

Current Issues of Suicidology

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

With 114 Figures and 160 Tables

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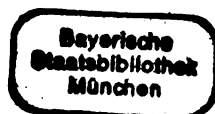
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Contents

Part I Epidemiology and Sociodemographic and Psychiatric Background Variables of Suicidal Behavior

Suicide Trends in 24 European Countries, 1972–1984 S. Platt. With 2 Figures and 9 Tables	3
Frequencies and Trends in Attempted Suicide in the Federal Republic of Germany: A Methodological Study A. Schmidtke, H. Häfner, H.-J. Möller, H. Wedler, and K. Böhme. With 1 Figure and 1 Table	14
Estimation of Suicidal Behavior in Representative Epidemiologic Studies D. Korczak. With 3 Tables	26
Birth Cohort Analysis of Suicide Mortality in Sweden P. Nordström and U. Åsgård. With 4 Figures and 2 Tables	33
Epidemiology of Suicide Events in a Hungarian County K. Ozsváth. With 6 Figures and 2 Tables	38
Psychiatric Aspects of Suicide in Budapest E. Demeter, M. Arató, Z. Rihmer, P. Sótönyi, G. Szuchovsky, and E. Somogyi With 2 Figures and 4 Tables	46
Deliberate Overdosage in a Hospital Catchment Area: Preliminary Results of a 7-Year Study J. G. Harvey and M. S. Christian. With 2 Figures and 2 Tables	50
Self-inflicted Death (1971–1985): Preliminary Results of a 15-Year Survey in an Inner City Area on the Incidence and Methods Employed D. R. Chambers and J. G. Harvey. With 1 Figure and 3 Tables	56
Suicidal Poisonings Caused by Pesticides in Voivodina (Yugoslavia) During 1960–1984 M. Šovljanski and R. Šovljanski. With 2 Tables	62

VIII Contents

Age-Related Differences in Patients Who Attempt Suicide G. Kockott. With 2 Figures and 2 Tables	65
Suicides Among Psychiatric Patients in Funen (Denmark) A. Linder and A. G. Wang. With 4 Figures and 3 Tables	70
Increasing Suicide Rate in Scandinavian Psychiatric Hospitals N. Retterstøl. With 1 Figure and 6 Tables	75
Suicide in Psychiatric Hospitals: Selected Results of a Study on Suicides Committed During Treatment in Five Psychiatric Hospitals in Southern Germany with Special Regard to Therapy Success and Presuicidal Symptoms M. G. Wolfersdorf, R. Vogel, and G. Hole. With 10 Tables	83
Suicides Among Patients Treated in a Ward Specializing in Affective Disorders R. Metzger and M. G. Wolfersdorf. With 1 Figure and 4 Tables	101
Types of Clinical Suicide J. Modestin. With 1 Table	109
Empirical Evaluation Studies Concerning Completed Suicide and Addiction A. Stötzer, W. Poser, and R. Becker. With 7 Tables	114

Part II Course and Prediction of Suicidal Behavior

Catamnestic Studies on Patients Who Attempted Suicide: An Overview H. Wedler. With 2 Tables	121
Sociopsychological Adjustment and Family History of Depressives with and without a Suicide Attempt T. Bronisch and H. Hecht. With 4 Figures and 1 Table	130
Psychopathological Course of Depressives with and without a Suicide Attempt H. Hecht and T. Bronisch. With 4 Figures and 1 Table	137
Preliminary Results of a Study Concerning the Evaluation and Evolution of Patients Having Attempted to Commit Suicide D. Cremniter, A. Meidinger, M. Thénault, and J. Fermanian With 4 Figures and 1 Table	144
A Follow-up Study 1 Year After Attempted Suicide: A Comparison Between Individuals Younger and Older Than 55 Years J. Bäuml, C. Wächtler, A. Pilz, and H. Lauter. With 6 Tables	153

Three-Year Follow-up of 150 Inpatients After a Suicide Attempt by Drug Overdose
 S. Bronisch, C. Bothge, and H.-J. Möller. With 2 Tables . . . 159

Survey and Follow-up of Patients Admitted to Innsbruck University Hospital for Attempted Suicide in 1983
 W. W. Fleischhacker, C. Haring, C. H. Miller, and C. Barnas
 With 3 Figures 164

Changes in Hopelessness and Dissimulation Tendencies in Patients After a Suicide Attempt
 A. Schmidtke and S. Schaller. With 1 Figure and 1 Table . . . 170

Validation of Six Risk Scales for Suicide Attempters
 A. Kurz, H.-J. Möller, A. Torhorst, and H. Lauter
 With 2 Tables 174

A Simple Method to Predict Crises After Suicide Attempts (Parasuicides)
 G. Sonneck and W. Horn. With 2 Tables 179

