Current Issues of Suicidology

Edited by H.-J. Möller,
A. Schmidtke, and R. Welz

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Measurements of Biogenic Amines and Metabolites in the CSF of Suicide Victims and Nonsuicides

G. KAUERT, T. ZUCKER, T. GILG, and W. EISENMENGER

Introduction

In 1983 we started a study by measuring biogenic amines in postmortem CSF from suicide victims and nonsuicides. What prompted us to do that? The reasons first were of a practical nature: In forensic medicine one of the main purposes is the detection of causes of death. However, in many cases there are no anatomic or morphological signs. Especially for those cases in which a differentiation between suicide and accident or homicide is not possible, other diagnostic criteria must be found. The monoamine hypothesis of the pathogenesis of depressive disorders and particularly the findings of decreased 5-hydroxyindoleacetic acid (5-HIAA) levels in lumbar CSF of depressive suicidal patients (Åsberg et al. 1976) prompted us to assess serotonin (5-HT) and other biogenetic amines in the postmortem CSF to look for quantitative differences in suicides and controls regardless of mental disorders. For detailed reviews about biological factors of suicidal behavior see Åsberg et al. (1986).

Analyzing the amines or neurotransmitters themselves instead of their acidic and neutral metabolites has practical reasons: We have a routine method for detecting biogenic amines simultaneously from biological fluids and tissues (Kauert 1986). Up to now measurements of biogenic amines in postmortem CSF have not been performed.

Methods

Sampling of CSF from the Corpse

The CSF samples were taken during legal autopsies. To obtain cranial CSF, the calvarium was opened, the chiasma opticum carefully cut avoiding injury to the arteriae carotis internae, the brain then slightly lifted, and with a long cannulated syringe CSF aspirated from the subarachnoid space in the area of the cisterna interpeduncularis and downward at the height of the pons (Fig. 1 a, b). We could not perform suboccipital puncture for forensic reasons.

The lumbar puncture was performed by ventral perforation of the intervertebral disk L3/L4 or L4/L5 after removal of the intestinal tract (Fig. 1 c). The volume of CSF ranged from 2 to 6 ml. The samples were kept frozen at $-20\,^{\circ}$ C until analysis.

Analyses

The isolation of biogenic amines from CSF consists of a combined ion exchange and derivatization step. In general, we took 2 ml of CSF and used etilefrine as an internal standard for quantification. The derivatives formed were detected by capillary gas chromatography mass spectrometry using an ammonia chemical ionization mode to get intensive quasi molecular ions for multiple ion detection (Fig. 2).

For more detailed description of the analytical procedure see Kauert 1986.

Casuistics

The causes of death for the controls and the modes chosen for suicide are listed in Table 1 according to the frequency of the number of cases in which we obtained cranial *plus* lumbar or cranial CSF alone.

Table 1. Frequency with which cranial *plus* lumbar CSF or cranial CSF alone was obtained

	No. of cases	
	Cranial	Lumbar
Causes of death in controls		
Cardiac	17	6
Pneumonia	4	1
Suffocation	4	1
Thrust of knife	3	2
Pulmonary embolism	2	–
Hyperglycemia	1	1
Electric shock	1	1
Thorax trauma	1	–
Carbon monoxide	1	1
Bathing accident	1	–
Drowning	1	–
Shooting	1	–
Total	37	13
Methods chosen for suicide		
Hanging atypical	21	10
Hanging typical	1	1
Cutting of wrist arteries	4	–
Shooting	2	–
Falling from height	1	–
Drowning	2	–
Suffocation (plastic bag)	–	1
Drug overdose	7	4
Cyanide intoxication	3	1
Carbon monoxide intoxication	3	4
Parathion intoxication	2	1
Total	46	22

