Efficacy and safety of antidotes for acute poisoning by cyanides

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European Centre for Ecotoxicology and Toxicology of Chemicals
2 Avenue E. Van Nieuwenhuyse (Bte 8), B-1160 Brussels, Belgium.

Efficacy and Safety of Antidotes for Acute Poisoning by Cyanides

CONTENTS

1. SUMMARY AND CONCLUSIONS

Cyanide poisoning is a very serious, albeit rare, event with possible neurological sequelae that may result in severe disability, and death. It can occur after exposure by ingestion, inhalation and dermal absorption of hydrocyanic acid, cyanide salts, or cyanogenic compounds such as acetone cyanohydrin, nitriles, biological substances like cassava or amygdalin (Vitamin B17), or sodium nitroprusside (SNP) (used as a medication for hypertension). Fire smoke inhalation may also cause cyanide poisoning under certain conditions of combustion.

Despite the 1993 overview on "Antidotes of Poisoning by Cyanide" (IPCS/CEC Evaluation of Antidotes Series) and the review of commonly used cyanide antidotes in the 2007 ECETOC Report in the JACC (Joint Assessment of Commodity Chemicals) series "Cyanides of Hydrogen, Sodium and Potassium, and Acetone Cyanohydrin" there has been no recognised consensus on the relative efficacy, efficiency, safety, and practicality of the various antidote treatments being used across the world. This situation although undesirable was sustained by the absence of any comprehensive review which was in turn due to the complexity of the clinical picture, since cyanide antidotes are employed in different circumstances of poisoning. In order to address this situation ECETOC established a new Task Force to review the available evidence on the efficacy, efficiency, safety, and practicality of antidote regimes under the different poisoning circumstances in which they were used. The four circumstances considered were direct poisonings with hydrocyanic acid or its salts, poisoning with cyanogenic compounds (above), fire smoke inhalation, and poisoning with initially unknown substances.

The Task Force reviewed the available literature until mid-2010 and collected further cases with a questionnaire from industry and Poison Control Centres. The Poisoning Severity Score (PSS), a well-established score system addressing all organ systems for grading of poisoning severity, was adjusted slightly to meet the specifics of cyanide poisoning (PSSa – 'a' denotes adapted).

The reported cases were then evaluated for antidote use. In the majority of cases single antidotes were given although in some combinations of antidotes were used. Cases were split into treatment groups based on the sequence in which different antidotes were administered, if more than one antidote was given. For each such sequence the efficacy based on the PSSa and the safety of the respective antidote or antidote combinations were described. This sequential view allowed in many cases for a statistical analysis of efficacy which was included in the single antidote assessments together with pharmaceutical data, pharmacokinetic data, safety/side effects, practicality, an overview of reported cases and case series. Single casuistic case reports shortly described in tables were collected in an appendix as summaries to make the antidote chapters more readable. An overall assessment was made for each antidote.

Pre-clinical antidotes were addressed based on data from animal experiments. In particular alpha-ketoglutarate and cobinamide, and to a lesser extent, dihydroxyacetone and pyruvate (pro-drugs) seem to have promising potential as cyanide antidotes.

It was apparent that although all direct acting antidotes (excluding sodium thiosulphate) may reverse cardiac arrest or at least facilitate resuscitation, they cannot prevent neurological sequelae due to hypoxia in all cases.

Sodium thiosulphate appears to act more rapidly than previously thought, but is effective, when administered alone, only in moderate poisoning (PSSa 2). Amyl nitrite, when administered alone, was found to be effective in moderate to severe poisonings (PSSa 2.5). Hydroxocobalamin, when administered alone, was found to be effective in severe poisonings (PSSa 3). The combinations of sodium nitrite and sodium thiosulphate, with or without amyl nitrite (PSSa 3), and of dimethylaminophenol and sodium thiosulphate (PSSa 2) were also found to be at least partially effective. These methaemoglobin forming agents all require combination with sodium thiosulphate to capture cyanide released during physiological methaemoglobin reduction. Dicobalt edetate has rarely been given, so that no comprehensive evaluation was possible. In any case, the intrinsic toxicity of dicobalt edetate significantly reduces its applicability. In regard of its effectiveness and low toxicity sodium thiosulphate can be given after all direct acting antidotes in situations when a delayed formation of cyanides cannot be excluded.

Oxygen, in contrast to the other antidotes discussed, has no effect on cyanide toxicokinetics, and is therefore not regarded as an antidote in its own right. Oxygen was only partially effective in severe poisonings (PSSa 3), and other antidotes were fully effective without oxygen. The administration of normobaric oxygen in combination with other antidotes to cyanide is very effective and possibly at least additive. It is however recommended that in conditions where oxygen is not available, treatment with antidotes to cyanide should not be delayed.

For the different circumstances of cyanide poisoning mechanism of cyanide formation, course, case series, case tables and an overview of antidote uses in the respective conditions were addressed, before recommending (a) specific antidote(s) for the condition. These recommendations could not only deal with efficacy and safety, but had also to account for practicility, e.g. cold storage requirement in a tropical country, or high price in developing countries. These combined aspects prompted recommendations for different situations / circumstances and severities shown in the following table.

Table 1: Antidotes[a] recommended for acute poisoning by cyanides

Circumstance[b]	Severity of poisoning	
	Mild or moderate	**Severe**
Direct		
HCN or its salts	None or STS (HOCO)	AN/SN or DMAP, followed by STS
Cyanogenic compound		
Cassava	None or STS	STS, in very rare most severe cases SN followed by STS
Laetrile/Amygdalin	None or STS	AN/SN/STS or HOCO(/STS) (or DMAP/STS)
Nitriles	None or STS	STS, in very severe cases SN/STS or HOCO(/STS) or DMAP/STS
SNP	STS	STS
Smoke		
CO and cyanide	None or STS	HOCO (followed by STS). Neither AN/SN nor DMAP
Unknown	None or STS	HOCO or AN, followed by STS. If then required SN/STS (or DMAP/STS)
Child	None or STS	HOCO and/or STS
Mass poisoning	None	AN (or DMAP i.m.)
First aider	None	AN

[a] STS, sodium thiosulphate; HOCO, hydroxocobalamin; AN, amyl nitrite; SN, sodium nitrite; DMAP, dimethylaminophenol; i.m., intramuscular (injection).
[b] HCN, hydrogen cyanide; SNP, sodium nitroprusside; CO, carbon monoxide.

The basis of cyanide poisoning treatment, independent of the source, are the usual measures of life support i.e. oxygen, and if required mechanical ventilation and safeguarding of adequate circulation by e.g. catecholamines.

ABBREVIATIONS

-	Not applied, to (in range)
ACH	Acetone cyanohydrin
AN	Amyl nitrite
ATC	Anatomical Therapeutic Chemical Classification System (World Health Organisation)
bw	Bodyweight
CAS	Chemical Abstracts Service (American Chemical Society)
CO	Carbon monoxide
COHb	Carboxyhaemoglobin
Co-EDTA	Dicobalt edetate
d	Day
DMAP	Dimethylaminophenol
FiO_2	Fraction of inspired oxygen
g	Gram
h	Hour(s)
h-	Hecto-
Hb	Haemoglobin
HCN	Hydrogen cyanide
HOCO	Hydroxocobalamin
i.m.	Intramuscular
i.o.	Intraosseous
i.p.	Intraperitoneal
i.v.	Intravenous
ICSC	International Chemical Safety Card (International Labour Organisation)
IPCS	International Programme on Chemical Safety
k-	Kilo-
KCN	Potassium cyanide
l	Litre
µ-	Micro-
m-	Milli-
MetHb	Methaemoglobin
NaCN	Sodium cyanide
NS	Not stated
Ph Eur	European Pharmacopoeia (Council of Europe)
p.o.	Per os (oral)
PSS	Poisoning Severity Score
PSSa	PSSAadapted to cyanide
s	Second
s.c.	Subcutaneous
SN	Sodium nitrite
SNP	Sodium nitroprusside

STS	Sodium thiosulphate
USP	United States Pharmacopeia
WHO	World Health Organisation

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APPENDIX A: QUESTIONNAIRE

A.1 Personal Cases

In order to have information on the occurrence of cyanide and nitrile poisoning a structured questionnaire was mailed to the EAPCCT members and to the industry.

The aim of this survey was to assess the number of poisonings reported to Poison Centres / hospital (intensive care unit) / Industry and to summarise their experience (Part A) and when available to obtain more detailed information on exposure, substances involved, antidotal treatment used and outcome (Part B).

The questionnaire was distributed and responses were received through personal contacts of the TF members and the EAPCCT secretariat, with information from the Cyanide Forum and WHO/IPCS.

The return on questionnaire Part A was limited to 20 respondents from 14 European countries; 2 respondents (from Estonia and the Netherlands) stated that no cyanide poisoning was reported and their reply is not included in the overview below.

Overview of responses to questionnaire

Country	Centre	Cases of poisoning	Number	Period (years)	Antidote(s) used	Reference
Belgium	PCC	Cyanide	15	2004 - 2008	Hydroxocobalamine	Smet, 2009
Belgium	PCC	Nitrile	2	2004 - 2008	-	Smet, 2009
Czech Republic	Hospital	Cyanide	3	1990 - 1996	AN, SN STS	Pelclová, 2009
Denmark	PCC	Cyanide	2	Since 2006	Hydroxocobalamine	Ebbehøj, 2009
Denmark	PCC	Nitrile	0	Since 2006	-	Ebbehøj, 2009
Finland	PCC	Cyanide	5 deaths	1998 - 2001	-	Hoppu, 2009
France	Hospital	Cyanide	12		STS HOCO, Co-EDTA	Baud, 2009
France	Hospital	Nitrile	2		Hydroxocobalamine	Baud, 2009
Germany	Hospital	Cyanide	9	Since 1985	4-DMAP, STS	Zilker, 2007
Germany	Industry	Cyanide	58	Since 1977	4-DMAP, STS, NAC, HOCO AN, SN Oxygen	Steffens, 2008 Rödelsperger, 2009 Küpper, 2009 Busch, 2010

Country	Centre	Cases of poisoning	Number	Period (years)	Antidote(s) used	Reference
Germany	Industry	Nitrile	116	Since 1977	4-DMAP, STS ,NAC Oxygen	Steffens, 2008 Rödelsperger, 2009 Küpper, 2009 Busch, 2010
Germany	PCC (Mainz)	Cyanide	88	1995 - 2009	DMAP, STS HOCO	Sauer, 2009
Germany	PCC (Mainz)	Nitrile	20	1995 - 2009	DMAP,STS, HOCO	Sauer, 2009
Germany	PCC (Mainz)	Cyanogenic glycoside	98	1995 - 2009	DMAP, STS HOCO	Sauer, 2009
Ireland	Hospital	Cyanide	4	Since 2004	-	Casey, 2009
Ireland	Hospital	Nitrile	3	Since 2004	-	Casey, 2009
Italy	PCC (Firenze)	Cyanide	4	Since 2000	SN, STS, HOCO, dimercaprol	Botti, 2009
Norway	Hospital	Cyanide	5 - 10 cases/y	Since 2004	Hydroxocobalamine	Jacobson, 2009
Norway	Hospital	Nitrile	0	Since 2004	-	Jacobson, 2009
Slovak Republic	PCC	Cyanide	1	9 y	DMAP, STS	Ficekova, 2009
Spain	PCC	Cyanide or nitrile, incl 5 cyanogenic glycoside and 4 fire smoke	27	2006 - 2008		Martinez Arrieta, 2009
UK	PCC (NPIS Edinburgh)	Cyanide	3	Since 1987	None	Whyte, 2009
UK	PCC (NPIS Edinburgh)	Nitrile	0	Since 1987		Whyte, 2009

In the global overview 482 cases of cyanide / nitrile / cyanogenic compound poisonings were reported of which 217 cases came with further details and are listed in an extended table in annex.

A wide range of antidotes was used. In spite of the availability of antidotes cyanide poisoning is a severe and uncommon disease and the problem remains in a selection of appropriate antidote based on efficacy and safety depending on the condition of poisoning.

A.2 The questionnaire

EUROPEAN CENTRE FOR ECOTOXICOLOGY AND TOXICOLOGY OF CHEMICALS

To: EAPCCT members

C054 Brussels, 13th December 2009

Dear Colleague,

Questionnaire on cyanide and nitrile poisoning – Further responses?

Following our questionnaire on cyanide and nitrile poisoning, announced on the EAPCCT website last April / May, we have received several replies from national Poison Centres, and from the cyanide and nitrile industries (list of responses enclosed [Note: By August 2010, there were 20 respondents in all, see list attached]). The statistics and information given by respondents so far are very useful for our Task Force in compiling a critical review of the efficacy and safety of cyanides antidotes (terms of reference enclosed [Section 2.1 of report]). A report is expected to be published early in 2011. It will also include a systematic evaluation, using the Poison Severity Score, of more than 300 cases to establish a cyanides toxidrome and assess the efficacy of various antidotes.

For these reasons, it seems prudent to repeat the questionnaire and we are soliciting PCCs and ICUs that have not been able to reply so far. As before, respondents are kindly invited to answer question A below to summarise their experience, and complete the attached form B for each poisoning case with the available information. All replies may be sent directly to the undersigned by e-mail: henk.vrijhof@ecetoc.org. All contributions will be acknowledged in the final publication.

A. Cases of poisoning	**Number**	**Period (years)**	**Antidote(s) used**
Cyanide?			
Nitrile?			

We look forward to receiving a positive reply.

Yours sincerely,

Ir. H. Vrijhof

Chemicals Programme Manager

encl. Form **B**, ToR, TF

B. ECETOC questionnaire on poisoning cases with cyanides

1	**Contact details**				
1.1	Centre:				
1.2	Contact person:				
1.3	Phone:				
1.4	E-mail:				
2	**Patient details**				
2.1	Internal ID number:				
2.2	Date of poisoning:				
2.3	Sex:				
2.4	Age:				
3	**Exposure details:**	**Yes / No**	**Remark**		
3.1	Suspected?				
3.2	Confirmed?				
	How confirmed:				
3.3	Cyanide in blood?				
3.4	Thiocyanate in urine?				
3.5	Cyanide container (e.g. bottle) presented?				
3.6	Smell of bitter almonds?				
3.7	Lactic acidosis?				
	Cyanide toxidrome (abnormal neurological				
3.8	status, respiratory pattern and heart rate)?				
3.9	Other?				
4	**Substance details**	**Yes / No**	**Oral**	**Inhalation**	**Skin**
4.1	HCN?				
4.2	Cyanide salt?				
4.3	Nitrile?				
4.4	Natural e.g. cassava, almond kernels, etc.?				
4.5	Sodium nitroprusside?				
4.6	Smoke?				
4.7	Intoxication mixed with …?				
5	**Poison Severity Score**	**Yes / No**			
5.1	Severe?				
5.2	Moderate?				
5.3	Minor?				
6	**Antidote treatment used**	**Yes / No**	**Dose**	**Time**	**Remark**
6.1	Oxygen 100%?				
6.2	AN?				
6.3	SN?				
6.4	4-DMAP?				
6.5	STS?				
6.6	Hydroxocobalamine?				
6.7	Dicobalt edetate?				
6.8	Other (specify)?				
7	**Outcome**	**Yes / No**	**Remark**		
7.1	Death?				
7.2	Recovery?				
7.3	Other (sequelae)?				

20 Responses received (10-Aug-10)

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Casey P. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 4 cyanide and 3 nitrile cases]. Personal communication. National Poisons Information Centre, Beaumont Hospital, Dublin, Ireland.

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Hoppu K. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 0 cases]. Personal communication. Poison Information Centre, Helsinki University Central Hospital, Helsinki, Finland.

Jacobsen D. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 5-10 cyanide cases per year]. Personal communication. Department of Acute Medicine. Ulleval University Hospital, Oslo, Norway.

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Sauer O. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 88 cyanide and 20 nitrile cases]. Personal communication. Giftinformationszentrum der Länder Rheinland-Pfalz und Hessen, Klinische Toxikologie, Universitätsklinikum, Mainz, Germany.

Smet H. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 17 cases]. Personal communication. Poisons Centre, Hospitaalcentrum van de Basis Koningin Astrid, Brussels, Belgium.

Steffens W. 2008. ECETOC questionnaire on cyanide and nitrile poisoning [response for 7 cases]. Personal communication. Bayer CropScience, Monheim, Germany.

Whyte I. 2009. ECETOC questionnaire on cyanide and nitrile poisoning [response for 3 cyanide cases]. Personal communication. Hunter Area Toxicology Service, Newcastle, UK.

Zilker T. 2007. ECETOC questionnaire on cyanide and nitrile poisoning [response for 8 cases]. Personal communication. Center Klinikum rechts der Isar, Toxikologische Abteilung. Technical University, Munich, Germany.

Zilker T. 2010. ECETOC questionnaire on on cyanide and nitrile poisoning [response for 1 cyanide case]. Personal communication. Klinikum rechts der Isar, Toxikologische Abteilung. Technical University, Munich, Germany.

APPENDIX B: DATABASE OF > 400 CASES

Please see separate spreadsheet issued with this report.

APPENDIX C: CASUISTICS

New actions:
GCS Glasgow coma scale

CONTENTS: **147**

Akintowa and Tunwashe, 1992*

Three persons ingested a cassava-based meal ('Gari') and died shortly after hospital admission. The patients vomited and complained of abdominal pain immediately after the meal. They were unconscious with renal failure and died of cardiopulmonary arrest. The cyanide levels in the blood were 1.15, 0.85 and 1.35 mg/l, respectively and urine averaged 0.54 mg/l. Cassava contains cyanogenic glycosides which slowly release cyanide and this may have been responsible for the death of these patients. There is an urgent need to establish maximum tolerable levels of cyanide in 'Gari' and other cassava food products. *Comment: No antidotes were applied, only symptomatic treatment for food poisoning was given with no effect.*

Baader and Wrbitzby, 2006

A cleaning worker in a railroad tank wagon has contained acrylonitrile. Neither acrylonitrile nor cyanide levels are reported as the paper deals with adduct monitoring in colleagues, rescue personnel and hospital staff, who had no symptoms of poisoning.

Bain and Knowles, 1967

A 61 year old patient employed in a factory dealing with cyanides was seen by the plant physician after a distinct collapse. He was semi-comatose, had seizures, a tachycardia of 120/min and was feeling nauseous and vomiting. Two doses of 150 mg of Co-EDTA (in total 300 mg of the antidote) were administered i.v. within a 10 minute period as his clinical state had aggravated. After the first dose the patient vomiting once more, became coherent and responded to questions, and the convulsions ceased. 2 hours afterwards, the patient had apparently completely recovered. A blood sample taken just before the administration of the antidote revealed a blood cyanide concentration of 5.1 mg/l. *Comment: A case of moderate poisoning, yet high cyanide level in blood.*

Ballesteros et al, 2003

A 1.5-month-old infant under SNP infusion (initial dose 1µg/kg/min, increased to 5.5 µg/kg/min after 12 hours with a total of 36 hours) developed somnolence under the infusion. Convulsions set in 6 hours after termination of the SNP treatment. Cardiorespiratory arrest required intubation and resuscitation. Catecholamines were needed to stabilise circulation. The infant developed signs of hypoxic encephalopathy. Under suspicion of cyanide intoxication, an unspecified dose of HOCO was given i.m. and prompted 'spectacular' improvement of the neurological signs. A mild residual paresis of the right arm and axial hypotension persisted. The cyanide level,

however, measured 8 hours after cessation of the SNP infusion, but before HOCO application, was not elevated. *Comment: This case does not enable any assessment due to insufficient reporting of data.*

Baud et al, 1986*; Bismuth et al, 1987*

A worker tried to repair a pump wearing neither breathing protection nor adequate protective clothing. He rapidly lost consciousness. After arrival in the intensive care unit, he was decontaminated and received i.v. 4 g of HOCO and 8 g of STS combined over 30 minutes. During the infusion, he regained consciousness and recovered. The blood cyanide level was 5.71 mg/l.

Baud et al, 2002*

A retrospective clinical study was made of 11 acute cyanide-poisoned patients. The aim was to test the hypothesis that plasma lactate concentrations could be of confirmatory value in patients with histories consistent with acute pure cyanide poisoning because immediate laboratory confirmation of suspected cyanide poisoning is rarely possible and because clinicians must rapidly decide whether to administer specific antidotes, which may have severe side effects.

All 11 patients were admitted to the intensive care unit from 1988 to 1999; fire victims were excluded. Before antidotal treatment, the median plasma lactate concentration was 168 mg/dl, the median blood cyanide concentration was 4.2 mg/l. Using Spearman's test, there was a significant correlation between plasma lactate and blood cyanide concentrations (r = 0.74, p = 0.017). Before antidotal treatment, plasma lactate concentration correlated positively with anion gap and inversely with systolic blood pressure, spontaneous respiratory rate, and arterial pH. During the course of cyanide poisonings, a plasma lactate concentration of ≥ 72 mg/dl (8 mmol/l) was sensitive (94%) and moderately specific (70%) for a toxic blood cyanide concentration (≥ 1.0 mg/l). The specificity was substantially improved in patients not receiving catecholamines (85%). The immediate and serial measurement of plasma lactate concentrations was considered useful in assessing the severity of cyanide poisoning.

