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Metabolic Changes in the Course of Severe Acute Brain Damage

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Summary. 27 Patients following severe brain injury were studied with regard to metabolic alterations. Metabolic rate was calculated from measured RQ, respiratory minute volume and oxygen consumption, as soon as the patient was able to breathe spontaneously. Catabolism was defined by determination of N-balance. It was concluded that metabolism following cerebral injury is particularly enhanced compared to common trauma. The neurological course of patients, who after midbrain syndrome recovered in contrast to those who developed apallic syndrome had little influence on metabolic rates, however, it distinctly seemed to modify catabolism. Thus catabolism throughout the apallic phase was found to be higher. The requirements for a favourable recovery from severe brain injury were defined as a well balanced nitrogen regime apart from a high amount of carbohydrate and fat calories.

Key words: Severe cerebral trauma, Midbrain syndrome, Apallic syndrome, Metabolic rates, Catabolism, High caloric nutrition.

I. Introduction, Objectives of Study

Following trauma certain almost obligatory metabolic changes have been reported to occur: a suppression of insulin production, (resulting in greatly reduced carbohydrate metabolism (Allison et al, 1968), increased lipolysis (Carlson and Liljedahl, 1971) and a marked catabolism of proteins for neoglycogenesis (Bünte, 1972). The degree of these alterations appears to correlate with the severity of the trauma, as can be indirectly concluded from the behaviour of free fatty acid levels (Birke et al., 1965) and from the excretion of catecholamines in the urine (Goodal et al., 1957; Frankson et al., 1954; Grashchenkov et al., 1965).

This hypercatabolism is even higher in patients with cerebral injury (Deligne, 1973) because of an increased demand on one hand (hyperthermia, hyperventilation, increased motor functions) (Steinbereithner, 1966; Bauer and Pia, 1966) and of an increased central stimulation on the other (Gerstenbrand, 1967). Clinically one gets the impression that metabolic changes are most severe and of longer duration in those patients who developed a midbrain syndrome and later became apallic. The notion was that there is a distinct difference in the metabolic behaviour between those patients with a midbrain syndrome who subsequently improve and those who become apallic.

Prior to the existence of intensive care units the classical apallic patient was bound to develop severe cachexia with grossly impaired further recovery and poor prognosis. High caloric parenteral nutrition was believed to suppress catabolic situations (Carlson, 1964; Haider and Steinbereithner, 1972).

However, compensation for the high protein loss, as well as parenteral high caloric nutrition in the immediate post-traumatic period can interfere with the necessity to prevent cerebral oedema through limitation of the fluid intake. Therefore it appears desirable to attempt to accurately define the influence of hypercaloric nutrition on the post-traumatic metabolism.

With regard to the above considerations, the aims of this study were: (a) to determine whether there is a particularly enhanced metabolic behaviour in patients following severe brain damage, (b) to clarify whether a specific neurological course (midbrain and apallic syndrome) causes particular metabolic alterations, (c) to show the impact of nutritional factors on the immediate post-traumatic period.

Table 1. Patients Studied and Collected Data

				days after injury							weeks				months					years	
				1	2	3	4	5	6	7	2	3	4	2	3	4	5	6	1	2	3 and longer
Class A: Patients studied immediately after brain injury																					
Group I: Brain concussion																					
Fo. G.	♂	29 a	subdural haematoma (op)	~	~	~	~	~	~	X~	T~	XT	X								
Group II: Mid brain syndrome (MBS) → recovery and defect																					
Bö. H.	♂	39 a	epidural haematoma (op)	~	~	~	~	~	~	~	~XTX~										
Ha. R.	♀	25 a	subdural haematoma (op)	X	~	~	†														
Sch. G.	♂	14 a	cerebral concussion	X	~	~	~	T	~	~	XT ~	XT ~	T	X							
Sd. S.	♀	16 a	subdural haematoma (op)	~	~	~	X	~	T	~	X ~	X									
Au. E.	♀	40 a	cerebral thrombosis	~	~	~	X	T	~	~	XT ~	X									
Schn. F.	♂	68 a	cerebral haematoma (op)	~	~	X	~	T	~	~	XT ~	XT ~	†								
Group III: Mid brain syndrome → apallic syndrome (AS)																					
Ru. R.	♂	6 a	epidural haematoma (op)	~	~	~	~	X	~	~	XT†										
Wo. P.	♂	27 a	cerebral concussion (severe)	~	~	~	T	~	~	~	XT	XT	X								
Mü. E.	♂	35 a	cerebral concussion (severe)	~	~	X	~	T	~	~	X ~	X ~	X ~	X							
Ti. K.	♂	49 a	cerebral shot wound (op)	~	~	~	T	X	~	~	XT†										
Class B: Late observations in 16 patients																					
Group I: Brain concussion – none																					
Group II: Defect stage after MBS																					
Hi. K.	♂	21 a	subdural haematoma (op)											X						X	
Ri. K.	♂	17 a	cerebral concussion								X										
Bo. J.	♂	40 a	cerebral concussion																		X
Group III: Apallic syndrome following MBS																					
Cz. G.	♂	12 a	brain abscess (op)												X	X					
Schi. R.	♂	6 a	epidural haematoma (op)															X	X		
Do. M.	♀	58 a	cerebral concussion																XX		
Hr. S.	♀	17 a	epidural haematoma (op)															X			
Ba. E.	♀	29 a	cerebral concussion													X ~	X				
Hr. B.	♀	11 a	cerebral concussion																X		
Pr. S.	♀	11 a	cardiac arrest														X ~		X		
Fr. K.	♂	29 a	cerebral concussion													XX ~					
Zm. St.	♂	31 a	cerebral concussion																	XX ~	
Ca. P.	♂	35 a	cerebral concussion															X ~			X
Ka. E.	♂	12 a	cerebral concussion																X	X	
El. F.	♂	14 a	cerebral concussion													X			X		
Ga. H.	♂	18 a	cerebral concussion											X							