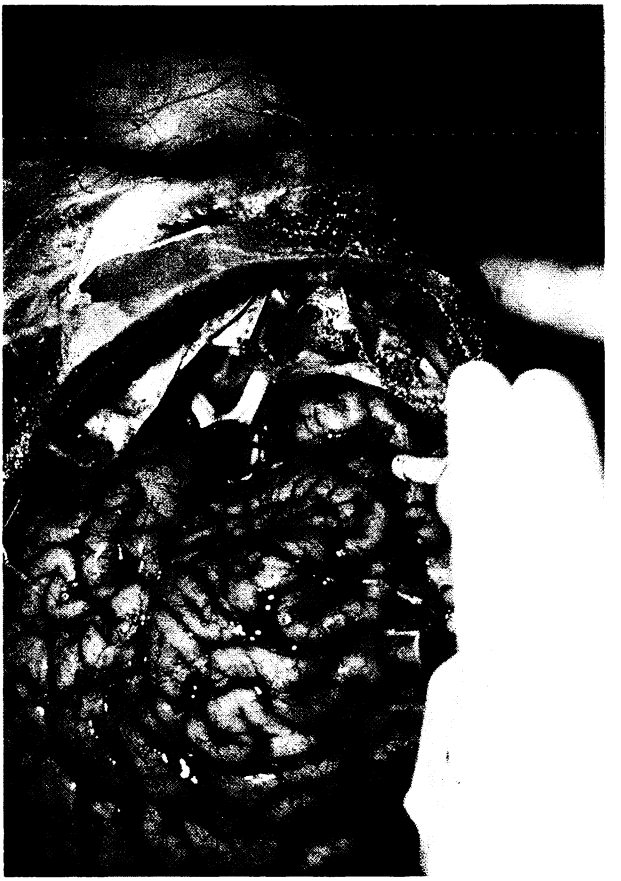

Fig. 1a

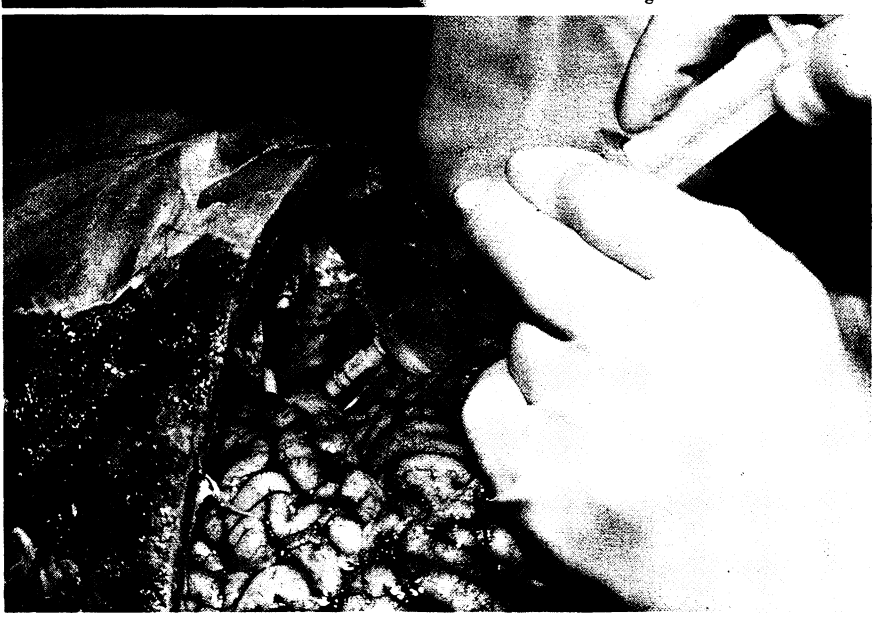

Fig. 1b

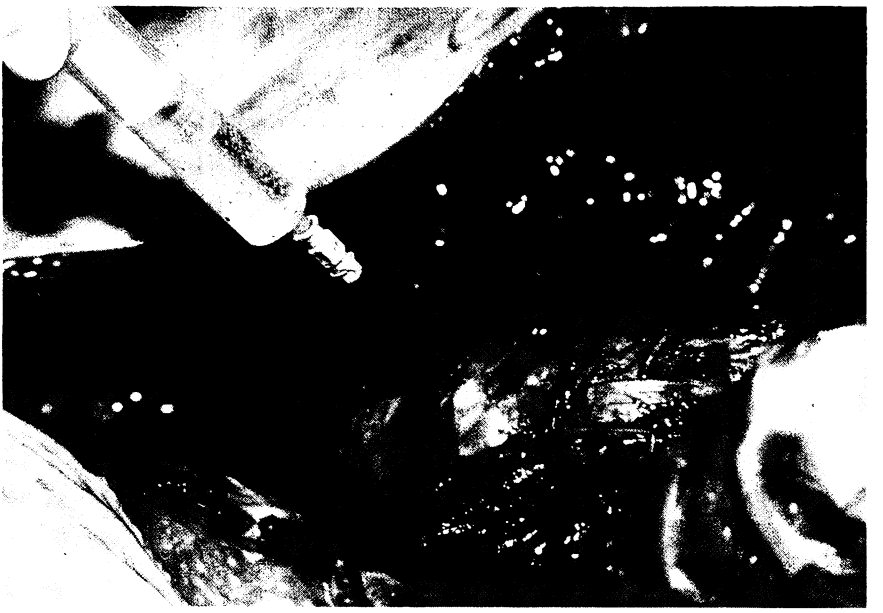

Fig. 1 c

Fig. 1 a – c. Technique of cranial puncture of CSF after opening of the scull (**a, b**) and technique of lumbar puncture of CSF (**c**)

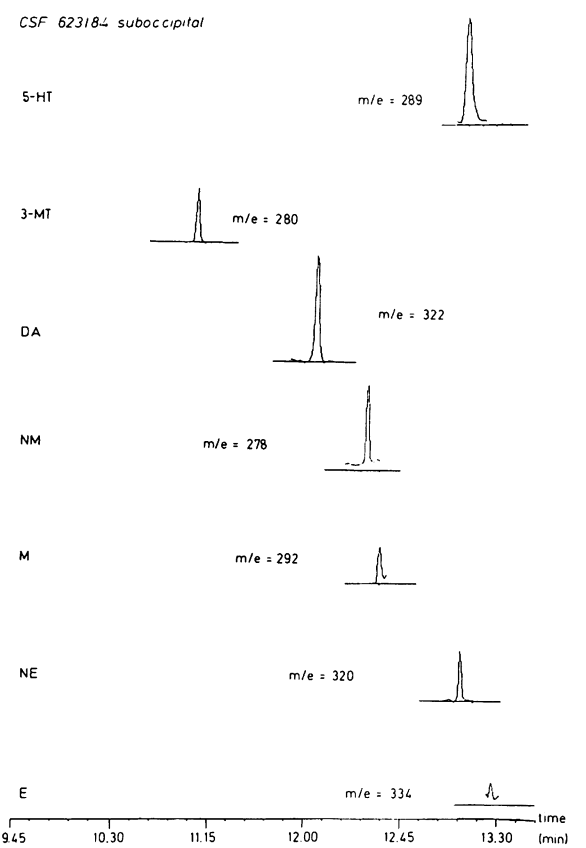

Fig. 2. Mass fragmentogram of biogenic amine derivatives isolated from CSF. *5-HT*, serotonin; *3-MT*, methoxytyramine; *DA*, dopamine; *NM*, normetanephrine; *M*, metanephrine; *NE*, norepinephrine; *E*, epinephrine ▶

Table 2a. Parameters of cases with 5-HT determination in cranial CSF

		Controls	Suicides	*P*
Age	$\bar{x}$/SD	51.9/20.1	51.3/18.9	NS
(yrs)	range	6–85	26–95	
	n	27	46	
Body weight	$\bar{x}$/SD	64.9/15.3	63.2/12.5	NS
(kg)	range	24.0–86.3	43.5–88.8	
	n	36	46	
Body height	$\bar{x}$/SD	168.4/11.4	167.4/9.6	NS
(cm)	range	126–186	145–185	
	n	36	45	
Postmortem delay	$\bar{x}$/SD	44.6/29.6	31.8/19.9	<.05
(h)	range	3–122	5.5–88	
	n	37	45	
Ethanol in blood	$\bar{x}$/SD	0.29/0.61	0.56/0.96	NS
(‰)	range	0.0–1.98	0.0–3.06	
	n	37	45	