Baud and Borron, 2008*

Case 1: A 42 year old male with smoke inhalation was found in cardiac arrest. He was resuscitated, intubated, mechanically ventilated with pure oxygen, and received colloidal fluids. Spontaneous circulation returned as well as consciousness. His blood pressure was 130/80 and plasma lactate was 12.8 mmol/l. He then received 5 g of HOCO prior to a session of hyperbaric

oxygen (HBTO) and survived. *Comment: In this case, improvement occurred before HOCO application.*

Case 2: A 37 year old male was also found in cardiac arrest after smoke inhalation. Resuscitation, intubation and mechanical ventilation with pure oxygen were installed, after which spontaneous circulation was achieved. Immediately after 5 g of HOCO spontaneous respirations returned but he remained comatose. He survived after administration of hyperbaric oxygen. *Comment: A case of sever poisoning in which symptomatic therapy improved the status, and further improvement was seen after HOCO. However, coma did not improve after HOCO, but did after hyperbaric oxygen.*

Beamer et al, 1983

This case of Laetrile poisoning is described as occurring as a result of a self administered medication error. A patient with heavy cancer had gone to Mexico to obtain Laetrile in the hope to treat his poignancy. As he had missed several days of his medication and took 12 to 18 tablets of Laetrile to catch up. He showed an acute onset of grand mal seizures. The time between intake and onset of symptoms is not stated. At admission, the blood pressure was 140/100 mm Hg and the heart rate 88 beats/min. He was tachypnoeic with a respiration rate of 32/min. The pupils were fixed and dilated, the skin was pink, and the patient was comatose. His arterial blood gas demonstrated a metabolic acidosis with ineffective respiratory compensation (pH: 7.09, PaO2: 135 mm Hg, PaCO2: 9 mm Hg, HCO3: 3 mEq). The Lilly Cyanide Antidote Kit was used. AN was administered for 30 sec/min by inhalation over 3 min. The patient was intubated and ventilated with 100% oxygen. Then he received 0.3 g of SN i.v. followed by 12.5 g of STS. The blood gases did not improve within the next 5 minutes, bicarbonate was given and a second dose of 0.3 g of SN was administered when the MetHb level reached 6.6%. Within 5 minutes, the blood gas improved significantly (pH: 7.21, PaO2: 562 mm Hg, PaCO2: 26 mm Hg, HCO3: 10 mEq). The MetHb level went up to 10.5%. Due to uncontrollable violent seizures, it was necessary to induce a neuromuscular blockade by pancuronium. The patient rapidly recovered full neurological function within 20 hours. *Comment: A case of severe poisoning in which treatment with SN and symptomatic measures were successful.*

Benaissa et al, 1995

Case 1: A female pharmacist (age 27) ingested mercury-oxy-cyanide. She developed chest and stomach pain and lost consciousness soon after. A quarter of an hour later the ambulance staff assessed the following parameters: blood pressure systolic 80 mm Hg, pulse 34/min, GCS 3 points and both pupils dilated and non-reactive to light. The patient was intubated and ventilated and because of cardio-circulatory failure cardiopulmonary resuscitation (CRP) was begun, which

was continued after admission to the ED. During the resuscitation, 4 g of HOCO and 8 g of STS were administered i.v. Half an hour later pulse and blood pressure had stabilised. 1 hour after the antidote application, she developed disseminated intravascular coagulation (DIC) with multiple internal bleedings followed by hypovolemic and haemorrhagic shock with anuria. The specific mercury antidotes dimercaptole and dimercaptosuccinate were given but without success. The woman died 5 hours later. The levels of cyanide and mercury had been determined once during the course with 6.99 mg/l (cyanide) and 0.44 mg/l (mercury). *Comment: A case of severe poisoning with initial cardiac arrest, HOCO prompted cardio-circulatory improvement, yet the patient died of DIC. The anuria probably was due to mercury poisoning.*

Case 2: A 14 year old boy had taken an unknown amount of mercury-cyanide. Approximately 50 minutes after the intoxication he was conscious with tachycardia (heart rate 120/min) and complained about diarrhoea, nausea, vomiting and stomach pain. During the next minutes he became agitated, hypotonic (decrease of the systolic blood pressure from 100 to 80 mm Hg) and more tachycardic (heart rate 140/min). The blood gases showed a pH of 7.33 and a lactate level of 19.6 mmol/l. The treatment consisted in emptying the stomach and administering i.v. fluids, dimercaprol (200 mg i.m. every 6 hours), dimercaptosuccinate (400 mg p.o. every 8 hours) and 5 g of HOCO. His condition improved temporarily. 7 hours later, he became anuric. He was given furosemide and dopamine, without any positive reaction. 5 days later peritoneal dialysis was initiated (creatinine of 1325 µmol/l). The cyanide level was determined 4 times, together with the lactate levels: at admission (0) and after 1, 5 and 11 hours: Cyanides (µmol/l): 217 , 35 , 4.6 and 13 (corresponds to 5.64, 0.95, 0.12 and 0.35 mg/l). He recovered completely and could be discharged after 26 days. *Comment: Apart from the anuria, this was only a case of mild cyanide poisoning, in which HOCO improved the status. The anuria was probably due to mercury poisoning.*

Berlin, 1970

A 17-month-old child drank about 7.5 ml of Drabkin's solution (contains sodium bicarbonate, potassium ferricyanide, and potassium cyanide), containing 50 mg/l of KCN which approximates 1/50th of a lethal dose. The child was rushed to the emergency department and given 0.3 g of SN followed by 12.5 g of STS (adult doses). Gasping resulted, interpreted as continuing cyanide poisoning. A second dose of 150 mg of SN was given with 12.5 g of STS. He vomited, had apnoeic spells and seizures. After treatment with bicarbonate, artificial ventilation, diazepam for seizures, he was noted to have brown blood. 15 mg of methylene blue was given i.v. then exchange transfusion begun. He suffered a cardiac arrest and died. Post-mortem cyanide concentration was 0.10 mg/l (10 µg/dl). *Comment: The MetHb concentration was not reported. However, this was a clear overdose of SN in a toddler that obviously had fatal outcome.*

Berumen, 1983

A 23 year old student saw his dog suddenly collapse and started external cardiac massage and mouth-to-nose ventilation effort. Moments later the dog died and the student felt nauseated, vomited, and lost consciousness. At the hospital an alert medical officer detected a bitter almonds odour on the patient`s breath and administered the accepted treatment for cyanide poisoning, after which he recovered. It turned out that the dog had accidentally swallowed cyanide, and the volatile poison eliminated through the dog's lungs had been inhaled by his master during the mouth-to-nose artificial respiration. *Comment: Probably a case of mild poisoning but no information which antidote was administered.*

Bismuth et al, 1984

Case 1: This patient was found in cardiorespiratory arrest, his cyanide level was later found to be 1.8 mg/l. Resuscitation and application of Co-EDTA and HOCO doses were without success.

Case 2: A patient in coma with cardiorespiratory arrest, convulsions and circulatory failure. Resuscitation and application of Co-EDTA and HOCO could not amend the coma, which persisted until the patient died 6 months later. Cyanide level had been 6.8 mg/l initially.

Case 3: A patient in coma with respiratory arrest, circulatory failure. Resuscitation, Co-EDTA and HOCO were applied. The patient recovered completely, though the initial cyanide level had been 6 mg/l.

Comment: In all 3 cases, it is impossible to assess the effect of HOCO, as Co-EDTA was also administered. In 2 cases, the antidotes could neither reverse cardiac arrest nor improve coma. In the third case, the patient survived without sequelaeindicating that the application of both antidotes was successful.

Bismuth et al, 1983

A retrospective study of 25 cases of cyanide poisoning brought to light the following points: cardiorespiratory arrests are frequent (7/25) and often inaugural; in severe intoxications (7/25), deep metabolic acidosis is the rule, and cyanide poisoning should always be suspected in cases of coma with severe acidosis; mild intoxications are frequently symptomless. Anxiety and agitation should not be considered as evidence of cyanide poisoning; they are merely due to fear in most cases. The present treatment of acute cyanide poisoning relies basically on symptomatic measures including sodium bicarbonate, cardiac massage and, above all, assisted ventilation with 100% oxygen. The author's experience did not support the concept of a lethal cyanide blood

level when patients can rapidly be transferred by a medical team to an intensive care unit. Survival depends more on prompt medical care than on the accessibility to sophisticated antidotes. *Comment:?????*

Boggild et al, 1990

A 22 year old female was found semiconscious at her place of work. On admission to hospital she was conscious. Her friends reported that she had probably taken an unknown poison approximately 8 hours before admission. She resisted gastric lavage. Clinical examination was unremarkable apart from transient sinus tachycardia. She remained drowsy but arousable over the next 18 hours, but then rapidly developed agitation followed by grand mal convulsions and asystolic cardiac arrest. Additionally a profound metabolic acidosis was found and the patient remained hypotensive. She died 30 hours after the supposed ingestion. Later investigation revealed that the patient had taken acetone and acetonitrile. Acetone is known to slow the metabolism of acetonitrile to cyanide. *Comment: A case of severe poisonng but no antidote was given.*

Bonsall, 1984

An accidental combined (skin and inhalation) cyanide intoxication occurred when a tank containing nitriles was washed with water. According to the authors HCN gas was liberated through this action. A male worker entered the tank to inspect it. He was wearing an overall and boots. Within 3 minutes he collapsed, lost consciousness and suffered from respiratory failure. His pupils were dilated. As no one was aware of the possibility of cyanide intoxication and decontamination was not performed, but he was ventilated mouth-to-mouth by a colleague. He arrived in the emergency department about 40 minutes after the incident. There he presented a coma with a positive Babinski's sign[a]. Then he was decontaminated and an unspecified dose of STS injected twice. As the patient convulsed and vomited he was intubated and ventilated. His condition improved over the next hours so that he could be extubated 48 hours after admission. 72 hours after the intoxication he was conscious and orientated. He was discharged 14 days later in complete remission. *Comment: A case of severe poisoning in which STS and supportive treatment were effective.*

[a] big toe goes up when the outside of the sole is stimulated

Borgohain et al, 1995

A 27 year old woman was admitted with stiffness and weakness of all 4 limbs making her more or less bedridden for the past 5 years. These symptoms started a month after she had attempted by ingesting approximately 300 mg of KCN procured from her father's place of work (goldsmith). She developed delayed onset of persistent generalised dystonia. Cranial CT scan showed bilateral putaminal hypodensities which were also seen on MRI scans to be hypotense on T1 and hypertense on T2 weighted images. Multimodality evoked potentials were normal. An improvement was noted with levodopa. No antidote was administered. *Comment: antidote efficacy cannot be judged as treatment was several years after intoxication and in any event no antidote was given.*

Borron, 1996, 2007

Borron reviewed a total of 18 clinical reports of the use of high dose hydroxoycobalamin including case reports cited by Yacoub (1974), Jouglard (1974), Bourneller (1971), Bismuth (1984(, Racle (1976), Motin (1970), Lutler (1971), Danel (cited by Broward 1987), Dougharty (1990), Tassan (1990) and Tartière (1992). HOCO was administered alone in 5 cases, and possible a further 2, or in combination with with EDTA (6), STS (2), EDTA/Nitrile (1) or EDTA/Nitrile/STS (1). Cyanide intoxication was confirmed in 7 cases by blood cyanide determinations. Recovery was complete in 15 cases, with sequalae in 1 (after 7gms of HOCO and STS) chronic coma in a second case and death in a third (both with CN poisoning confirmed and after 4gms of HOCO and EDTA).

Bouachour et al, 1990 (Harry et al, 1985)

A 54 year-old woman ingested 200 mg of NaCN and was found 30 minutes later in deep coma. The patient was intubated and ventilated with 100% oxygen. 1 hour after the incident and upon arrival in hospital, the patient presented a low score (3 points) on the GCS, hypotonia (blood pressure 70/40 mm Hg), hypothermia (33˚C), tachycardia (heart rate 120/min), and high levels of lactic acid (29.5 mmol/l) and blood cyanide (10 mg/l). The patient was given 4 g of HOCO and 8 g of STS i.v. This resulted in a normal peripheral resistance, a normal aortic pressure and an improvement on the GCS (increase to 9 points), while lactate decreased to 7.5 mmol/l. Additional hyperbaric oxygen was initiated. Under this treatment, the patient's neurological status normalised completely (GCS 15). The cyanide level during further hospitalisation dropped to 1.2, 0.3 and 0.25 mg/l after 4, 6 and 20 hours, respectively. (The case was communicated earlier by Harry et al, 1985.) *Comment: A case of severe poisoning in which HOCO and STS applied together were partially successful for the neurological symptoms.*

Bourrelier and Paulet, 1971

During an accident implicating molten NaCN, both exposed persons were treated with two ampoules and more of Co-EDTA. One of the victims, a 50 year old male, was in a coma and received 2 ampoules of Co-EDTA but had regained consciousness by the end of the infusion. He received oxygen and glucose. The other, a 27 years old male, was in a profound coma, apnoeic and in a state of intense shock. He immediately received 100% oxygen after intubation and initially three ampoules of Co-EDTA and then two further ampoules. In addition, he received two doses of 100 mg of HOCO. The cardiovascular state normalised. 1 hour after administration of the five ampoules adverse effects attributed to the Co-EDTA were noted including a phase of tachycardia with hypertension and profuse sweating. 2½ hours later, the gasping had disappeared being replaced with irregular spontaneous respirations which subsequently regularised. He recovered but had some difficulties with speech. *Comment: A case of severe poisoning which indicated that 200 mg of HOCO in an adult represents an inefficacious dose. Co-EDTA was fully effective for the cardiovascular, but only partially for the neurological status.*

Brian, 1990

A case of a 7 year old girl year old that developed weakness, tremor and vomiting one hour after eating cassava was reported from Papua New Guinea. After 4 to 65 hours later she was conscious, but still had tremor and restlessness. She was given 2 ampoules of Co-EDTA without clinical effect/improvement, but consecutive facial oedema. *Comment: A case of mild poisoning in which no cyanide or thiocyanate measurements were performed and Co-EDTA was not effective.*

Brivet, 1983

A 43 year old male analytical chemist attempted suicide by ingesting KCN (unknown quantity). Ten minutes after ingestion he developed a headache and 10 minutes later his blood pressure dropped and he became comatose. The first treatment comprised the application of 100% oxygen and gastric lavage. The patient developed seizures followed by cardiac arrest. He was resuscitated by CPR and administered with catecholamines, sodium bicarbonate and i.v. fluids. This resulted in a blood pressure of 80/50 mm Hg and a heart rate of 130/min. The pH was 6.8 at admission and 7.34, 7.49 and 7.47 after 2, 12 and 18 hours respectively. The lactate level was measured at the beginning and after 12 hours with 20 mmol/l and 1.96 mmol/l respectively. The patient developed a mild rhabdomyolysis probably due to the reanimation procedure. He recovered completely during the next 20 hours. *Comment: No antidote was used. Comment: A case of moderate poisoning in which HOCO reversed the neurological symptoms.*

Brown et al, 1987

A 24 year old man was cleaning a vat containing silver cyanide when he was overcome by fumes and collapsed into several inches of slurry. Upon arrival in the causalty emergency department 15 minutes later he was unresponsive, apnoeic, and hypotensive. His skin was brick red, which is indicative of cyanide toxicity. After resuscitation, administrations of 300 mg of Co-EDTA were given at 1, 2, 3, and 7 minutes. Spontaneous respiration resumed, although he remained haemodynamically unstable. 24 hours after admission urine output and blood pressure were satisfactory. Electroencephalograms obtained at this time and 48 hours after admission showed no cerebral activities. As he remained haemodynamically stable and blood cyanide values were rapidly falling to non-toxic concentrations the possibility of organ donation was considered. Both kidneys were perfused immediately after removal and transplanted within 24 hours. *Comment: A case of sever poisoning in which respiration and cardiovascular status could be improved by Co-EDTA, yet the brain damage could not be reversed.*

Brueske, 1997

A 35 year old man was exposed to copper cyanide gas while attempting a valve repair. He developed nausea, dizziness and lethargy/disorientation. A nurse practitioner administered AN for 10 minutes. After about 45 minutes he was taken to hospital. In the ED, he was dizzy and short of breath, with hypertension, tachycardia and tachypnoea. He remained lethargic and disoriented. He was given 300 mg of SN followed by 12.5 g of STS. His level of consciousness rapidly improved, but he remained tachycardic. He was admitted to the ICUwhere he stayed for 24 hours before being discharged the following day without sequelae. *Comment: A case of moderate poisoning in which the "Lily-Kit" was effective.*

Bryson, 1978

This is a comment to a publication in Lancet (1977; **11**, p1167) in which casualty officers were instructed to offer Kelocyanor for all patients displaying symptoms of cyanide poisoning. Bryson responded saying that in the past 2 years several c ases of unpleasant reactions to Kelocyanor had (600mg) been referred to him and cautioned the need for clinical evaluation and case history before its use (see also Edwards and Thomas 1978).

Buchanan, 1976

A 23 year old goldsmith attempted suicide by ingesting KCN. He was found collapsed only 1.5 hours later. In the ED he presented a coma, with shallow respiration and a pink coloured skin.

The heart rate was weak at 40-50/min and the systolic blood pressure was 110 mm Hg. Five minutes after the clinical examination respiration ceased making intubation and mechanical ventilation necessary. The bradycardia persisted and the blood pressure dropped to 0 mm Hg systolic. The young man received dextrose, sodium chloride, noradrenaline, cortisone, aramine and methoxamine but this did not improve his condition. With adrenaline and isoprenaline infusions, a blood pressure of 80 mm Hg could was established. After 2.5 hours a blood level of 5 mg cyanide/l was determined he was treated with 0.3 g of Co-EDTA and 4 g of glucose. 1 hour later the patient was also given 0.5 g of SN and 25 g of STS. The antidotal treatment was completed with 3 g of HOCO 30 minutes after the nitrite application. There was no change the man's condition and 17.5 hours after the suicide attempt cardiac arrest occurred.CPR failed and the man died 37.5 hours after the incident because of irreversible asystolia. *Comment: A case of severe poisoning in which the delayed application of several antidotes, including HOCO, could not save the patient.*

California Bureau of Communicable Disease Control, 1972

In California a couple shared a drink of pureed apricot kernels (total of 30) and dried apricots. 1 hour later the woman complained of abdominal discomfort, tachycardia and a strange feeling, her husband also developed headache, light-headedness, tachycardia and a strange feeling. *Comment: This is a case of mild poisoning but no antidotes were given norcyanide measurements reported.*

California Bureau of Communicable Disease Control, 1975

In California a 56 year old woman ate a handful of apricot kernels and developed a headache, light-headedness, tachycardia, flushing and weakness 1 hour later. Vomiting was induced and the vomit was found to contain 1.7 mg/l cyanide, while she had no cyanide in blood. *Comment: This is a case of mild poisoning but no antidotes were given.*

Campbell and Jones, 2001

A 30 year old male ingested KCN. 1 hour later he suffered cardiac arrest and was resuscitated. SN at an unspecified dose was not successful, so he then received 2.5 g of HOCO. While the cardiovascular situation improved, there was no CNS improvement, and he was declared brain dead after 5 days. This was attributed to cardiac arrest hypoxia. Cyanide in blood had been found to be above 4 mg/l. *Comment: In this case HOCO improved the cardiovascular, but not the neurological situation after cardiac arrest.*

Caravati and Litowitz, 1988

A case of dermal/inhalation uptake is reported in a 2 year old child, surviving a cyanide level of 6 mg/l.

2 cases of paediatric accidental ingestion of an acetonitrile-containing cosmetic are reported. 1 of the children, a 16-month-old boy, was found dead in bed the morning after ingesting the product. No therapy had been undertaken, as the product was mistakenly assumed to be an acetone-containing nail polish remover. The second child, a 2 year old boy, experienced signs of severe cyanide poisoning, but survived with vigorous supportive care. Both children had blood cyanide levels in the potentially lethal range. The observed delayed onset of severe toxic reactions supports the proposed mechanism of acetonitrile conversion to inorganic cyanide via hepatic microsomal enzymes. *Comment: These are cases of severe poisoning but no antidotes were given.*

Carella, 1988

A 46 year old woman and her husband were brought to hospital after drinking from a bottle of a soft drink which had been adulterated with cyanide. The man died before arriving at the hospital. On admission, his wife was found to be unconscious, readily reacting to pain and moving her limbs without obvious symmetry. Babinski's sign was present on the right. An analysis of gastric aspirates from the patient and her husband confirmed the presence of cyanide. CT scan and cerebrospinal fluid examination were normal. EEG showed occasional decerebrate posturing, but she slowly recovered after 2 weeks. She was transferred to a rehabilitation unit where she was found to have dystonia and dysarthria, slight plastic limb hypertonia, slight hemiparesis with right Babinski's sign and diffuse hyperactivity of tendon reflexes. 1 year later, the patient was admitted to another hospital because of increasing speech and swallowing difficulties, drooling and unsteadiness of gait. Subsequently, drooling and dysphagia became more marked and sustained abnormal postures of the mouth and tongue developed. 5 years after poisoning, the patient was readmitted to the rehabilitation unit. In conclusion this case confirms that Parkinsonism, dystonia and lid opening apraxia may evolve after cyanide poisoning.

Cesion and Jurrling, 2009

The authors report on a 54 year old man suffering from smoke inhalation. The COHb was 29% and although HCN poisoning was suspected,this was not confirmed by measurement. Lactate was high 16 mmol/l. The patient was treated with hyperbaric oxygen and 5 g of HOCO. The course is not reported. C*omment: As it is unclear, whether the patient really suffered cyanide poisoning, the efficacy of HOCO cannot be assessed in this case. No side effects were reported.*

Cetnarowski and Conti, 1986

A patient received a total dose of 215 mg (3.07 mg/kg)of SNP over 33 hours. The patient showed unexplained and worsening nausea, vomiting, mental alterations and metabolic acidosis with dramatic improvement after the admission of amyl nitrite. The authors commented that the administered dose was well below any recommended total maximum dose. *Comment: A case of moderate poisoning in which AN showed improvement.*

Chen and Rose, 1952

Chen and Rose reported 16 cases of cyanide poisoning treated with parts of their cyanide antidote kit, among which 10 received SN after exposures to HCN gas/fumes.

Case 1: A 24 year old man tried to start a syphon by mouth and thereby accidentally swallowed a solution containing 2.3% silver cyanide and 6.9% NaCN. He became weak, had a burning sensation in the throat and felt his heart pounding. 45 minutes later he showed fine tremors together with a very rapid pulse and received 10 ml of a 3% SN (300 mg $NaNO_2$) and 50 ml of 12.5% STS solution (6.25 g STS). He was discharged from the clinic after 24 hours. *Comment: A case of mild poisoning only and recovery is not necessarily attributable the use of antidotes.*

Case 2: A man was found unconscious in a HCN fumigated barracks and given artificial respiration and an unknown amount of methylene blue. Despite absence of respiration and being regarded as hopeless he was later given 0.3 g of SN and 1 g of STS i.v. Both antidotes were applied for a second time about 90 minutes later. He began to respond to stimuli and was fully conscious the next day. After 1 week he developed acute excitement, restlessness, and mania. After 2 months, he was discharged with persistent athetoid movements of the hands and personality changes. *Comment: A case of severe poisoning with delayed treatment. CNS symptoms not fully reversed.*

Case 3: A male patient was accidentally poisoned with cyanide. He received SN and STS and reportedly recovered. *Comment: The patient's recovery was attributed to the medication. This case is inconclusive as not enough data about the condition of the patient were available.*

Case 4: A customs inspector lost consciousness about 1 hour after he had stayed in a room with 460 sacks of mung beans that had been HCN fumigated the previous day. Immediately after he had been given AN pearls for inhalation he recovered but felt weak for one month. *Comment: A case of moderate poisoning with successful treatment with AN.*

Case 5: A 64 year old man was overcome by HCN fumes and was found unconscious, cyanotic and pulseless. He received artificial respiration and AN, resulting in

'hypermotor activity'. One hour later he received 0.3 g of SN and 12.5 g of STS i.v. AN inhalation was continued together with oxygen. He became conscious about 2 hours after exposure. The patient was sent home 4 hours later. *Comment: A case of severe poisoning in which quick application of AN, but delayed application of SN/STS were effective.*

Case 6: A 22 year old female was exposed to cyanogen chloride by inhalation. She was found unconscious and flaccid, followed by restlessness and hypermotor activity. 15 to 20 pearls of AN and 300 mg of SN were simultaneously administered. STS was not given (technical difficulties). Epinephrine was injected subcutaneously due to low blood pressure. 3 hours later she regained her rationality and was discharged with complaints of persistent abdominal, chest and back muscle tenderness the next day. *Comment: A case of moderate poisoning in which AN and SN were effective after some delay.*

Case 7: A 61 year old man was overcome by HCN. He was found unconscious, cyanotic, and in cardiorespiratory arrest. 20 minutes later he was treated with AN by inhalation, artificial respiration, and additionally with nikethamide intramuscularly. When he regained consciousness approximately 45 minutes later 0.3 g of SN and 12.5 g of STS were administered to him, along with epinephrine, caffeine, and sodium benzoate. He was taken home 2 hours after exposure. *Comment: A case of sever poisoning. From the description it is not fully clear if the patient regained consciousness before the administration of SN. If so AN was effective in a severe poisoning case.*

Case 8: A 67 year old man was overcome by HCN. He was found unconscious, cyanotic, and in cardiorespiratory arrest. The man was unconscious several minutes after exposure to hydrocyanic acid. He was pale and his pulse was weak and irregular. Respiration was normal. He responded well to AN only. *Comment: A case of moderate poisoning in which AN was effective.*

Case 9: A man, aged 44, was accidentally exposed to HCN gas. He became unconscious for a few minutes. AN was administered from several pearls, one at a time. He was nauseated, vomited several times, and developed a severe headache. He was sent home in an ambulance. *Comment: This case is inconclusive as the moderately poisoned patient may have regained consciousness before he got treatment. The headache was probably due to the AN. Specific treatment was not really appropriate in this case. However, AN was tolerated well.*

Case 10: A man was exposed to HCN from a cake of precipitate. He became dizzy and vomited. This continued for approximately 2 hours, at which time he arrived at the first-aid station. He was immediately given 0.3 g of SN and 12.5 g of STS. After 45 minutes the patient had recovered and was discharged 32 hours later. *Comment: This is a case of mild poisoning. SN/STS would not be conclusive at all as there was no indication that nitrite treatment had been required.*

Summary of the cases presented by Chen and Rose: Assessment was difficult as symptoms were often insufficiently described: 10 mild, 2 moderate and 2 severe intoxications. Four out of 10 patients received AN alone; the remaining 6 received the nitrite antidote kit. The moderately and severely poisoned patients all received SN, 3 in combination with STS, and 1 with AN. They all recovered completely.

