Europ. J. Intensive Care Medicine 1, 1, 115–123

Urinary Catecholamine Excretion and Thyroid Hormone Blood Level in the Course of Severe Acute Brain Damage

W. Haider[1], H. Benzer[1], G. Krystof[1], F. Lackner[1], O. Mayrhofer[1], K. Steinbereithner[1], K. Irsigler[2], A. Korn[2], W. Schlick[2], H. Binder[3], F. Gerstenbrand[3]

Intensive Care Unit of the Institute of Anaesthesiology[1] and the 2nd Surgical Clinic, 1st Clinic of Internal Medicine[2], and Neurological Clinic[3] of Vienna University, Austria

Abstract. Urinary catecholamine excretion and thyroid hormone blood level were studied in 16 patients following severe cerebral trauma. Increased excretion rates of epinephrine and norepinephrine were found. There was no significant difference in the catecholamine excretion when compared with generally traumatized patients. The relationships between catecholamine excretion, increased metabolic rates, and negative nitrogen balance indicate that in patients with a midbrain syndrome there exists an additional diencephalic metabolic factor, which leads to a rise in fat oxidation and perpetuation of catabolism.

Early high caloric parenteral nutrition seems to inhibit the initial increase of catecholamine excretion and thus protects the body from an unnecessary breakdown of its own reserves. If the course is classified according to neurological stages, it can be shown that patients with a traumatic apallic syndrome in poor condition have a high increase of catecholamine excretion. Secretion of thyroid hormones is not influenced significantly by cerebral trauma.

Keywords: Severe cerebral trauma, Midbrain syndrome, Apallic syndrome, Catecholamines, Fat oxidation, Thyroid hormones, High caloric total parenteral alimentation (TPA).

Introduction

In patients with severe brain injury a deviation of the metabolic functions of a similar nature as in patients with severe general trauma can be observed. In a previous study we described the metabolic changes following severe brain injury (Haider *et al.*, 1974), and showed that the metabolic rates were markedly elevated. This elevation was of a higher degree and of longer duration than the rise seen in general trauma (Cuthbertson and Tilstone, 1969; Kinney, 1962). It was also shown that urinary nitrogen excretion was increased for at least three weeks after trauma and that the degree of catabolism was related to the extent of the cerebral damage.

Since increased metabolism can be an indicator of increased fatty acid turn-over (Challoner and Steinberg, 1966) and catabolism (increased break-down of protein for gluconeogenesis) is associated with increased fatty acid mobilisation (Deligne *et al.*, 1973), the increased fat metabolism can be considered as a manifestation of these two conditions.

In general trauma catecholamines (Carlson, 1964) and endogenous stimuli generated by "relative hunger" (Wadström, 1959) determine the post-traumatic metabolism which is characterized by an increase of fatty acids, whilst in cerebral trauma apparently there is an additional autonomic disturbance (Gerstenbrand, 1967).

This additional metabolic effect, which obviously is seen only in patients with an acute midbrain syndrome (MBS) can, via reticulo-thalamic action lead to an activation of hypothalamic centers (Fossati *et al.*, 1973) with ACTH-production which, like catecholamines (Sutherland and Robison, 1966) activates cAMP (Westermann, 1966) and thus leads to an increase of lipolysis.

The problems are still unsolved, as to how endogenous hormone excretion changes after severe brain trauma,

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 5 patients																					
Group I: Brain concussion – none																					
Group II: Defect stage after MBS																					
Group III: Apallic syndrome following MBS																					
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
Ba. E.	♀	29 a	cerebral concussion													X	X				

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

particularly the excretion of catecholamines and thyroid homones, and whether there are any relationships between hormone levels, and metabolic rates or nitrogen balance (Haider *et al.*, 1975). It is the aim of this study to contribute to the solution of these questions.

Material and Methods

16 subjects were studied. They were divided into two classes (A and B). Class A consisted of 11 patients who were examined immediately after the brain injury. The 5 patients of Class B were studied between two months and three years after the trauma. Table 1 shows all patients, the diagnoses and the schedule of examinations.