Estimating Suicide Risk Among Inpatients Treated for Depressive Disorders
 R. Vogel and M. G. Wolfersdorf. With 1 Figure 182

Clinical Prediction of Suicidal Behavior Among High-Risk Suicide Attempters
 M. W. Hengeveld, J. van der Wal, and A. J. F. M. Kerkhof . . . 189

Typology of Persons Who Attempted Suicide with Predictive Value for Repetition: A Prospective Cohort Study
 A. J. F. M. Kerkhof, J. van der Wal, and M. W. Hengeveld
 With 3 Figures and 3 Tables 193

Path Analytical Models for Predicting Suicide Risk
 M. Kuda. With 4 Figures and 1 Table 204

A Computer Program for Exploring Depressive Patients with Suicidal Thoughts
 R. Kalb, I. Fehler, R. Grabisch, and J. Demling
 With 2 Tables 211

Part III Biological Factors of Suicidal Behavior

Biological Correlates of Suicidal Behavior
 M. Åsberg and P. Nordström. With 2 Tables 221

Postmortem Neurochemical Investigation of Suicide
 M. Arató, A. Falus, P. Sótónyi, E. Somogyi, L. Tóthfalusi, K. Magyar, H. Akil, and S. J. Watson
 With 3 Figures and 1 Table 242

Corticotropin-Releasing Factor in Depression and Suicide C. M. Banki, G. Bissette, C. B. Nemeroff, and M. Arató With 2 Tables	247
Measurements of Biogenic Amines and Metabolites in the CSF of Suicide Victims and Nonsuicides G. Kauert, T. Zucker, T. Gilg, and W. Eisenmenger With 8 Figures and 3 Tables	252
Plasma Amino Acids and Suicidal Behavior: An Investigation of Different Groups of Patients Who Attempted to Commit Suicide J. Demling, K. Langer, W. Stein, R. Höll, and R. Kalb With 1 Table	263
Review of Imipramine Binding in Platelets from Psychiatric Patients: Its Relevance to the Biology of Suicide D. Marazziti and G. M. Pacifici. With 1 Table	270

Part IV Psychosocial Factors of Suicidal Behavior

Broken Home and Suicidal Behavior: Methodological Problems S. Schaller and A. Schmidtke. With 1 Table	279
“Broken-Home”-Related Data from Patients Following an Attempted Suicide: Comparison with Data from Psychiatric and Nonpsychiatric Patients. T. Riehl, A. Kurz, A. Torhorst, and H.-J. Möller With 3 Tables	296
Life Events, Current Social Stressors, and Risk of Attempted Suicide R. Welz. With 3 Figures and 3 Tables	301
Recent Life Events and Suicide Attempts J. Tegeler and M. Platzek. With 3 Tables	311
A Description of Behavioral Patterns of Coping with Life Events in Suicidal Patients C. Thomssen and H.-J. Möller. With 2 Figures and 3 Tables	316
Social Support and Suicidal Behavior R. Welz, H. O. Veiel, and H. Häfner. With 3 Tables	322
Description and Prognostic Value of Standardized Procedures for Determining Remarkable Personality Traits in Patients Following a Suicide Attempt T. Dietzfelbinger, A. Kurz, A. Torhorst, and H.-J. Möller With 9 Tables	328

Imitation Effects After Fictional Television Suicides A. Schmidtko and H. Häfner. With 2 Figures	341
Background Factors of Parasuicide in Denmark U. Bille-Brahe. With 6 Figures	349
Suicide and Unemployment in Italy P. Crepet and F. Florenzano. With 5 Tables	356
The British Anomaly: Suicide, Domestic Gas and Unem- ployment in the United Kingdom N. Kreitman. With 5 Figures and 1 Table	364
Individuals and Social Factors in the Psychodynamics of Suicide B. Temesváry. With 8 Figures	372
Suicidal Patients' Comprehension of Significant Others' Attitudes Toward Them D. Wolk-Wasserman. With 1 Table	381
Suicide: Beyond the Regulation of Emotion H. Gutscher. With 2 Figures and 1 Table	390

Part V Treatment Evaluation

Effectiveness of Suicide Prevention: A Short Review of the Literature A. Kurz and H.-J. Möller	399
On the Effect of Crisis Intervention in Suicidal Patients K. Böhme, M. Gast, C. Kulessa, and E. Rau With 1 Figure and 8 Tables	405
Evaluation of Two Different Strategies of an Outpatient Aftercare Program for Suicide Attempters in a General Hospital A. Kurz, H.-J. Möller, F. Bürk, A. Torhorst, C. Wächtler, and H. Lauter. With 2 Figures and 2 Tables	414
Comparing a 3-Month and a 12-Month-Outpatient Aftercare Program for Parasuicide Repeaters A. Torhorst, H.-J. Möller, A. Kurz, W. Schmid-Bode, and H. Lauter. With 3 Figures and 2 Tables	419
Are There Differences Between Patients Who Were Seeking Help in a Suicidal Crisis and Patients Who Were Referred to a Suicide Prevention Center After Having Attempted Suicide? I. Winter, A. Vogl, H. P. Breucha, and H.-J. Möller With 8 Tables	425