X = metabolic rate, T = thyroid function, ~ = urinary excretion of nitrogen and catecholamines respectively

II. Patients Studied and Methods

11 patients were studied immediately after brain injury (class A) and a second group of 16 patients was examined from two months to three years following injury (class B). Table 1 shows initials, sex, age and diagnosis of all patients studied. The time schedule of the collected data is given in symbols on the right half of the table.

Basal metabolic rates where determined at weekly intervals, urinary excretion of catecholamines was measured during the first three days and subsequently every third day, N-excretion for 21 days and thyroid hormone blood levels weekly. The results of the study on catecholamine excretion and on the thyroid function tests will be presented in a paper to be published later.

The patients of class A (studied immediately after the injury) and group B (studied later) were divided into three groups according to their neurological course. Group 1 consists of patients with primary brain damage of traumatic or other origin, who did not show any neurological complications. Group 2 includes all patients who developed an acute midbrain syndrome in its different stages or a bulbar brain syndrome. After passing through the different phases up to the full stage in these patients a remission of the midbrain symptomatology occured. Consequently they did not develop an apallic syndrome. However, to pass through all different phases was not obligatory for all patients. In group 3 also in nearly all patients an acute midbrain syndrome or a bulbar brain syndrome was observed. Following this, however, within a characteristic span of time a transitory stage towards an apallic syndrome developed, and finally, the full picture of an apallic syndrome appeared. This again in some patients showed a remission up to a defect stage of differing extent. Several patients could not be followed up because they died in the full stage or at an early stage of remission.

As to the aetiology of acute midbrain syndrome (MBS) or apallic syndrome (AS) respectively, it must be pointed out that in the majority of cases there was a cranio-caudal displacement caused by oedema of the brain and only in a few patients by an intracranial haematoma. The consequence of this was a tentorial or foraminal herniation which lead to this secondary midbrain and bulbar brain symptomatology. The same mechanism may be assumed in the apallic cases. However, morphological studies (Jellinger et al., 1966; Gerstenbrand, 1967) showed the occurrence of local lesions in the upper brain stem, also of secondary aetiology (Peters, 1966).

As soon as the patient was able to breathe spontaneously, oxygen consumption and CO_2 production were measured and the respiratory quotient was calculated. Measurements were performed in an open system using a pneumotachograph with volume integration for the assessment of respiratory minute volume. From every breathing cycle a portion of expiratory gas was removed by a breath synchronized pump and examined for its oxygen and CO_2 content in a respective gas analyzer (O_2 analyzer Hartmann & Braun, paramagnetic principle, CO_2 analyzer Hartmann & Braun, infra-red absorption). From these data the metabolic rate was calculated. The metabolic rate was expressed in calories per minute and in calories per hour per square meter body surface and compared to the standard value of every patient (using the standards of Biological Data).

Total daily urinary nitrogen excretion was determined by a Micro-Kjeldahl method. Protein catabolism was calculated from the amount of excreted nitrogen, multiplying nitrogen by the factor 6.25.