Cheok, 1978

A report from Sarawak describes two children having eaten a bitter cassava cake – so bitter that the parents did not eat more than a bite. The cake was later found to contain 3 ppm cyanide (as CN^-). Both children (1.5 and 2.5 years of age) fell ill 7 and 7.5 hours after eating the cake with vomiting, drowsiness, and weakness, in one case also cyanosis. As no SN, which the authors recommend for treatment followed by STS, was available, the children were treated with STS alone (50 ml of 25% solution, 2.5-5 ml/min i.v.) and one with oxygen via nasal catheter. Both children recovered quickly and fully. *Comment: No measurements of cyanide in blood or for thiocyanate are available; in both cases gastric aspirate was negative for cyanide. The clinical improvement after antidote application is not conclusive in the absence of measurements cyanide detection.*

Cherian and Richmond, 2000

A fatal accident occurred aboard a stern freeze trawler 40 miles off the coast of West Africa. The warm temperature and failure to clean the storage tank immediately after its previous use had left a mixture of sea water and fish to rot. After opening the door of this salt water tank to flush it out, the crew member collapsed immediately, apparently having been overcome by toxic fumes. Unaware of the danger several crew members went to help him and were also overcome. 3 men died within 1 minute and 6 suffered other injuries, such as blackouts, seizures, chest pain, vomiting and difficulty in breathing. Several volatile agents were detected in the blood and whole lung. Besides cyanide (blood level <50 µg/l) and MetHb (14%) methane could also be identified. *Comment: A case of severe poisoning although involvement of cyanide is doubtful, the use of antidotes is not described.*

Chin and Calderon, 2000

A 19 year old woman drunk from an open soda bottle contaminated with cyanide (cyanide could be detected) by her boyfriend who had been working in a jewellery business for some time. He called EMS and on their arrival she was apnoeic and unresponsive. She was intubated and taken to hospital, unresponsive (GCS = 5); apnoea, tachycardia of 110/min, hypotonia of 90 mm Hg systolic blood pressure and a blood-pH of 7.01 and with 10 mmol/l lactic acid were observed.

She received naloxone, thiamine, glucose and i.v. fluids, together with activated charcoal and sodium bicarbonate. Thirty minutes after administration of 12.5 g of STS, the pH increased to 7.17 and the MetHb counted for 9.5% (no MetHb forming drug had been administered until that time; the TF believes that the blood samples had been mixed up). 300 mg of SN were administered and half the initial dose of both antidotes was repeated 60 minutes later "to avoid excessively high levels of methaemoglobinaemia yet continue to treat the patient's acidosis". The patient made a good recovery and could be extubated after 5 days. 8 days after the incident she was discharged. 5 months after the intoxication she complained about short-term memory problems as well as a weakness in the lower legs. *Comment: A case of moderate intoxication in which the victim could be rescued with the nitrite-kit but neurological and motor sequelae persisted. The elevated MetHb level before SN remains unexplained.*

Cliff and Coutinho, 1995

One 3 year old child died, probably from aspiration pneumonia. Cyanide poisoning was diagnosed by the history of eating cassava and the similarity of the clinical picture with acute cyanide intoxication. *Comment: No measurements at all were done.*

Coentrão et al., 2010a (Coentrão and Moura, 2010b)

A 50-year-old goldsmith who tried suicide by ingestion of a jewellery cleaner solution containing approximately 1.2 g of potassium cyanide presented unconsciousness, with severe lactic acidosis and arteriolisation of venous blood gases. Following hydroxocobalamin treatment, neurologic and metabolic disorders rapidly improved. He was discharged home 4 days later, without neurological sequelae.

Coentrão and Moura, 2011

A retrospective chart review of all admissions for acute cyanide poisoning by ingestion for the years 1988 to 2008 was conducted in a tertiary university hospital serving the largest population in the country working in jewelry and textile facilities. Of the 9 patients admitted to hospital during the study period, 8 (7 males, 1 female; age 36 ± 11 years, mean ± SD) attempted suicide by ingestion of potassium cyanide used in their profession as goldsmiths or textile industry workers. 5 patients had severe neurologic impairment and severe metabolic acidosis (pH 7.02 ± 0.08, mean ± SD) with high anion gap (23 ± 4 mmol/L, mean ± SD). Of the 5 severely intoxicated patients, 3 received antidote therapy (sodium Thiosulphate or hydroxocobalamin) and resumed full consciousness in less than 8 hours. All the patients survived without major sequelae. *Comment by the authors: Metabolic acidosis with high anion is a good surrogated marker of severe cyanide poisoning. STS and HOCO are both safe and effective antidotes.*

Dall and Hannah, 1964

As examples, Dall and Hannah (1964) reported on "Oxygen therapy in cyanide poisoning" in a 39-year-old male accidentally poisoned by oral ingestion of a solution of KCN. After having 'called for medical attention' he was found in deep coma, hypopnoea, seizures, respiratory and cardiac arrest. In addition to oxygen, reported treatment included the administration of a number of ampules of AN, external cardiac massage, artificial ventilation after endotracheal intubation, STS (25 g) and SN (300 mg). The patient fully recovered. However, the causal relationship between oxygen administration and survival is complicated by the associated treatment, the efficacy of which might have been underestimated. Additionally, Bismuth et al (1984) emphasised about the priority of oxygen in cyanide poisoning and reported details of treatment in 4 out of 25 cyanide poisoning. One of the four with minor poisoning recovered with oxygen alone. Among the three remainders two died while one survived without sequelae after having received in addition to oxygen and supportive treatment, including cardiopulmonary resuscitation for transient cardiac arrest, Co-EDTA, and HOCO. *Comment: It seems rather difficult to draw any definitive conclusion from these complex and severe cases receiving multiple combined treatments.*

Daunderer et al, 1974

A 26 year old female working in a chemical laboratory tried to commit suicide by drinking about 10 g of KCN solved in sour vine. She vomited and then collapsed directly after having drunk the solution. In the hospital she presented the following symptoms: good general condition, irregular breathing, mild peripheral cyanosis, red shining parts of the skin, odour of bitter almonds, deep coma, no reaction to painful stimulus, wide pupils, minimal reaction to light, blood pressure 140/90 mm Hg, HF 130/min and weak pulse. She was intubated, ventilated and gastric lavage was performed. 45 minutes after ingesting the poison she was administered 250 mg of DMAP together with 300 mg of Cobalt-EDTA i.v. No adverse effects were seen and a short time after this therapy the pupils became reactive to light again and deep tender reflexes as well as reaction to pain could be triggered. About 45 minutes after that treatment 60 ml of a 10% STS-solution were applied (6 g). During this infusion the patient became awake and orientated. In the former course metabolic acidosis, hypotonia, tachycardia and a general oedema occurred. All these side effects could be managed within a few days. Methaemoglobin was not measured. *Comment: A case of severe poisoning in which the combination of DMAP, Cobalt-EDTA (both given in the recommended doses) and STS worked well. The generalised oedema can be correlated to the Cobalt-EDTA. Fortunately all adverse effects and poisoning consequences could be managed.*

Davies, 1975

A 40 year old male received SNP while anaesthesized during surgery. The blood pressure could not be lowered sufficiently, so the SNP dose was increased. Approximately 400 mg had been given in total. After cessation of the infusion the blood pressure dropped, and the skin was pink. Bradycardia necessitated administration of gatropin but blood pressure dropped further. Refractory cardiac arrest followed. The level of cyanide in blood was 5 mg/l.

Davis and Ewer, 1988

A 22 year old electrician drank a solution containing cyanide to commit suicide. A short time later he told his father. On admission to the ED 45 minutes later he presented a GCS of 5 points, dilated and fixed pupils, a hypertonia (blood pressure 160/80 mm Hg), a hyperreflexia, tachypnoea, tachycardia, a flush and facial oedema (*note: this was prior to being given the cobalt-EDTA*). There was no reaction to painful stimuli. 2 ampoules of AN were administered by inhalation together with 100% oxygen. Then he received 300 mg of Co-EDTA together with 50 ml of a 50% glucose solution, 0.3 g of SN and 12.5 g of STS. Furthermore gastric lavage was performed and activated charcoal administered. As his condition did not change during the next hour, the cobalt-EDTA-treatment was repeated and hyperbaric oxygen initiated. At this time the patient was still unconscious with loss of reaction to pain, but the pupils were now reactive to light and he made spontaneous movements. After the second dose of Co-EDTA lactic acidosis he quickly recovered and was orientated within 20 minutes. He vomited and was given sedatives as he had become agitated. Furthermore, facial oedema was detected *(Note: this was already mentioned with the initial symptoms, one does not know, when it really occurred).* He finally recovered completely. *Comment: Ths is a case of severe poisoning. No blood-cyanide-levels were measured. The Co-EDTA was given in high dose, as the first antidote and the patient made a remarkable recovery only after the second cobalt-dose, but the authors attribute the good course to the hyperbaric oxygen (which was initiated at the same time as the second antidote application).*

Dawood, 1969

According to a report from Singapore a 3.5 year old girl had started to vomit ten hours after ingestion of cassava and arrived in hospital in a state of shock with acidosis, drowsiness, irritability, shortness of breath and paleness. The eyes were staring, the pupils were dilated and non-reactive to light, blood pressure was hypotonic, and reflexes were missing. Neither cyanide nor thiocyanate could not be detected in the gastric aspirate. The tapioca contained 94 mg/kg hydrogen cyanide. The child recovered without specific treatment, no cyanide or thiocyanate measurements were done. *Comment: Cyanide poisoning was only suspected, and no antidotes were given.*

De Busk and Seidel, 1969

Case 1: A 22 year old student in chemical engineering took 1.5 g of NaCN in a suicide attempt. He presented a flush, arrhythmia and was unresponsive to stimulation. As the vomit smelt like bitter almonds and a suicide note was found, cyanide intoxication was quickly suspected. The ECG was altered, consisting of a short QT time, an increased ST-line and a loss of the P-wave. The cyanide blood level or other blood-parameters were not determined. Primarily intubation and ventilation as well as gastric lavage were performed and AN pearls were crushed over the respirator. Afterwards he was administered 300 mg of SN (10 ml of a 3% solution). The estimated interval from cyanide ingestion to i.v. SN administration was 20 minutes. Cardiac arrest occurred but could be reversed with CPR. Then 12.5 g of STS (50 ml of a 25% solution) were injected resulting in spontaneous movements and vital parameters being re-established. As the mental state did not improve a course of repeat AN/STS treatment was administered resulting the cyanosis clearingcompletely and the patient fully recovering. *Comment: A case of severe poisoning in which the patient recovered from a high dose of cyanide even though he had to be resuscitated. Resuscitation was required after the first nitrite dose, and only STS and a repetition of AN/STS led to full recovery.*

Case 2: A laboratory technician presented respiratory distress, followed by collapse and a possible seizure during the half-hour preceding admission. On arrival in hospital she was comatose, the extremities were flaccid and deep tendon reflexes were absent. No blood pressure was obtainable and the femoral pulse was weak with a pulse rate of 110/min. Respiration was irregular at 6-10 breaths per minute. The pupils were dilated and unresponsive to light. The ECG showed atrial fibrillation, occasional ectopic ventricular beats with T waves originating high on the R wave. Treatment included intubation, ventilation with 100% oxygen, gastric lavage and metaraminol (probably a catecholamine) i.v. for circulatory support. Because of a smell of almonds acute cyanide poisoning was suspected and 0.3 g of SN was given over four minutes and 17.5 g of STS (both agents were taken from the Lilly cyanide Kit). The patient became moderately cyanotic (MetHb). After 15 minutes she had regained consciousness and could be asked about what had happened. She said she had taken 4-6 g of KCN in a suicidal attempt. The cyanide came from her laboratory. The approximate time between ingestion of cyanide and treatment with SN was 50 minutes. Recovery was uneventful with prompt reversal of the ECG changes. No cyanide or MetHb measurements were taken. *Comment: This case is conclusive as regards the prompt improvement after nitrite treatment that followed the respiratory and circulatory insufficiency caused by a high cyanide dose.*

Dequidt et al, 1974

Case 1: A 19 year old man was accidentally exposed to acetonitrile vapours. After 4 hours GI symptoms developed with convulsive coma accompanied respiratory pauses by 12 hours

followed by collapse and cardiac arrest by 24 and 26 hours respectively. From the time of collapse he had been intubated and had received gastric lavage. He survived the cardiac arrest with symptomatic treatment but with persistence of an unstable blood pressure and repeated convulsions. 41 hours after the exposure, cyanide poisoning was confirmed by a free blood cyanide concentration of 1.12 μg/ml. He received 600 mg of Co-EDTA. On the following day he remained in a profound coma. About 65 hours after the exposure, reactivity to painful stimuli reappeared and the patient then received 4 g of HOCO. The patient was subsequently diagnosed as brain dead. *Comment: Antidotal treatment of this severe poisoning was delayed for more than 24 hours so Co-EDTA or HOCO efficacy cannot be assessed.*

Case 2: There is a second case report of death due to inhalation in spite of administration, after more than 24 hours, of Co-EDTA (2x600 mg) and HOCO (4 g) in a worker after he had cleaned with acetonitrile and hot water. The first symptoms developed after about 12 hours. 24 hours after admission to hospital the blood cyanide level was 11.2 mg/l and the maximum level of acetonitrile in blood was 311 mg/l. *Comment: Again, antidotal treatment of this severe poisoning was delayed for more than 24 hours so Co-EDTA or HOCO efficacy cannot be assessed.*

DiNapoli et al, 1989

A 29 year old man was found unresponsive a few minutes after self-injecting undetermined amounts of KCN and $NaAs_2O_3$ (arsenite) i.v. in a suicide attempt. On arrival at ED he was unconscious and unresponsive to stimuli. He was tachycardic with deep gasping respiration. He was given naloxone, dextrose and thiamine but his condition did not change. AN pearls were administered followed by a return of the spontaneous movements. In the blood gases a pH of 7.12 and a pO_2 of 54 mm Hg were detected. After infusion of 0.3 g of SN and 12.5 g of STS the patient regained consciousness within seconds and the blood pH increased to 7.28 and the pO_2 to 85 mm Hg. The MetHb level was 7.5%. The anion gap counted 24 mmol/l and the cyanide level 4.4 mg/l before antidotal treatment. The cyanide level fell to 0.18 mg/l 12 hours after the nitrite had been administered. After recovering from coma the patient had visual problems and was treated empirically for methanol poisoning, which was subsequently not confirmed. Blood and urine arsenic concentrations were also quite elevated, so the patient received BAL and later D-pencillamine. He was ultimately released from the hospital about 3 weeks later. *Comment: Though the arsenite level was not increased the man received specific treatment for arsenite intoxication (chelators). None of the antidotes, neither for arsenite nor for cyanide intoxication, seems to have caused adverse effects. In the end he made an uneventful and complete recovery after effective treatment of the cyanide poisoning.*

Dodds and McKnight, 1985

A 43 years old worker was completely immersed in a bath of hot copper cyanide for 3 minutes by accident. Only 15 minutes later he lost consciousness, became cyanotic, his respiration was irregular and the blood pH counted only 6.9. The base excess was 20 mmol/l. He was ventilated, the clothes were removed and 300 mg of Co-EDTA administered over 3 minutes. As this did not show a prompt improvement, the antidote administration was repeated. He developed massive facial oedema which commenced in the peri-orbital area. As his condition still did not improve, the nitrite antidote kit was additionally administered (AN pearls, SN and STS). It took 16 hours before he regained consciousness and recovered completely. *Comment: The antidotal therapy consisting of Co-EDTA (twice) and nitrites did not result in fast recovery but in the end the treatment seemed to have been efficient (however, as 16 hours had passed the recovery could also be attributed to physiological detoxification because normally the antidotes should have worked more quickly).*

Edwards and Thomas 1978

This is primarily a comment to a publication from Graham et al, Arch Intern Med 1977, 137:1042 (see also **Bryson, 1978)**, but they describe an additional case.

A 48 year old chemist was presented deeply unconscious with unrecordable blood pressure; he had a cardiorespiratory arrest on arrival. He was asystolic and was resuscitated by a combination of intubation and ventilation with 100% oxygen, cardiac massage, and intracardic injection of 1 mg of adrenaline. Direct-current (DC) shock was used to revert ventricular tachycardia to atrial fibrillation and 10 mg of practolol i.v. to control rapid ventricular rate. He remained hypotensive (60 mm Hg systolic, central venous pressure zero) and his arterial pH was 6.92. Serum *Comment: There is little to mindicate that this was cyanide poisoning other than he was a chemist.*

Eiró Gonsalves et al, 1956

6 cases of cyanide intoxication as a result of ingestion of wild cassava were admitted to the emergency ward of the pediatric clinic during a 5 month period.

Case 1: A six year old boy became unconscious about 2½ hours after the meal. He survived after i.v. application of a total of 90 ml of STS 2% and 50 ml of SN (concentration not reported).

Case 2: Another six year old boy had abdominal pain and was vomiting 5 hours after a meal and became somnolent. He recovered after i.v. adminsitration of 2 ampoules of SN 2% and 2 ampoules of STS 10% (amount not reported).

Case 3: A five year old girl became somnolent and started vomiting 8 hours after a meal. She, too, recovered after 2 ml of SN 2% and 20 ml of STS 10%.

Case 4: A nine year old boy presented with initial tetanic convulsions of the extremities, followed by flaccid paralysis and inability to speak. He recovered after 15 ml of SN and 15 ml of STS (concentration not reported). The time between ingestion and treatment was not reported.

Case 5: A six year old boy was vomiting and feeling weak four hours after a meal. He inhaled an ampoule of AN and then received 5 ml of SN 10% and 60 ml of STS 10%, after which he recovered.

Case 6: After the same meal a four year old girl also was feeling weak and vomited. She too recovered after 5 ml SN of 10% and 30 ml of STS 10%. *Comment: No cyanide or thiocyanate measurements were performed, cyanide intoxication can only be assumed due to the symptoms observed, which are more or less indicative only, the effect of the antidotal treatment is not clearly described – all children had recovered on the following day, which may or may not be due to the antidotes given.*

Espinoza at al, 1992

8 male children between 8 and 11 years of age developed vomiting and excessive weakness after ingesting bitter cassava. On arrival at ER they presented respiratory failure, bradycardia, hypotension and cardiovascular collapse. Two children developed generalised seizures with relaxation of sphincters. All recieved 100% oxygen. The more serious half of the children were treated with 3% SN (0.2ml/kg bw) and 25% STS (0.04ml/kg). The less serious 4 were treated with 500mg hydroxocobalamin in dextrose soluton. All 8 survived without sequalae.

Favarel-Garrigues, 1982 / Dreykluft et al, 2004 (case 1)

Case 1: A 36 year old chemical-technical assistant was presented to the hospital in a coma with tachypnoea (32/min), tachycardia (100/min) and wide, non-reactive pupils. The P_aCO_2 was only 19 mm Hg and he showed a severe acidosis (pH 6.96, lactate 20 mmol/l, anion gap 25 mmol/l). The patient had a history of multiple suicide attempts. His treatment included gastric lavage application of activated charcoal and sodium bicarbonate, because of the unknown poison. 9 hours after the incident the man became responsive again and recovered completely. He then

confirmed the suicide attempt with KCN. *Comment: No specific antidote was given. Documentation a bit confusing because the publication was part of a quiz.*

Case 2: A 27 year old woman ingested 2 g of KCN in a suicide attempt. 30 minutes later the ambulance staff found her comatose, hypotonic with dilated pupils, failed respiration and convulsing. She was ventilated with 100% oxygen and given drugs for haemodynamic support (it has not been mentioned which drugs). In the ED an unspecified amount of STS was administered together with sedatives. This resulted in intermittent improvement of the haemodynamic situation but 5 days later she died due of cerebral damage. *Comment: Information is incomplete although it would appear that in this case of severe poisoning STS alone was eventually not effective.*

Feihl et al, 1982

After ingesting 600 mg of KCN a 60 year old chemist arrived in hospital comatose and bradypnoic with severe metabolic acidosis. In addition to supportive care, he received SN and STS and recovered without sequelae 'despite excessive methaemoglobinaemia' (value not provided). The authors note an extraordinary blood cyanide concentration of 40 mg/l including a fraction bound to inactive cyanMetHb. *Comment: The actual free cyanide level is unclear. However, in this case of moderate poisoning SN/STS was effective.*

Feldmann, 1990

A 28 year old man was presented at the emergency department after ingesting 800 mg of KCN in a suicide attempt. He survived only with intensive medical and psychiatric intervention, and went on to develop severe Parkinsonian symptoms, including profound micrographia and hypersalivation. Magnetic resonance imaging showed bilateral and symmetrical basal ganglial abnormalities. Survival following cyanide (*TF insert: severe*) poisoning is rare; the clinical, radiologic, and neuropathologic sequelae are reviewed in other documented cases.

Fernández et al, 2008

A 38 year old man was exposed by inhalation and dermal to pentenenitrile solvent solution at work. Several hours afterwards he developed progressive weakness prompting him to decontaminate in a shower for 20 minutes. He became syncopal. EMS found him in atrial fibrillation at a rate if 112 bpm that increased to 177 upon arrival at hospital where he presented headache, nausea, vomiting and metablic acidosis. He was treated with 100% oxygen,

ondensetron and AN followed by SN and STS. He had a confirmed serum thiocyanate level of 7.5 µg/ml and recovered without sequalae.

Finelli, 1981

A 30 year old man was found unconscious, apnoeic, and cyanotic after he had ingested an unknown quantity of a cyanide-containing insecticide. After resuscitation and the application of STS, he regained consciousness. He remained mute for 12 days and developed choreiforme (involuntary spasmodic) movements. After 16 days he was normal apart from mild dysarthria and minor movement disorders. A computer tomography revealed a bilateral symmetrical infraction of the globus pallidus and an infarction of the left cerebellar hemisphere. *Comment: In this case of severe poisoning neurological sequelae persisted after effective treatment with STS, but these were probably due to cardiorespiratory arrest.*

Fortin et al, 2007

A 47 year old male with third-degree burns and (later) proven cyanide poisoning from smoke inhalation was found in cardiac arrest. He was resuscitated and received 10 g of HOCO in two stages. After the first 5 g of HOCO the cyanide level in blood was 3.4 mg/l and after the second 5 g it was below 1 mg/l. While cardiac activity resumed he had to be declared brain dead on day 3. *Comment: A severe poisoning case in which HOCO improved the cardial, but not the neurological situation after cardiac arrest.*

Froldi et al, 2001

This was a rare case of suicide by oral ingestion of 6 g of SNP in a 41 year old woman found dead. At autopsy total cyanide in blood was 5 mg/l. *Comment: Fatality, no antidote use.*

Gaillard, 1991

After a domestic dispute with his wife a 33 year old man tried to commit suicide with a solution containing cyanide. The ambulance staff found a semi-comatose, agitated, tachycardiac (heart rate 135/min), tachypnoeic, dizzy patient with a bilateral miosis (not mydriasis). They administered i.v. fluids and initiated ventilation with 100% oxygen by face-mask. At admission at the ED the patient was fully conscious with spontaneous respiration but after developing seizures had to be treated with clonazepam and intubated. Ventilation and sedation were performed. Gastric emptying was undertaken and activated charcoal administered. Additionally,

sodium bicarbonate and 4 g of HOCO were given about 2 hours after poison intake. A cyanide level of 150 µmol/l (4.05 mg/l) was measured at admission and decreased to 15 µmol/l (0.41 mg/l) after treatment. The HOCO-treatment was repeated twice within the next three days. The man was extubated on the fourth day in hospital and discharged on the seventh day. He recovered completely. *Comment: The clinical course is not well described, but obviously HOCO significantly decreased the cyanide blood level.*

Gambaro et al, 2007

Case 1: A 26 year old woman was taken to hospital presenting slowed respiration, gasping breath and bradycardia. She died more than 1 hour later. Symptomatology suggested cyanide poisoning, which remained unconfirmed.

Case 2: A 30 year old man was found dead his car. Laboratory analysis of the liquid in a bottle found nearby revealed cyanide. Forensic evaluation could not establish the time interval between ingestion and death. The analysis of the stomach content and postmortem blood samples was positive for cyanide.

Comment: cases of severe poisoning with fatal outcome but no antiotes were used.