XII Contents

Influence of Crisis Intervention Telephone Services (“Crisis Hotlines”) on the Suicide Rate in 25 German Cities T. Riehl, E. Marchner, and H.-J. Möller. With 2 Figures . . .	431
Suicidal Callers on the Telephone Crisis Line in Ljubljana, Yugoslavia O. Tekavčič-Grad and A. Zavasnik. With 9 Figures	437
Effect of Deregistration of Drugs and Prescription Recommendations on the Pattern of Self-Poisonings Ø. Ekeberg, D. Jacobsen, B. Flaaten, and A. Mack With 1 Table	443
Experiences with a Routine-Documentation-System for Parasuicides H. Wedler and F. Klein. With 3 Tables	446
General Structure of Single Interviews Coping with a Suicidal Crisis P. Szabó. With 8 Figures	451
Motivation for Compliance with Outpatient Treatment of Patients Hospitalized After Parasuicide A. Torhorst, F. Bürk, A. Kurz, C. Wächtler, and H.-J. Möller	458
Attitudes Toward Suicide: Development of a Suicide Attitude Questionnaire (SUIATT) R. F. W. Diekstra and A. J. F. M. Kerkhof. With 8 Tables . .	462
Subject Index	477

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XX List of Contributors

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

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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 biogenic 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°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. 1c

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

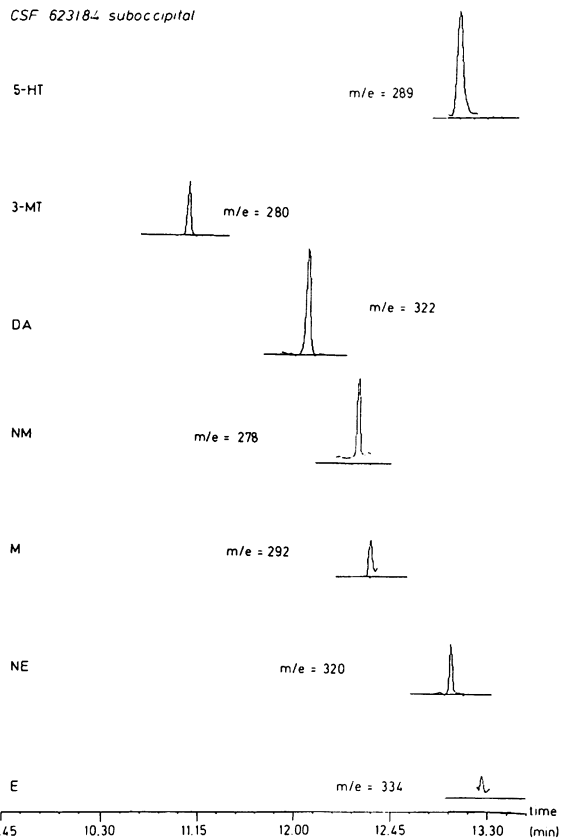


Fig. 2. Mass fragmentogram of biogenic amine derivatives isolated from CSF. 5-HT, serotonin; 3-MT, methoxytyramine; DA, dopamine; NM, normetanephrine; M, metanephrine; NE, norepinephrine; E, epinephrine

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

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

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

		Controls	Suicides	<i>P</i>
Age (yrs)	\bar{x} /SD	42.9/20.5	46.9/16.6	NS
	range	6–85	25–95	
	<i>n</i>	13	22	
Body weight (kg)	\bar{x} /SD	61.4/16.7	64.3/10.4	NS
	range	24.0–78.3	44.7–88.8	
	<i>n</i>	12	22	
Body height (cm)	\bar{x} /SD	167.3/15.0	168.5/7.9	NS
	range	126–186	145–185	
	<i>n</i>	12	22	
Postmortem delay (h)	\bar{x} /SD	45.1/34.7	38.3/26.8	<.05
	range	3–122	5.5–88	
	<i>n</i>	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	
	<i>n</i>	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 similar 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.