Nutrition. It was attempted to start high caloric parenteral nutrition as quickly as possible after trauma. If the liver function was found to be fairly good, a high caloric intake regime was instituted with the aim to give at least two to three thousand calories per day to the adult patient. The main calorie carrier was dextrose which was used as a concentrated solution with insulin added to it. The idea was to compensate for the endogenous insulin suppression that exists during this stage of shock, by supplying a large enough amount of exogenous insulin (Haider et al., 1973). In contrast to other recommendations (Berg, 1973) based on previous experiences, we were using intravenous fat emulsions, not only starting on the fifth to seventh day, but already on the first day following trauma. Presuming that heparin increases the activity of lipoprotein lipase and in doing so accelerates fat clearance 2500 units of heparin were added to each 500 cc of intralipid. Within the regime of complete intravenous nutrition protein was supplied using amino-acid solutions. Since a mixture of aminosol, glucose and alcohol was used in order to increase the calorie content of the single bottle, the actual percentage of protein did not exceed 15 g per bottle, which meant that by giving two bottles per day only 30 g of protein were administered to the patients. Tube feeding was gradually increased in accordance with normalisation of bowel movements, however, intravenous nutrition was maintained throughout the study „class A".

Other common therapeutic measures like antibiotic cover, use of aldosterone antagonists, and diuretics, as well as all therapeutic steps of contemporary intensive care were also applied to these patients.

III. Results

1. Metabolic Rate (Calorimetry)

Table 2 shows the patients of both studied classes, acute (A) and late observations (B), grouped according to their neurological course. On the specified days following trauma we determined the respiratory minute volume, respiratory quotient, oxygen consumption, basal metabolic rate and basal metabolic rate in per cent of normal (100 per cent being the normal value).

Table 2. Metabolic Parameters

	Initials	days after trauma	minute vol. (1/min)	Resp. Quo-tient	Oxygen con-sumpt (ml/min)	BMR (cal/h/m²)	BMR in % of normal
Class A							
Group I	Fo. G.	7	7,8	0,82	390	57,6	164
		13	12,9	0,82	568	83,9	240
		22	7,9	0,72	363	52,3	149
Group II	Ha. R.	1	14,5	0,76	551	94,7	270
	Schu. G.	1	6,8	0,69	397	56,8	130
		8	7,8	0,81	332	62,3	142
		17	8,6	0,81	361	70,9	162
		40	7,5	0,71	311	56,8	130
	Sd. S.	4	10,9	0,76	545	95,9	254
		8	10,5	0,69	441	76,3	202
		18	7,5	0,78	400	70,7	188
	Au. E.	4	10,7	0,82	417	76,8	219
		10	8,3	0,75	332	63,8	182
		18	12,8	0,87	384	71,8	205
	Bö. H.	9	10,9	0,74	589	95,8	278
		12	9,2	0,77	432	70,6	205
Group III	Ru. R.	5	5,0	0,75	255	69,1	128
		10	11,6	0,58	302	78,3	145
	Wo. P.	14	13,7	0,91	589	88,6	253
		20	12,2	0,82	622	91,4	261
		26	8,2	0,79	385	56,0	160
	Mü. E.	3	7,9	0,77	339	46,8	127
		10	9,8	0,79	373	51,8	140
		17	13,9	0,75	446	61,3	166
		24	12,1	0,73	494	67,5	183
		31	8,6	0,83	353	49,5	134
	Ti. K.	5	16,3	0,79	391	47,1	130
		11	14,1	0,93	409	64,1	177
	Schn. F.	3	6,0	0,69	336	51,5	151
		8	7,1	0,76	298	46,6	137
		15	8,5	0,90	262	42,3	124
Class B							
Group II	Ri. K.	10	8,7	0,92	293	49,7	119
	Hi. K.	57	8,0	0,80	288	38,4	104
		482	8,5	0,89	307	53,5	143
	Bo. J.	1015	12,7	0,84	483	41,0	112
Group III	Cz. G.	72	5,7	1,00	159	39,1	86
		112	4,9	0,85	132	31,3	69
	Schi. R.	165	5,1	0,82	169	54,8	104
		192	4,8	0,77	159	51,5	95
	Do. M.	198	4,4	0,91	190	34,8	99
		214	8,3	0,75	261	68,5	176
	Hr. S.	172	9,0	0,82	351	60,4	150
	Ba. E.	125	19,8	0,95	430	70,6	202
		155	17,4	1,05	385	55,0	157
	Ar. B.	460	7,0	0,80	195	58,1	131
	Pr. S.	155	6,0	0,90	128	65,4	148
		196	5,9	1,00	106	32,0	73
	Fr. K.	103	8,1	0,80	202	31,7	84
		123	9,7	0,95	270	42,9	114
	Zm. St.	534	26,2	0,95	430	52,0	132
		545	21,2	0,70	428	49,5	126
	Ca. P.	144	14,9	1,05	251	39,3	107
		896	6,3	0,82	247	32,4	88
	Ka. E.	215	12,3	0,86	441	51,7	114
		387	6,1	0,87	184	34,1	75
	Ei. F.	97	12,7	0,80	477	84,3	192
		366	6,6	0,88	225	40,6	93
	Ga. H.	36	11,9	0,89	452	79,3	196

