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Ethanol-induced ketoacidosis as a possible neglected cause of sudden death in chronic alcohol consumers

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SUMMARY

Alcohol accounts for a great amount of deaths per year either due to the acute intoxication or due to the secondary impacts of acute or chronic alcoholism. Commonly, in large amount of such fatal cases blood alcohol concentration is low or absent and fatty liver disease is frequently the only pathological finding detected at the autopsy of alcohol consumer. We offer a short case report of a case with the following analysis of why we decided to consider alcoholic ketoacidosis in our differential diagnosis.

Keywords: metabolic disturbance – alcoholic ketoacidosis – chronic alcoholism – sudden death

Etanolom-indukovaná ketoacidóza: potenciálna príčina smrti u chronických alkoholikov

SOUHRN

Súdni lekári sa v každodennej praxi stretávajú s mnohými prípadmi náhlych úmrtí, ktoré musia zhodnotiť z viacerých aspektov, zatiaľ čo ich hlavnou úlohou je stanoviť príčinu smrti. Často sa stáva, že pitevný nález je nešpecifický a aj po vykonaní histologickej diagnostiky a laboratórnych vyšetrení ostáva príčina smrti nejasná. V zahraničí je bežnou rutinou vykonať toxikologické vyšetrenia so skríningom prchavých látok a čo najvyťažujúcejší biochemický rozbor dostupných biologických materiálov. Avšak, z mnohých dôvodov, paušálne vyšetrenie v takomto rozsahu si pracoviská u nás nemôžu dovoliť. Preto lekár priamo pri pitve čelí dôležitému rozhodnutiu: aký materiál odoberie a najmä, aké vyšetrenie navrhne vykonať. Keďže mnohé chorobné stavy sa prejavujú len rozvratom vnútorného prostredia organizmu, morfologický korelát v podobe orgánového nálezu môže pri pitve chýbať. Autori predloženého článku chcú preto pripomenúť, že najmä v prípadoch úmrtí jedincov s anamnézou chronického konzumu alkoholu, treba myslieť na možnosť alkoholickej ketoacidózy. Tento špecifický stav si za účelom konfirmácie vyžaduje vykonať cielené laboratórne vyšetrenia, výsledky ktorých, ak sú „naordinované" správne, poskytnú lekárovi možnú príčinu smrti. Na potvrdenie diagnózy nestačí len stanoviť koncentráciu acetónu, ktorý býva zvýšený aj pri iných typoch ketóz (diabetes mellitus, hladovanie), ale je treba stanoviť koncentráciu betahydroxybutyrátu v krvi, ktorý je prvoradým markerom tohto typu metabolickej dysbalancie.

Kľúčové slová: alkoholická ketoacidóza – chronický alkoholizmus – náhle úmrtia

Soud Lek 2014; 59(4): 48–50

CASE REPORT

A 48-year-old man with a history of chronic alcoholism was found lifeless at his home address, lying in the bed. There was a bucket with vomited remnants found near the bed and an empty bottle of alcalic mineral water. Because of unclear cause of death, medicolegal autopsy was ordered. Both, externally and internally the body did not show any signs of mechanical violence. The only pathological findings at the autopsy comprised very mild generalised atherosclerosis with no stenosis of blood vessels that would be of a hemodynamic importance, configuration of the heart with the weight of 400 g was physiological, with no signs of chronic venostatic changes on the internal organs. The deceased had diffuse alcoholic steatosis of the liver. The brain was mildly swollen. Results of alcoholimetric analysis performed by GC-FID (gas chromatography – flame ionization detection) method confirming the Widmark´s method were absolutely negative for the presence of ethanol in blood or urine. Toxicological analysis did not notice any psychoactive substances or medicaments, besides of positive acetone in blood, level of which was more than 400 mg/l, while acetonuria was also very high. As we are aware of the fact that alcoholic ketoacidosis can be a consequence of methanol or ethyleneglycol poisoning, we performed the analysis by the GC-FID method, which showed negative results. With the help of Department of clinical biochemistry we tried to perform experimental approximate informative measurement of betahydroxybutyric acid (BHBA) in the venous blood and tested it by the glucometer Free Style Optimum commonly used for POCT – point of care testing which is calibrated also for detection of BHBA in the capillary blood (correlation coefficient is 0,98 for the concentrations of ketone bodies between 0,07–5,2 mmol/l and accuracy of the measurement reflects in SD (standard deviation) form 0,03 mmol/l for low measured concentrations up to 0,2 for the high ones) BHBA concentration was increased but still cannot be considered reliable evidence as it is used in clinical practise for diabetic patients and forensic toxicology in our republic has no possibilities for measuring this very important marker of ketosis which seems to be the most important default of forensic toxicology especially in cases of fatal metabolic disturbances