Gaultier and Le Breton, 1952

A male in custody at a police station developed hand ataxia, followed by trembling. 30 minutes later convulsions, somnolence/coma and irregular respiration were observed, followed by respiratory arrest and death 3 hours after the start of the ataxia. Autopsy revealed cyanide poisoning. A tube with cyanide was found in the duodenum. *Comment: Cyanide poisoning was not recognised and no antidote was administered.*

Geller et al, 1991

A patient received gastric lavage and activated charcoal administration 30 minutes after ingesting acetonitrile. Hours later he developed mental status abnormalities. He vomited prior to a generalised seizure. The blood cyanide level documented a significant exposure (intoxication). Following the administration of STS, the patient made an uneventful recovery. *Comment: A case of moderate poisoning with apparent delayed effects in which STS was effective.*

Ghiringhelli, 1955

One case from Italy has been reported – a young man drank by mistake a few mls of adipic nitrile. Symptoms were weakness, headhache, vertigo, nausea/vomiting, cyanosis, tachypnea, hypotension, mydriasis, somnolence, tonic-clonic convulsion, inability to stand independently. All these quickly resolved after application of STS. *Comment: a case of nitrile poisoning in which STS was completely effective.*

Glenn et al, 1994

A 54 year old man deliberately drank a potassium-gold cyanide solution (300 ml) that contained about 1.65 g of KCN and survived after treatment with the Lilly antidote kit and hyperbaric oxygen. When he arrived in hospital 6½ hours later vomiting with diarrhoea and was gradually disorientated. He was given oxygen and received antidotal treatment from the Lilly-kit. There was no proper response and the SN and STS treatment was repeated. Despite these measures, he became progressively obtund, with hypotension refractory to pressors. 8 hours after ingestion, the blood cyanide level was 0.09 mg/l, i.e. within normal limits. *Comment: The case is not conclusive. It seems that cyanide poisoning was not the main problem, as the patient was alive 6½ hours after ingestion and the cyanide level in blood was very low. The whole case probably describes gold salt poisoning.*

Goodhart, 1994

A 54 year old man working in a watch factory intentionally drank 300 ml of a gold-solution containing 1650 mg of KCN. 6 hours and a half later he was presented in the emergency centre with vomiting, diarrhoea and an altered mental status. The blood cyanides were measured eight hours after poisoning with 0.09 mg/l. The vital signs included a systolic blood pressure of 50 mm Hg and a heart rate of 118/min. He was given 100% oxygen by face mask, activated charcoal and sympathomimetics. Under this treatment he became comatose, cyanotic, anuric and apnoeic. The nitrite-antidote-kit (AN pearls, 0.3 g of SN and 12.5 g of STS i.v.) was administered and since the patient did not respond to the first dose this was repeated. Additionally six cycles of hyperbaric oxygen were performed. The blood cyanide level remained below the measurable range and the lactate only rose to 3.4 mmol/l. He stayed with on respirator for 18 days, developed ARDS, pneumonia, jaundice and had to be temporarily dialysed. Despite all this he could be discharged 41 days later in complete remission. *Comment*: *The clinical presentation was serious, but the blood-cyanide levels were quite low. Probably it was a gold intoxication.*

Graham et al, 1977

A young man (aged 21) ingested KCN with suicidal intent. He became cyanotic and lost consciousness. The vital signs included a respiration frequency of 24/min, heart rate of 68/min (arrhythmic) and blood pressure of 168/112 mm Hg. Both pupils were reactive to light. In the blood samples an anion gap of 35 mmol/l together with a mild acidosis (pH 7.27 under 5 l oxygen per minute) could be detected. Furosemide was administered for the treatment of pulmonary oedema (noticed in the chest X-Ray). Further therapy consisted of sodium bicarbonate and sodium-chloride i.v. as well as intubation and ventilation. 10 hours later the suspicion of cyanide intoxication was substantiated (the authors do not report how, but after recovery the patient confirmed that he had taken 200 mg KCN). Since so much time had passed between the intake of the cyanide and the diagnosis of intoxication no specific antidote was applied. 12 hours after the suicide-attempt the blood-cyanide-level was measured with 2.0 mg/l and fell down to 1.6 mg/l after 22 hours and 1.2 mg/l after 84 hours respectively. 24 hours after the incident the young man could be extubated and was discharged on the ninth day post exposition. *Comment: A case of moderate poisoning with supportive treatment only. Even if the time span between intoxication and diagnosis was long, a specific antidote that supports the excretion of the poison (e.g. STS) might have accelerated the recovery.*

Grunske, 1949

In 1949, the death of a child was reported following pest control operations with CAN. Two more similar cases have not been published. The child had lacrimation, nausea, tonic seizure 1 hour after exposure in a room ventilated after pest control operator treatment. The next morning the child was in respiratory distress and coma with tachycardia and pallor. She died on transport to hospital. *Comment: A case of sever poisoning but no antidote had been given and cyanide measurement was not done.*

Gunder et al, 1969

A 3.5 year-old Israeli year old girl ingested an unknown amount of apricot pits and developed later pallor, fast pulse, rapid breathing, dilated pupils, vomiting and temporary unconsciousness. A bitter almond smell was noticed. Gastric lavage and paraffin application prompted full recovery. *Comment: Cyanide poisoning after amygdalin intake can be supposedly based on symptoms. No specific treatment was done*.

Hall et al, 1986

A four year old boy with Down syndrome and an eye seizure disorder ingested 12 Laetrile tablets (equivalent to 300 mg amygdalin) after a meal of fruits, vegetables and nuts. Within 1.5 hours he slowly became unresponsive and had multiple seizure episodes. The child was transported from the local hospital to a referral hospital but in both hospitals no complete antidote set (Lilly-Kit) was available. The child was presented in the second hospital with bradycardia (60/min) and thus measurable blood pressure. He was intubated and ventilated. Gastric lavage was performed and 15 g of activated charcoal was instilled. The blood gases showed severe metabolic acidosis with ineffective respiratory compensation, pH: 6.85, pCO_2: 15 mm Hg, pO_2: 169 mm Hg, anion gap: 16 E/l. Initially only AN pearls were administered by intermittent inhalation. This increased the blood pressure to 100/50 mm Hg. The blood pH could only be improved a little by sodium bicarbonate. The full antidote-kit (probably Lilly-Kit) was available 6 hours post ingestion. During the last 2 hours the child was maintained on AN (the dose was not specified). Bradycardia and hypotension improved during periods of inhalation and worsened during periods of noninhalation. The antidote-kit, 5 ml of SN 3% (150 mg) and 25 ml of 25% STS (6.25 g) were sequentially administered. Within 30 minutes the child recovered with normal blood pressure seizures. The child was extubated 15 hours postingestion and was alert and awake. The blood gases improved rapidly and were normalised 15 hours after ingestion. The highest whole blood cyanide level was 16.3 mg/l after the antidote administration and 8.2 mg/l at admission to the second hospital. *Comment: A case of severe cyanide poisoning after Laetrile ingestion. AN was partially effective, but allowed to bridge the time until availability of SN/STS which was fully effective.*

Hall et al, 1987

A man (34 years old) took 1 g of KCN in a suicide attempt. He reported the incident after about half an hour and became lethargic. A further 15 minutes later he lost consciousness and was ventilated with an anaesthesia bag by the ambulance staff. Upon arrival at the ED, 1 hour after the intoxication, he presented apnoeic so was intubated and ventilated with 100% oxygen. Sodium bicarbonate was administered to correct an acidosis (pH 7.11 to 7.33). His pupils were fixed and dilated. Quarter of an hour after his admission 300 mg of SN and 12.5 g of STS were administered over 45 minutes. As this was finished the patient showed spontaneous respiration and movements and the pupils were again reactive to light. 4 hours after the suicide attempt the man was alert and was extubated 6 hours later. The cyanide levels in the blood samples were determined at 1, 1.5, 2, 2.5, 3, 5, 11 and 17 hours after the exposure as 5.5, 8.72, 15.65 (extremely high value!), 9.93, 1.74, 9.82 (*no explanation can be given for this new increase, after the blood cyanide had already fallen*), 0.62 and 0.53 mg/l. A level of 2% MetHb was measured after the application of the antidotes (*this is below the level that is generally considered as therapeutically effective*). The patient fully recovered. *Comment: This is a case of severe*

poisoning as confirmed by high cyanide blood levels with respiratory arrest in which quite high doses of SN/STS were fully effective, albeit MetHb levels remained low.

Hall et al, 1989a

After smoke inhalation a 78 year old patient was found comatose, apnoeic and asystolic. He could be resuscitated on site. In hospital he was treated with 300 mg of SN i.v., which induced severe hypotension. MetHb elevated very little. 4½ hours after hospital admission the patient was treated with hyperbaric oxygenation (hyperbaric oxygen-chamber) without success. The patient died the next day in irreversible circulatory failure. The cyanide level in blood was 0.34 mg/l on admission. *Comment: This was no significant cyanide poisoning as shown by the blood level. In the absence of therapeutical benefit the side effect of SN (hypotension) prevailed.*

Hall et al, 2009

In a review article, Hall et al (2009b) quoted a case report from Person (1993). Two children were found comatose in a fire. They received oxygen by inhalation and 400 mg/kg bw of STS and were transferred for hyperbaric oxygen treatment. Both children recovered completely. The treatment was well tolerated. The cyanide levels were 1.15 mg/l in one child and 1.1 mg/l in the other. *Comment: Two cases of moderate poisoning that reacted well to STS alone, while CO poisoning was probably concurrently treated with hyperbaric oxygen.*

Hantson, 1996

A woman, aged 38, ingested one teaspoon of potassium ferrocyanide ($K_3Fe(CN)_6$) in a suicide attempt. Half an hour later she arrived in hospital fully conscious, tachycardiac (heart rate 110/min) and hypertonic (blood pressure 150/80 mm Hg). The physician on duty suspected cyanide poisoning and performed intubation, ventilation and application of 5 g of HOCO i.v. 1 hour after the incident the patient was still conscious, the vital signs were all within the normal ranges and a cyanosis could not be detected. She also had not developed adverse effects because of the antidote. The blood gases only showed an elevated anion gap of 13 mmol/l. 12 hours after the intoxication the patient was extubated and could be discharged soon after. During her time as an in-patient the cyanide level was determined after 1, 3, 4, 6 and 8 hours as 20 (0.54), 15.4 (0.42), 12.3 (0.33), 8.85 (0.24) and 3.6 (0.1) µmol/l (mg/l). MetHb, pH or the lactate were altered at any time. *Comment: As potassium ferrocyanide does not liberate cyanide, this was no cyanide poisoning. The antidote was not justified, but did not show any side effects.*

Harry, 1985

A 54 year old female laboratory assistant ingested 200mg of sodium cyanide. After 5 minutes she was confused and uncomfortable. 30 minutes later she eas found comatose and unreactive and was ventilated with oxygen. At 60 minutes she was resusciated and was in hypertonic coma (class 3/15 on Glasgow score), temperature 33ºC, blood pressure 70/40 mmHg, rate 120/min with lactic acidosis (29.5 mmol/l). Blood analysis showed vascular shock and collapse. At 20 minutes HOCO (4gms) and STS (8gms, Cyanokit) normalised systemic arteriol resistance and perfusion with 500ml plasma resumed moderate capillary pressure. The antidotes partially ameliorated the neurological state (Glasgow score 9/15) and decreased the lactic acidosis. Administration of hyperbaric oxygen at 2.5 ATA normalised venous oxygen levels and neurological status. Haemodynamic analysis revealed a 48% reduction during the first 5 hours that was not improved by HOCO and STS administration without clinical or radiological signs of pulmonary oedema. Total cyanide levels were measured as being 10mg/l at 1 hr, and 1.2, 0.3 and 0.25 mg/l at 4, 6 and 20 hours respectively, with an apparent distribution volume of 0.4L/kg.

Heintz et al, 1990

A chemistry student (aged 23) ingested 1500 mg of KCN in a suicide attempt (he admitted it after recovering). He developed a seizure and lost consciousness. His respiration was fast and the blood gases demonstrated a lactic acidosis (pH 6.96, BE -28 mmol/l and lactate 22 mmol/l). The blood cyanide was determined 6 hours after the incident as 6 mg/l. Until then he had received supportive treatment only, since the poison was not known. The therapy consisted of gastric lavage, administration of activated charcoal and as carthatic sodium-sulphate, ventilation with 100% oxygen and sodium bicarbonate. After the result of the blood cyanide level was available, he was given 1 g/hour STS over 24 hours (24 g in a whole). At the end of the antidote administration he was conscious and could be extubated. The pH normalised within 9 hours of therapy, the BE within 12 hours, the respiration parameters within 24 hours and the blood lactate level was within the normal range after 2 days. So the STS showed a good efficacy, despite of having been administered six hours after the intoxication. The patient was discharged on the 9th day post exposure. *Comment: A case of mild poisoning based upon the clinical presentation although blood cyanide levels were high. STS alone was effective when accompanied by supportive treatment.*

Herbert, 1979

In 1940, a 14 year old Indian school girl was hospitalised with stupor, convulsions, fibrillary muscular twitching, pale cyanosis, laboured respiration, fever, hypotension and tachycardia. She died within 24 hours. Autopsy revealed large amounts of choke cherries and pits in the

gastro-intestinal tract. Crushed pits fed to guinea pigs caused convulsions and death. *Comment: Cyanide measurements were not done, however, in regard of the symptoms and the animal experiments cyanide poisoning is probable in this case. Some further cases have only been mentioned in a critical overview on Laetrile as cancer cure.*

Hill, 1942

A young female swallowed a quantity of SNP in water, possibly 5 grams. Death seems to have occurred after about 1 hour. The post mortem status resembled cyanide poisoning. Cyanide was found in the stomach contents (0.013 g). *Comment: A case of severe poisoning without use of antidote.*

Hillman et al, 1974

A 68 year old man accidentally drank from a bottle filled with a sodium-cyanide solution. He collapsed 3 minutes later and was found by the GP 15 minutes later cyanotic with respiratory difficulties. 55 minutes after the incident the patient was brought to the emergency department with apnoea, a pinkish complexion and acrostic. Due to these signs and symptoms, cyanide intoxication was suspected and the man was treated with CPR, sodium-bicarbonate and 300 mg of Co-EDTA. In a blood-sample taken before the therapy was started, the cyanide level could be measured at 5.5 mg/l (results were only available later on). Immediately after the treatment the vital signs stabilised (pulse palpable with sinus-tachycardia, systolic blood-pressure of 60 mm Hg), but the apnoea persisted. The antidote was given twice again after 10 and 15 minutes. However, this did not result in a clinical improvement. Gastric lavage and mechanical ventilation were initiated 2 hours after the exposure. 30 minutes later the patient regained spontaneous breathing. After a further hour he started hyperventilation resulting in an alkalosis (pH 7.47), hypocapnia (pCO_2 17 mm Hg) and an increased pO_2 (95 mm Hg). The systolic blood pressure decreased from 90 to 60 mm Hg and ectopic cardiac activity could be seen in the ECG. These symptoms led to a fourth administration of Co-EDTA followed by a further drop of the blood pressure and a reactive tachycardia. 10 hours after the intoxication the antidote was given a fifth time because of a new respiratory arrest. No reaction to the therapy could be observed so mechanical ventilation was initiated again. A sixth antidote-dose was injected 12 hours after the incident and after that 9 doses had been given, one every 30 minutes. Despite of all these efforts the patient died of cardiac arrest 44 hours after the intoxication. The cyanide levels were measured 4 times during the trearment and were always below 1.0 mg/l. *Comment: The blood cyanide level on admission was 5.50 mg/l which decreased to 0.3 mg/l after administration of 1,800 mg of Co-EDTA over 12 hours. In total there was a massive overdose of Co-EDTA administered, which may have been the cause of the fatal outcome.*

In an addendum, Hillman et al (1974) reported another case of a patient admitted after ingestion of a small amount of NaCN who collapsed several minutes later but without loss of consciousness. He received one ampoule of Co-EDTA and another one 4 hours afterwards. He then developed diaphoresis, anginal pain, ventricular extrasystoles, and intense nausea and vomiting. The antidote was stopped and the symptoms disappeared. These symptoms were attributed to Co-EDTA. 36 hours afterwards, although he was well, he developed a red maculo-papular rash on the thorax and the arms which cleared over the following day. This eruption was attributed to the antidote. *Comment: In this mild poisoning case the side effects of Co-EDTA were in the foreground.*

Hoang The Dan et al, 1981

A female chemical assistant (25 years old) attempted to commit suicide with KCN. 30 minutes after intake she was in deep coma, showed gasping respiration with intermittent apnea, bilateral mydriasis, pupils reactive to light and hypotonia with 80 mm Hg systolic blood pressure. She was ventilated with 100% oxygen and 2 mg of HOCO was administered. When she reached the ED 30 minutes later no reflexes could be triggered and the pupils were not reactive to light. Blood pressure and heart rate could not be detected and the ECG showed alternating brady- and tachy-cardia (cardio-circulatory instability / failure). The treatment continued with ventilation, application of 500 ml plasma *(resulting in acute heart insufficiency, treated with dobutamine)*, sodium bicarbonate as well as 600 mg of Co-EDTA without glucose and infusion of STS *(20 g infused during the next 12 hour*s). The blood gases showed severe acidosis (pH 7.08, anion gap 33 mmol/l). The levels normalised within 4 hours after the antidote administration. The cyanide blood levels were determined as 5.25 mg/lupon admission and 0.5 mg/l after 16 hours. The patient could be extubated after 12 hours as an in-patient and recovered completely. *Comment: A case of severe poisoning in which Co-EDTA and STS were fully effective. The very low HOCO dose was ineffective.*

Humbert et al, 1977; Braico et al, 1979

A 11-month-old child ingested from one to five 500 mg amygdalin tablets used by her father for cancer treatment and became listlessness and vomited after half an hour. In hospital breathing became irregular, and somnolence set in. 1 hour after ingestion, the girl was in shock and coma, and had Kussmaul (laboured and deep) respirations and acidosis. The level of cyanide in blood was determined as 0.29 mg/l. 3.5 hours after ingestion of 1.5 ml SN (3%) and 6 ml of STS (25%) were given a maximum MetHb of 20% and improving signs and symptoms was observed. However, 14 hours after ingestion respiratory arrest occurred, and the child died after 72 hours in spite of 2 more doses of SN (0.8 ml 3%) and STS (3 ml 25%). Before the respiratory arrest no cyanide and no MetHb were detectable in blood, and after the first re-treatment MetHb was only

1%. *Comment: As the initially measured cyanide level was not in a life-threatening range, and as no cyanide was detectable when the deterioration occurred, it seems doubtful that this death was indeed due to amygdalin/cyanide. Methaemoglobin can also be ruled out as cause of death.*

Humphrey, 1978

A 66 year old woman with severe hypertension received SNP infusions. After a total of 490 mg and 28 hours the patient became agitated and tachypnoeic. SNP was discontinued after lactic acidosis was found. She recovered uneventfully after bicarbonate application. *Comment: No cyanide antidote was given in this assumed cyanide poisoning from SNP.*

Hung et al, 2009

A 32 year old Taiwanese woman drank from a soda-bottle that had been contaminated with KCN, either with criminal intent or accidentally. At once she lost consciousness and tendon reflexes, and had to be intubated and ventilated. Her oxygen-saturation could be improved from 79% at the beginning to up to 100%. She exhibited alkalosis (pH 7.846, *possibly due to artificial hyperventilation and a base excess of 26 mmol/l*). 1 hour later her blood pressure dropped to 85/46 mm Hg and she became acidotic (pH 7,096, BE 21mmol/l, lactate 14 mmol/l and anion gap 37.6 mmol/l). She was given 2.5 g HOCO which led to a quick improvement, however, the antidotal therapy was repeated 1 hour later because acidosis persisted. The cyanide level in the blood was measured once at 288.6 µmol/l corresponding to 7.8 mg/l. The woman could be extubated the next day and made an uneventful recovery. *Comment: In this case of severe poisoning HOCO was effective.*

IPCS, 1993a

Four cases are specified in "Antidotes for Poisoning by Cyanide (Volume 2)" without citing the sources. 2 cases of cyanide intoxication (cyanide uptake by ingestion) were confirmed by measurements of the blood levels (9.9 mg/l and 3.7 mg/l respectively). In the third case, also cyanide uptake by ingestion, the cyanide concentration after therapy with STS was 0.26 mg/l. The fourth case (two 2.5 year old twins) was a combined cyanide and carbon monoxide intoxication with cyanide levels of 1.15 mg/l and 1.1 mg/l respectively and carbon monoxide concentrations of 5.7% and 1.4%. All cases were successfully treated with STS; no sequelae are described.

IPCS, 1993b

A 32 year old woman who suffered from a hemangioblastoma received Laetrile treatment while in Mexico and arranged for a continuing supply of Laetrile in Canada. In a suicidal attempt she ingested 9 g of the parenteral preparation of Laetrile. On admission at the hospital she revealed severe metabolic acidosis and hypoxemia. Her clinical condition and her blood gases were not stated in the paper, but the cyanide levels (3.85 mg/l) were in the lethal range. She was treated with AN by inhalation and 300 mg of SN i.v., then a further 50 mg of SN was given i.v. and 50 mg orally. The patient survived. *Comment: A case of mild poisoning in which SN treatment was effective without side effects.*

Jacobs, 1984

The lower part of a male worker's body was splashed with liquid HCN and the worker inhaled the gases evolved. When he wanted to leave the room he fell into a coma and had to be rescued by first-aid staff. The contaminated clothes were removed. He did not show any signs of spontaneous respiration, pupils were not reactive to light, pulse was weak and fast and blood pressure was low. The cyanide level in blood was 6.5 mg/l in the first evaluation rising to 16.9 mg/l after 45 minutes and dropping to 3.0 mg/l after 4 hours. The treatment consisted of ventilation and one ampoule of DMAP i.v. together with 60 ml of a 25% STS solution (12.5 g). The patient started to breathe spontaneously 2 minutes after the DMAP administration. At first he recovered quite well (pupils reactive to light again, reaction to pain positive, spontaneous breathing through tubus) but 2 hours later he developed cerebral seizure activity. Because of that he was anaesthetised and ventilated. He was diagnosed with a 'non-spontaneous transitory [psychotic] syndrome' (*German: 'Aspontanes Durchgangssyndrom'*). He stayed in a coma for 4 weeks and after neurological rehabilitation some permanent CNS-sequelae such as psychomotoric retardation and organic psychosyndrome remained. *Comment: In this case of severe intoxication by both dermal and inhalation exposure in which DMAP and STS initially showed very good efficacy. The author gave no reason for the secondary sequelae-development which may be due to delayed absorption of HCN through the skin.*

Jaeger et al, 1977

This is a case of a suicidal ingestion of 40 g acetonitrile by a 26 year old man. Symptoms appeared after a 3 hour delay and included vomiting and convulsions followed by coma with respiratory insufficiency, severe metabolic acidosis (pH = 6.40) and a state of shock with 2 recovered cardiac arrests. After 10 hours he was treated with oxygen ventilation, correction of shock and acidosis, and administration of the antidotes Co-EDTA, SN, STS, and HOCO. The notable later course of events was a coma for 6 days and a syndrome of multi-organ failure.

Muscle enzymes were especially elevated and histological examination showed a process of muscular destruction followed by regeneration. The very slow elimination of acetonitrile is confirmed by the persistence of thiocyanate in the urine 20 days after intoxication. The patient recovered fully after three months. *Comment: A case of severe poisoning in which the combined administration of Co-EDTA, SN, STS, and HOCO did not improve the neurological status for 6 days. Despite multi-organ failure the patient survived.*

Jamali, 1993

A 27 year old male presenting apnea episodes that was treated with oxygen, HCO3, dobutamin and administered 4g of HOCO and 8g of STS but did not survive. Personal communication with Prof. Baud.