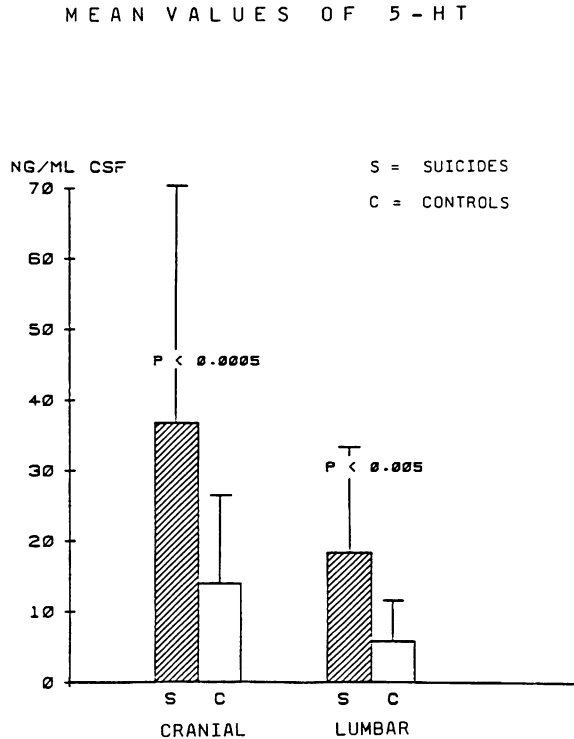
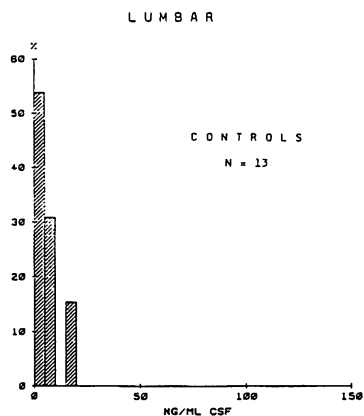


Fig. 3. Serotonin concentrations in cranial and lumbar CSF of suicide victims and controls

RELATIVE FREQUENCY DISTRIBUTION OF 5-HT LEVELS IN CSF



RELATIVE FREQUENCY DISTRIBUTION OF 5-HT LEVELS IN CSF

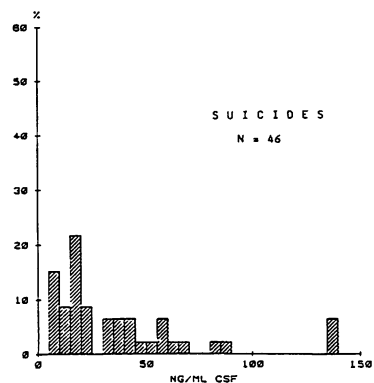
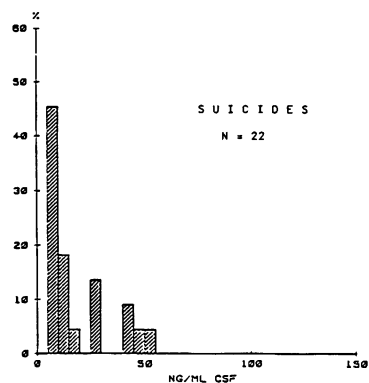
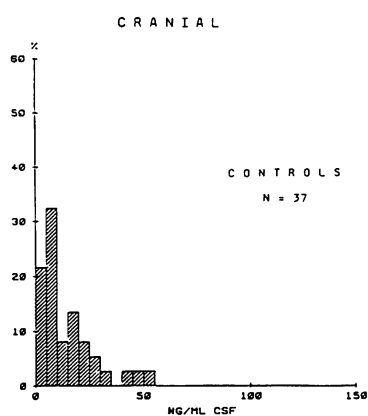


Fig. 4. Distribution of relative frequency of the cranial serotonin concentrations in suicide victims and controls

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 skepticism 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

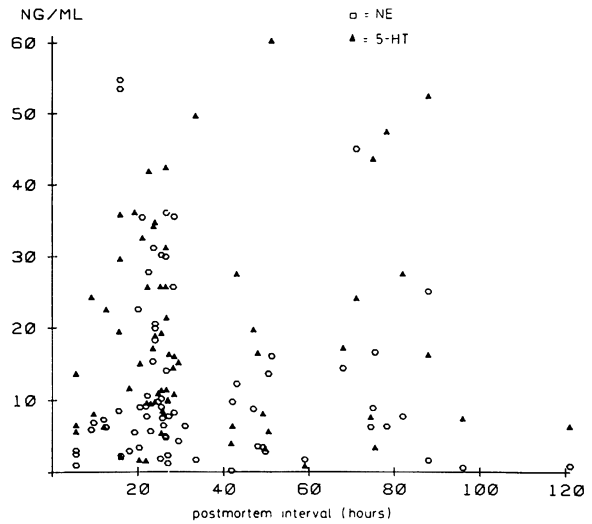


Table 3. Theoretical and practical aspects of factors influencing postmortem biogenic amine levels in CSF