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analysed by forensic experts. We are aware of the fact that GC-MS is the most suitable methodology for estimation the BHBA concentrations in blood but it requires the standards that are not available in any forensic toxicology lab in Slovakia, as this substance is not commonly being analysed. While analysing the case mentioned above, a few problems arose to be discussed, which were as follows:

1. Alcohol-related ketoacidosis as a potential "killer"

Alarming aspects of excessive long-term alcohol consumption and its fatal outcomes reflecting in mortality of our population have been recently reported by several authors (1,2). In forensic assessment, chronic alcohol abuse has wide-ranging implications including alcohol-related multiple organ dysfunction, acute alcohol intoxication, high suicide risk, predisposition to trauma-related deaths or even proneness to criminal activity (3–5). Recent studies provide an increasing amount of evidence that rather than being benign, the alcoholic ketoacidosis (AKA) and/or related metabolic disturbances may be a cause of sudden death in individuals with chronic alcohol dependence (6,7). These kinds of death typically occur in white males older than 50 years of age (8). Autopsy findings are often nonspecific and the only pathological change might be fatty liver disease (9–12).

Severe derangements of potassium and/or magnesium ions together with markedly elevated levels of betahydroxybutyric acid (BHBA) may be involved in some of the unexplained sudden deaths of alcoholic patients (13,14). AKA is specific BHBA dominated type of ketoacidosis related with enhanced glucagone/insulin and NADH/NAD ratios (15). In an extent study of cases of sudden deaths in chronic alcoholics, the statistically significant result has been presented, that measured BHBA levels may be even 10 times higher in these cases, than in alcoholic patients in whom another cause of death was found (16). *Denmark* (9) noted elevated BHBA levels in a few unexplained deaths and hypothesised that AKA may have caused fatal hypoglycaemia which could have lead to death. *Thomsen et al* theorised that the acidosis itself causes metabolic disruption of vital functions leading to death, proposing the term "ketoalcoholic death" (13). Then the authors subsequently reported ketoalcoholic deaths in 7 % of sudden deaths in alcoholic patients in a prospective series (14). Similarly, *Pounder et al* detected very high levels of total ketone bodies, suggesting profound AKA, in 10 % of sudden unexplained deaths in alcoholic patients (17). Up to these results of previously mentioned international studies, it should be concluded that AKA itself may be a cause of sudden death in severe drinkers due to its direct toxic effects on physiological metabolic pathways.

2. The importance of AKA in differential diagnosis

A reduced blood pH, resulting from the production of beta-oxidative ketone bodies (ketoacidosis) as a result of alcoholism (AKA) or diabetes (diabetic ketoacidosis, DKA), may play the main role in many fatalities. However, in cases of abdominal pains and few vomiting periods, blood pH can vary due to interference with partial metabolic alkalosis after vomiting and respiratory alkalosis caused by severe pain and consequent *Kussmaul* breathing pattern (18,19). Analytical evidence can be used as a dominant support of a pathological diagnosis, or it itself may provide a possible cause of death in the absence of other pathologically significant postmortal findings (20). However, results of postmortem biochemical analyses must be interpreted very carefully because of numerous factors affecting their reliability.

AKA must be taken into account in the differential diagnosis of any suddenly deceased alcoholic patient with acidosis, irrespective of the blood glucose concentration (21). Measuring blood glucose levels is not very useful in forensic practice as glucose levels tend to fluctuate in the short post-mortem period because the glycolytic processes remain still active. Therefore, some authors suggest estimation of blood glucose level prior to death by calculating the sum of the glucose and lactate level in the vitreous humor or cerebrospinal fluid which correlates tighter with the blood glucose levels as there are few very metabolically active cell elements in comparison with blood (22). So postmortem hypoglycemia can only be an artificial phenomenon but hyperglycemia in the postmortem specimen always speaks for increased glucose levels in the period before death. Although the association between alcohol ingestion and nondiabetic ketoacidosis has been initially speculated for the first time by Dillon long ago, it has only recently been further documented and studied as its occurrence may not be infrequent in cases of chronic alcoholism (23,24). The fundamental reason of AKA development in chronic alcohol drinking persons is that they undergo a period of binge drinking, associated with a negligible intake of solid food. After the characteristic development of abdominal pain and severe vomiting - due to pancreatitis, gastritis, or acute hepatitis - the person usually stops drinking. So if the drinker dies subsequently, the presentation of ethanol in blood is frequently undetectable in the sample taken *post mortem* during the autopsy (20). The combination of AKA and hypoglycemic coma in non-diabetic persons has not been described in the literature as a clinical entity, but still it is potentially serious consequence of chronic alcohol intake. Although its treatment is simple and effective, this entity may have a fatal outcome and therefore must be thought of in suddenly deceased chronic alcohol consumers (22).