The patients of Class A were divided into three groups according to their neurological course. Group 1 consisted of patients with primary brain injury without acute brain stem symptomatology. Group 2 included patients who developed an acute midbrain syndrome in its different phases or bulbar symptomatology. In these patients a remission of the midbrain symptomatology occurred after they had passed through the different phases up to the full stage. Group 3 consisted of patients who at first showed acute midbrain or bulbar symptomatology and then changed to a characteristic transitory stage, which finally led into the typical apallic syndrome.

The patients of Class B were all related to Group 3 as far as the neurological course was concerned. A few of them showed a varying remission towards a defect stage. Several of these patients died at an earlier stage of remission.

24-hour urinary excretion of catecholamines was measured daily during the first three post-traumatic days and then every third day. The blood level of the thyroid hormones was measured weekly. The catecholamine determination was performed on 24-hour urine samples, to which 6-n-hydrochloric acid was added, using a modified technique of the fluorimetric method of Euler and Lishajko (1961). The extraction was performed by adsorption on aluminium-hydroxide and the differentiation between epinephrine and norepinephrine was made by reading at two different wave lengths. The measurements were performed with a Perkin-Elmer spectral fluorimeter; the interference of dopamine was smaller than 2 p.c. The normal values of the laboratory are < 20 μg per 24 hours for epinephrine and < 50 μg per 24 hours for norepinephrine.

For the estimation of thyroid hormones the T_3 test (in vitro test with radioactive L-trijodthyronine, modified by Hamolsky), the T_4 resin-uptake (Abbott) and the effective thyroid ratio (ETR by Buck-Malinkrodt) were made.[1] The normal values are, T_3: 90–110 μg/100 ml, T_4: 5.5–12.5 μg/100 ml, and ETR: 0.87–1.13. Because of the high standard deviations the statistical significances have not been tested.

Nutrition

Attempts were made to start high caloric total parenteral alimentation (TPA) as soon as possible after trauma (Steinbereithner, 1966; Gerstenbrand and Galanti 1972). Total parenteral nutrition was initiated on the day of admission and amounted to a minimum of 3000 calories per day. The main caloric carrier was 33 p.c. dextrose solution with insulin added regularly in order to compensate for the posttraumatic disturbance of glucose utilisation (Haider *et al.* 1974); in addition 500–1000 ml 20 p.c. fat emulsion was also given (Intralipid, containing 20 g soybean-oil per 100 ml) already during the first 24 hours. This procedure has been considered valuable by some workers (Carlson, 1964; Haider and Steinbereithner, 1972), but has not found universal favour (Berg, 1973). With regard to liver function no disturbances are to be expected (Jeejeebhoy *et al.*, 1973, Zumtobel, 1974). However, to our knowledge, with the exception of McCarthy, fat emulsions have not been used immediately following trauma, at least not in patients with brain injury. For replacement of protein a 3.3 p.c. aminoacid solution with addition of alcohol (Aminosol – Laevulose-Ethanol) was used. This way only about 30 g protein per day could be administered, however, there were additional calories of different origin available to prevent breakdown of muscle proteins for gluco-neogenesis. During the further posttraumatic course an increasing amount of nutrients was administered through a naso-gastric tube. In addition, other therapeutic measures for severely injured patients were used, as necessary.

Results

1. Catecholamines

In Table 2 the data of epinephrine and norepinephrine excretion are summarized for all the patients. Fig. 1 demonstrates the course of the mean values and standard deviations for epinephrine excretion. The curve shows an initial increase of epinephrine excretion of 30 μg per 24 hours followed by a decline towards normal over the first 3 days (15 μg per 24 hours). Within the following two weeks, however, daily epinephrine excretion rises again up to double of the normal values. Within the fourth and fifth week there is an increase of the means up to 70 μg per 24 hours. Mean values of class B cases who were observed later in their posttraumatic course, show that epinephrine excretion has returned to normal within six months after the trauma.

[1] The determinations were kindly performed by the Radionuclear Laboratory of the Ist Internal Clinic.