Jeannin et al, 1961

A 4 year old girl was found with headache, fatigue and increasing somnolence. Upon arrival in hospital, she was in a coma, sweating, breathing slowly, without palpable pulse and with tonic contractions of the extremities. Gastric lavage revealed fruit pits and a bitter almond smell. She had played with her sister and prepared a mixture of about 30 ground peach pits with sugar which she ate about 30 minutes before the onset of the symptoms. She was treated with oxygen and 50 ml methylene blue 1%, which led to full recovery. *Comment: Cyanide in blood was not analysed, but cyanide poisoning is likely due to thesmell of bitter almonds. It remains unclear whether the methylene blue was effective beyond the oxygen treatment.*

Jerretin, 1963

A 24 year old man ingested 1.5 g of KCN in a suicide attempt. He arrived in hypertonic coma with mydriasis, cardio-vascular collapse (blood pressure 50 mm Hg and pulse 100/minute) and respiratory depression. He was intubated and ventilated with 100% oxygen, and underwent gastric lavage. He received 200 mg of SN 1 hour after ingestion, a second dose of SN combined with STS 5 hours and 30 minutes after ingestion as well as one ampoule (about 20 ml) of Co-EDTA six hours after ingestion. The next day he received another ampoule of Co-EDTA. The coma cleared after injecting the Co-EDTA. The patient remained amnesic until the injection of the second ampoule of Co-EDTA. He finally recovered without sequelae. *Comment: A case of severe poisoning in which despite of application of several antidotes recovery took more than a day, so efficacy of the antidotes remains doubtful.*

Johnson and Mellors, 1988

A 30 year old male laboratory supervisor presented to the ED with stupor and a rapid, irregular pulse. The patient ingested approximately 24 ounces of beer, 35 mg clorazepate dipotassium, 7.5 g of acetoaminophen, and 3 g granular NaCN. Shortly after the ingestion of the cyanide the patient vomited and called an ambulance. Ipecac was administered and he vomited again. Initial vital signs were blood pressure 90/60 mm Hg, pulse irregular at 160/min, and respirations 20/min. No focal neurological findings were observed. No bitter almond odour was detected. An arterial blood gas on 100% oxygen was pH 7.35, pCO_2 39 mm Hg; a simultaneous venous blood gas drawn from the median antecubital vein was pH 7.35, pCO_2 22mEq/l. An ECG showed atrial fibrillation at a rate of 160/min and ST segment depression in the inferior and lateral leads. 2 ampoules of AN were administered by inhalation, followed by 0.3 g of SN i.v. and 12.5 g of STS. A few minutes later the patient became alert and conversant. Gastric lavage was performed and magnesium citrate and charcoal were instilled. 4 hours after ingestion the serum thiocyanate level was 1.5 mg/dl, MetHb level was 3.6%, ethanol level was 56 mg/dl and the acetaminophen level was 28 µg/ml. *Comment: A case of moderate poisoning in hich the 'Lilly-Kit' was fully effective.*

Johnson et al, 1989

A woman, aged 24, attempted suicide with an unknown amount of KCN after a domestic dispute. She lost consciousness, developed a metabolic acidosis and was unresponsive to all stimuli. Blood pressure, heart rate and respiration were all within the normal range. The therapeutic actions included administration of 2.0 mg of naloxone, intubation, ventilation, AN pearls, 300 mg of SN (followed by a mild decrease of the blood pressure from 124/80 to 100/80 mm Hg) and 12.5 g of STS. Methaemoglobin levels were 7.1% during inhalation of AN and 9.2% about 10 minutes after injection of SN, decreasing to 3.6% about 5 hours, and 0.2% about 18 hours, post SN. The patient became alert within 15 minutes and recovered completely (gastric lavage was additionally performed and activated charcoal was administered). 45 minutes after antidotal treatment the pH had normalised (7.4) and the MetHb was still below 10% (exactly: 9.2%). This decreased further (to 0.2%) during the following 18 hours. The cyanide level was determined once before the nitrite application as 13 mg/l (*surprisingly high in contrast to the mild clinical presentation*) and dropped to undetectable levels by the next day. The patient was extubated and transferred to the psychiatric service. She was seen in follow up three months later with no physical or neuropsychiatric sequelae. *Comment:* A *case of severe poisoning in which the 'Lilly-Kit' was effective and caused only minor side effects (blood pressure drop).*

Jones, 1992

A 48 year old man and a 53 year old female were found dead at their home. Both victims had vomited during the night. Circumstantial evidence indicated that the couple might have ingested ethanol and/or methanol. But analytical examinations identified acetonitrile at concentrations of 0.8 g/l in blood, 1.0 g/l in urine, and 1.3g/l in stomach contents. The cyanide concentrations in the victims were 2.4 mg/l (female) and 4.5mg/l (male), respectively. *Comment: a case of severe poisoning without the use of antidotes.*

Jouglard et al, 1974

A 34 year old male chemist attempted suicide by ingesting 1.5 g of KCN. He arrived at the hospital in deep coma with mydriasis and hardly palpable pulse. He was given 3 g of HOCO (one quarter of the ampoule lost during its manipulation) in STS. 10 minutes later he reacted to pain stimuli. Several sudden episodes of cyanosis occurred prompting intubation and mechanical ventilation. Severe coma reappeared, so a further 4 g of HOCO were given finally prompting recovery. *Comment: A case of sever poisoning in which the combination of HOCO and STS improved the neurological status; a second dose was fully effective.*

Jourdan et al, 1993

After suicidal poisoning with 800 mg of KCN, a 28 year old was diagnosed with cerebral oedema and bilateral mydriasis. 5 hours after poisoning he received antidotal treatment combined with 100% oxygen, 0.6 g of Co-EDTA, and 4 g of HOCO (probably with 8 g of STS) followed by 10 hyperbaric oxygen treatments, but the clinical outcome was a persistent vegetative state which still persisted four years later. *Comment: A case of severe poisoning in which the neurological situation after cerebral oedema could not be improved by the antidotes.*

Kampe et al, 2000

A young man, aged 29, drank 12 bottles of beer in which he had solved 1250 mg of KCN. 10 minutes later he was in a coma, tachypnoeic (respiration frequency 35-45/min), with dilated but reactive pupils, and only three points on the GCS. The blood pressure was low (80/60 mm Hg) and the heart rate was high (95/min). He was intubated and ventilated immediately. Intravenous fluids were infused and 250 mg of DMAP was administered together with 9 g of STS. since the ambulance staff had found remains of the cyanide powder and the clinical presentation fitted to a cyanide intoxication (respiratory insufficiency without signs of cyanosis). The blood cyanide level was 6.9 mg/l (results available only hours later). The MetHb-level was measured 2 hours

after the specific treatment with the MetHb-forming agent as 9.9%. The metabolic alterations were comparatively mild (pH only 7.3). Additionally, gastric lavage was performed and activated charcoal, laxatives and furosemide were administered amongst others. The blood ethanol level was 270 mg/dl (2.7 g/l). 2 days after the suicide attempt the patient could be discharged in a good state of health. *Comment: A case of severe intoxication in which DMAP and STS was effective.*

Kasamo, 1993

A 31year old male technician in an electroplating factory, who had been suffering from temporal lobe epilepsy for 24 years and from hypertension for 2 years attempt to commit suicide by taking an unknown amount (apparently above the lethal dose) of KCN. He was treated successfully and survived without any neurological sequelae. The electroencephalograms and the nature of the seizures were not different before and after poisoning. The T_2-weighted magnetic resonance images at 9 and 51 days after poisoning showed bilateral elevation of signals in the caudate nuclei and the putamina. On the 143^{th} and 286^{th} days, T_2-weighted high-resonance areas were restricted to the lateral portion of the putamina. The T_1-weighted images on the 51^{st} day showed abnormal signal elevations in both putamina, while those on the 9^{th}, 143^{th} and 286^{th} days were mainly normal. Selective vulnerability of the putamen and the caudate nucleus may be due to their specific structural properties of high oxygen and glucose utilisation, and enzyme distribution. Both chronological changes of striatal damage and the absence of neurological sequelae in this patient suggest the possibility that anti-epileptics and a calcium antagonist played a neuroprotective role in the acute cyanide intoxication.

Kim et al, 1982

A 43 year old man received SNP infusion for hypertensive encephalopathy and renal insufficiency. A total dose of 2,878 mg was applied over 14 days. Peritoneal dialysis was performed initially, haemodialysis from day 9 onwards. On days 4 and 8 brief episodes of respiratory arrest occurred with spontaneous recovery. When not on SNP momentarily, a third respiratory arrest occurred on day 11. In spite of resuscitation he fell into a coma. One day later resuscitation from cardiac arrest was successful. After bilateral nephrectomy on day 15 no further SNP was required. The patient remained semi-comatose to comatose except for 4 lucid days (d 25-29), and he died on day 47. Though there were no cyanide measurements, cyanide encephalopathy was diagnosed at autopsy. *Comment: This case of encephalopathy, which the authors relate to cyanide toxicity from SNP treatment, is dubious. A cyanide antidote was not given.*

Kirk et al, 1993

Case 1: A 20 year old man was recovered from a fire scene. The paramedics found him unconscious and in convulsions. 15 percent of the body surface was burned. The heart rate was 110 beats/min. The first laboratory results showed a blood pH of 6.69, a COHb of 4.9% (3 hours later 0%) and a cyanide level in blood of 130 µmol/l (3.48 mg/l). 10 hours later the cyanide level had dropped to 22 mol/l (0.59 mg/l) and another 4 hours later to 10 µmol/l (0.27 mg/l). The patient was intubated and received 0.3 g of SN plus 12 g of STS after the laboratory results were available. 4 hours after being discovered the patient received this treatment which was repeated 6 hours later due to persistent metabolic acidosis. The highest methaemoglobin level after the antidotal therapy was 8-9% which normalised within 6 hours. The patient recovered completely. No severe side effects were reported. *Comment: In this case of severe smoke inhalation SN/STS were effective without any side effects.*

Case 2: A 39 year old man was found lifeless after exposure to smoke. He was treated with SN plus STS after he had received successful CRP. He stayed in apnoea, developed tachycardia (heart rate: 140/min) and exhibited metabolic acidosis (pH 7.17). The COHb amounted to 30% at the beginning. It normalised after 5 hours. The blood cyanide level amounted to 3.16 mg/l and was no longer detectable after 5 hours. Methaemoglobinemia peaked 2 hours after the administration of SN at 10%. The patient died 6 days later due to hypoxia of the brain. *Comment: A case of severe hypoxic brain damage causing the patient's death, probably before antidotal treatment was commenced,. The antidotes could not improve the cerebral situation.*

Case 3: A 64 year old man was found after smoke inhalation. Nothing is stated about his consciousness. The heart rate was 154/min, the respiratory rate 30/min. COHb amounted to 22% and normalised after 7 hours. Cyanide levels in blood were 0.96 mg/l at the beginning and fell to 0.5 mg/l after the next 6 hours but could no longer be detected after 13 hours. The maximum MetHb level reached 10% 1 hour after administration of the Lilly-Kit. A bundle block was seen in the ECG. The patient was put on artificial respiration after intubation. He survived and could be discharged after 5 days. *Comment: With missing data this appears to have been moderate poisoning in which the 'Lilly-Kit' was effective.*

Case 4: A 26 year old woman was found without reaction after exposure to smoke. She was tachycardic (heart rate: 106/min) and showed metabolic alkalosis (pH: 7.45). COHb was 18.3% at discovery and 7% two hours later. Ethanol in blood was measured with 79 mmol/l cyanide with 0.7 mg/l at first and was not detectable after 9 hours. Methaemoglobine reached 10% 1 hour after the application of the Lilly antidote and normalised 15 hours later. The patient recovered completely. *Comment: This was no significant cyanide poisoning, so the antidote efficacy cannot be judged.*

Case 5: A 29 year old man was exposed to a fire. 25 percent of his body surface was burned. Obviously he was not in a coma, showed tachypnoea, tachycardia and hypothermia. Laboratory results gave a carbon monoxide level of 28.7% in haemoglobin, an ethanol level of 60 mmol/l (270 mg/dl or 2.7 g/l) in blood and a cyanide level of 1.5 mg/l in blood. The patient received the nitrite antidote due to persistent metabolic acidosis and recovered completely. *Comment: A case of mild poisoning in which SN/STS seems to have been effective.*

In this case series two more patients were included. They were not treated with any antidote. One survived on supportive treatment only, the other one survived with severe neurological sequelae.

Kläui et al, 1984

Case 1: An 18 year old male ingested 300 mg of KCN and arrived in hospital in coma with a blood pressure of 100/70 and respiratory failure. After intubation he received AN. 1 hour after ingestion 0.6 g of Co-EDTA were injected, followed by another 0.3 g after which he awoke. In addition he received 10 g of STS and 0.3 g of SN. This caused a drop in blood pressure requiring catecholamines. 10 g of HOCO were given as infusion. The patient recovered fully. *Comment: Improvement was achieved by Co-EDTA, the hypotension was probably due to the SN, and HOCO did not contribute to the positive course, as given after neurological recovery. Except for hypotension from SN no side effect occurred.*

Case 2: The 16 year old friend of case 1 ingested 900 mg KCN. He had to be resuscitated upon arrival in hospital; however the pupils remained wide and fixed. High catecholamine doses could not bring the blood pressure higher than 60/30. 600 mg of Co-EDTA initially stabilised the blood pressure; after a recurring drop 300 mg of Co-EDTA were given again. This did not change the neurological status. 10 g of STS and 0.3 g of SN were given, then 10 g of HOCO. The patient had to be declared brain dead. *Comment: The 4 antidotes given could not save the patient after initial cardiac arrest.*

Case 3: A 24 year old man ingested an unknown amount of KCN. He was resuscitated after cardiorespiratory arrest by lay people until hospital arrival. He was still in respiratory arrest, in deep coma and had wide fixed pupils. In spite of catecholamine application the blood pressure could not be measured. 0.3 g of SN and 12.5 g of STS were without effect. 0.6 g of Co-EDTA normalised the blood pressure, due to electro-mechanical dissociation. An extra 0.3 g of Co-EDTA was given. No neurological improvement could be seen. 0.3 g of SN and 12.5 g of STS were given again. This patient, too, had to be declared brain dead. *Comment: The three antidotes given could not save the patient in the end. After initial cardiac arrest, SN/STS could not reverse the cardiocirculatory problems.*

Klöver and Wenderoth, 1965

A 20 year old student bought a bag of almonds which contained only bitter almonds. Though it tasted bitter she ate 30-40 pieces. A few minutes later she felt sick, developed a headache, angina pectoris and nausea. Though she felt very weak, she made her way home (1 hour) and became unconscious 40 minutes after the purchase of the almonds. She arrived at hospital by ambulance. At admission her skin was rosy, her respiration was fast and deep but sometimes stopped. The pulse rate was 92/min, blood pressure with 110/70 mm Hg, she was unconscious with seizures and smelt of bitter almonds. A gastric lavage was performed. The patient was treated with an i.v. injection of 0.3 g of SN solution (10 ml/3%) and 10 g of STS (100 ml/10%). As the respiration was insufficient, she got analeptica and had not to be intubated. 20 minutes after the antidote therapy the patient was reactive, after 50 minutes she was awake. *Comment:* A *case of moderate poisoning in which SN/STS was efefctive.*

Krefft, 1955

2 cases due to inhalation have been reported in Germany. Acetone cyanohydrin (ACH) was spilled in a plant and immediately evaporated, the condensate showed 20% HCN. A worker collapsed while trying to escape, another worker tried to help and collapsed, too. They were found 15 minutes later unconscious on the floor – one recovered fully, one died upon arrival in hospital 1 hour later. *Comment: 2 cases of severe poisoning, presumably to HCN gas, although levels of ACH or cyanide were not measured and no antidote had been given.*

The same author also reports percutaneous poisoning in a 51 year old woman. 19 litres of acetone cyanohydrin (ACH) were spilled when a glass container broke, and they splashed on her face and her clothes, which were not fully changed. 5 minutes after the accident she vomited, had dyspnoea and became comatose; 10 minutes after the incident she had convulsions. She died upon arrival in hospital. *Comment: A case of severe poisoning although levels of ACH or cyanide were not measured and no antidote was given.*

Krieg and Saxena, 1987

Case 1: A young man aged 23 accidentally drunk from a juice-bottle containing a metal cleaning solution (6.2 mg/ml cyanide). The ambulance found a comatose man with 100-120 heartbeats/min, a respiration of 30/min and a blood pressure of 80/40 mm Hg as well as a severe metabolic acidosis (pH 703 and pCO_2 17 mm Hg). The oxygen-saturation was 95%. He coughed up pink sputum, the colour of his skin was pink and a pulmonary oedema could be seen in the chest-X-ray. The supportive treatment consisted of intubation and ventilation, gastric lavage, naloxone, dextrose, furosemide i.v. and morphine. The specific therapy was undertaken

with the nitrite antidote kit (0.3 g of SN and 12.5 g of STS) and was repeated after 5 hours with half the initial dose. 1 hour after the second antidote-application the patient regained consciousness and became responsive. Independently a haemoperfusion was applied for 4 hours. While it was performed the young man could be extubated. He could be discharged on the fourth day after the accident in complete remission. *Comment: A case of moderate poisoning in which SN/STS was effective.*

Case 2: A 2.5 year old girl who had drunk from a metal cleaning solution (6.2 mg/ml cyanide) was comatose, with a blood pressure of 90/30 mm Hg, tachycardia (120/min) and a tachypnoea (28/min). A smell of bitter almonds could be detected. She received AN pearls and her health state improved even before being administered a reduced dosage of the nitrite – antidote kit (76 mg of SN and 2.5 g of STS). Thereupon the patient immediately regained consciousness and was discharged with no sequelae 2 days later. *Comment:* A case of *moderate poisoning in which AN prompted partial improvement and SN/STS were fully effective.*

Küpper, 2009

Case 1: In this case of hydrogen cyanide intoxication (skin absorption and inhalation), the cyanide level prior to therapy was 1.5 mg/l. 4 hours after application of 12.5 g of STS the cyanide level was 0.12 mg/l and 24 hours later the intoxication cyanide was not detectable in the patient's blood. The patient recovered clinically very fast and uneventful after therapy. Neither side effects of the therapy nor sequelae were observed. *Comment: As the clinical signs have not been reported, it is unclear what degree of poisoning was effectively reversed by STS alone.*

Case 2: Acetone cyanohydrin was taken up by inhalation and skin absorption in the second case. Initially the cyanide blood level was 3.5 mg/l. 5 hours after therapy with 12.5 g of STS, the level decreased to 0.69 mg/l and 24 hours after intoxication it was lower than 0.1 mg/l. After application of STS clinical parameters, the patient's health, improved quickly. Neither side effects nor sequelae were observed after the therapy. *Comment: As clinical signs are not reported, it is unclear what degree of poisoning was effectively reversed by STS alone.*

Case 3: Likewise the exposure to a combination of acetone cyanhydrin and hydrogen cyanide was the cause for the third intoxication case. The initial cyanide level was 2.07 mg/l. 30 minutes after therapy with 12.5 g of STS the level decreased to 0.42 mg/l. Then 125 mg of DMAP were administered. Measurements of the cyanide levels performed subsequently showed levels lower than 0.2 mg/l. The therapy was followed by a quick improvement of the clinical status. No side effects of the therapy or sequelae were observed. *Comment: As clinical signs are not reported, it is unclear, what degree of poisoning was effectively reversed by STS and DMAP.*

Kurt et al, 1990

A two year old child (15.8 kg) ingested 5 to 10 ml of liquid containing 84% acetonitrile. In the ED she was asymptomatic and was discharged. 12 hours later she awoke restless, vomiting and this was followed by 3 seizures 2 hours later. Upon arrival in hospital she was comatose with a respiratory rate of 36 and pulse rate of 136, blood pressure 104/89 and marked acidosis. She received oxygen and the 'Lilly-Kit' (3.3 ml SN 3%, 17 ml STS 25%). She awoke within minutes and fully recovered. *Comment: A case of severe poisoning in which SN/STS were fully effective.*

Lam and Lau, 2000

Case 1: A young lady, aged 19, who was working as a secretary in a chemical-trading company, stayed in a room in which HCN had been liberated. After being in there for 4 hours, she collapsed and was brought to the ED. There she presented a blood pressure of 77/65 mm Hg, a heart rate of 120/min and only 3 points on the GCS. The BGA showed severe acidosis (pH 7.07), a bicarbonate concentration of 11 mmol/l, a base excess of 18.4 mmol/l and an anion gap of 42.6 mmol/l. Treatment included oxygenation, intubation, application of sodium bicarbonate (to combat the metabolic acidosis) and administration of the nitrite antidote kit (AN for inhalation and 50 ml of a 25% STS solution (12.5 g) i.v.). As the blood pressure was quite low SN was withheld. A short time after the antidote administration the patient developed cerebral seizure activity that could be controlled with midazolam and phenytoin. The patient regained consciousness soon afterwards but short-term memory and retentiveness problems persisted. *Comment:* A case of *severe intoxication in which STS (and AN) showed efficacy although, the patient retained memory problems for unknown reasons. No BCN determination was reported.*

Case 2: A physicist (the owner) of the company for which Case 1 was working rescued her and stayed in the environment for 10 minutes. He was admitted to the ED with severe dizziness, near syncope, tachycardia (heart rate 110/min) and tachypnoea (AF 25/min), with a GCS of 14. Under oxygenation with 100% oxygen the blood pH was 7.58. As he developed these symptoms after one of his employees had been intoxicated by cyanide gas he was preventively given the nitrite antidote kit (AN for inhalation, 10 ml of a 3% solution of SN and 50 ml of a 25% solution of STS(). He recovered without any complications and was discharged home three days later. *Comment:* A *case of mild poisoning in which the intense antidotal treatment consisting of the whole nitrite antidote kit was actually not indicated althoughthe overtreatment resulted in a prompt and complete recovery and does not seem to have caused any adverse effects.*

7 cases of hydrogen cyanide gas poisoning which occurred in an industrial building in Hong Kong are presented here. 2 of them were more severely injured and required specific antidotal treatment. The other 5 were mild and responded to supportive treatment alone. All

patients except one recovered completely. Cyanide poisoning was reported to be relatively uncommon in urbanised area, so high index of suspicion is important for early diagnosis and treatment. The authors believed that prevention of cyanide poisoning can be achieved by proper storage of chemicals, and by enforcing rescuers to wear special chemical protective clothing to avoid systemic poisoning because of dermal absorption of hydrogen cyanide gas. They recommendedthat as there are newer and safer cyanide antidotes available, each emergency department should have a stock of updated products such as HOCO.

Lang and Stintzy, 1960

Case 1: This is a French report on a 19 year old man having worked with acetone cyanohydrin and developing headaches and nausea/vomiting at work. He went home, and after further deterioration (opisthotonos) he was taken to hospital. 3 hours after leaving work he arrived there in deep coma with cyanosis, hypotension, and slow, deep respiration. Later tonic-clonic seizures set in but recovery followed. *Comment: No specific treatment was done, as the possibility of acetone cyanohydrin poisoning was stated only one day after the incident, when the patient was already recovering. No measurements of cyanide in blood were done.*

Case 2: A further case has been reported in a 23 year old laboratory worker filling acetone cyanohydrin from a rail tanker into steel drums. One of his cotton gloves was drenched, which he did not notice. He put the glove into his pocket. 5 minutes later nausea set in, vomiting occurred, and 10 minutes afterwards he lost consciousness and collapsed. AN and artificial respiration led to short improvement, but then coma and convulsions set in. The use of SN and STS also improved the status for a few minutes only. After the glove had been found, clothing had been changed, and a decontamination bath had been performed, SN and STS led to an immediate and persistent improvement [Cited by Thiess and Hey, 1969]. *Comment: The initial non-satisfactory efficacy of AN/SN/STS was due to continued absorption. After decontamination, the antidotes SN/STS were fully effective.*

Lasch and El Shawa, 1981

Two episodes of cyanide poisoning occurred in children after ingestion of apricot kernels. The first episode involved eight children who exhibited typical signs and symptoms of cyanide poisoning two hours after having ingested a large amount of apricot kernels. Seven children recovered. One died soon after admission. The second episode involved 16 children who had eaten a sweet prepared from such kernels. The symptoms and signs were identical with those in the first group but appeared one-half hour after the ingestion and were much more severe. Thirteen children recovered, two died shortly after admission, and a third child died two hours later. Apricot kernels contain a cyanogenetic substance called amygdalin, which after hydrolysis,

liberates hydrocyanic acid. This activation usually occurs only after ingestion. In the second instance hydrolysis probably occurred during the preparation of the sweet, explaining the short interval between the ingestion and the appearance of the signs of poisoning.

Lawson-Smith et al, 2010

Case 1: One patient was removed from a fire in a house. The COHb in blood was 40%, the lactate 8.2 mmol/l. Ethanol could be found in blood. He was intubated, sedated and ventilated and brought to hospital. On admission the following laboratory parameters could be determined: pH 7.33, COHb 17%, lactate 4.2 mmol/l, cyanide 58 µmol/l = 1.5 mg/l. The patient received an unspecified dose of HOCO prior to 5 cycles of hyperbaric oxygen-treatment. After the first turn of hyperbaric oxygen therapy the COHb had dropped to 2.8%. Cyanide blood levels had gonee down to 28 µmol/l (0.76 mg/l) and to 23 µmol/l (0.62 mg/l) after 24 hours respectively. The patient was extubated on day five and recovered completely.