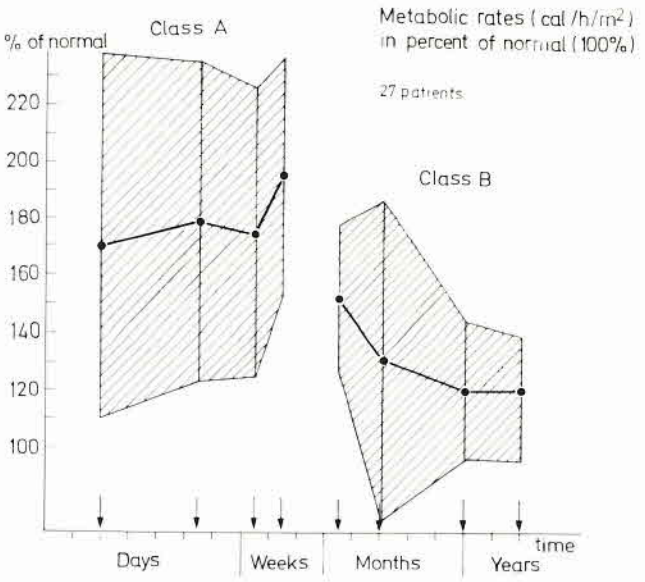

Fig. 1

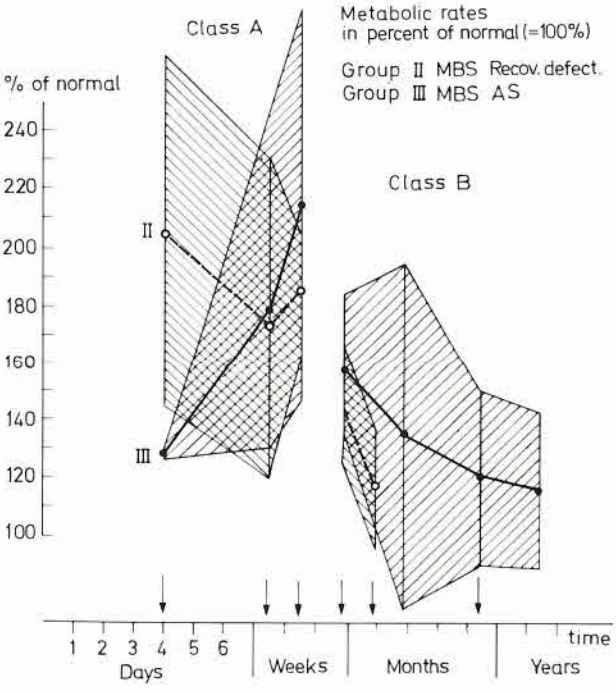

Fig. 2

Mean values of all patients studied are given in Fig. 1. The periods of time are on the abscissa, whereas on the ordinate the mean values including deviations from the standard can be seen. The metabolic data were obtained by comparing the respiratory minute volume, which had

been measured by means of calorimetry for the respiratory quotient and the oxygen consumption per minute. This way the metabolic rate per hour and square meter was calculated and compared with normal values for every single patient. The metabolic rate on the ordinate is expressed in per cent of normal values.

It can be seen that mean values of the metabolic rates at the beginning were markedly elevated (170 per cent). In the further course they rose slowly up to the third week, at which time a maximum of nearly 200 per cent was reached. Later observations in class B show a slow and persistent decline towards normal values (120 per cent). Measurements were taken over a period of two years.

In Fig. 2 the metabolic rates are arranged according to the neurological course of the patients. The declining tendency of the metabolic rates in group 2 progressing towards recovery was more marked than in group 3, which was developing the apallic syndrome. However, there were no significant differences between the two groups because of the high standard deviations. Observation of group 2 terminated after two months since these patients due to fast recovery could not be studied any longer.

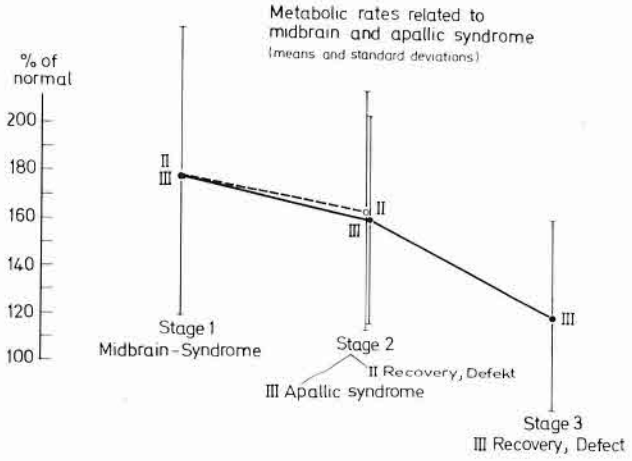

Fig. 3

Fig. 3, in which the obtained metabolic rates of the different groups are arranged in accordance with the neurological stages, shows that groups 2 and 3 remain closely together during the stage of acute midbrain syndrome at a level of about 180 per cent. For group 2 progressing towards recovery or defect stage and group 3 moving on to the apallic syndrome, the values remain still close together, but are somewhat lower than during the midbrain syndrome stage. However, when group 3 proceeds towards recovery or defect stage, its mean value is only about 120 per cent.