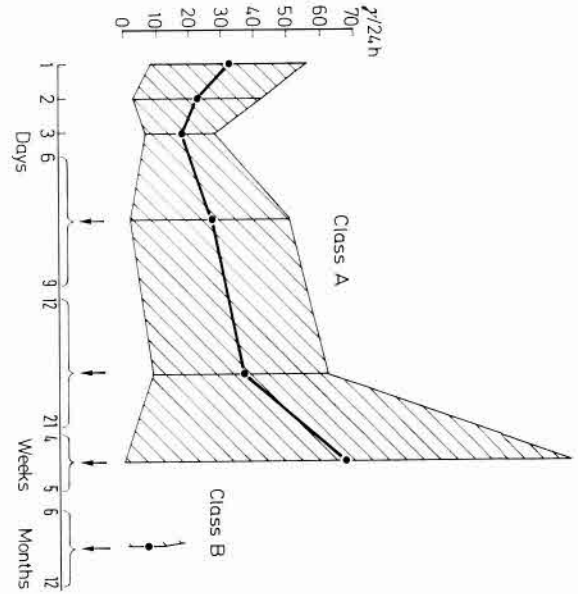

Fig. 1. Course of urinary epinephrine excretion throughout 5 weeks (class A) respectively the second term (class B). Contractions of daily, weekly and monthly levels are expressed as mean values

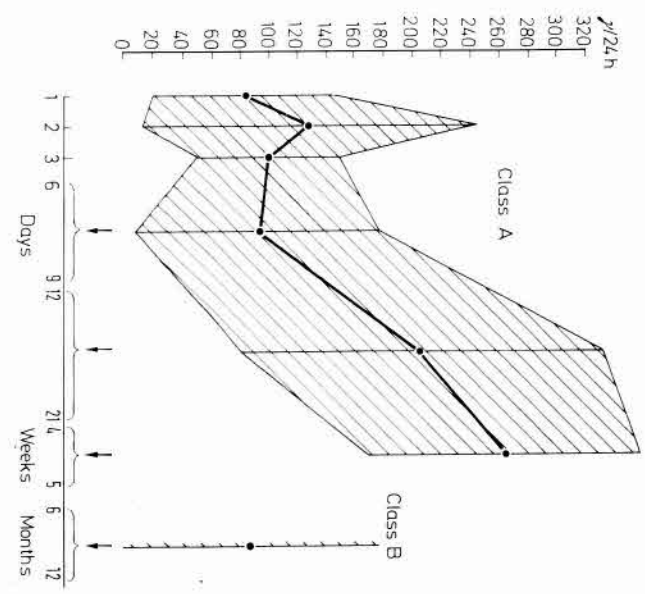

Fig. 2. Course of urinary norepinephrine excretion. Construction like figure 1

Table 2. Urinary Excretion of Epinephrine (E) and Norepinephrine (N) (γ per 24 Hrs)

Class A	1st Day		2nd Day		3rd Day		6th Day		9th Day		12th Day		15th Day		18th Day		21st Day		Later on
	E	N	E	N	E	N	E	N	E	N	E	N	E	N	E	N	E	N	
I Fo. G.	24,6	87,4	68,9	405,6	30,4	185,8	84,6	335,0			80,9	252,7	28,6	111,4	36,8	152,1			
II Bö. H.	2,3	52,3	12,2	112,3	12,8	122,3		110,0	34,2	32,2									
Ha. R.	31,7	97,2	9,5	34,2	1,7	37,8													
Schu. G.	49,1	81,8	12,4	61,3	32,7	96,7													
Sd. S.	21,0	250,0	36,9	216,2	8,3	49,5													
Au. E.			15,1	98,8	21,2	157,5													
Schn. F.		26,0				56,2		64,4		30,7		91,8							
III R. R.	72,8	69,9	24,2	83,6	26,9	39,1	4,1	35,3	34,8	69,1	23,0	80,4							
Wo. P.	56,3	81,8	8,3	67,9	25,7	138,4	11,1	78,2	22,1	123,4	15,0	264,7	13,2	237,3	80,9	402,2	64,5	315,0	
Mü. E.	8,1	43,8	22,7	72,9	17,3	105,2	29,2	117,1											N 377, 193, 212, 189, 354 E 185, 46, 69, 36, 6
Ti. K.	63,5	47,3					2,0	35,3			65,4	24,5							

Class B	E	N
III Pr. S.	15,3	54,1
Fr. K.	3,0	32,8
Zm. St.	2,7	29,2
Ba. E.	6,9	78,7
Ca. P.		235,0