Case 2: A severely injured patient was rescued from a burning apartment. He had 2-3 degrees of burns and only a GCS of 3. He was intubated and ventilated. On admission to hospital the COHb amounted to 33.5% and blood lactate was up to 9.0 µmol/l. An unknown dose of HOCO was administered. Prior to hyperbaric oxygen treatment the blood pH was 7.32, COHb 10.2%, lactate 4.0 mmol/l and the blood cyanide level 39 µmol/l (1.0 mg/l). After the first hyperbaric oxygen cycle pH was 7.3, COHb 3.9%, lactate 2.5 mmol/l and cyanide 14 µmol/l (0.38 mg/l). Despite all this therapy the patient died 5 days after the incident in septicaemia, MOF, hyperthermia with irreversible cardiac arrest. *Comment: In both patients the COHb were not critical, and the cyanide levels comparable. However, the outcome was contradictory (death vs. recovery). As the HOCO dose and the clinical course of its application have not been not reported, the efficacy cannot be assessed.*

Lazarus-Barlow and Norman, 1941*

Case 1: A woman aged 23 showed a pulse rate of 132 and respiration of 26. She was drowsy, the pupil were slightly dilated. After stomach lavage she was unconscious. Her respirations became gradually slower and more laboured, the pulse rate had fallen to 45, the pupils were fully dilated, and trismus was present. She died within 1 hour. Postmortem analysis showed traces of HCN and SNP.

Case2: A young surgeon was found dead. He had taken a large dose of arsenic before SNP. It was suggested, that death must have taken place very quickly and that the arsenic played no role. *Comment: In both cases of severe poisoning the individuals were found dead and therefore no antidotes had been administered.*

Lee et al, 1982

An 18 year old man with schizophrenia and cancerophobia following an alleged nutritional and metabolic antineoplastic diet and health foods had ingested 30 tablets of Laetrile (3 grams) in a suicide attempt. He arrived in hospital deeply comatose, cyanotic, diaphoretic, with dilated pupils and hypotension. Gastric lavage was performed with activated charcoal, and he underwent mechanical ventilation with 100% oxygen. 5 hours after admission his status improved and he recovered without sequelae. Cyanide levels in blood were not measured. *Comment: No specific antidote was given.*

Lee-Jones et al, 1970

Case 1: A 25 year old man arrived at the accident and emergency department at midnight, deeply unconscious. He was pulseless and apnoeic, but the skin was warm and the mucous membranes were pink. He was in cardiac asystole and, because cardiac action was not restored by external cardiac massage and intravenous isoprenaline a transvenous bipolar pacemaker catheter was introduced into the right ventricle. Cardiac response to the pacemaker impulse was intermittent and he died 30 minutes after admission. Forensic examination showed cyanide in blood and tissues. *Comment: This case will not be part of the antidotal statistics as no specific antidote was given*

Case 2: A 35 year old man was found unconscious in the street. On admission in hospital he was deeply unconscious, with fixed dilated pupils and absent deep tendon reflexes. The blood pressure was unrecordable, the heart rate 40/min. He was not cyanosed, respiration was shallow and infrequent. Ventilation was manually assisted via a cuffed endotracheal tube. As the diagnosis was unclear at the time, lumbar puncture was performed. The patient went into asystole. Resuscitation with external cardiac massage, isoprenaline and a transvenous pacemaker restored circulation only temporarily. As at that time a suicide note with links to cyanide poisoning was found, 0.3 g of SN and 12.5 g of STS were given i.v. Nevertheless asystole recurred and the ventricle failed to respond to the pacemaker impulse. *Comment: The time that had passed between the intoxication and the specific treatment was not stated explicitly. It might have been long as the patient died a short time after the antidotal therapy because of cardiac arrest. This case describes fatal cyanide intoxication as treatment started too late. It points to the reality that after cardiac arrest reanimation does not seem possible even if nitrite is used. This may differ from DMAP where resuscitation under these circumstances was possible in four cases but not that did not occur without severe sequelae.*

Case 3: A 25 year old technician accidentally drank from a bottle about 1 g of KCN. An 18-minute treatment was started. The patient was drowsy, his heart rate was 120/min and regular, blood pressure 110/80 mm Hg, tachypnoea: 30/min. He was treated with inhalation of AN plus

0.3 g of SN, 12.5 g of STS i.v. Aspiration and lavage were then performed and a further 12.5 g of STS were left in the stomach. The patient survived uneventfully. *Comment: No cyanide levels nor MetHb levels after treatment and no blood gases are given. This makes the determination of the severity difficult. In terms of effectiveness of the antidote used it is not conclusive if nitrite changed the outcome, but early treatment is imperative.*

Case 4: A 14 year old girl either intentionally or accidentally drank a solution which was acquired for electroplating. The exact amount of cyanide in it stayed unknown. Twelve minutes later the girl was admitted to hospital. She was fully conscious with a regular heart rate of 120/min and a blood pressure of 110/70 mm Hg. She was treated with AN by inhalation, 0.3 g SN, 12.5 g STS i.v. and after gastric lavage with the same amount STS orally. The outcome was uneventful. *Comment: The only conclusion that can be drawn from this case is that the treatment did not do any harm.*

Leor et al, 1986

After i.v. injection of ampoules of 3 g each of amygdalin, followed by oral application of a third ampoule on the next day, a 65 year old woman developed diarrhoea two hours later, followed by progressive stupor and convulsions. In hospital she was in deep coma, and received oxygen 100% and antidotal treatment with SN (0.6 g) and STS (dose not specified). Within eight hours she improved and regained consciousness. However, she died 11 days after admission of liver failure, the underlying disease being hepatocellular carcinoma and cirrhosis. Cyanide in blood was 0.23 mg/l two hours after admission, so cyanide poisoning is certain. *Comment: The antidotes were primarily effective; death was due to hepatic necrosis on the basis of underlying disease and hypoxemia.*

Liebowitz and Schwartz, 1948

After taking an estimated 3-5 g KCN in a suicide attempt, a 60 year old chemist vomited once half an hour later and arrived in hospital one hour after ingesting the poison. At that time no history about the poisoning was available. He was comatose, did not react to painful stimuli and showed generalised muscular rigidity. Blood pressure was 120/80, pulse rate 140 and respiratory rate 40/min. Respiration was stertorous [heavy snoring], deep and rapid, with an expiratory grunt [deep guttural sound]. The skin was pink, cold and clammy, and the pupils were dilated. The pulse was very weak and the heart sounds were inaudible. Gastric lavage was performed immediately and an infusion of 1000 ml of 5% glucose was started. Half an hour later the patient regained consciousness. Two hours after admission the CO_2 combining power of the plasma was 25 volumes per 100 ml, and rose to 41 volumes on the third day. Blood cyanide determinations revealed 20 mg HCN/100 ml leading to an estimated total of 1.2 g of HCN in the total blood

volume. Thirteen hours later the tests revealed the presence of 0.25 mg HCN/100 ml of blood. A qualitative test for MetHb was negative, as well as the blood thiocyanate determinations on the first, second, fourth and fifth day. Free cyanide in the second twenty-four hour specimen of urine was found to be 0.116 mg in 1160 ml of urine. Eight hours after admission he was alert, well orientated and cooperative. His only complaints were weakness and nausea. The patient was discharged on the fifth day. *Comment (WM): After ingestion of an unusually large amount of KCN, the patient recovered promptly and uneventfully without any specific therapy.*

Litovitz et al, 1983

A 23 year old female chemistry student intentionally took a teaspoon of KCN and was brought in with seizure activity, tachycardia, mild hypotony (blood pressure 92/50 mm Hg) and clonic spasms of the extremities. The reaction to painful stimuli was preserved. Therapy was initiated about 30 minutes after the intake of the poison with oxygen, naloxone, dextrose, and as cyanide antidotes AN pearls were crushed and SN (0.3 g) as well as STS (12.5 g) were injected. A metabolic acidosis could be seen in the blood gases (determined immediately after treatment had begun, and moreover after 20 and 30 minutes) with a pH of 7.17, 7.26 and 7.36 respectively. One ampoule of sodium bicarbonate was given between the later two blood gas examinations. The pCO_2 also increased from 18 mm Hg via 33 to 45 mm Hg. Due to the dextrose infusion she developed hyperglycaemia with a blood sugar of 720 mg/dl. The blood cyanide level was measured once with 6.1 mg/l. Despite this high level and the clinical and metabolic alterations she recovered within two hours and later only complained about nausea and vomiting. *Comment: The Lilly-Kit was effective in this moderate poisoning case.*

Nineteen months later the same young woman was seen with tonic seizures after a collapse. She developed respiratory arrest and mechanical ventilation was initiated. The heart rate increased from 10/min (at arrival) to 110/min 17 minutes after admission. During this time span the cyanide antidote kit (AN pearls, 0.3 g SN and 12.5 g STS) had been administered. An odour of bitter almond could not be detected by several medical persons. Due to the circulatory distress, cyanosis and dilated, fixed pupils, she was intubated and CPR was performed. Dopamine was injected and caused the blood pressure to rise (80/50 mm Hg) and respiration recovered. Acidosis (pH 7.19) could be seen in the blood gas analysis. The coma persisted, so that the patient received multiple cycles of hyperbaric oxygen. The initial blood cyanide level (ca. 40 minutes after cyanide ingestion) was 683 µg/dl (68.3 mg/l) and dropped down to 12 µg/dl (1.2 mg/l) cyanide (two hours after the hyperbaric treatment began) with a corresponding MetHb level of 14.3%. Based on the fact that the results of the blood samples were only available eight hours later, a second dose of STS (12.5 g) was administered two hours after the hyperbaric treatment had begun. Despite all these efforts the woman died four days later because of multiple brain damages, as the authors suppose. *Comment: This severe poisoning case with respiratory*

arrest could not be reversed with the Lilly-Kit, and hypoxic brain damage was probably the cause of death.

Lorz, 1950

The death of a child was due to lice treatment with an acrylonitrile product. During autopsy a strong bitter almond smell was noticed. *Comment: As this child was found dead, this case cannot contribute to antidote evaluation.*

Losek et al, 1991

After ingestion of approximately 60 ml Super Nail Off (containing acetonitrile) a 23-month-old boy vomited three times post ingestion, but he did not seem to be ill. His blood pressure was 96/60 mm Hg, his pulse rate 144 beats/min, and his respiratory rate 40 breaths/min, blood pH 7.43 and anion gap 13. Eight litres/min of oxygen by mask was initiated and the patient was admitted. Twenty-four hours post ingestion the patient began having staring episodes and was not responding to his mother. Vital signs were heart rate 130 beats/min; blood pressure 110/50 mm Hg, respiratory rate 30 breaths/min. Oxygen saturation by pulse oximetry was 93% on 8L/min of oxygen mask. The low oxygen saturation prompted the administration of amyl nitrite, but without success. An arterial blood gas analysis revealed the following values: pH 7.43, PCO_2 26.4 mm Hg; pO_2 167.6 mm Hg, HCO_3 17 mm Hg, oxygen saturation 99%. Lactic acid concentration was 50.1 mg/dl and MetHb level was 0.7 g/dl. A later treatment included activated charcoal with magnesium citrate, intravenous sodium thiosulphate1.65 ml/kg every four hours. After two doses of STS, the lactate level decreased to 14.2 mg/dl (normal 4.0 to 15 mg/dl) and arterial blood gas values were pH 7.42, PCO_2 33.8 mm Hg, pO_2 435.8 mm Hg HCO3 21.8 mEq/l and oxygen saturation 99.9%. During his hospital stay, the patient received five doses STS every four hours. The patient was discharged on the third hospital day. → The blood cyanide levels were measured to be 2.1mg/l.

Lundquist, 1992

Case 1: A 28 year old young man tried to commit suicide by drinking 10 to 50 ml of a metal cleaning solution that contained 9% cyanide. He collapsed, vomited and fell in a coma. At admission to the ED one hour later he showed dilated pupils, the heart rate was 90/min, the systolic blood pressure 160 mm Hg and the blood gas analysis showed metabolic acidosis with a pH of 7,12 and a BE of -22,8mmol/l. He was intubated and ventilated and gastric lavage was performed. Moreover, sodium bicarbonate and STS (95 mmol) were applied i.v. This was followed by a tachycardia (140/min) with elevation of the ST segment on the ECG. About

30 minutes after he had ingested the poison he received 0.3 g Cobalt-EDTA. Although the drug was not combined with glucose as it is generally recommended, the patient did not develop any adverse effects, but became responsive 15 minutes after the administration of the antidote. The cyanide level was measured in a whole blood sample which contained 105 µmol/l (according to 2.84 mg/l) on the same day and 4.5 µmol/l (0.12 mg/l) on the next day. The plasma levels were 1.8 µmol/l, corresponding to 0.05 mg/l on the first day and 0.6 and 0.7 µmol/l, corresponding to 0.02 mg/l on the second and third day post exposition. Eleven hours after the incident he was extubated and recovered completely. *Comment: This moderate or severe case (depth of coma is unclear) was effectively treated with Co-EDTA and STS.*

Case 2: A 28 year old man was found comatose and convulsing. On admission at the ED one hour later he was intubated and ventilated because of insufficient respiration. Moreover, he showed dilated pupils, hypotonia (systolic blood pressure 65 mm Hg) and hypothermia of 34˚C. The blood pH was 7.11 and the BE -16.5 mmol/l. The patient was given naloxone and cortisone, though without any positive reaction. A brain CT showed small bleeding in basal ganglia and probable brain oedema (confirmed in a second CT, 12 hours later). About six hours after the incident KCN powder was detected in the man's flat. The blood cyanide level was 139 µmol/l (3.76 mg/l) at this time (samples were analysed on day 11 after the incident). Gastric lavage was performed and 120 mmol of STS (corresponding to about 30 g) were administered i.v. This resulted in prompt recovery, the patient regained consciousness and could be extubated, but 15 hours after the intoxication he developed – for whatever reason – diffuse cerebral oedema and died after five days. *Comment: This was a severe case of poisoning; however, the patient was – initially – effectively treated with STS alone. Cyanide levels were probably even higher shortly after ingestion, and the brain oedema had begun to develop early in the clinical course. The developing brain oedema could obviously not be reversed and progressed.*

Lurie, 1953

A man, aged 47, entered a room which had been fumigated with hydrocyanic acid. Two or three minutes after exposure, he temporarily lost consciousness but 15 minutes after the incident he was presented at the EC fully conscious. However, he was pale, bradycardic, with weak peripheral reflexes and complained about shortness of breath and nausea. He was given methylene blue, nitrites and calcium thiosulphate. Despite this treatment he lost consciousness again and became cyanotic a short time after the antidote administration. ACTH was then applied, resulting in a prompt improvement of his clinical condition. Three hours later the ACTH therapy was repeated and 12 hours after the intoxication the patient was completely orientated. According to the author no changes were detectable in the blood parameters. *Comment: The patient was initially given antagonistic antidotes (methylene blue and nitrites/calcium thiosulphate). Perhaps this was why no changes in his condition could be seen. The author then attributes the outcome of the case to ACTH, but he does not explain how this substance should be*

able to antagonise cyanide (ACTH increases the amount of suprarenal hormones). As the ACTH was administered only a short time after the other antidotes, it might be suspected that the effect of methylene blue had regressed until then and that the nitrites could develop their efficacy. As nitrites were the main 'accepted' cyanide antidotes, the case report is cited in this chapter. It seems as if the patient was orientated most of the time – though the author explicitly states the awareness at the end of the case report – so it could be discussed whether it was a mild or a moderate intoxication (I decided on a moderate one).

Lutier et al, 1971

Case 1: A 23 year old man lost consciousness and developed seizures and intermittent apnoea after the ingestion of an unknown amount (1-10 g?) of KCN dust during 1.5 hours. The initial therapy consisted of intubation and ventilation. As the patient developed circulatory instability without measurable blood pressure, aramine and sodium bicarbonate were injected. This resulted in spontaneous respiration but the coma persisted. Therefore he was given 4 g HOCO in a solution of STS. A systolic blood pressure of 130 mm Hg could be established. Four and a half hours after intoxication 10 ml methylene blue, vitamin, vitamin B1 (200 mg) and one ampoule Co-EDTA were administered after the patient had recovered. *Comment: HOCO/STS were effective in this probably severe poisoning, as Co-EDTA was given after recovery.*

Case 2: One hour after ingesting 1 g KCN a chemical engineer presented with profound coma, rigidity with trismus, bilateral mydriasis, and polypnoea with respiratory pauses. The systolic blood pressure was 140 mm Hg and the pulse was 140/minute. He was intubated and ventilated with 100% oxygen. After nitrite and STS and one ampoule Co-EDTA (300 mg in 20 ml) his condition had not improved and he received an ampoule with 4 g HOCO in STS. In the middle of the infusion, the neurological state improved, the gasps disappeared, and consciousness was regained at the end of the infusion, about one hour after the beginning of the treatment. *Comment: The application of HOCO/STS was obviously effective, though it is unclear, whether the other antidotes given before HOCO may have contributed to the outcome.*

Case 3: A 58 year old man intoxicated by KCN presented profound coma with the appearance of bouts of hypertension. He received 600 mg of Co-EDTA (2 ampoules) combined with AN inhalation. Because of the lack of improvement, he received 4 g of HOCO/STS a quarter of an hour later, and hyperbaric oxygen therapy during one hour. The coma persisted. He was extubated at the 14th hour and complete recovery occurred by 24 hours after poisoning. C*omment: HOCO/STS slowly led to recovery. But other antidotes (dicobalt-EDTA, AN) were used, which makes it difficult to assess HOCO efficacy with certainty.*

Case 4: A patient was hospitalised, following ingestion of KCN, in a profound coma with axial hypotonia, clonic movements, and mydriasis. He presented polypnoea and then more and more

significant apnoeic episodes. The hemodynamic state was initially conserved, but then degraded with unobtainable blood pressure. He received 4 g of HOCO in solution in STS. Spontaneous movements reappeared at the conclusion of the infusion; the patient regained his reflexes and spontaneous respiration. Four hours and 30 minutes after poisoning he received: 10 ml of methylene blue, 1 g of vitamin C, and 300 mg of Co-EDTA which were well tolerated. The blood pressure was 120/80 mm Hg. Recovery was complete two hours after receiving Co-EDTA. *Comment: The HOCO/STS mixture prompted partial recovery in this severe case, Co-EDTA contributed to the final recovery.*

Further cases: A man of unspecified age was found by the ambulance in a deep coma with his respiration turning from tachy- to apnoea and vice versa but with sufficient circulation (respiration rate 140 mm Hg systolic). He was intubated, ventilated and received SN in an uncharted quantity, two doses of STS (amount not mentioned either) as well as 600 mg Co-EDTA (twice the recommended dose). No significant improvement occurred, so he was given 4 g HOCO and 8 g STS. He recovered completely within a short time. *Comment: HOCO/STS seem have been most effective in this severe case, although the polypragmatic approach including Co-EDTA, SN and STS made the assessment difficult.*

This consideration can also be applied to another case of KCN poisoning (patient's age and sex, as well as respiratory or circulatory parameters were not mentioned). No supportive treatment was established (unless it was simply not reported). The antidotal therapy consisted of 600 mg cobalt-EDTA, AN, STS, 4 g HOCO and hyperbaric oxygen. This second patient also recovered from the incident. *Comment: Due to lack of data, it is unclear, which antidote (combination) was effective. The severity also is unclear.*

Mannaioni, 2002

An 80 year old diabetic patient reached the ED after a sudden collapse. His son suspected that his father might have drunk from an open bottle from the family's jewellery-factory and containing a potassium-cyanide-solution. The patient was in a coma, he was non-reactive to painful stimuli, gasping and sweating. His skin was pale, although the face was slightly pinkish. The vital signs were within the normal range and both pupils were reactive to light. In the blood samples a lactate of 9.2 mmol/l was detected, but the patient was taking oral diabetes medication. The blood gases showed a severe acidosis (pH 7.15) with a high pCO_2 (52 mm Hg) and a low pO_2 (28.3 mm Hg). The first therapeutic actions were mechanical ventilation with 100% oxygen, the infusion of liquids and sodium-bicarbonate as well as the application of activated charcoal. As cyanide-antidotes the doctors used 1 g of HOCO together with 0.3 g SN, both about 40 minutes after his admission. This did not result in a better clinical condition, so 15 minutes later a second dose of nitrite, now in combination with STS (3 g) was applied. Furthermore, 3 g of HOCO were infused within the next 24 hours. Under this treatment the elderly man's

condition improved steadily, and he started breathing spontaneously. Twelve hours after admission, he was fully conscious and he did not develop any neurological damage. During the course of his recovery different parameters were measured in the blood samples: The pH fell from 7.15 at the time of admission to 7.03 10 minutes later but could be increased and stabilised at 7.38 during the next two hours with supportive and specific treatment. Lactic acid increased from 9.2 mmol/l to 15 mmol/l 20 minutes later and then decreased to 7 mmol/l at the time of the second antidote administration. The cyanide level was 27.2 µmol/l (corresponding to 0.74 mg/l) at the beginning and dropped down to 5 µmol/l (0.14 mg/l) within 24 hours and to 2.5 µmol/l (0.068 mg/l) after 48 hours. The levels of thiocyanate rose from 280 µmol/l at the first analysis up to 310 µmol/l 24 hours later and then fell to 175 µmol/l during the following 24 hours. Ten minutes after the second antidote-dose the MetHb amounted to 4%, 10 minutes later to 9% and to 10% one hour after the nitrite application. Three days after the accident the patient could be discharged without any sequelae. *Comment: This was a severe case of cyanide intoxication. The rise of the thiocyanate levels indicates the detoxification of the cyanide in the liver. The combination of several antidotes was effective, no assessment as to which was the main cause for recovery can be made.*

Marbury, 1982

A 58 year old female with congestive heart failure and aortic valvular insufficiency received SNP to lower her blood pressure. This was effective, but SNP dosage had to be increased. On the third days she became confused and combative. SNP doses were lowered, other antihypertensives added to the regimen. Two days later she was lethargic with Cheyne-Stokes respiration. Two more days later her cyanide blood level was 5 mg/l, thiocyanate 24 mg/l. This prompted the application of SN 300 mg and STS 12.5 g. Haemodialysis was started, but it removed thiocyanate only, not cyanide. The total dose of SNP had been 1 g over six days. The patient survived. *Comment: SN/STS were effective in a case of moderate cyanide poisoning.*

Martín-Bermúdez, 1997

A 19 year old woman presented a coma (GCS: 7 points) and with mydriasis on both sides, which was non-reactive to light. She developed a hemodynamic instability (treated with i.v. fluids and dopamine), metabolic acidosis (with a lactate level of 5.6 mmol/l thirty hours after admission, therefore bicarbonate was administered), ARDS (oxygenation was started), renal failure with polyuria and hypernatriuria as well as a rhabdomyolysis. Additionally, gastric lavage was performed and the patient received activated charcoal and the nitrite antidote kit (SN 0.3 mg and STS 12.5 g). Blood had been taken before starting the therapy and six hours later the results came in: the cyanide level was 3.04 mg/ml *(note: The units of mg/ml cited may be an error as usually the dimension unit is mg/l rather than mg/ml and 3.04 mg/ml would correspond to 3040*

mg/ and it is not possible to achieve such a value). The specific treatment was repeated (because of the blood cyanide level, whatever that means for this amount) and the patient could be discharged seven days later without any sequelae. *Comment: Severe intoxication (the patient developed diverse complications), SNS/STS were effective.*

Martinelli et al, 2008

A 51 year old female inadvertently ingested 2500 mg of Laetrile that she was using as cancer remedy. In the ED where she went, when realising she had overdosed Laetrile, she suffered diaphoresis, apnoea, bradycardia, and generalised seizures. After 5 g HOCO she quickly recovered. Whole blood cyanide was 0.4 mg/l. *Comment: This was a moderate poisoning case, in which HOCO was effective.*

Mascarenhas et al, 1969

An 18 year old man was brought to the ED 45 minutes after ingesting a calcium cyanide insecticide. He was comatose, tachycardic, with gasping respiration. He shortly suffered several tonic-clonic seizures. AN pearls were administered until a solution of 3% SN was available. 10 ml were administered, followed by 20 ml of 10% STS. His cyanosis deepened. MetHb concentration was 5.9 g/dl or about 40%. Metaraminol was administered to maintain the blood pressure over 100 mm Hg (unreported drop from the original 130/30 mm Hg). Seventy-five minutes after SN therapy, 20 ml of methylene blue were administered. Fifteen minutes later, the relatively profound cyanosis began to abate. Three hours after methylene blue, the MetHb was 0.5 g/100 ml. On admission, BCN was measured at 2.6 mg/l. The patient was discharged from hospital on day three without sequelae. *Comment: This was at least a moderate case of poisoning. The Lilly-Kit led to cyanosis, however it probably also led to recovery.*