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

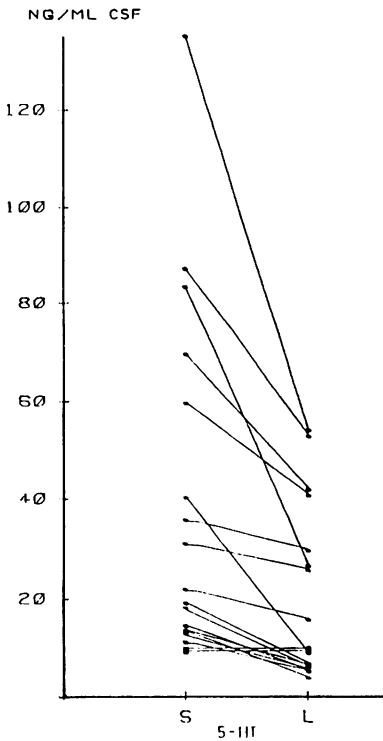


Fig. 7. Postmortem craniocaudal gradient of CSF serotonin concentrations. S, Suboccipital; L, lumbar CSF

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

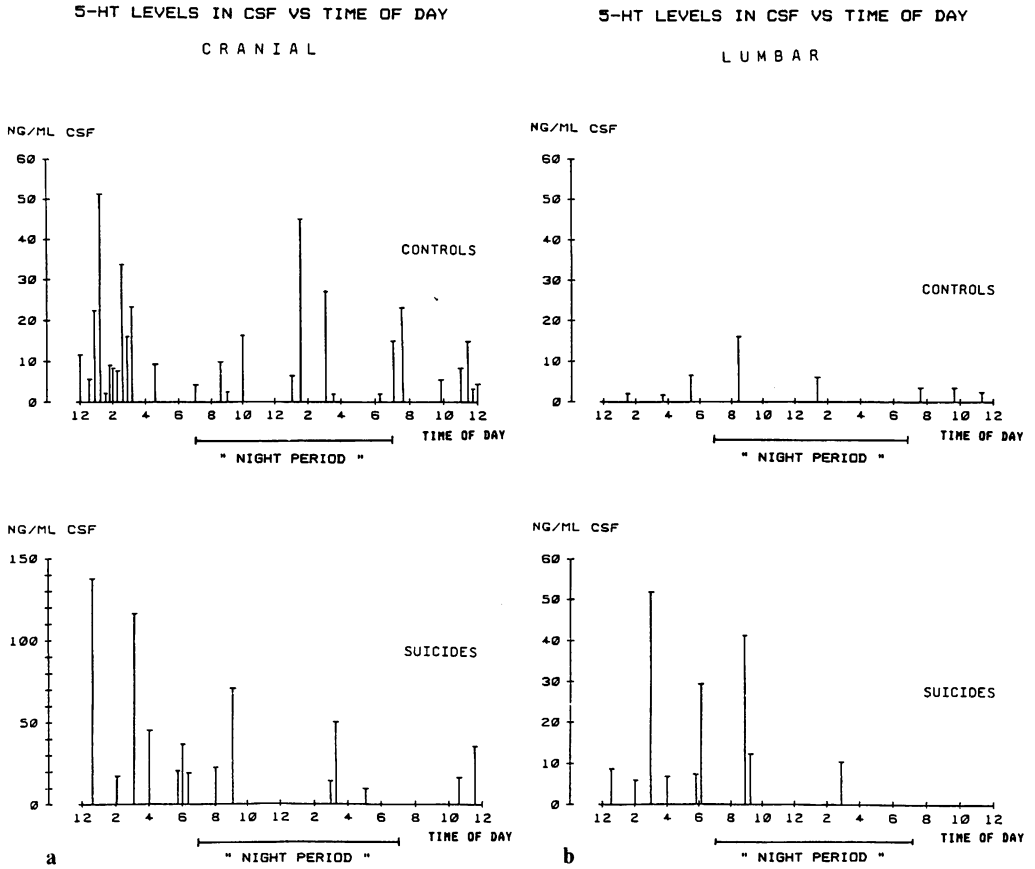


Fig. 8a, b. Distribution of serotonin levels in cranial (a) and lumbar (b) CSF vs time of day