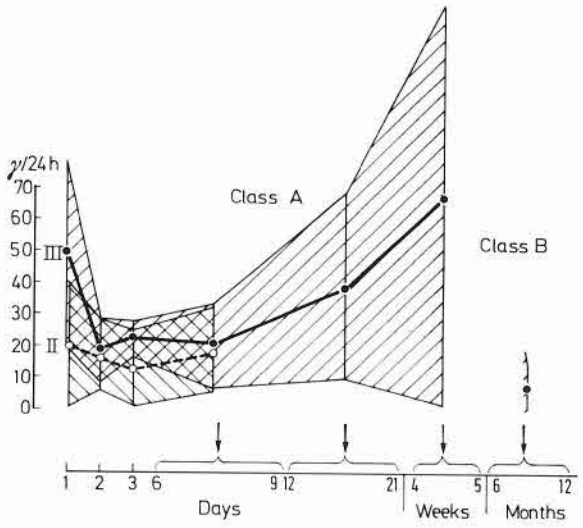

Fig. 3. Course of mean excretion of epinephrine separated into group II and III. Group II: Midbrain syndrome resulting in recovery of defect stage respectively. Group III: Midbrain syndrome terminating in an apallic syndrome. Construction like figure 1

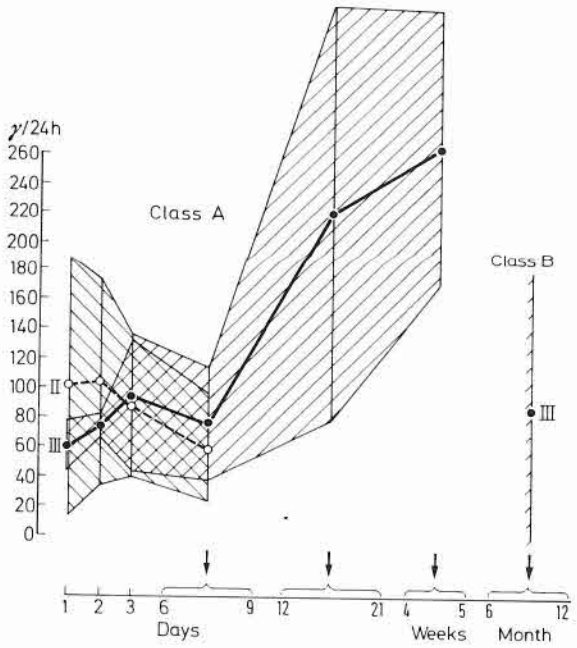

Fig. 4. Course of norepinephrine excretion separated into group II and III (see figure 3)

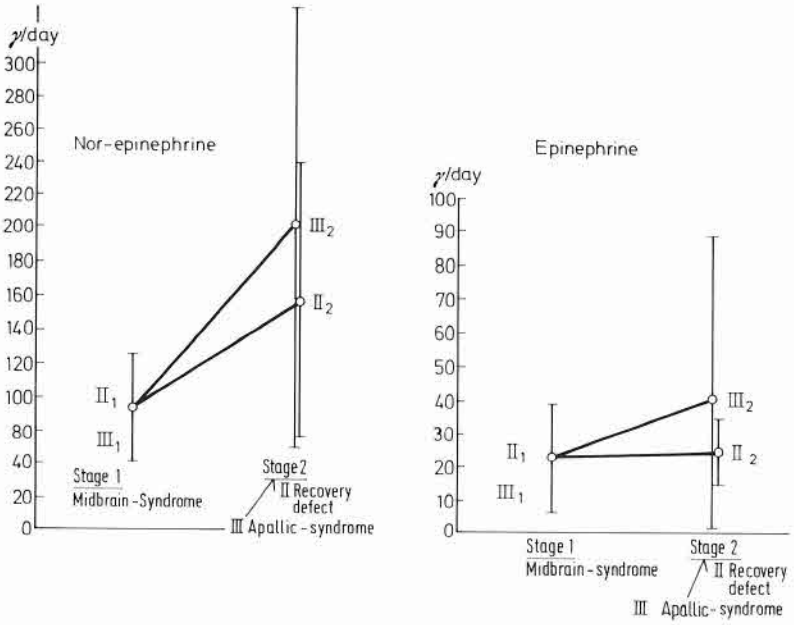

Fig. 5. Course of urinary epinephrine and norepinephrine excretion in a time-independent fashion according to neurological stages. II_1 MBS resulting later in recovery or defect stage (II_2). III_1 MBS later leading to an apallic syndrome (III_2)