Table 2b. Parameters of cases with 5-HT determination in cranial CSF

		Controls	Suicides	*P*
Age	$\bar{x}$/SD	42.9/20.5	46.9/16.6	NS
(yrs)	range	6–85	25–95	
	n	13	22	
Body weight	$\bar{x}$/SD	61.4/16.7	64.3/10.4	NS
(kg)	range	24.0–78.3	44.7–88.8	
	n	12	22	
Body height	$\bar{x}$/SD	167.3/15.0	168.5/7.9	NS
(cm)	range	126–186	145–185	
	n	12	22	
Postmortem delay	$\bar{x}$/SD	45.1/34.7	38.3/26.8	<.05
(h)	range	3–122	5.5–88	
	n	13	22	
Ethanol in blood	$\bar{x}$/SD	0.39/0.67	0.68/0.99	NS
(‰)	range	0.0–1.75	0.0–2.81	
	n	7	9	

In the controls about half of the cases derived from cardiac failures and in the suicides the largest group were due to atypical hanging. Besides other violent methods, we also included cases from drug overdoses as well as intoxications by poisons such as cyanide, carbon monoxide, or organophosphates.

A comparison of the demographic data in suicide victims and controls separated according to cranial and lumbar 5-HT determinations is demonstrated in Table 2a, b.

Results

The most striking findings among the amines investigated showed 5-HT both in cranial and lumbar CSF (Fig. 3). In suicides the 5-HT levels were significantly increased by a mean factor of about 3 for both cranial and lumbar CSF.

The data available from the literature for basal 5-HT levels in lumbar CSF are very inconsistent, obviously because of different analytical methods employed. The most probable level is in a range of 1 ng/ml. The distribution of frequency of 5-HT levels is demonstrated in Fig. 4. In cranial CSF there was a rather broad overlapping of values between suicides and controls, but we found that 50% of the values of the controls showed levels lower than 10 ng/ml and only 15% of the suicide values were within this range.

Moreover, there was no case of suicide below the 5-ng limit. The relative frequency of distribution of 5-HT levels in lumbar CSF is plotted in Fig. 5. Again we found a simular situation compared with the cranial 5-HT levels: about 50% of the control values were below the 5-ng limit, and no case of suicide could be found there.

The other amines which we analyzed, i.e., dopamine, epinephrine, norepinephrine, and the 3-methoxy metabolites of these amines, showed no or no significant differences between the groups.

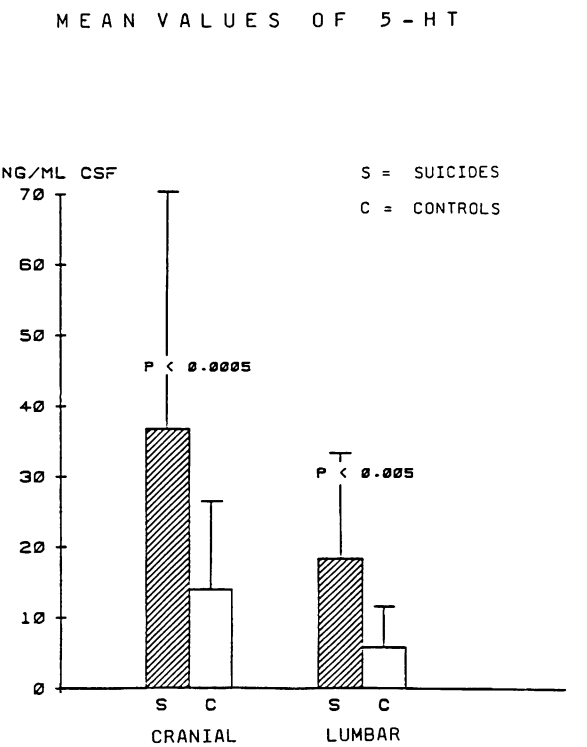

Fig. 3. Serotonin concentrations in cranial and lumbar CSF of suicide victims and controls