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

- accident 57
- ACTH 243, 244, 247
- addiction 31, 114, 115, 157, 165
- admissions 19, 77
- affective disorder 101, 104
- aftercare 42, 80, 126, 161, 189
- age 41, 47, 51, 65, 91, 102, 104, 105, 110, 153
- age distribution 33
- age-specific rates 8, 12, 17, 23, 153
- aggregate data 28
- aggression 213, 235
- aggressiveness 385
- alcohol 53, 60
- alcohol problems 285
- alcoholics 109ff, 115, 180
- alcoholism 40, 109, 114, 115, 132, 135, 146, 155, 161, 165, 180, 193, 199
- ambivalence 384, 451
- AMS 138
- anomia 354
- anomic theory 46
- anonymity 28
- antisocial behavior 175
- anxiety 31
- arousal, emotional 390
- attempted suicide (see also suicide attempter) 296, 301ff, 311, 316, 322, 373, 381
 - incidence 349
 - risk factors 303, 354
- attitude
 - components 465ff
 - Hungarian 379
 - scale 462
 - to suicide 374ff, 381, 462f
- autopsy studies 233
- availability model 369

- basal ganglia 233
- BDI 189
- behavioral problems 199
- bereavement 53
- biogenic amines 252, 253, 255, 258, 259
- biological correlates 222, 233
- biological (biochemical)
 - marker 261, 268, 270, 272
- birth cohort 33
- BPRS 145
- British anomaly (see also suicide) 364ff
- broken home 31, 132, 165, 185, 205, 290, 296, 298f, 373, 427
 - definition 280f, 296
 - meta analysis 290
 - methodological problems 286f

- carbon-monoxide 366
- case register 16, 27
- case studies 27
- catamnestic studies 121, 407
- catecholamines 259
- children 15, 22
 - adoptive 283
 - emotionally disturbed 282
- citizenship 66
- city districts 28
- class, social 351, 285
- classification problems 14
- clinic suicide 75ff, 83, 109, 116
- cohort analysis 33ff
- cohort effects 23
- common cause hypothesis 115
- community psychiatric service 399
- compliance 126, 149, 161, 415, 418, 446, 458ff
- computer program 211ff
- coping behavior 302f, 319, 322, 390, 451
- coping checklist 317
- cortisol 221, 224, 225, 228, 229, 231, 232, 235, 243, 244, 247, 249, 250, 267
- course of illness 103
- CRF 247ff
- crisis 180
- crisis intervention 121, 127, 405, 419, 431
 - in general hospital 447f
 - CSF 223ff, 231ff, 242ff, 247ff, 252f, 256ff, 263f
- cumulative suicide incidence 33ff

- day patient 71
 death wish 385 f
 delinquency 289
 dependency need 383
 depression 95, 109 ff, 182, 196, 248 ff, 263, 270, 272
 depression subtypes 101
 depressive mood 31
 depressive patients 313 f
 depressive reaction 130, 137
 depressivity 421
 deviant behavior 286
 DIS 138
 disabled 110
 discharge 106
 disintegration 322
 disorientation 139
 dissimulation 170
 diurnal rhythms/biorhythms 259
 divorce 28, 42, 66, 282 ff
 domestic gas
 detoxification 367
 dopamine 255, 257
 drug abuse 390, 409
 drug dependence 165, 193
 drugs 114
 DST (Dexamethasone Suppression Test) 224, 228 ff, 235, 249 f
 duration of illness 111

 early social adjustment 111
 ecological analysis (see also geographic distribution) 399
 ECT 71
 education 42
 effect of treatment 122, 126, 161
 egoism 354
 elderly 28, 65 ff, 153
 endogenous opioids 242
 β -endorphin 242–244
 ENR 160
 environmental reaction 166
 epinephrine 255, 257
 evaluation 435
 expert ratings 27
 expert system 214
 extraversion 135

 family factors 31
 family history 49, 130 ff
 family history of suicide 184
 family, incomplete 280, 290 f
 disharmonious 282, 284
 member 381
 structure 372 f
 family status 66, 91, 102, 104, 110, 131, 198
 family structure 28, 66, 102, 155

 FBS 173, 205
 feelings after attempt 165
 follow-up 123, 149, 159, 166, 201
 foreigners 66
 future perspective 205

 genetic 234
 geographic distribution 399
 Giessen-Test 205
 Global Assessment Scale 418
 grandiose expansiveness 139

 health system 42
 help-seeking behavior 425
 help-seeking strategies 23
 hereditary 341
 heroin 114
 5-HIAA 223 ff, 233 ff, 242 ff, 252, 260, 263, 270
 high-risk groups 125
 history of suicide 184
 homovanillic acid (HVA) 223, 225, 227, 234
 hopelessness 96, 102, 156, 170, 184, 205, 208, 314
 hopelessness scale 172
 hormone 242
 hospital suicide 75 ff, 92
 hostility 137, 139
 4-hydroxy-3-methoxyphenylglycol (HMPG) 223
 5-hydroxytryptophan 225, 232
 hypersensitivity
 serotonin receptors 228
 hypothalamic-pituitary-adrenal axis (HPA axis) 224, 228 ff, 235, 242, 247, 250
 hypothalamus 233