Table 3. Thyroid Hormone Blood Levels

Class A 11 Patients	1st Week		2nd Week		3rd Week		4th Week	
	T_3	T_4	T_3	T_4	T_3	T_4	T_3	T_4
Ru. R.	–	–	84	13,6	†	–	–	–
Au. E.	97	10,3	94	7,0	–	–	–	–
Ha. R.	93	4,8	†	–	–	–	–	–
Sd. S.	106	9,4	–	–	–	–	–	–
Schu. G.	87	7,7	90	7,1	1,05 (Etr)	7,6	94	11,0
Wa. P.	98	7,1	1,1 (ETR)	5,5	93	14,6	–	–
Fa. G.	91	8,2	0,95 (ETR)	5,0	90	13,3	–	–
Bö. H.	1,24 (ETR)	7,6	83	7,0	–	–	–	–
Schn. F.	103	8,3	93	10,9	92	5,4		–
Mü. E.	107	10,8	–	–	–	–	–	–
Tip. K.	0,89 (ETR)	–	0,88 (ETR)	6,6	†	–	–	–

Normal Range		
	T_3 (Triiodinethyronine)	90–115
	T_4 (Thyroxine)	5,3–13,5
	ETR (Effective Thyroid Ratio)	0,87–1,13

Fig. 2 shows the course of norepinephrine excretion. The initial values on the first and second day of 80–120 µg/24 hrs are markedly elevated above the normal values given in the literature. In the further course there is a slight decline towards the end of the first week but normal values were not reached. Towards the end of the second week and until the fifth week the mean values for the norepinephrine excretion rose to 260 µg per 24 hours. The cases of class B observed later have mean values of about 80 µg per 24 hours.

In Fig. 3 the posttraumatic course of mean values and standard deviations of group 2 (patients who recovered from midbrain syndrome) and group 3 (patients who developed apallic syndrome) are shown separately. The epinephrine excretion in group 2 is somewhat smaller than in group 3, but there is no statistically significant difference between the two groups either in the absolute size of the values or in the tendency of the course. The course after this first week is practically the same as could be shown for all the three groups together (Fig. 1), since no measurements were done in group 2 patients after the first week.

Fig. 4 demonstrates the same comparison for norepinephrine. The discrepancy of the two curves on the first and second day can be attributed to a very high single value in group 2. Until the end of the first week mean values for the patients of group 2 again are somewhat lower that in group 3. They reach at 60 µg per 24 hours the lowest values of all patients studied. The further course from the second week on is again identical with the collective description shown in Fig. 2.

In Fig. 5 the mean values of patients studied immediately after trauma are classified only according to their neurological course and are not related to the time after injury. In the beginning only one mean value was calculated because at that time all patients had a midbrain syndrome. The final mean values for all patients who developed the apallic syndrome were 60 p.c. higher for epinephrine excretion and 30 p.c. for norepinephrine excretion, when compared to the mean values of the patients who recovered from a midbrain syndrome.

2. Thyroid hormones

Table 3 shows the values of thyroid hormone measurements (triiodinethyronine, tyroxine and effective thyroid ratio = ETR) for 11 patients studied in the early posttraumatic phase (class A). All the values were within normal limits.

Discussion

1. Overall Catecholamine Excretion

First the course of catecholamine excretion in the immediate post-traumatic phase will be discussed.
a) Acute Phase (1.–2. day). The epinephrine and norepinephrine excretion of out patients was markedly elevated on the first and second posttraumatic day. Normal excretion by Goodall *et al.* (1957) was stated to be 15.7 µg per 24 hours for epinephrine and 32.3 µg per 24 hours for norepinephrine, which is in the same range as the results of other authors (Starlinger *et al.*, 1969). Generally norepinephrine excretion is about the two to threefold of epinephrine excretion. While emotional stress can increase catecholamine excretion only up to 50 p.c. (Starlinger *et al.*, 1969), in our patients the increase was about double for epinephrine and threefold for norepi-

nephrine. This fact correlates well with similarly increased values of metabolic rates measured in brain injury patients after trauma that were described in an earlier study (Haider *et al.*, 1975). Grashchenkov *et al.* (1965) measured in severe brain injury patients urinary excretion of 18 μg per 24 hours for epinephrine and 50 μg per 24 hours for norepinephrine. These excretions seem to be lower than our values, however, they were related to much lower normal values (4 and 23 μg per 24 hours respectively), so they represent a fourfold increase for epinephrine and a twofold for norepinephrine excretion.