3. Signs which may lead to diagnosis of AKA

Changes in metabolic processes in patients with alcoholic ketoacidosis are unique. Ketone levels are much higher than those found in normal subjects, even after prolonged fasting (24). Physiological levels of ketone bodies in blood ranges up to 30–45 mg/l, equivalent to 0.3–0,5 mmol/l but they can reach variable values depending to the method used (25,26). On the other hand, the hyperglycemia and glycosuria that accompany severe ketoacidosis in diabetes are absent in AKA. In physiological conditios, ketosis is part of the metabolism's response to carbohydrate deprivation and as such is normally subject of strict and immediate metabolic control (24). Sex related variations could arise from hormonal and/or nonhormonal mechanisms. Women tend to develop fasting ketonuria and ketonemia more rapidly and more severely than men (27). Non-hormonal sex differences could also influence the susceptibility to fasting and alcoholic ketoacidosis. The mean postabsorptive free fatty acids (FFA) level in healthy women is significantly higher than in men (27). These sex differences do not appear to be explained on the basis of differences in age, weight or insulin requirement (28,29).

4. Diagnostic procedures of AKA available in forensic practice

The basal problem while testing autopsy blood samples using standard clinical laboratory methods is the postmortal haemolysis. That is why such investigations in forensic practice should be interpreted *cum grano salis* only, as to obtain properly interpretable results. Vitreous ketone levels show good correlation with blood and pericardial fluid levels, suggesting that it could be used as an alternative autopsy specimen for the analysis as it can be obtained easily (30). A gas chromatography-mass spectrometry (GC-MS) is the most suitable method for determination of total blood level of ketone bodies but for the all in one analy-

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sis of β-hydroxybutyrate, acetoacetate and acetone the sample must undergo previous modification. BHBA must be oxidated and together with acetoacetate must then undergo a process of decarboxylation and finally total acetone can be estimated in blood serum by GC-MS and previous concentrations of each ketone body must be back-caculated (30,31). The ketone bodies in the cerebrospinal fluid show the best correlation to blood, while this concentration dependence is mainly due to ketone bodies being utilized by the brain during normal nutritional state. In vitreous humour, the dependence is mainly due to protein bindings of acetoacetate and beta-hydroxybutyrate in blood and the difference in dry matter between blood and vitreous humour (32).

Data from post-mortem blood BHBA, acetone concentrations and vitreous humour glucose concentrations have been collected to determine the markers required to identify and distinguish between AKA, DKA and hyperosmolar hyperglycaemic state (HHS) (17,20,30,32,33). Blood BHBA concentrations higher than 250 mg/l were considered significant, so it was supposed to be the preferred and stable marker of ketoacidosis (33). Cases with significant BHBA detected also had the acetone present, demonstrating that the acetone can be used as an initial marker to identify ketoacidosis, thus the acetone can be used to indicate whether BHBA detection is necessary (31). Vitreous humour glucose concentrations above 6.9 mmol/l are considered high and indicative of hyperglycaemia prior to death. In cases of AKA postmortem glucose level can be normal or lightly decreased. So also the glucose concentrations can be used to distinguish between DKA and ketoacidosis from other causes and to identify deaths due to HHS (34).

Additional laboratory tests measuring hormonal and other ketone levels may be helpful for recognizing between DKA and AKA. Results of clinical studies of treated and therefore surviving alcohol consuming patients with DKA and AKA disclosed that these two pathological entities differ in their metabolic parameters more than in the hormonal profile. The metabolic profile of DKA is characterized by a higher plasma glucose concentration together with a lower BHBA to acetoacetate and lactate to pyruvate ratios compared with patients with AKA, while the initial hormonal profile in both ketoacidotic states is characterized by similarly decreased insulin levels and elevated levels of counterregulatory hormones (34,35).