 illegal drugs 115
 imipramine binding sites 222, 225, 233, 236, 270 ff
 imitation 341 ff
 IMPS 138
 impulsiveness 147
 incidence of suicide 3, 6, 11, 26, 38, 46, 51, 64
 inpatient 70, 71, 77, 83, 105, 109, 182
 integration 44, 322, 353
 intent (ionality) 222
 interpersonal relationships 154
 interview 43
 isolation, social 306, 322

 labor market 351
 lethality 170
 lethality risk 15
 life events 111, 121, 144, 155, 165, 167, 175, 185, 187, 195, 301 ff, 311 ff, 316 ff, 421

- depressive patients 311, 313
- mediating factors 316
- physical illness 301
- psychiatric disorder 301
- risk for attempted suicide 301, 303 ff.
311 ff, 318
- lifetime prevalence 29
- living conditions 155, 195
- LNA (large neutral AA) 264 ff
- long-term patients 90
- lumbar puncture 248, 252

- magnesium 228
- male-female ratio 7
- MAO 222, 225, 236, 260, 270
- marital status 102, 111
- mass media 341
 - effects on suicidal behavior 341
- mass suicide 342
- medical history 61
- melatonin 228, 259
- mental disorder
 - incidence 322
- mental illness 46
- meta-analysis 123
- methodological problems 221, 223
- migration tendencies 168
- MMPI 30
- model-suicidal behavior 342 ff
- monoamines, monoamine metabolites 223,
242, 247, 261
- mortality rates 39
- motives 66
- multiple suicide attempts 23

- narcissistic theory 44
- network 80, 133
- neurochemical 242, 245
- neuroendocrine 221, 245
- neurotransmission 221 f, 235
- norepinephrin 255, 257 ff, 270
- noxious substances 53

- obsessive-phobic behavior 139
- occupational problems 155, 161
- official statistics 14, 26
- open wards 70, 79
- outpatient 71, 77
- outpatient aftercare program 414 ff
- outpatient care 425
- outpatient treatment 70
- overdose 50, 159

- parasuicide 15, 121, 153
 - incidence 350 f
 - rates by age 351
 - rates by sex 351 f
- parental conflicts 165, 168, 169
- parental loss 281 f
- partner problems 165
- path analysis 204
- patient status 72 ff
- PDS 138, 159
- perceptual distortion 139
- personality 43, 135, 205
 - correlation between personality
dimensions 333
 - defect 325
 - diagnosis 330
 - profiles 331
 - prognostic value 336
- pesticides 62
- pharmacological treatment 71
- physical health 154, 175
- physical illness
 - incidence 283
- platelet 270, 271
 - binding sites 222, 225
 - MAO activity 222
 - markers 225, 236
- poisoning 62
- police statistics 16, 17, 21, 27
- polytoxicomania 116, 117, 155
- postmortem 242, 244, 252, 258 ff, 268, 272
- PPI 130
- prediction 93, 121, 161, 167, 177, 179, 182,
190, 193, 204
- predictors of suicide 222
- pregnancy 167
- presuicidal syndrome 96, 213, 374
- prevalence of suicide 26, 43
- prevention 121
- previous suicide attempts 51, 103, 104
- problems
 - emotional 283
 - financial 307, 314, 351
 - interpersonal 384
 - legal 314
 - partnership 307, 314
- processual cause hypothesis 114
- prognosis 96, 103, 144, 151, 193, 208
- prophylactic measures 80
- psychiatric diagnoses 66, 72 ff
- psychiatric illness 46, 75, 130, 175
- psychiatric patients 70, 75 ff, 83 ff, 182
- psychiatric symptoms 53, 60, 66, 72, 90,
92, 94, 109, 154, 185
- psychiatric treatment 193
- psychoanalytic theory 212
- psychobiological 221, 234
- psychological tests 182
- psychosomatic disorders 31