It is attempted to compare our findings with regard to those found in different kinds of general trauma (operations, myocardial infarction, burns). Patients after uncomplicated operations have practically normal catecholamine blood levels (Franksson *et al.*, 1954), only occasionally slightly increased norepinephrine values were found. Complications during and after the operation however (fever, bleeding, shock) were accompanied by a significant increase of epinephrine and norepinephrine excretion. Similar results were shown by Prakash *et al.* (1972) for myocardial infarction, where complications like cardiac failure, arrhythmias, and cardiogenic shock showed a marked increase of catecholamine levels up to threefold when compared to uncomplicated cases.

Burns cause a much greater increase of the epinephrine and norepinephrine excretion during the first two days. Birke measured threefold increases of epinephrine and norepinephrine excretion, and Goodall found three-to sixfold elevations of epinephrine and five-to tenfold increases of norepinephrine excretion (Birke *et al.*, 1958; Goodall *et al.*, 1954).

We found that in the first posttraumatic phase catecholamine excretion in patients with brain injury was not much different from that seen in generally traumatized patients. Furthermore it appeared to correlate to the severity of the trauma and the symptomatology.

Catecholamines probably have a dominating influence on the disposition of calories (Havel *et al.*, 1964) because of their lipolytic and metabolic effects (Spitzer, 1971, Challoner and Steinberg, 1969) so that the catecholamine level can be taken as an indicator of the metabolic situation. It is perhaps surprising that catecholamine excretion in brain trauma is not increased more than in general trauma, that, however, metabolic rates and catabolism (negative nitrogen balance) are markedly increased in brain trauma compared to the figures given in the literature for general trauma. This difference on one hand can be interpreted as an expression of the fact that our patients immediately after trauma received sufficient parenteral nutrition which somehow suppressed the endogenous catecholamine excretion.

This statement would have to be proven by measurements of a group of patients without high caloric nutrition, however, this would not be ethically justified.

On the other hand these findings underline the assumption that cerebral dysfunction (Gerstenband, 1967, Deligne there is a decrease of catecholamine excretion, which is faster and more marked for epinephrine than for norepinephrine, and apparently corresponds to recovery from the acute shock phase. Comparison with generally traumatized and burnt patients shows that in these, the catecholamine excretion returns to normal after the third day (Goodall *et al.*, 1957; Birke *et al.*, 1958).

c) Period after the First Week. In brain injury patients catecholamine excretion in the urine shows a second increase starting in the second week with a peak showing markedly evaluated values in the third and fourth week. The epinephrine and norepinephrine excretion increased to five and eight times normal respectively, corresponding with a similar course of metabolic rates. These values from the second to the fourth week, however, represent only the group of patients who became apallic, and generally these were in a poor condition frequently suffering from chronic infections. Again comparison with severe burns is instructive: Birke states that patients with complications, such as infection and hypotension have a second increase in epinephrine and norepinephrine excretion. Goodall reports that in extreme impairment of general condition, particularly in the preterminal phase, catecholamine excretion rises up to 26 fold of normal (Birke *et al.*, 1958; Goodall *et al.*, 1957).

d) Late Phase. The decline of epinephrine and norepinephrine excretion in patients studied six months to one year after the trauma corresponds with a stabilisation of the situation. In this phase also the metabolic rates tend to normal (Haider *et al.*, 1975).

2. Grouped Catecholamine Excretion

From comparison of the different groups of patients (Fig. 3 and 4) it can be seen that epinephrine excretion in the acute phase in group 3 (patients who developed an apallic syndrome following a midbrain syndrome) was twice as high as in group 2 (patients who recovered from midbrain syndrome or went into a defect stage). From this one could conclude that patients who became apallic suffered more severe injuries.