2. Nitrogen Balance (Catabolism)

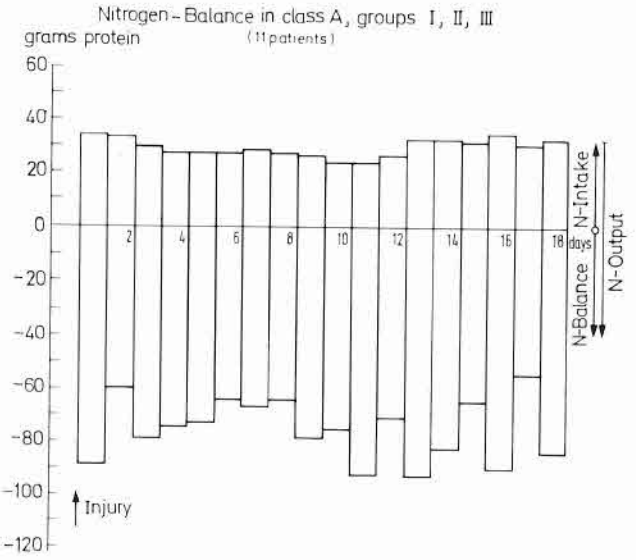

Fig. 4

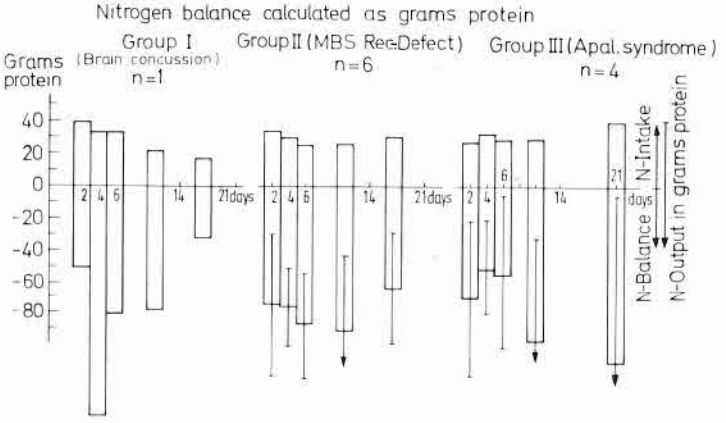

Fig. 5

The nitrogen balance, mean values of 11 patients of class A, is shown in Fig. 4. Columns above the abscissa give the amount of nitrogen intake in grams of protein, columns below show the nitrogen balance. The total length of the column represents the nitrogen output, whereas on the abscissa the days following trauma are listed. There appears to be a highly negative nitrogen

Table 3. Nitrogen-Balance (Means and standard dev.) (calculated as Grams Protein)

	1st & 2nd day	3rd & 4th day	5th & 6th day	2nd week	3rd week	
Group I n = 1	29,0	32,5	32,5	21,0	16,3	N-intake
	−50,5	−144,5	−81,5	−79,3 ± 15,7	−33,0	N-balance
	79,5	177,0	114,0	100,3	49,3	N-output
Group II n = 6	33,6 ± 14,0	30,0 ± 19,6	24,9 ± 15,6	25,4 ± 12,3	31,1 ± 11,9	N-intake
	−74,6 ± 45,0	−82,2 ± 25,0	−91,7 ± 33,6	−89,1 ± 48,6	−55,5 ± 35,7	N-balance
	108,2 ± 41,9	112,2 ± 82,9	116,6 ± 38,8	114,5 ± 47,8	86,6 ± 29,3	N-output
Group III n = 4	27,3 ± 11,0	32,2 ± 17,7	28,8 ± 17,8	29,1 ± 15,3	39,4 ± 18,1	N-intake
	−82,9 ± 49,2	−50,8 ± 30,3	−54,1 ± 47,5	−97,8 ± 65,2	−110,6 ± 104,2	N-balance
	110,2 ± 52,2	83,0 ± 40,7	83,0 ± 61,7	126,9 ± 74,9	150,2 ± 99,4	N-output

balance throughout the first three weeks at an average protein intake of about 30 g per day.