Maxwell, 1978

Out of curiosity on what would happen a 60 year old man ingested twice a double dose of his daily Laetrile®, and in both cases he went to hospital with weakness, light-headedness, palpitations and headaches beginning one hour after each ingestion. Upon arrival (half an hour transport) he was well in both cases. Cyanide measurement on the second presentation showed 0.6 mg/l blood. *Comment: Mild poisoning without antidote application.*

McKiernan, 1980

A male worker was contaminated with a KCN solution on 8% of his body surface. His clothes were put off and the man was washed. Although he was fully conscious, the physicians administered 20 ml of Co-EDTA i.v. The patient developed larynx oedema and vomited. He was discharged after three days in hospital and no chronic damages persisted. *Comment: In this case of mild cyanide intoxication Co-EDTA was administered without indication, resulting in larynx oedema and vomiting as adverse effects.*

MacRae, 1974

A 42 year old woman had hypotensive anaesthesia with 0.01% SNP solution. Blood pressure fell slower than usual. Over 90 minutes 250 mg were infused. During surgery the respiratory rate increased and became laboured. Extrasystoles appeared before the end of surgery. Her lips and ears were pink; the skin was warm and sweating. After cessation of the last anaesthetics spasticity in the arms and flaccid legs were noted. Lumbar punction was normal. Bicarbonate infusion improved the symptoms. Recovery was complete. No cyanide was measured. *Comment: Potentially mild poisoning, though unproven. No antidotes administered.*

Mellino, 1980

A 52 year old female patient received SNP during treatment of myocardial infarction. The total dose was 1093 mg over 34 hours. After 32 hours laboured respiration was noted, nausea, vomiting and irritability set in. She became unconscious, and had severe lactic acidosis and a thiocyanate level of 25 mg/l. SNP was discontinued, bicarbonate was given, which prompted recovery. *Comment: Mild poisoning. No antidotes administered.*

Merryfield, 1974

A 20 year old male died after surgery with hypotensive anaesthesia. 750 mg SNP had been infused over five hours. Tachyphylaxy developed. In the recovery ward his respiration rate slowed and blood pressure decreased. Cardiac arrest in asystole followed. External cardiac compression was successful, but periods of tachycardia and asystole occurred repeatedly until he died 32 hours after cessation of anaesthesia. No cyanide was found in urine, serum or brain, and thiocyanate was less than 10 mg/l. Severe acidosis may have been the cause of death. *Comment: Probably not cyanide poisoning.*

Messing, 1991

A 29 year old student of chemistry took 50 ml of a 1% potassium cyanide solution (500 mg) in attempted suicide. He became comatose, mydriatic and was admitted to hospital in an apneic state. He woke up after seven hours and developed Parkinsonism in the following weeks. This regressed slowly in the second month after the poisoning apart from dysarthria, bradykinesia of the upper limbs and very brisk monosynaptic reflexes. Three weeks after the intoxication, CCT was largely normal, and there was CSF-dense hypodensity in both putamina after five months. Sharply delimited signal elevation in T2 corresponding to the two putamina was detected in the MRI eight weeks and five months after ingestion of the poison

Michaelis et al, 1991

A previously healthy young male (body weight 60 kg) ingested 5 ml of acetonitrile without severe symptoms of intoxication. After a STS bolus application the cyanide blood levels decreased rapidly to 10% of the initial value. *Comment: Mild poisoning, in which STS was effective.*

Michenfelder and Tinker, 1977

Twelve hours after cessation of SNP infusion in a 13 year old boy who had become obtunded and tachypnoeic and rapidly deteriorated further, until he finally died, a cyanide level of only 0.18 mg/l was found. Therefore cyanide intoxication had been ruled out. *Comment: No cyanide intoxication, no antidote treatment.*

Miller and Toops, 1951

A 62 year old drug dealer tried to commit suicide with 6 g KCN (quite a high quantity) after he had been arrested. Only three minutes later he lost consciousness, developed seizure activity and a shock. Ten minutes after taking the poison he became cyanotic, bradypnoeic (respiration frequency 4/min) and hemodynamically instable (heart rate and blood pressure could not be detected). Fifteen minutes after the incident gastric lavage was performed and four ampoules of AN were crushed over his face. He received 50 ml of STS i.v. (the concentration was not reported, so it is not known whether 12.5 g or 25 g had been injected), coramine and oxygen. Half an hour later his conditions had stabilised (respiration frequency 10/min and a weak pulse palpable). As the patient convulsed more than once, the antidote was repeated in the same quantity and dextrose was administered so that he recovered within 30 minutes. *Comment:*

Severe intoxication (hemodynamic instability, no pulse/blood pressure measurable), treatment with AN and STS was effective and successful.

Mlingi et al, 1992

An extensive outbreak of acute intoxications, with nausea and vomiting as major symptoms, occurred after ingestion of bitter cassava in a drought stricken district in southern Tanzania in 1988.

Case 1 : A 7 year old girl was admitted with abdominal pain and vomiting and was released the following morning without specific treatment. She recieved antibiotic treatment and recovered in less than 24 hrs.

Case 2 : A 4 year old girl was admitted after intensive vomiting. On admisison she was semi-conscious, dehydrated without fever, and routne neurological examination was normal.

Case 3 : A 10 year old boy was admitted unconscious after sudden onset of intoxication symptoms. Due to unavailability of antidotes he was treated with dextrose saline infusion and corticone but died 3 hours later. *Comment: No antidote was given in any case.*

Montoliu et al, 1979

A 42 year old male developed malignant hypertension with renal insufficiency and cardiac failure. SNP infusion was started. After 90 minutes and a total of 13 mg SNP, his blood pressure dropped and the infusion was stopped. However, the blood pressure fell further in spite of treatment with fluids and catecholamines. In parallel severe metabolic acidosis developed, and the patient became obtunded. About two hours after the infusion was stopped, a refractory cardiac arrest occurred. *Comment: No cyanide measurement was done, and no antidote was applied.*

Morse et al, 1979

In Mexico, a treatment with Laetrile® via i.v., intramuscular, oral and rectal routes in a 48 year old woman resulted in cold sweats, headaches, nausea, lethargy and dyspnoea. Upon hospital arrival she had Kussmaul respiration, but improved after fluid replacement. A combined cyanide blood sample (2 ml at admission, 4 ml 6 hours later), showed 1.16 mg/l, which makes cyanide poisoning certain. *Comment: A mild poisoning case without antidote use.*

Morse et al, 1981

A 32 year old woman used Laetrile for four years. In an emotional crisis she ingested 9 g of the parenteral preparation of Laetrile in an attempt at suicide. She was immediately taken to hospital, where a gastric lavage was performed. AN was administered by inhalation and 300 mg of SN were given i.v., then 50 mg of STS were given i.v. and another 50 mg was administered through nasogastric tube (Eli Lilly Kit). After her admission metabolic acidosis and hypoxemia were observed; the serum cyanide level was 3.85 mg/l. Twelve hours later the cyanide serum level was 3.5 mg/l and the thiocyanate serum level was 0.22 mmol/l (1.3 mg/dl). She made a full recovery. *Comment: The repeated combination of AN and STS was effective, however the severity of poisoning is unclear.*

Moss et al, 1981

A 32 year old woman with von Hippel-Lindau's disease ingested 9gms of Laetrile in a suicide attempt. She was immediately hospitalised and underwent gastric lavage. AN was administered and 300mg SN and 50mg STS i.v. then a further 50mg STS by nastrogastric tube (Lilly kit). At administration she had severe metabolic acidosis and hypoxemia and a blood cyanide level of 143µmol/L (385µg/l). 24 hrs later it had dropped 13 µmol/L (35µg/l) andf the serum thiocyanate 0.22 µmol/L (1.3µg/l). After coredtion of her metabolic abnormalities she recovered without sequalae.

Motin et al, 1970

A 54 year old man ingested 1 g of KCN in a suicide attempt. While initially showing only mild signs of poisoning, upon arrival in hospital he had lost consciousness, but he still reacted to pain. Trismus and mydriasis were present, while breathing was irregular. Mechanical ventilation was used after intubation and gastric lavage. Then 300 mg SN, 2 g STS and 300 mg Co-EDTA were given. As this did not improve the status 4 g HOCO in 500 ml 10% STS (50 g) were given, which led to rapid and full recovery. *Comment: One more case of recovery after HOCO/STS application, while the other antidotes given before had not been effective.*

Mueller and Borland, 1997

A 39 year old woman was admitted after swallowing 25 g of acetonitrile two hours previously in a suicide attempt. She vomited and physical examination was unremarkable. Eleven hours after ingestion, she became nauseated, confused, sweaty and tachycardic (140 beats/min). She developed Kussmaul respiration and became rapidly comatose. She was given 20 ml of 3% SN

and 200 ml STS intravenously as bolus dose. Grand mal fits occurred, she was intubated and ventilated. Two hours later the methaemoglonbin level was 15%. On day 2 she was stable but 32 hours following the ingestion, she became hypotensive and tachycardic and a further 30 ml of SN and 3% STS were administered. A continous infusion of 3% SN at a rate of 2.5 ml/min was administered from day 4 to day 5. She was finally discharged 26 days following admission. Blood levels of acetonitile (max. 640 mg/l) and cyanide (max. 2.7 mg/l) were measured. The halftime of acetonitrile was 36 h and of cyanide 44 h and the harmful blood cyanide level persisted for over 24 hours after ingestion. *Comment: The delayed occurrence of symptoms after ingestion of acetonitrile and the delayed reoccurrence after the first treatment required an intensive observation of the patient during two days. The repeated i.v. administration of SN and STS was successful.*

Muraki et al, 2001

Neither acetonitrile nor cyanide could be measured in a new case of suspected, yet unproven, acetonitrile poisoning describing the development of massive rhabdomyolysis and acute renal failure. *Comment: From the report it is not clear, whether and to what degree an uptake of acetonitrile from the described exposure had occurred.*

Musshoff et al, 2002

Case 1: A 38 year old goldsmith was found dead in a large pool of blood. In the closet a plastic vial labelled “Fabvergoldungsbad” (gold plating solution) was found. The analysis identified pure NaCN. An autopsy was performed three days later. The chemical analysis of different specimens yielded the following cyanide concentrations: blood 80.9 mg/l, gastric content 1.26 mg/l, lung 46.3 mg/kg, brain 4.8 mg/kg, kidney 0.89 mg/kg, liver 17.6 mg/kg, bile 21.3 mg/l. The distribution of cyanide in the specimens examined indicated oral ingestion. *Comment: Patient was found dead, no treatment.*

Cases 2 and 3: A physician and his wife, both 71 years old, were found dead. The examination of two vials found besides them yielded positive results of cyanide. Analysis of the blood samples quantified cyanide levels of 6.1 and 8.6 mg/l, respectively, which indicated severe intoxication and provided adequate evidence to attribute both deaths to cyanide toxicity and to be a suicide. *Comment: The patients were found dead, no treatment.*

Case 4: The 70 year old wife of a chemist had ingested a spoonful of substance which had earlier been described by her husband as highly toxic. A few minutes later she experienced convulsions and, despite immediate attempts at resuscitation, she died two hours later. The powder in the

vials taken in at the scene proved to be NaCN. The cyanide levels were: blood 4.2 mg/l; brain 2.54 mg/l; gastric contents 1.2 g/l. *Comment: Patient was found dead, no treatment.*

Case 5: A 29 year old woman, who worked as a laboratory technician in a metal processing factory experienced seizures and collapsed, after drinking a cappuccino with her colleagues. A few minutes later she was already comatose. Four hours afterwards the woman was admitted to hospital, specimens of blood and stomach were obtained for toxicological examination. The cyanide blood level was 3 ml/l. Death occurred four days later. The colleague who had given her the cappuccino was convicted of murder. *Comment: Patient died; no antidote treatment.*

Mutlu, 2002

An 83 year old retired biochemistry professor and owner of a small chemical firm was found unresponsive in the bathtub by his wife (he was dressed in his pyjamas and his arms were crossed on his chest). The ambulance men saw a hypopnoeic patient, with a barely palpable pulse and no measurable blood pressure. During transportation to hospital pulseless electric activity occurred for which he received CRP in combination with epinephrine and atropine (1 mg each). Spontaneous circulation could be re-established within 10 minutes of resuscitation. In the ED the weak pulse and no measurable blood pressure persisted; moreover, no spontaneous respiration was detectable, the patient was hypothermic (32°C) and both pupils were mydriatic and non-reactive to light. The ECG showed atrial fibrillation (history of ischemic cardiomyopathy and a stroke) and the cardiac enzymes were elevated (hinting to myocardial infarction). Pulmonary oedema was seen in the chest X-ray. Blood gases revealed a pH of 7.1, a venous oxygen saturation of 62 mm Hg and a lactate of 6.2 mmol/l (whether the pH and lactate were also measured in venous blood is not clear). The patient was intubated and started on catecholamines. This resulted in a stabilisation of the blood pressure, but after transfer to the coronary care unit, he had become flaccid, unresponsive to external stimuli (sound, light, pain) and had lost the cranial nerve functions. His relatives found a suicide note in the bathroom, together with an empty potassium-cyanide bottle. Notifying this he was administered STS (dose not reported on) with a subsequent stabilisation of the blood pressure. Eighteen hours after the suspected cyanide intoxication the blood cyanide level was 1.0 mg/l. *Comment: Severe and finally fatal intoxication in which the delayed application of STS alone was not effective. Although the blood level is not explicitly given, it seems to be probable that the antidote was applied at the same time (18 hours post ingestion), that means much too late. As the cranial nerve function could not be re-established, nor could the peripheral reflexes, the patient was declared brain-dead the next day. Severe intoxication, died under STS treatment only.*

Nagler et al, 1978

Three workers suffered from hydrocyanic acid intoxication because of the open addition of a CN^--salt to an acid.

Case 1: The first worker, a 42 year old man, was standing about 1 metre away from the tank that contained the solution. He became semi-comatose a short time after inhalation of the gas had begun. As the application of one ampoule of AN resulted in tachycardia (120/min), he was administered Co-EDTA together with 20 ml of a 30% glucose solution. This antidote was repeated a few minutes later (as the first dose had not shown any efficacy). This resulted in a prompt improvement of his status (he regained consciousness) but adverse effects also occurred (the patient vomited, became agitated and developed facial and peripheral oedema for 24 hours). Moreover, the blood pressure rose to 200 mm Hg systolic. *Comment: A case of moderate cyanide poisoning with effective application of both AN and Co-EDTA. However, the latter one prompted adverse effects.*

Case 2: A second worker was standing about 3 metres away from the toxic solution. He later complained about a sore throat and was given one ampoule of AN for inhalation. Two hours later he became bradycardic and one dose of the cobalt-EDTA/glucose combination was administered. He immediately complained about nausea, retro-sternal chest pain and oedema. He was discharged 3 days after the incident and treatment. At this time all adverse effects had gone. *Comment: In this case of mild cyanide intoxication the CO-EDTA was actually not required. Altogether, the treatment showed good effectiveness, however, adverse effects occurred.*

Case 3: A 46 year old worker was standing 10 metres away from the tank. As he also developed mild symptoms of cyanide poisoning (headache, nausea and a sore throat), he was given one ampoule of AN and one dose of Co-EDTA as well. He suffered from adverse effects of this antidote as did his workmates: chest pain, vomiting, tachycardia and oedema. He also had to stay in hospital for 3 days. *Comment: Another case of mild intoxication that was treated with cobalt-EDTA without indication. The patient also developed adverse effects of the antidote but recovered in the end.*

Nakatani et al, 1992

A 31 year old man presented deep coma and seizure activity. The respiration frequency decreased within a few minutes from 15/min at admission to 6/min. He was intubated and ventilated. The BGA showed acidosis (pH 7.049, BE -20 mmol/l). Gastric lavage was performed and activated charcoal administered. Half an hour after admission an empty bottle that had formerly contained KCN was found in his flat. So specific treatment for cyanide poisoning was initiated: 3 ampoules of AN for inhalation every 3 minutes (result: mild drop of systolic blood

pressure to 80 mm Hg) and 10 g of STS i.v. over 10 minutes. Half an hour later he had completely recovered. The MetHb was always below 2%, the pH increased from 7.02 to 7.5 five hours later together with normalisation of the lactate that had been up to 12.4 mmol/l before. The blood cyanide level was measured twice: 1.2 mg/l after 2 hours and 0.1 mg/l after 3 hours. *Comment: This was a severe case of poisoning, so the cyanide levels analysed 6 months later from stored samples are nor reliable. AN/STS were effective.*

Naughton, 1974

Case 1: A 35 year old prisoner was seen while taking a capsule containing cyanide salt. He fell in a coma and developed tachycardia 10 minutes after the intake. The reflexes could be triggered normally and he was responsive to painful stimuli. He was intubated, ventilated and given i.v. fluids together with sodium bicarbonate to counteract the metabolic acidosis, recognisable in the BGA (pH 7.25, pCO_2 15 mm Hg and BE -2 0mmol/l). Blood cyanides accounted for 1.0 mg/l. About one hour after intoxication the patient was given 11 pearls of AN for inhalation (despite of this high dose, the author does not report any adverse effects such as hypotonia or peripheral vasodilatation). This was followed by 600 mg of Co-EDTA (twice the recommended dose) together with 50 ml of a 50% glucose solution, 1 hour later. This resulted in a flush, atrial fibrillation and ventricular extra systoles. Therefore digitalis was administered to him. The patient regained consciousness within the next 2.5 hours and could be extubated afterwards. 4.5 hours after the incident he was completely responsive and made an uneventful recovery. The blood cyanide was 0.6 mg/l. *Comment: AN and Co-EDTA were effective in this moderate poisoning, however, the latter had significant side effects.*

Case 2: A man (age not mentioned) tried to commit suicide after he had found his wife in a comatose status (she had tried to kill herself with an overdose of sedatives). The ambulance found an unconscious, tachypnoeic patient. As he smelt of bitter almonds and an empty bottle with the same odour was found beside him, cyanide intoxication was suspected. In the blood gas analysis, mild acidosis (pH 7.3), hypocapnia (pCO_2 16 mm Hg) and a BE of -16mmol/l were detected. He was intubated, ventilated with 100% oxygen and gastric lavage was performed together with the application of activated charcoal. Furthermore, he was given sodium bicarbonate and 300 mg of Co-EDTA twice (20 and 45 minutes after admission), each together with 50 ml of a 50% glucose solution. Two hours after the suicide attempt he was conscious again and orientated; he only complained about facial oedema *(side effect of the double antidote dose).* The cyanide amounted to 0.2 mg/l in the blood sample. He recovered completely. *Comment: Co-EDTA was effective in a moderate poisoning, but prompted side effects.*

Nicoletta et al, 2007

A 58 year old man with a long history of hypertension was admitted with aortic dissection, and put on SNP infusion. The dosage had to be increased until he developed psychomotor agitation, hallucinations and speech impairment. SNP infusion was stopped. No cyanide measurements were done, nor is the total or the maximum SNP dose reported. *Comment: Cyanide poisoning was unproven, no antidotes were given.*

O'Brien et al, 2005

A 32 year old woman ingested 6 amygdalin tablets and was found shortly thereafter by her father with a rapid progression from abnormal behaviour to gait disturbance and writhing on the floor plus somnolence. She recovered fully over 8 hours with symptomatic treatment and without antidote application, though nephrogenic diabetes insipidus had occurred. No cyanide measurement was made, thiocyanate in blood was 445 µmol/l, which indicates probable cyanide poisoning. *Comment: Mild poisoning, no antidotes applied.*

Ortega and Creek, 1978

A 3 year old boy was given Laetrile® enemas for 3 days, each with 3.5 g Laetrile®. After the second application vomiting and diarrhoea occurred, after the third dose the child became lethargic, unresponsive, tachypnoeic and cyanotic. He recovered under oxygen therapy. The blood cyanide level was 2.14 mg/l, so cyanide poisoning is certain. *Comment: Moderate poisoning, no antidotes given.*

"Patient S.T. was first admitted at the age of $2^{10}/_{12}$ years with a two-month history of constipation and back pain. Physical examination revealed a hard, nontender spherical mass in the right upper quadrant of the abdomen. Initial evaluation revealed a normal complete blood count, urinalysis, chest radiograph, skeletal survey, bone, liver and spleen scans, and bone marrow. An i.v. pyelogram (urography) revealed an extra-renal calcified mass in the right flank close to the kidney. Abdominal exploration resulted in the excision of a tumor immediately below the hilum of the right kidney. Pathology examination revealed neuroblastoma with invasion of the capsule and regional lymph nodes. Ten days after surgery, chemotherapy consisting of vincristine, cyclophosphamide, and dimethyltrianzeno imidazole carboxamide was initiated. However, after receiving three doses of the latter and one dose of cyclophosphamide, the patient was lost to follow-up. It was subsequently learned that the patient was receiving treatment with Laetrile, 500 mg daily by mouth and 3.5 g i.v., daily.

Because of increasing technical difficulty in administrating the drug i.v., the mother was advised to use the parenteral form of Laetrile as an enema. Following administration of the second daily enema of 3.5 g Laetrile in 10 ml, the patient developed vomiting and diarrhoea which persisted into the third day of treatment. Shortly after receiving the third Laetrile enema, the patient, who previously had been irritable, became progressively lethargic and unresponsive, tachypnoeic, and cyanotic. He was then taken to a local hospital where O_2 therapy and i.v. hydration were rapidly initiated.

Because of the history of Laetrile administration, the possibility of cyanide poisoning was suggested and, five hours after admission, cyanide blood concentration levels were obtained and reported as 214 µg/dl. (The lethal range is 260 to 3,000 µg/dl). The child recovered slowly over the next 12 hours. Subsequent evaluation revealed recurrence of intra-abdominal disease with several bone metastases."

Orušev et al, 1972

A report from Yugoslavia described a 35 year old worker exposed to acrylonitrile vapours in the open for one hour who developed some neurological symptoms like intention tremor, but recovered without specific therapy. Blood levels for acrylonitrile or cyanide are not reported. *Comment: Mild poisoning, but it is unclear whether cyanide was involved. No antidotes given.*

Pardee, 1947

There is a report from 1947 on a boy dying after ingestion of large amounts of choke (wild) cherries for days including kernels. One morning he complained about epigastric pain, vomiting and convulsions and died within an hour. *Comment: Cyanide poisoning unclear, but probable. Fatal case, no antidotes given in time.*

Patel, 1986

Case 1: A 62 year old woman had surgery for coronary bypasses and cardiac aneurysmal resection. Her blood pressure required SNP therapy after surgery. The SNP dose had to be increased progressively. In parallel her clinical status deteriorated. On day 3 she became unresponsive to pain stimuli. Total SNP had been 721 mg over 80 hours. SNP infusion was stopped and a blood sample taken. Cyanide levels were 0.585 mg/l on day 3 and 0.8 mg/l on day 4. STS was given at doses of 12.5 and 6.25 g. This prompted improvement. *Comment: In spite of relatively low cyanide levels this may have been severe poisoning, but this is unclear. STS was effective.*

Case 2 in text (4 in table): A 65 year old man with myocardial infarct had quadruple coronary bypasses and mitral valve replacement. SNP (and dopamine) were required with an increasing need for SNP. After 30 hours and 157 mg SNP total he became restless and did no longer respond to commands. SNP was discontinued and a blood sample for cyanide and thiocyanate taken. Lactic acidosis was seen. Heart rhythm and hemodynamics became unstable. Cardiac arrest occurred late on day 2 and could not be reversed. Cyanide level had been 0.5168 mg/l and thiocyanate 16 mg/l. STS had been given, though the dose is not reported. *Comment: In spite of a low cyanide level, there was cardiac arrest. STS was not effective.*

Paulet, 1965

Case 1: A 32 year old chemical engineer unintentionally swallowed a small amount of a solution containing potassium- and copper-cyanide while working. A few minutes later he developed nausea, palpitations and paraesthesia. 300 mg Co-EDTA were administered 20 minutes after the incident. This resulted in vasodilation with a flushed face, in accordance with the fact that cobalt-EDTA, when being administered without strict indication (as in this case) may cause adverse effects, even in the recommended doses. All the symptoms and adverse effects subsided within half an hour. *Comment: Mild intoxication treated with Co-EDTA, which was effective, but caused mild side effects.*

Case 2: A male patient, aged 24, had taken 1.5 g of KCN in a suicide attempt. Forty-five minutes later he was seen in the ED with pink-coloured, cool skin and dilated pupils. He was in a deep coma, respiration was shallow and weak and the systolic blood pressure was only 50 mm Hg together with a tachycardia heart rate of 100/min. The treatment consisted in 200 mg of SN, STS (dose not mentioned) and – as this nitrite-antidote treatment did not show any positive reaction – 300 mg of Co-EDTA were given twice (solved in 20 ml each time), 5 hours after admission as well as on the next day. The patient regained consciousness straight after the first dose of Co-EDTA (Co-EDTA) and made an uneventful recovery. *Comment: In this severe case of poisoning SN/STS were obviously not effective, while Co-EDTA was. The reason for the application of the second dose is not clear.*

Pentore et al, 1996

In Italy a 56 year old woman came to hospital with nausea, vomiting, respiratory problems and sleepiness. Her pupils were dilated. As she rapidly fell into a deep coma, artificial respiration was conducted, she became conscious again, but remained confused for 14 days. Later she developed signs of a parkinsonian syndrome, retrobulbar neuritis and sensory-motor neuropathy. MRI showed abnormal signal intensities involving the basal ganglia. She had eaten choke cherries steeped in alcohol (cyanide in cherries up to 15 mg/kg, in the spirit 45 mg/kg).