For norepinephrine excretion a discrepant behaviour was found which was, however, caused by an extremely high single value of a patient in the second group and is therefore difficult to explain.

If the patients are classified according to their neurological stage, transition into an apallic syndrome seems to correlate with an increase of epinephrine and norepinephrine excretion. In apallic patients epinephrine excretion was 60 p.c. higher, and norepinephrine excretion 30 p.c. higher, as compared to patients who recovered. Obviously the poor condition of patients with traumatic apallic syndrome is expressed by a prolonged increase of catecholamine levels. It should be mentioned, that the two patients who could be examined over a long period of time were in a poor condition.

Differentiation of the metabolic rates (Haider *et al.*, 1975) according to the neurological stage shows, that transition into an apallic syndrome is not accompanied by an increase of metabolic rates. To explain this increase of catecholamine excretion without a corresponding increase in metabolic rate, one could speculate that in the apallic patient metabolism does not react in a proper way to an increase in the blood catecholamine level; Metabolism of the apallic patient seems to be fixed and not capable of responding with an increase.

3. Thyroid Hormones

Thyroid hormone levels (thyroxine T_4 and triiodinethyronine T_3) in our patients were within normal limits in the first, second and third week after the trauma. In this way it could be excluded that the metabolic changes were due to a preexisting disturbance of thyroid function. In the literature concerning post-traumatic metabolism a similar minor role is attributed to the thyroid gland and Caldwell showed that in rats with burns the metabolic changes could not be prevented by a previous thyroidectomy (Caldwell *et al.*, 1959).

Conclusions

1. Catecholamine excretion is markedly increased in patients with severe cerebral trauma; this increase resembles, in extent and course the increase of severe general trauma and is exceeded in burnt patients. There seems to be a positive correlation between catecholamine excretion and severity of the condition. The excretion of norepinephrine is always nearly three times as high as that of epinephrine.
2. Besides stimulation of metabolism by catecholamines and endogenous stimulation by "relative hunger" in patients with a midbrain syndrome, there seems to be an additional factor of dysregulation at reticulo-thalamic and hypothalamic level, which is capable of further stimulating metabolism, fat oxidation and maintaining catabolism.
3. Patients with a traumatic apallic syndrome in poor general condition experience a second rise of catecholamine excretion, however, they seem to have a fixed metabolism.
4. As the postulated central factor probably cannot be influenced by any therapeutic measure, the early application of high caloric parenteral alimentation should also be capable of inhibiting initial rises of catecholamine excretion since this satisfies the endogenously increased needs of the body.
5. Neither the course nor the neurological symptomatology show any influence upon the secretion of thyroid hormones.

References

Berg, G.: Discussion at the "5. Gemeins. Tagung der Deutschen und Österreichischen Arbeitsgemeinschaft für Internistische Intensivmedizin", Wien, September 1973

Birke, G., Duner, H., Liljedahl, S. O., Pernow, B., Platin, L. O., Troell, L.: Histamine, catecholamines and adrenocortical steroids in burns. Acta chir. Scand. 114, 87 (1958)

Caldwell, F. T., Osterholm, J. L., Siwer, N. D., Moyer, C. A.: Metabolic response to thermal trauma of normal and thyroprivic rats of three environmental temperatures. Ann. Surg. 150, 976 (1959)

Carlson, L. A.: Deposition, mobilisation and utilization of fat. Acta chir. Scand. Suppl. 325, 5 (1964)

Challoner, D. R., Steinberg, D.: Oxidative metabolism of myocardium as influenced by fatty acids and epinephrine. Amer. J. Physiol. 211, 897 (1966)

Cuthbertson, D. P., Tilstone, W. J.: Metabolism during the post-injury period. Adv. clin. Chem. 12, 1 (1969)

Deligne, P.: Le retentissement metabolique du traumatisme cerebral le catabolisme. Ann. Franc. Special 1, 150 (1973)

Euler, U. S., Lishajko, F.: Improved technique for the fluorimetric estimation of catecholamines. Acta physiol. Scand. 51, 348 (1961)