CONCLUSION

The reason why the authors decided to write this article is an experience from their own practice when the diagnostic process of the autopsy itself was not capable of providing the satisfactory cause of death in healthy young individual. Auxiliary laboratory methods were used to uncover an abnormality in the metabolic status of the deceased, therefore the differential diagnosis of AKA was taken in mind. As proven above, AKA really has irreversible effect on vital functions, if not treated right or on time. Although the human organism did not suffer from any severe disease, it still is very vulnerable to the effects of starving after even a mild alcohol intake. That is why forensic practitioners have to use additive laboratory tests combined with the macro- and microscopic findings and fuse them all to a complete puzzle of before separate elements. As in such cases as this one, estimation of ketone bodies may be the only helpful information to rely on in the diagnostic process responsible for concluding the cause of death in young healthy alcohol consumers.

REFERENCES

1. **Straka Ľ, Žúbor P, Novomeský F, et al.** Fatal alcohol intoxication in women: A forensic autopsy study from Slovakia. *BMC Public Health* 2011; 11: 924.
2. **Kováč P, Bobrov N, Fiala P**. Súdne lekárstvo pre právnikov. 1. vyd. Bratislava. Iura Edition, 2005. 332 s.
3. **Straka Ľ, Novomeský F, Macko V, Krajčovič J.** The ritual suicide of Japanese girl in Slovak Republic. *Leg Med* 2009; 5(S1): 506–507.
4. **Hejna P, Šafr M, Zátopková L, Straka Ľ.** Complex suicide with black powder muzzle loading derringer. *Forensic Sci Med Pathol* 2012; 8(3): 296–300.
5. **Krajčovič J, Janík M, Novomeský F, et al.** *Suicidium consummatum:* alkohol ako rizikový faktor. *Fol Soc Med Leg Slov* 2012; 2(2): 98–101.
6. **McGuire LC, Cruickshank AM, Munro PT.** Alcoholic ketoacidosis. *Emerg Med J* 2006; 23(6): 417–420.
7. **Yanagawa Y, Hirano Y, Takemoto M et al.** Characteristics of Severe Alcoholic Ketoacidosis with a Reversible Visual Disturbance. *J Clinic Toxicol* 2011; S7: 001.
8. **Copeland AR**. Sudden death in the alcoholic. *Forensic Sci Int* 1985; 29: 159–169.
9. **Denmark LN**. The investigation of beta-hydroxybutyrate as a marker for sudden death due to hypoglycaemia in alcoholics. *Forensic Sci Int* 1993; 62(3): 225–32.
10. **Kadiš P, Balažic J, Marolt VF.** Alcoholic ketoacidiosis: a cause of sudden death of chronic alcoholics. *Forensic Sci Int* 1999; 103(1): S53–S59.
11. **Straka Ľ, Štuller F, Novomeský F**. Letálne intoxikácie alkoholom a úmrtia v ťažkom stupni opitosti v regióne severného Slovenska v rokoch 1994–1996 a 2003–2005: súdnolekárska komparatívna štúdia. *Soud Lek* 2008; 53(1): 2–7.
12. **Straka Ľ, Novomeský F, Krajčovič J, Štuller F**: Súdnolekárska alkohológia, Martin, Osveta, 2011: 146.
13. **Thomsen JL, Simonsen KW, Felby S et al.** A prospective toxicology analysis in alcoholics. *Forensic Sci Int*. 1997; 90(1-2): 33-40.
14. **Thomsen JL, Felby S, Theilade P et al.** Alcoholic ketoacidosis as a cause of death in forensic cases. *Forensic Sci Int* 1995; 75(2–3): 163–71.
15. **Sibaï K, Eggimann P**. Alcoholic ketoacidosis: not rare cause of metabolic acidosis. *Rev Med Suisse* 2005; 1(32): 2106, 2108–10, 2112–2115.
16. **Iten PX, Meier M**J. Beta-hydroxybutyric acid--an indicator for an alcoholic ketoacidosis as cause of death in deceased alcohol abusers. *Forensic Sci* 2000; 45(3): 624–632.
17. **Pounder DJ, Stevenson RJ, Taylor KK**. Alcoholic ketoacidosis at autopsy. *J Forensic Sci* 1998; 43(4): 812–816.
18. **Fulop M.** Alcoholic ketoacidosis. *Endocrinol Metab Clin North Am* 1993; 22(2): 209–219.
19. **Caspar CB, Risti B, Iten PX et al.** Alcoholic ketoacidosis. *Schweiz Med Wochenschr* 1993; 123(41): 1929–1934.
20. **Elliott S, Smith C, Cassidy D.** The post-mortem relationship between beta-hydroxybutyrate (BHB), acetone and ethanol in ketoacidosis. *Forensic Sci Int* 2010; 198(1–3): 53–57.
21. **Thompson CJ, Johnston DG, Baylis PH et al.** Alcoholic ketoacidosis: an underdiagnosed condition? *British medical journal* 1986; 292: 463–465.
22. **Platia EV, Hsu TH.** Hypoglycemic coma with ketoacidosis in nondiabetic alcoholics. *West J Med* 1979; 131: 270–276.
23. **Cristian Palmiere, Patrice Mangin**. Post-mortem chemistry update part I. *Int J Leg Med* 2012; 126(2): 187–198.
24. **Cooperman MT, Davidoff F, Spark R et al.** Clinical Studies of Alcoholic Ketoacidosis. *Diabetes* 1974; 23(5): 433–439.
25. **Gilles Gary.** How to Read Blood Ketone Test Results. [online] [cit. 12.11.2013] Dostupné na: http://type1diabetes.about.com/od/technologyandequipment/p/How-To-Read-Blood-Ketone-Test-Results.htm
26. The National Academy of Clinical Biochemistry, Laboratory Medicine Practice Guidelines. Guidelines and Recommendations for Laboratory Analysis in the Diagnosis and Management of Diabetes Mellitus. [online] [cit. 25.2.2014] Dostupné na: http://www.aacc.org/members/nacb/LMPG/OnlineGuide/PublishedGuidelines/diabetes_update/Documents/Chapter%208.pdf
27. **Deuel HJ, Gulick M.** Studies on ketosis. I. The sexual variation in starvation ketosis. *J Biol Chem* 1932; 96: 25–34.
28. **Moorhouse JA, Steinberg J, Rosen NJ.** Sex difference in serum free fatty acid levels in di-

