR ANOTHER, YOU STRETCH THE MAINSPRING OF YOUR HEALTH TO THE VERY MAXIMUM. AND HOW LONG DO YOU THINK YOU CAN CONTINUE TO DO SO? REMEMBER THAT YOU HAVE ONLY ONE HEALTH AND FINALLY MAKE UP YOUR MIND TO GRANT IT, AT A VERY REASONABLE PRICE, WHAT IT DESERVES: COMPLEX TREATMENT AT ONE OF THE OLDEST AND THE MOST WIDELY RECOGNIZED SPAS IN EUROPE.

CZECHOSLOVAK SPAS — OASES OF HEALTH,
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