Mean values of nitrogen balances grouped according to the neurologic course are depicted in Fig. 5. In groups 1 and 2 it tends to be highly negative between the third and eleventh day and becomes less negative after three weeks. Nitrogen balance in group 3, however, becomes progressively negative with a maximum on the 21st day after trauma.

Nitrogen balances calculated as gram protein are listed in Table 3. Means and standard deviation can be seen in groups 1 to 3, listing the intake, the balance and the output.

IV. Discussion

From the clinical point of view one can expect that metabolic rates following brain injury would be elevated. This is on one hand caused by a general metabolic factor common to all trauma. Cuthbertson (1972) described in the course of overall metabolic activity following general trauma an "ebb phase" and a "flow phase". The first being of very short duration shows a decreased rate of metabolism. This is followed by a period of increased metabolism which is characterized by a high metabolic rate and elevated excretion of nitrogen in the urine. These alterations reach a peak 4 to 8 days after general trauma (Cuthbertson, 1969). A rise in oxygen consumption after trauma was described by Cope et al. (1943) in burned patients, with an elevation of metabolic rates of 30 to 80 per cent, and by Kinney (1962) after surgical procedures, with the metabolic rate elevated by 7 to 15 per cent.

On the other hand, in cerebral injury there appears to be an additional factor involved which is caused by a vegetative dysregulation (Gerstenbrand, 1967; Gerstenbrand and Galanti, 1972). For this reason the metabolic rate of these patients exceeds the metabolic rate of common trauma patients, a fact which has also been pointed out by Deligne, 1973.

Our patients have, as can be seen from Fig. 1, an increased metabolic rate which is raised 70 to 100 per cent, remaining at about the same high level for the first four weeks, even slightly rising during this period. As far as the extent of the metabolic changes following cerebral injury are concerned, our results show that they are about twice as marked as in ordinary trauma. In relation to the duration, however, it is about three times as long compared to that of trauma in general. As can be seen from the metabolic rates of class B patients, metabolism is gradually decreasing in these patients, but still remains at a slightly elevated level.

As for the comparison of the increase in metabolism related to the severity of trauma in general based on the indirect evidence of elevation of free fatty acids (Birke et al., 1965; Carlson and Liljedahl, 1971) and of the catecholamines (Goodall et al., 1957; Birke et al., 1958) there is a positive correlation. Grashchenkov et al. (1965) found for cerebral injury corresponding values of catecholamine excretion in accordance to the severity (Lorenz, 1973). Grouping our patients according to their neurological course one would expect that patients who pass from MBS to AS show a higher elevation than patients who just came out of the MBS and recovered.

If one looks on our metabolic rates grouped according to time (Fig. 2), there appears to be an uncharacteristic course. One gets the impression that metabolic rates of patients in group 2 are getting back to normal values sooner. Possibly this is just the expression of the shorter and milder course of disease in this group. If groups 2 and 3 are observed only in accordance to the neurological course disregarding the time factor, it appears (Fig. 3) that there is no difference in metabolism between patients who are in the recovery stage following midbrain syndrome and patients who following midbrain syndrome became apallic. Obviously, the autonomous stimulation of metabolism is not that strong or the response of metabolism to the neurological course is less sensitive. It has to be taken into consideration that interpretation of the metabolic rate is more difficult since daily fluctuations apparently overrule the neurological stimulus and, due to fast recovery, group 2 patients have not been studied in a stabilized recovery stage.

Respiratory quotient was ranging between 0.7 and 0.82 during the first week. If RQ is considered an indicator as to which kind of nutrients are metabolised, RQ values below 0.82 would indicate a predominance of lipolysis either of exogenous or body fat, in spite of the large amount of glucose which had been administered and metabolized. Hyperventilation was also seen in most of the patients during the first week and must be considered as a factor which lowers the respiratory quotient. For this reason it is very difficult to comment on the rather inconsistent values, as with increased ventilation RQ is not considered a true metabolic indicator. Measurements of RQ several months or even years after brain injury show, however, that at this time RQ values tend to be higher.

Catabolism – nitrogen balance. After trauma the N-balance is usually negative. Cuthbertson (1969) has found that in man the protein loss within the first 10 to 12 days after general trauma can reduce the total nitrogen content of the body by 7 per cent. Concerning the influence of nutrition upon posttraumatic protein loss, Fleck and Munroe (1963) found in rats that those animals which had had a diet rich in protein, showed a significant loss of nitrogen in the urine and also a loss in weight, whereas animals which were on a protein free diet did not show these alterations. This would indicate that protein excretion is somehow dependant on the administration of protein solutions. Schlick et al. (1972) had shown, however, in overweight patients who were fasting completely, that nitrogen excretion per day amounts to 6.4 g and remained at the same level for three weeks. This is equivalent to a daily loss of protein of less than 50 g. The administration of 50 g of protein per day did not increase nitrogen excretion and completely balanced nitrogen metabolism. In connection with the influence of caloric content of nutrition on the protein loss Calloway and Spector (1954) showed that the average protein loss amounted to 12.4 g nitrogen per day, when protein was administered in the diet. Using a protein free diet of 400 to 700 calories a day, average nitrogen loss was only 8.4 g and remained at the same level, even when the calorie intake was increased to 3000 cal. per day (Huguenard and Roujas, 1970).