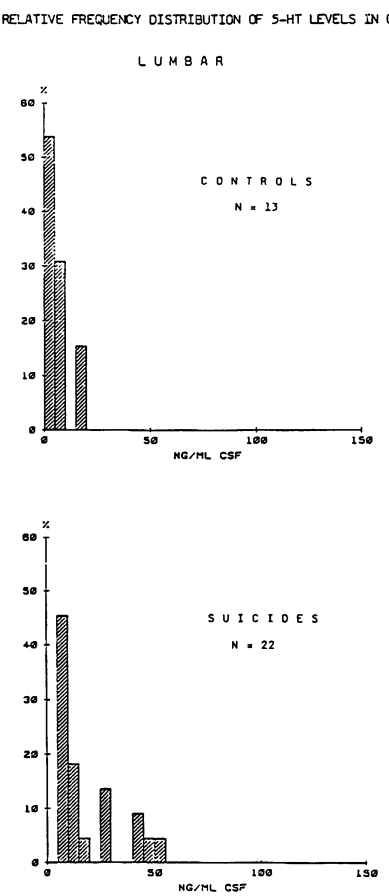

Fig. 4. Distribution of relative frequency of the cranial serotonin concentrations in suicide victims and controls

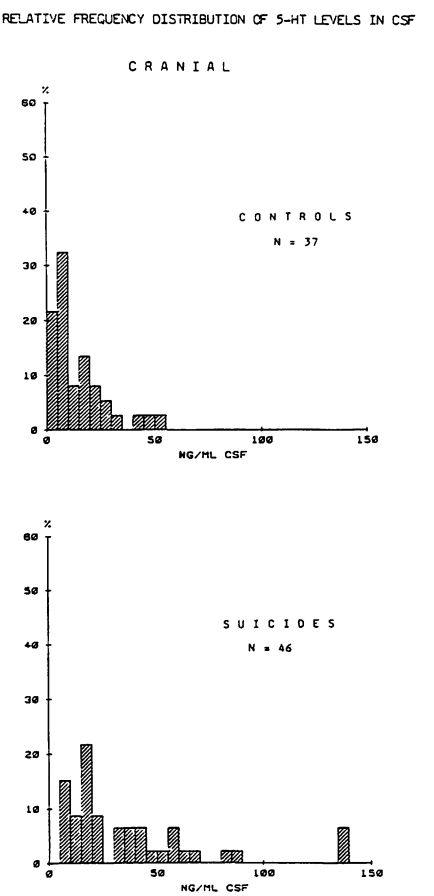

Fig. 5. Distribution of relative frequency of the lumbar serotonin concentrations in suicide victims and controls

Discussion

Postmortem biochemical investigations of mental disorders often are regarded with skepsis by some clinicians because of potential artifacts. In fact, for many endogenous substrates there is evidence for postmortem alterations, e.g., because of sustained enzymatic activities. Table 3 lists some possible factors of influence on CSF of biogenic amines.

The most often discussed and greatest factor of influence is believed to be the postmortem delay, which either may include an enzymatic activity within the pathway or a chemical degradation process of the amines. However, we could not find any correlation between postmortem delay and the concentrations, for instance, of 5-HT and/or norepinephrine (Fig. 6), although we have to make one limitation: we were not able to observe the time immediately after

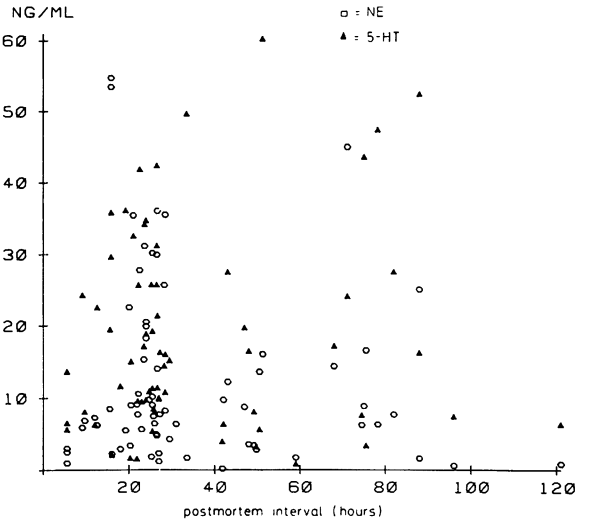

Fig. 6. Postmortem interval (= time elapsed between time of death or discovery of the corpse and autopsy) vs cranial CSF serotonin and norepinephrine concentrations of both suicide victims and controls. *NE*, norepinephrine; *5-HT*, serotonin