Fossati, P., Cappoen, J. P., Dessaint, J. P., Isnard, C., Durand, Y., Laine, E.: La fonction hypophysocorticotrope du traumatise cranien. Ann. Anesth. Franc. Special 1, 65 (1973)

Franksson, C., Gemzell, C. A., von Euler, U. S.: Cortical and meduallary adrenal activity in surgical and allied conditions. J. clin. Endocrinol. 14, 608 (1954)

Gerstenbrand, F.: Das traumatische apallische Syndrom. Wien, New York: Springer-Verlag 1967

Gerstenbrand, F., Galanti, T.: Die Bedeutung der Ernährung für die Prognose des apallischen Syndroms. In: Parenterale Ernährung, p. 166. Bern, Stuttgart, Wien: Huber 1972

Goodall, Mc. C., Stone, C., Haynes, Jr., B. W.: Urinary output of adrenaline and noradrenaline in severe thermal burns. Surgery 145, 479 (1957)

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 3rd International Congress of Neurological Surgery. Copenhagen, August 23–27. Excerpta Medica Foundation 119, 1965.

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. Intensivmedizin 11, 207 (1974)

Haider, W., Steinbereithner, K.: Das Verhalten der Lipidfraktionen im Plasma während langdauernder parenteraler Fettzufuhr bei Patienten mit schwerem Schädel-Hirn-Trauma (SHT). Hrsg. von G. Hartmann und H. Berger. Bern, Stuttgart, Wien: Huber 1972

Haider, W., Lackner, F., Schlick, W., Benzer, H., Gerstenbrand, F., Irsigler, K., Korn, A., Krystof, G., Mayrhofer, O.: Metabolic ıe changes in the course of severe acute brain damage. Europ. J. Intensive Care Med. 1, 19 (1975)

Havel, R. J., Carlson, L. A., Ekelund, L.-G., Holmgren, A.: Studies on the relation between mobilisation of free fatty acids and energy metabolism in man: effects of norepinephrine and nicotinic acid. Metabolism 13, 1402 (1964)

Jeejeebhoy, K. N., Zohrab, W. J., Langer, B., Phillips, M. J., Kuksis, A., Anderson, G. H.: Total parenteral nutrition at home for 23 months, without complication, and with good rehabilitation. Gastroenterology 65, 811 (1973)

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. Berlin, Göttingen, Heidelberg: Springer-Verlag 1962
McCarthy, G.: Personal Information, 1970
Prakash, R., Parmley, W. W., Horvat, M., Swan, H. J. C.: Serum cortisol, plasma free fatty acids and urinary catecholamines as indicators of complications in acute myocardial infarction. Circulation 45, 736 (1972)
Spitzer, J. J., Spitzer, J. A.: Endogenous control of FFA metabolism. In: Progr. biochem. Pharmacol. Vol. 6, 242. Basel: Karger 1971
Starlinger, H., Hawel, W., Rutenfranz, J.: Untersuchungen zur Frage der Katecholaminausscheidung im Harn als Kriterium für emotionalen Streß unter verschiedenen Umgebungsbedingungen. Int. Z. angew. Physiol. 27, 1 (1969)
Steinbereithner, K.: Spezielle Fragen der künstlichen Ernährung schwer Schädel-Hirn-Verletzter. Fortschritte der parenteralen Ernährung. Symp. d Internat. Soc. of Parenteral Nutrition, p. 96. Lochham bei München: Pallas-Verlag 1966
Sutherland, E. W., Robison, G. A.: The role of cyclic 3-5-AMP in response to catecholamines and other hormones. Pharm. Revue 18, 145 (1966)
Wadström, L. B.: Plasma lipids and surgical trauma. Acta chir. Scand., Suppl. 238 (1959)
Westermann, E.: Drugs affecting the mobilisation of free fatty acids. In: Pathophysiologische und klinische Aspekte des Fettstoffwechsels. Hrsg. G. Schettler, R. Sanwald, S. 38. Stuttgart: Thieme 1966
Zumtobel, V.: Indikationen für eine parenterale Fettgabe in der Chirurgie. Infusionstherapie 1, 531 (1974)

Dr. W. Haider
Inst. of Anaesthesiology
of Vienna University
Spitalgasse 23
A-1090 Vienna IX
Austria