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abetic subjects. *J Clin Endocrinol Metab* 1963; 23: 1080–1089.
29. **Komáreková I**, Straka Ľ, Novomeský F, Hejna P. Gender differences in alcohol affection on an individual. *Soud lek* 2013; 58(3): 76–79.
30. **Holm KM, Linnet K, Rasmussen BS et al.** Determination of ketone bodies in blood by headspace gas chromatography-mass spectrometry. *J Anal Toxicol* 2010; 34(9): 549–554.
31. **López-Soriano FJ, Argilés JM.** Simultaneous determination of ketone bodies in biological samples by gas chromatographic headspace analysis. *J Chromatogr Sci* 1985; 23(3): 120–123.
32. **Felby S, Nielsen E, Thomsen JL.** The postmortem distribution of ketone bodies between blood, vitreous humor, spinal fluid, and urine. *Forensic Sci Med Pathol* 2008; 4(2): 100–107.
33. **Hockenhull J, Dhillo W, Andrews R et al.** Investigation of markers to indicate and distinguish death due to alcoholic ketoacidosis, diabetic ketoacidosis and hyperosmolar hyperglycemic state using post-mortem samples. *Forensic Sci Int* 2012; 10;214(1–3): 142–147.
34. **Umpierrez GE, DiGirolamo M, Tuvlin JA et al.** Differences in metabolic and hormonal milieu in diabetic- and alcohol-induced ketoacidosis. *J Crit Care* 2000; 15(2): 52–59.
35. **Mihai B, Lăcătuşu C, Graur M.** Alcoholic ketoacidosis. *Rev Med Chir Soc Med Nat Iasi* 2008; 112(2): 321–326.

ZPRÁVA ZE SJEZDU



Ve dnech 15. – 16. května 2014 se konal již tradiční, 4. společný sjezd soudního lékařství a toxikologie. Tentokrát Praha se ujala pořadatelství Česko-Slovenského sjezdu s mezinárodní účastí, konkrétně všechna čtyři pražská pracoviště soudního lékařství. „Zkušeným" prezidentem sjezdu byl prof. MUDr. Miroslav Hirt, CSc. Praha zvolila za místo konání hotel Duo, který se nachází na severním okraji hlavního města, s dobrou dostupností Pražské integrované dopravy i s možností bezproblémového příjezdu po vlastní ose osobní dopravou.