Table 3. Theoretical and practical aspects of factors influencing postmortem biogenic amine levels in CSF

1. Postmortem delay	5. Age, sex, height, weight
2. Duration of death agony	6. Chemical stability
3. Handling of the corpse	7. Sampling technique
4. Previous drug treatment	8. Seasonal/diurnal rhythms

death. The duration of agony may influence the amine levels. We were able to find in previous investigations of peripheral postmortem blood that catecholamines are extremely high in prolonged agony (Kauert 1986). If that would be the case, however, we must assume that people committing suicide respond more powerfully to agonal stress than those who died without any intention.

The position of the corpse and its handling before autopsy are potential factors influencing the biogenic amine levels, particularly in regard to the craniocaudal gradient of concentration. One would expect that an equilibration process during transfer of the corpse may occur. On the other hand, from our results we were able to show a marked gradient between cranial and lumbar CSF levels of 5-HT (Fig. 7). Because of the involvement of 5-HT in dark-light-dependent pathways of the pineal gland hormone melatonin, an influence of this biorhythm on CSF 5-HT was thought to exist by Åsberg (personal communication, 1985). Taylor et al. (1985) reported about diurnal rhythms of serotonin in Rhesus monkey CSF rising at the beginning of the dark period and falling to base line at the appearance of light. However, the authors mentioned that there is a marked variation in the amplitude of the serotonin elevations. We therefore examined a possible influence of biorhythms on our results and plotted the levels of cranial and lumbar CSF vs clock time of death so far as this was possible (Fig. 8). Only those cases are illustrated in Fig. 8 for which we

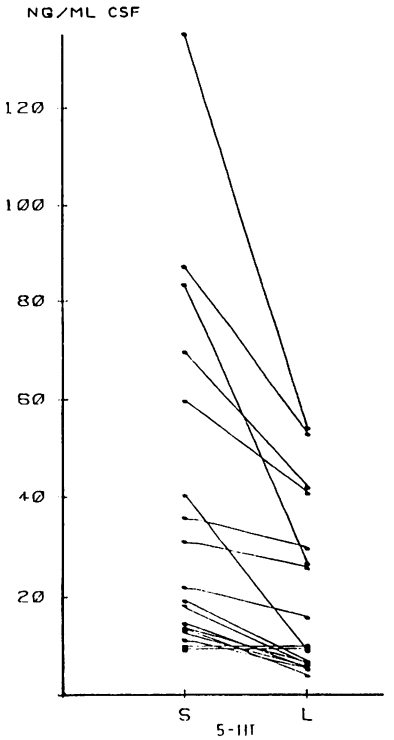

Fig. 7. Postmortem craniocaudal gradient of CSF serotonin concentrations. *S*, Suboccipital; *L*, lumbar CSF

have nearly exact time data, and there were no hints of any correlation between dark-light periods and 5-HT levels, either in cranial or in lumbar CSF. Beyond that, there is no evidence that suicides are preferably committed at night.

On examination of all factors we have discussed, we now have to take into account that all but a previous drug treatment, especially with antidepressives, have to be referred to both suicide victims and controls so that none of these factors gives an explanation for the differences of 5-HT levels in CSF of suicides and controls. The demographic data for both collectives listed in Table 2a, b do not differ with the exception of postmortem delay, which is shorter in suicide cases because autopsies are performed as soon as possible after discovery for criminalistic investigations.