Hallberg (1966) showed that higher calorie supply was in good correlation with a lower negativity of nitrogen balance. In our patients nitrogen balance (Fig. 4) remained highly negative for at least three weeks following trauma at a nearly constant level. From the fact that the N-balance of group 3 in contrast to groups 1 and 2 (see Fig. 5) becomes increasingly more negative, it can be concluded that catabolism following severe brain injury in these patients who had become apallic, is higher and lasts for a longer period of time. A higher rate of complications (decubital ulcers, urinary tract infections etc.) is perhaps also of some significance, since Abbott and Albertson (1963) demonstrated in surgical patients that the extent of catabolism was more closely related to the occurrence and severity of complications than to the extent of the surgical trauma.

Parenteral nutrition itself is certainly of great influence on metabolism. High protein administration as well as high caloric feeding are most imperative in hypercatabolic conditions. In our study not all the possibilities of high protein administration had been made use of. The significance of this is, however, disputable, if one takes into consideration the specific dynamic effect of protein. Apart from the amount of calories, early administration of parenteral nutrition also seems to be of importance, because we feel that by this the establishment of a hypercatabolic situation can be mitigated. To what extent high caloric nutrition is really able to suppress catabolism can eventually be demonstrated by the rate of excretion of catecholamines in the urine. The data on this will be published in a separate paper. Therefore we recommend to prolong the period of sedation up to the third week in order to slow down metabolism, inhibit motor hyperactivity and lower the body temperature. Simultaneous administration of protein and calories, starting within the first few hours following trauma, should be increased.

From the results of our study we concluded that:

a) Metabolism in patients with severe brain injury shows a distinctly higher increase in comparison to that following trauma in general. This is true for metabolic rates, which are markedly above normal up to the fourth posttraumatic week and remain slightly elevated for at least one year. It also applies to catabolism which is considerably higher and stays at the same level for at least three weeks.

b) The neurological course of patients who after midbrain syndrome recovered in contrast to those who developed on apallic syndrome has little influence on metabolic rates. Catabolism expressed by N-balance in patients with an apallic syndrome is higher and lasts for a longer period of time than in patients who only passed through the midbrain syndrome.

c) From a clinical impression hypercaloric nutrition in the immediate post-traumatic period seems to be of substantial benefit. Since a control group is lacking, we are at this time not able to quantify this beneficial effect. The protein intake given, however, was not large enough to balance the hyper-metabolism, and though a certain suppressive action on catabolism by a sufficiently high administration of carbohydrate and fat calories can be assumed, it seems important to further increase the protein intake.

References

1. Abbott, W. E., Albertsen, K.: The effect of starvation, infection and injury on the metabolic processes and body composition. Ann. N. Y. Acad. Sci 110, 941 (1963)
2. Allison, S. P., Hinton, P., Chamberlain, M. J.: Intravenous glucose tolerance, insulin and FFA levels in burned patients. Lancet 1113 (1968)