Záštitu nad sjezdem přijaly tři Lékařské fakulty UK Praha a Fakulta vojenského zdravotnictví Univerzity obrany v Hradci Králové, dále čtyři pražské fakultní nemocnice, kde jsou v Praze dislokovány Ústavy soudního lékařství. Vlastní odborné jednání bylo otevřeno významnými osobnostmi, konkrétně prorektorem Univerzity Karlovy prof. MUDr. Janem Škrhou, DrSc., MBA a přednesenou zdravicí významného hosta, který se bohužel omluvil několik dnů před zahájením sjezdu, ale jejíž text přednesl prof. Hirt. Tou významnou osobností, která již delší dobu projevuje velký zájem o dění na české a slovenské soudně lékařské scéně, byl prezident IALM (International Academy of Legal Medicine) prof. Ferrara. Před vlastním zahájením jednání byla ještě předána významná ocenění osobnostem našich odborných společností. MUDr. Aleně Lysenkové a prim. MUDr. Michalu Beranovi, Ph.D. předali diplom čestného členství naší odborné společnosti její předseda doc. MUDr. František Vorel, CSc. a prezident sjezdu prof. MUDr. Miroslav Hirt, CSc. Medaili společnosti převzal čestný host prof. MUDr. František Novomeský, Ph.D.


Prof. Fryc při závěrečném hodnocení kongresu

Po oficiálním zahájení nastal maraton odborných sdělení, který byl rozdělen v průběhu dvou dnů do celkem 9 bloků. První a poslední bloky byly společné ve velkém kongresovém sále hotelu Duo, dalších 6 bloků proběhlo paralelně ve dvou sousedících sálech, které umožnily účastníkům sjezdu volbu programu podle vlastního uvážení. V průběhu dvou jednacích dnů zaznělo neuvěřitelné množství 55 orálních sdělení. Čtvrteční odpoledne bylo věnováno diskusi v posterovém bloku, kde se živě rokovalo před 40 postery, za velkého zájmu posluchačů i autorů. Díky profesionálně moderované diskusi prim. Zeleným a Ing. Černou bylo možné strávit příjemné pracovní odpoledne, které bylo ozdobeno účastí autorů z 8 zemí, včetně Bulharska, Itálie, Japonska, Maďarska, Spolkové republiky Německo a Turecka. Hladkému průběhu všech bloků ústních sdělení přispěli všichni předsedající i řečníci, kteří dodržením desetiminutového limitu pro referáty umožnili nejen přednesení řady zajímavých poznatků, ale přenesení diskuse z vyhrazeného časového prostoru i do kuloárů. Hlavními tématy našeho společného sjezdu byla náhlá a násilná úmrtí, dopravní nehody, forenzní a klinická toxikologie, vojenská toxikologie, dále forenzní a molekulární biologie. Zahajující přednáškou byla „Virtuální pitva" doc. Hejny, pomyslné otevření okénka do budoucnosti. Tou budoucností můžeme predikovat zájmem řady mladých autorů velmi dobrých odborných sdělení, která byla vhodně doplněna velmi kvalitními referáty již zkušených kolegů.

Jaký byl zájem o 4. Česko-Slovenský sjezd lze dovodit z účasti 230 registrovaných osob, což organizátory mile potěšilo i zavazovalo. Kromě soudních lékařů, toxikologů, genetiků a dalších medicínských pracovníků se sjezdu účastnili i studenti lékařských fakult, dále policisté, státní zástupci, soudci a další. Na tomto místě je vhodné poděkovat i bohaté účasti zástupců z řad odborných firem, bez jejichž podpory by bylo nemyslitelné zorganizovat takovou mimořádnou a dle ohlasu mnohých i odborně hodnotnou a společensky příjemnou akci. Závěrečné a příznivé zhodnocení sjezdu přednesl prof. MUDr. Oldřich Fryc, který vyzvedl nejen vysokou odbornou úroveň všech sdělení, ale i přátelskou a pracovní náladu, která se linula celým průběhem kongresu. Za zdárný průběh sjezdu všem účastníkům děkujeme.

Za organizační výbor
plk. MUDr. Miloš Sokol, Ph.D.