What can we conclude from our studies up to now? First of all, we may suggest that in suicide cases a deficit of 5-HT does not exist *at least* in CSF. In contrast, some authors reported *decreased* postmortem 5-HT levels in specific brain sections (Lloyd et al. 1974; Korpi et al. 1983; Pare et al. 1969). We are presently not able to confirm whether or not these observations are perhaps negatively correlated with our findings. A decreased monoamine oxidase activity, as observed by some authors in suicidal behavior and suicides with previous alcoholism, does not seem to be responsible for our findings when we keep in mind the results of increased 5-HIAA levels in suicides reported by Arato (1986).

It might be that suicide has its own nosologic criteria, which are independent of mental disorders; however, up to now we are not able to give an

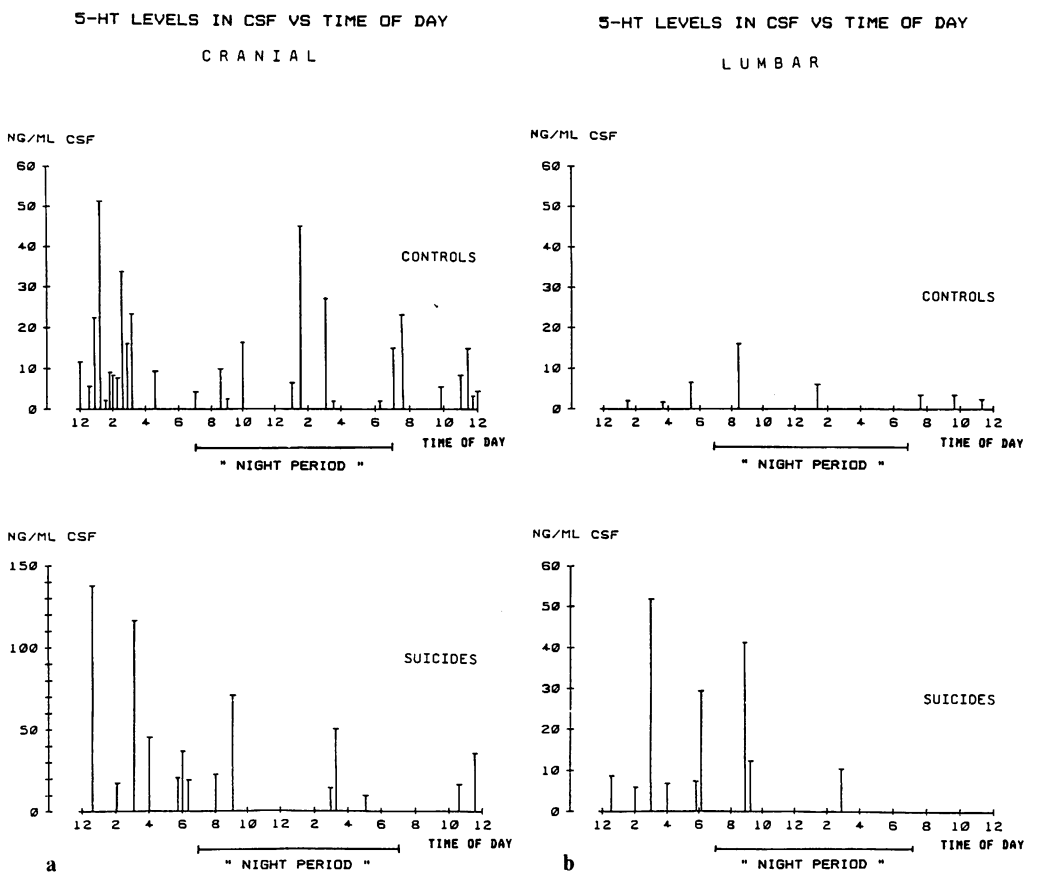

Fig. 8a, b. Distribution of serotonin levels in cranial (**a**) and lumbar (**b**) CSF vs time of day

explanation for our findings with regard to the monoamine hypothesis of depressive disorders. Many potential influences, e.g., drugs and poisons on serotonin turnover, remain to be clarified which is why we are going to enlarge the number of cases to obtain sufficient collectives for statistical work. For forensic purposes as mentioned at the beginning of our paper, we are hopeful of having available a biochemical marker for differentiation between suicide and nonsuicide.

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Subject Index