3. Bauer, B. L., Pia, H. W.: Parenterale Ernährung in der Neurochirurgie. In: Lang, K., Frey, R., Halmagyi, M.: Springer, Berlin – New York 1966
4. Berg, G.: Discussion at the: 5. Gemeinsame Tagung der Deutschen und Österreichischen Arbeitsgemeinschaft für Internistische Intensivmedizin, Wien, Sept. 1973.
5. Birke, G., Duner, H., Liljedahl, S. O., Pernow, B., Platin, L. O., Troell, L.: Histamine, Catecholamines and Adrenocortical Steroids on Burns. Acta. chir. Scand. 114, 87 (1958)
6. Birke, G., Carlson, L. A., Liljedahl, S. O.: Lipid metabolism and trauma III. Acta. Med. Scand. 178, 337 (1965)
7. Bünte, H.: Klinik der postoperativen Störung des N-Metabolismus und Elektrolythaushaltes. Band: Stoffwechsel, Frey, Kern, Mayrhofer 58, 99 (1972)
8. Calloway, D. H., Spector, H.: Nitrogen balance as related to caloric and protein intake in active young men. Amer. J. Clin. Nutr. 2, 405 (1954)
9. Carlson, L. A.: Deposition, mobilisation and utilization of fat. Acta. chir. scand. Suppl. 325, 5 (1964)
10. Carlson, L. A., Liljedahl, S. O.: Effect of treatment with intravenous fat emulsions on plasma lipids, proteins and clinical condition of burned patients. Acta. Clin. Scand. 137, 123 (1971)
11. Cope, O., Nathanson, I. T., Rourke, G. M., Wilson, H.: Symposium on the management of grave burns at Massachusetts General
12. Cuthbertson, D. P., Tilstone, W. J.: Metabolism during the post-injury period. Adv. clin. Chem. 12, 1 (1969)
13. Cuthbertson, D. P., Fell, G. S., Smith, C. M., Tilstone, W. J.: Nutrition in the Post-Traumatic Period. Nutr. Metabol., Vol. 14, Suppl. 92 (1972)
14. Deligne, P.: Le retentissement metabolique du traumatisme cerebral le catabolisme. Ann. Franc. Special 1, 150–157 (1973)
15. Fleck, A., Munro, H. N.: Protein metabolism after injury. Metabolism 12, 783 (1963)
16. Frankson, C., Gemzell, C. A., von Euler, U. S.: Cortical and meduallary adrenal activity in surgical and allied conditions. J. clin. Endocrinol. 14, 608 (1954)
17. Gerstenbrand, F.: Das traumatische apallische Syndrom. Springer Verlag, Wien – New York 1967
18. Gerstenbrand, F., Galanti, T.: Die Bedeutung der Ernährung für die Prognose des apallischen Syndroms. In: Parenterale Ernährung, S. 166–171, Verlag H. Huber 1972
19. Goodall, Mc. C., Stone, C., Haynes, B. W., Jr.: Urinary output of Adrenaline and Noradrenaline in severe Thermal Burns. Surgery 145, 479 (1957)
20. Grashchenkov, N. I., Boeva, E. M., Irger, J. M., Kassil, G. N., Kamenetskaya, B. I., Fishman, M. N.: Clinical and pathophysiological analysis of acute closed cranio-cerebral injury. In: Proceedings of the third International Congress of Neurological Surgery. Copenhagen, 23–27 August, International Congress, Series n° 110, Excerpta Medica Foundation, 119–159 (1965)
21. Haider, W., Lackner, F., Skudrzyk, I., Tonczar, L.: Das Verhalten der Kohlenhydrattoleranz bei gleichzeitiger Verabfolgung von hohen Dextrose- und Insulinmengen zur parenteralen Ernährung von Intensivpatienten mit gesteigertem Kalorienbedarf. 1973, in print
22. Haider, W., Steinbereithner, K.: Das Verhalten der Lipidfraktionen im Plasma während langdauernder parenteraler Fettzufuhr bei Patienten mit schwerem Schädel-Hirn-Trauma (SHT). Herausgegeben von G. Hartmann und H. Berger, Verlag Hans Huber, Bern, Stuttgart, Wien 1972
23. Hallberg, D., Schubert, O., Wretlind, A.: Parenterale Ernährung. Dt. Übersetzung aus: Läkartidningen 65, 4563 (1968)
24. Huguenard, P., Roujas, F.: Essai clinique systematique d'un nouvelle assoziation des L amino acides. An. Anesth. Franc. XI, I, 31 (1970)
25. Jellinger, K.: Zur Pathogenese und klinischen Bedeutung von Hirnstammschäden nach gedecktem SHT. Acta 25 Conv. Neuro. Psych et EEG. Hung. Budapest 303, 1966
26. Kinney, J. M.: Protein metabolism in human pathological states: The effect of injury on human protein metabolism in "Symposium on Protein Metabolism. Influence of growth hormone on anabolic steroids and nutrition in health and disease". F. Gross ed., p. 275, Springer-Verlag, Berlin 1962
27. Lorenz, R.: Wirkungen intracranieller raumfordernder Prozesse auf den Verlauf von Blutdruck und Pulsfrequenz. Acta neurochir. Suppl 20, Springer-Verlag 1973
28. Peters, G.: Morphologische Forschung in der Neurologie und Psychiatrie. Nervenarzt 37, 429 (1966)
29. Schlick, W., Kloucek, H., Lageder, H., Irsigler, K.: Energieumsätze unter Nulldiät. Medizin und Ernährung 13, 215 (1972)
30. Steinbereithner, K.: Spezielle Fragen der künstlichen Ernährung schwer Schädel-Hirn-Verletzter. Fortschritte der parenteralen Ernährung. Symp. d. Internat. Soc. of Parenteral Nutrition, Pallas-Verlag, Lochham bei München, S. 96, 1966

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