- psychotherapy 72
 duration 429
 motivation 459
 public services 165
- questionnaire 28, 30
- railway suicide 343
 raphe nuclei 233
 rating scales
 BPRS 311
 Hamilton Scale for Depression 311
 paranoic-depressive-scale 311
 recent visit to a doctor 60
 receptor binding sites 222, 225, 233
 referral strategies 23
 rehabilitation 80
 relationship, confidential 317 ff, 354
 relatives 81
 repeated suicidal behavior 43, 194, 201
 repeated suicides 123
 repetition of suicidal behavior 458
 repetition rate 123, 126
 risk factor 417
 for attempted suicide 303, 354
 for suicide 354, 362, 405
 risk groups 161, 164, 189, 193, 204
 identification 450
 risk lists 211
 risk scales 174
 Rosenzweig Test 30
 runaway from home 31
 rural districts 38
- SADS 46, 233
 Samaritans 402
 SC-L 138, 147, 159
 schizophrenia 95, 109 ff, 155
 screening methods 29
 seasonal variation 47
 self-destructive behavior 43, 57
 self-harm 15
 self-poisoning 50, 443
 self-reports 20, 29
 serotonin, serotonergic 221 ff, 228, 233,
 235 f, 242, 244 f, 252, 255 ff, 263, 268, 270,
 272
 serotonin-uptake inhibitors 234
 sex 34, 39, 41, 51, 65, 78, 102, 104
 sex/age ratio 56, 59
 sex-specific rates 15, 18, 22
 single 42
 SIS 130
 social class 131
 social desintegration 27
 social desirability 171
 social environment 185
 social integration 130
 social isolation 67
 social learning 463
 social mobility 207
 social network 322 ff, 381, 386
 social problems 193, 215
 social relations 213
 social status 285, 351
 social support 132, 133, 194, 198 ff, 302, 314,
 316, 322 ff, 421
 and attempted suicide 317, 322 ff
 definition 323
 social support-index 317
 sociobiography 205, 209
 sociodemographic 41
 socioecological approach 27
 somatic complaints 103, 104
 statistical trends 15, 18
 stress model 316
 students 30, 204
 suboccipital (cisternal)
 puncture 243, 252
 substances 53
 suicidal behavior
 classification 222
 definition 221
 documentation scale 447
 incidence 2
 prevalence 26
 suicidal callers
 characteristics 438 ff
 problems of 439 f
 suicidal communication 383, 451
 intent 314, 318, 441
 measure of 317
 measure threats 350
 threats 350
 suicidal ideas 207
 suicidal impulses 44
 suicidal intent 101
 suicidal risk scales 30
 suicidal signals 81
 suicidal thoughts 29, 207, 211, 383, 425, 429
 suicide 322, 341, 391
 suicide age specific rates 7
 suicide attempt 14 ff, 23, 26, 29, 41, 103,
 109, 130, 137, 144
 suicide attempt repetition 201
 suicide attempt severeness 161
 suicide attempters
 history 427
 personality profiles 328, 331 ff
 psychiatric diagnosis 297, 382, 410 f
 recidives 408, 412
 suicide/attempt ratio 21, 41, 153
 suicide attempts – trends 14
 Suicide Attitude Scale 462 ff

- suicide definition 222
 suicide epidemic 13
 suicide frequency 3
 suicide history 90, 92, 156, 167, 386
 suicide ideas 31
 suicide impulse 182
 suicide incidence 350
 suicide in the family 185, 374
 suicide indicators 185
 suicide intention 171, 189
 suicide method 58, 90, 94, 369
 suicide mortality 33 ff
 suicide motive 66, 154, 195
 suicide prediction 49, 144, 174, 179, 189, 211
 suicide prevention 79, 81, 103, 388, 399, 405, 421 f, 437
 clinical investigations 400 f
 effectiveness 399
 suicide prevention programs 81
 suicide rate 38, 46, 56, 62, 71, 75, 83
 British anomaly 364 ff
 by sex 6, 8, 9, 358
 by unemployed 358 f, 366
 in European countries 364
 in Hungary 372
 in Italy 358
 in West Germany 431 f
 suicide risk 28, 30, 33 ff, 90, 101–103, 111, 174, 179, 182, 185, 189, 204, 208, 211
 suicide risk factors 354, 362, 405
 suicide risk scales 174
 suicide secular trend 14
 suicide seriousness 14
 suicide sex ratio 7
 suicide statistics 3, 16, 26, 41
 reliability 357
 suicide trends 3

 suicide trends Europe 3
 survey technique 28

 teaching programs 81
 telephone service 431 f, 437 f
 testosterone 235
 therapy 148 ff
 therapy outcome 126
 thyroid-stimulating hormone (TSH) 228
 thyrotropin-releasing hormone (TRH) 228, 233
 time series analysis 18
 trait marker 242
 treatment 72, 89, 179
 treatment effect 94
 treatment methods 79
 treatment prevalence 28
 tryptophan 263 ff
 tryptophan pyrrolase 268
 TV suicide model 342 ff
 typology 193

 unemployment 53, 61, 161, 162, 216, 354, 356, 365 f
 unnatural death 77
 urban areas 38
 utilization strategies 23

 visit to doctor 52
 vulnerability 235 f, 268, 270

 Weintraub criteria 452
 Werther Effect 373
 WHO 3

 youth suicide 282 f, 343 f

 zeitgeist 23