Comment: Cyanide levels in blood were not measured; neither antidotes, nor oxygen probably, were administered.

Perschau et al, 1977

A 14 year old boy was scheduled for a Harrington rod correction of a thoracolumbar scoliosis in association with spastic paraplegia below the level of T-6. Under anaesthesia, sodium nitroprusside (0.1 mg/ml in 5% dextrose) was infused. A total dose of 130 mg nitroprusside was administered over five hours. As the arterial systolic blood level fell below 30 torr, a cardiopulmonary resuscitation was performed. Because of the patient's severe intrapulmonary shunting, hypotension, and arterial hypoxemia, not to administer inhalation or i.v. nitrites, in order to avoid further compromising the patient by inducing elevated MetHb levels and reducing available oxygen. Therefore it was decided to administer 150 mg/kg STS (25%) and thereafter 75 mg/kg every 3 hours. By 12 hours after the cardiovascular collapse, he was responding appropriately to command. *Comment: This seems to have been a severe poisoning, STS was effective, although delayed, which would have been expected.*

Peters et al, 1982

A 31 year old chemist wanted to commit suicide by cyanide and informed his wife at 5.30. One hour later he was found in his laboratory and brought to hospital at 7.20. In hospital he showed central and peripheral cyanosis, a respiratory rate of 2/min, a heart rate of 72/min, and an arterial blood pressure of 140/70 mm Hg. He was intubated and respirated with 100% O_2. At 8.00 he arrived at the ICN still comatose and cyanosed. Bilateral leg clonus and dilated pupils were observed. The patient was again ventilated with 100% oxygen and received 0.3 g SN and then 25 g STS ('Lilly-Kit') i.v. Blood samples were taken for estimation of cyanide, MetHb and other biochemical/haematological parameters. The metabolic acidosis was corrected by 200 ml 8.4% sodium bicarbonate. At admission the patient had severely displaced: pH: 7.07 ↓, pO_2: 41.2 kPa ↓, BE: -21 mmol/l ↓↓, bicarbonate: 9 mmol/l ↓↓, pCO_2: 4.1 kPa ↓↓. In the recovery phase which started at 9.45, the patient was hyperventilating, had seizures which had to be treated by diazepam and even by neuromuscular relaxation. At 10.15 the patient got a second dose of SN, and STS because of the presence of cyanide in the gastric aspirate. In addition 150 g STS in 300 ml water were infused in the stomach. As no MetHb could be measured SN/STS treatment was repeated together with AN. Twenty-four hours after admission neuromuscular blockade was reversed and spontaneous respiration established. The total amount of KCN taken was 8 ml of 5% solution = 400 mg. The blood cyanide levels were 2.6, 1.9 and 9.7 mg/l at 10.5, 14.5 and 26.5 hours after ingestion, respectively. The authors explained the last increase by incorrect sample collection. *Comment: Severe intoxication, treated with SN/STS.*

MetHb formation could not be sufficiently achieved for unknown reasons in spite of repeated dosing. However, in the end SN/STS was probably effective, but the patient survived with sequelae.

Pijoan, 1942

Case 1-3: One person, 15 years old, came to hospital with assumed choke cherry ingestion and stupor/somnolence and fibrillar muscular twitching. He recovered after gastric lavage. So did a person, 54 years old, with slight stupor, listlessness and fibrillar muscular twitching, and another person, 14 years old, with somnolence, dizziness and fibrillar muscular twitching. All three had pink cyanosis; initially all had had vomiting and epigastric pain. *Comment: In these cases cyanide poisoning can be only regarded as possible, no antidotes were given.*

Posner, 1977

In an olicuric patient (male, 66 years old) with hypertensive crisis, SNP was given at a total dose of 238 mg over 38 hours. Hypotension persisted and the patient died 24 hours later. Maximum cyanide level was 2.99 mg/l. *Comment: No antidotes were given.*

Potter, 1950

Case 1: A 52 year old man worked in a HCN-processing company. He inhaled hydrocyanic acid while trying to clean a conduct that led to a tank containing HCN. Actually he was wearing gloves and a respirator but he had to take the latter off because the fresh air flow was not working properly. A short time after he had left the room he became dizzy, dyspnoeic, developed a headache and a collapse. As first line treatment he was given oxygen and one ampoule of AN for inhalation. Five minutes after the incident the man presented a deep coma with irregular, laboured breathing, tachycardia of 110/min, non-reactive pupils and convulsions. Therefore he was administered the cyanide antidote kit consisting of 300 mg of SN and 50 ml of a solution containing 50% STS (25 g). His breathing normalised immediately, he showed spontaneous movements, murmured and the pupils were again reactive to light. Ten minutes after the antidote application he was fully conscious and made a good recovery. The author reports that the cyanide level in the blood samples was elevated (however, the exact amount is not given). *Comment: In this severe case the nitrite antidote kit showed a prompt and satisfying efficacy.*

Case 2: A man, aged 41, was employed in a company working with HCN. When he was trying to clean a gas-pipe for HCN he was hit by liquid prussic acid on his hand, as he had put off the gloves he had worn before to be able to work better. Two minutes after the accident the man complained about dizziness and dyspnoea, 5 minutes after the exposure he lost consciousness.

He was given oxygen at once and one ampoule of AN was crushed over his face. His respiration was irregular, his face was flushed, he was tachycardic (heart rate 100/min), his pupils were fixed and dilated and no deep tensor reflexes could be triggered. As 2 more ampoules of AN and coramine injection did not influence his conditions but he rather became cyanotic, 300 mg of SN and 50 ml of a 50% STS solution (25 g) were administered i.v. After this special treatment his clinical status improved quickly, he moved spontaneously, murmured, the respiration normalised and the tachycardia ceased. *Comment: In this severe poisoning AN obviously did not prompt improvement, while SN/STS were effective.*

Prieto, 2005

A middle aged woman (30 years old) injected herself with a cyanide-solution s.c. in a suicide attempt. She was found lying on the floor with only 3 points on the GCS, bilateral mydriasis responsive to light and hypotonia. Local necrosis could be detected at the points of injection. In the emergency department the patient was intubated and i.v. fluids were administered, in combination with dopamine and noradrenaline. In the blood gases a pH of 6.74 (indicating severe acidosis) was seen, with a lactate of 19.3 mmol/l and an anion gap of 33mmol/l. Sodium bicarbonate was infused and increased the blood pH (7.13). The blood cyanide level was determined at 4.6 mg/l (which shows that the resorption after s.c. injection occurred quickly and completely). A further treatment consisted of haemodialysis (lasting four hours) resulting in a pH of 7.3 and a rise in the pCO_2 (from 19.4 mm Hg to 32.8 mm Hg). A few hours later the woman reached 15 points on the GCS and could be transferred to the psychiatric unit days days later. *Comment: This is an example of severe cyanide intoxication which could be controlled with supportive treatment only.*

Queisser, 1966

This is a cassava poisoning report from an island off the coast of Tanzania. Half an hour after a meal of fresh cassava, which tasted bitter, two children (aged 7 and 9) fell ill with nausea, vomiting, chest oppression and dizziness. Half an hour later one child died after becoming unconscious. A further 30 minutes later the second child died, too, and a third child developed dizziness and nausea. Three qualitative hydrocyanic acid tests applied during *post-mortem* examination gave positive results for the gastric content of both children. *Comment: In these two severe and one mild poisonings no antidotes were given.*

Quinlan et al, 2008

A 14 year old girl had renal transplantation and developed hypertension after surgery. SNP was given, but increasing doses were needed. A total of 200 mg was given over 56 hours. Blood cyanide was found to be 3.1 mg/l and SNP was discontinued. 0.3 g SN and 19 g STS were applied and haemodialysis to remove thiocyanate. Cyanide fell quickly, and the patient recovered fully. *Comment: In spite of the cyanide level this obviously was mild poisoning, in which SN/STS may not have been required, but was effective.*

Rachinger et al, 2002

A 35 year old laboratory worker tried to commit suicide with a cyanide salt. Ten minutes after the ingestion she presented unconscious and was intubated and ventilated. DMAP and STS were given in unspecified doses. The patient developed an intermittent akinetic mutism with agitation and an irreversible cerebral ischemia. *Comment: Clinical details were only partially communicated. This seems to have been moderate poisoning, so that it is unclear, why brain ischaemia developed. DMAP plus STS have at least not been fully effective.*

Racle et al, 1976

A 68 year old man ingested 5 g of KCN in a suicidal attempt. Upon arrival in hospital 30 minutes later he was in a deep coma, had convulsions and mydriasis as well as cardiovascular collapse and tachypnoea. Mechanical ventilation was installed after intubation, and he was given 4 g HOCO in an 8 g STS solution. This led to recovery. *Comment: HOCO/STS were effective and saved the patient. The cyanide level was not measured, but the clinical course was typical.*

Ram et al, 1989

A 53 year old with intracerebral haematoma, which was removed surgically, needed SNP to control his blood pressure postoperatively. After initial improvement he became drowsy on day 7, had metabolic acidosis, and fell into a coma. Under the assumption of cyanide poisoning 250 mg DMAP and 12.5 g STS were given, and SNP was discontinued. Haemodialysis and oxygen ventilation were started. The patient's condition improved; neither cyanide nor thiocyanates were measured. *Comment: This is possible, but unproven, cyanide poisoning, probably moderate. DMAP and STS in combination with haemodialysis and oxygen were effective.*

Ruangkanchanasetr et al, 1999

Nine hours after ingestion of boiled cassava, two children, a 4 year old girl and a 1.5 year old boy, vomited and became comatose. They required intubation and ventilatory support (FiO2 = 1). The girl needed antidotes (SN 3% 4 ml, and STS 2.5% 250 ml), the MetHb was measured at 3.95%, gastric lavage with application of activated charcoal and liquid paraffin was also done. The cyanide level before antidote application was 0.56 mg/l (19 hours after ingestion). The boy could be kept on supportive treatment only, though his circulation was initially instable. He had a bitter almond odour on breath, and the cyanide level in blood (23 hours after ingestion) was 0.32 mg/l. Metabolic acidosis and lactic acidemia were observed in both children. Comment: Two cases of moderate poisoning, in one case SN/STS, in the other case supportive treatment, were effective.

Rauscher, 1978

A 27 year old female with renal transplant developed a gall bladder rupture requiring surgery. Hypertension developed, which was difficult to control and required SNP therapy. Over 48 hours 1,120 mg were given. Then consciousness shifted between agitation and coma, acidosis was found, the hemodynamic status became unstable. In spite of intensive treatment, cardiac arrest in asystole occurred. Resuscitation and bicarbonate were successful, so the patient survived. Cyanide was not measured. *Comment: It is unclear, whether this was cyanide poisoning. Anyhow, no antidote was given.*

Riudavets et al, 2005

A 17 year old man drunk a beverage laced with KCN by another individual. He became unresponsive within a few minutes of ingesting the cyanide and suffered cardiorespiratory arrest. Resuscitation efforts were implemented. Then he was taken to a local hospital, where he received antidotes for cyanide. A blood test revealed the presence of thiocyanate (25.5 mg/l). He remained in a coma, on ventilator, and was pronounced brain dead 4 days later. *Comment: After cardiac arrest the patient succumbed to cyanide poisoning. The antidotes given were not specified, so this case cannot be evaluated.*

Robinson, 1959

Case 1: A young woman, who accidentally took 1.04 grains of hydrogen cyanide walked 150 yards, was found unconscious 5 minutes later, became comatose within 20 minutes, and died within 30 minutes of taking the poison.

Case 2: A student nurse died 2 hours after the start of symptoms with nausea, followed by coma and convulsions. 2.5 grains of SNP and 54 mg of cyanide were found in the stomach.

Case 3: A worker fell down and was found in coma with fixed, small pupils. He died in the hospital receiving room 35 minutes later. A suicide note was found and 170 mg of KCN were found in the stomach.

Comment: In all three fatal cases no antidote was given.

Roedelsperger, 2009

One case was probable inhalation by a worker working close to an acetone cyanohydrin leakage, who complained about a bad taste. Again, cyanide in blood was below the limit of detection, and except for the taste there were no symptoms. 2.5 g HOCO were given.

A skin contact (about 1 palm size) with a mixture of acetone cyanohydrin and HCN did not result in symptoms either. Cyanide in blood was no determined. 2.5 g HOCO were given.

A similar case occurred (2 palms area affected); cyanide in blood was about 1 mg/l and decreased quickly after antidote (HOCO 2.5 g) application (1.5 h: 0.15 mg/l). This worker had no symptoms either.

Comment: Antidotes were applied in all cases. However, as their application does not seem to have been required, the cases are not contributive for assessment of antidote efficacy. In one case a cyanide level of about 1 mg/l was found, however, this patient, too, was asymptomatic, and the applied antidote efficacy cannot be assessed either [personal communication].

Rosenberg et al, 1989

A 46 year old man ingested 1,500 mg of KCN in a suicide attempt. He survived, but later on developed a severe parkinsonian syndrome. The MRI revealed multiple areas of low-signal intensity in the globus pallidus and posterior putamen. A 6-fluorodopa PET study revealed bilateral decreased uptake in the basal ganglia. This evidence of functional impairment of dopaminergic nigrostriatal neurons is related either to direct toxicity of cyanide or to the effects of cerebral hypoxia secondary to cyanide intoxication.

Rubino and Davidoff, 1979

A 49 year old woman with lymphoma ingested 20-40 apricot pits as a surrogate lunch, not for cancer treatment. After 30 minutes, headache, weakness, disorientation, nausea and vomiting set in. In hospital she was given AN, SN and STS. Her cyanide blood level was 3.2 mg/l. Recovery was complete. *Comment: In this mild poisoning case, SN/STS were effective, but would probably not have been required.*

Sadoff et al, 1978

After having injected 12 g of Laetrile i.v. per day for a month without problems, a 17 year old girl ingested a 10.5 g dose orally, as injection was not possible. Shortly after the ingestion, she developed headache and dizziness, collapsed, had tetanic contractions of the hands, and generalised convulsions. Breathing was laboured; she became comatose after 10 minutes, and died 24 hours later in hospital after gastric lavage and mechanical ventilation. No antidotes were given. Cyanide in blood was normal, but the sample was taken 36 hours afterwards, and the analysis was delayed by one month. Though evidence was circumstantial only, cyanide poisoning was suspected to be the cause of death. *Comment: In this fatal supposed cyanide poisoning no antidotes were given.*

Saincher and Swirsky, 1994

A chemistry student (aged 23) took 100 mg KCN in a suicide attempt. Three hours later he became pale, incontinent, disorientated, showed uncoordinated movements and murmuring. The vital and biochemical parameters were as follows: heart rate 135/min, accelerated respiration 36/min, pH 7.02 (7.26 with oxygen), pCO_2 24 mm Hg (34 mm Hg under oxygenation), BE −24 mmol/l and anion gap 33 mmol/l. The blood cyanide was measured at 4.65 mg/l. However, the patient was not comatose and because of this no antidote was applied though the patient reported cyanide poisoning. He was only given oxygen and 150 mmol sodium bicarbonate. A forced alkaline diuresis was performed and pneumonia treated with antibiotics. The young student became alert and responsive. The acidosis cleared (pH 7.26) and the pO_2/pCO_2 normalised. An intermittent weakness of the legs and the pulmonary affection were both reversible. *Comment: Mild intoxication in spite of high cyanide level. No antidotes were applied.*

Sarikaya, 2006

Parkinsonian syndrome developed in a young male following a suicide attempt with cyanide. MRI was reported as showing bilateral decreased signal on the T1 image and increased signal on

the T2 image in both lentiform and caudate nuclei as well as a minimally increased signal on T1 image in the cerebellar white matter consistent with hypoxic-ischemic changes. F-18 FDG PET/CT scan of the brain demonstrated absent FDG accumulation in both putamens. There was also diminished FDG uptake in both caudate nuclei and the cerebellum. The CT scan demonstrated hypodense areas representing encephalomalacic changes both in the putamens as well as in the caudate nuclei. Similar areas were also present in the cerebellum.

Sartorelli, 1966

A 22 year old chemist was exposed to acrylonitrile vapours (distillation) in a small room for two hours. He developed headaches, nausea and dizziness, followed by vomiting, dyspnoea, tremor, incoordination, and convulsions without loss of consciousness. No specific treatment or blood measurements for acrylonitrile or cyanide were performed, the patient recovered fully. *Comment: Alleged cyanide intoxication, symptoms might be due to acrylonitrile, too. No antidotes applied.*

Schulz et al, 1979

A 42 year old man was treated with a combination of SNP and STS. During long-term infusion of SNP the patient developed toxic cyanide levels (3.6 µg/ml erythrocyte cyanide concentration). The patient was treated successfully with STS; erythrocyte cyanide levels decreased to 0.5 µg/ml within 7 hours. *Comment: Effective lowering of intra cellular cyanide level by STS.*

Schulz et al, 1982

Nuremberg Case: This is a short note in a table on the death of a 29 year old female after a dose of 480 mg SNP.

Cologne Case: See below, a total dose of 28 mg SNP.

Schulz and Roth, 1982

A 10-day-old infant received SNP at 2-5 µg/kg for increasing hypertension. After 30 hours, the blood pressure dropped and acidosis developed. An erythrocyte cyanide level of 11.3 mg/l was found, STS applied and SNP infusion suspended. SNP was recommenced after three days as infusion together with STS (100 mg/kg). Cyanide levels remained low. However, two days after SNP/STS were started again, the child died from respiratory failure. *Comment: A case of*

moderate poisoning, STS (and stop of SNP) was effective. Later the combination STS/SNP was given, so that no further cyanide poisoning can be assumed. Reason for respiratory failure is unclear.

Scolnick et al, 1993

Two workers were accidentally exposed to propionitrile fumes from a waste slurry. One worker collapsed after seven hours of exposure, and had to be resuscitated on site. Upon arrival in the hospital he was in coma with eye deviation, and later developed seizures. A case of poisoning with carbon monoxide, cyanide, or hydrogen sulphide was suspected and the cyanide antidote kit with 300 mg SN and 12.5 g STS was applied i.v. Thereafter the seizures stopped and the patient regained conscience. A further treatment with hyperbaric oxygen was started. Forty-eight hours later the patient was dismissed with a resolving pneumonia. Laboratory results arrived later and revealed a cyanide concentration in blood of 5.0 mg/l. The patient complained of severe headaches and dizzy spells over 30 more days. *Comment: Effective treatment with SN/STS in severe poisoning.*

The other worker complained of headache, nausea, and dizziness after two hours exposure. He vomited and then lied down in the cafeteria, where he was found five hours later in a confused and disoriented state. His nausea persisted, as did his headache. The blood cyanide level was 3.5 mg/l. He also received the cyanide antidote kit described above, and was discharged 25 hours later. *Comment: Mild poisoning, in which SN/STS was effective.*

Selden, 1990

A man, 31 years old, took a capsule containing KCN in a suicide attempt. He vomited at once, was unresponsive to external stimulation except pain, and showed bradycardia of 44/min and tachypnoea of 32/min. Twenty-two minutes after ingestion of the poison he showed cyanosis, although he was ventilated with 100% oxygen. The patient was mildly acidotic (pH 7.29) and was behaving himself. He was intubated, ventilated and received AN for inhalation. Moreover, 0.3 g SN and 12.5 g STS were administered twice within four minutes. This caused the MetHb to rise to 9.8% after eight minutes. The maximum MetHb was measured after 90 minutes with 13.4%. A short time after application of the antidotes the man was alert and had to be sedated because of his aggressive behaviour. Before being extubated 75 minutes after admission, gastric lavage was performed. The cyanide levels were determined 30 minutes, 60 minutes, 120 minutes, 3.5 hours and 6 hours after the incident at 5.12 (dissociation between high blood cyanide and mild clinical presentation) – 6.93 – 3.09 – 0.67 and 0.18 mg/l. The man recovered completely. *Comment: Moderate intoxication in spite of high blood cyanide level. AN/SN/STS were effective, though a double dose of SN/STS was given.*

Shragg et al, 1982

A 67 year old woman with colon cancer took 4-5 ground bitter almonds. Thirty to forty-five minutes later she became light-headed and felt nauseous, started vomiting and had abdominal cramps. After the symptoms subsided overnight she crushed and ingested 12 bitter almonds. Fifteen minutes later she had severe abdominal cramps and collapsed. Upon arrival in hospital she was in deep coma and incontinent. As bitter almond smell was detectable she was given AN, SN (0.3 g) and STS (12.5 g), and gastric lavage was done, a cathartic and activated charcoal were given, intubation was done for oxygenation. Twenty minutes after the antidote application the patient became responsive again, however pulmonary oedema developed. She made a full recovery. Later on the cyanide content of one bitter almond was determined as 6.2 mg and cyanide was highly positive in a qualitative check of the gastric contents. *Comment: A case of severe poisoning, in which SN/STS were effective.*

Singh et al, 1989

A 24 year old man was working in a silver processing plant. While cleaning a tank that had contained silver cyanide he fell into it and lay in the remains of the toxic solution for an unknown time. The cyanide concentration in the room was 200 ppm. On arrival in the ED he was in coma, apnoeic and a smell of bitter almonds could be detected. Furthermore, he was tachycardic (120/min), the blood pressure could not be measured, both pupils were fixed and dilated and he did not react to painful stimulus. So intubation was performed and 0.3 Co-EDTA together with 50 ml 50% glucose solution were administered. The antidote was given twice within one minute. The treatment resulted in spontaneous respiration but this only persisted for seven minutes. So a third dose of Co-EDTA was applied, now leading to an oedema of the whole body. Cardiac ischemia was seen in the ECG and the BGA showed a severe acidosis of 7.08. It was only now that the patient was decontaminated, the stomach was washed and he was given i.v. fluids. As vasopressors were continuously administered the systolic blood pressure could be established at 100 mm Hg. Despite all these efforts the young patient died 24 hours later.

During his hospitalisation, different biochemical parameters were monitored and are summarised in the following table:

Time (h) post exposure	0,5	2	4	12	18	24
CN^- (µmol/l) / (mg/l)	804/21.7	819/22.1	608/16.4	23/0.62	15/0.,41	15/0.41
SCN^- (µmol/l)	-	147	172	345	281	267
pH	7.21	7.23	7.27	7,33	7.40	7.41
Lactate (mmol/l)	17.5	13.0	5.4	3.8	2.4	2.2

Comment: In this severe cyanide intoxication case the Co-EDTA was not able to rescue the patient though it had been given 3 times. The antidote was not effective and it even prompted adverse effects.

Sipe et al, 2001

A 78 year old woman was involved in a motor vehicle collision. At the scene she had a decreased level of consciousness and was intubated. At the hospital she was started on esmolol and nitroprusside doses in the range of 4.4 µmol/kg/minute to control her blood pressure. The Ativan (lorazepam) infusion was discontinued because of her altered mental status. She received a total of 1300 mg nitroprusside over the five days of infusion. A cyanide level was checked on post-trauma day 4 (2.0 mg/l or 80 µmol/l). At this time the nitroprusside infusion was discontinued and 4 mg/kg (10 ml) of 3% SN and 50 ml of 25% STS were administered. The same regimen was repeated nine hours later. She did not improve neurologically. An electro-encephalogram showed theta waves slowing down. The family decided to withdraw support on post-trauma day 20. The patient died on post-trauma day 21. *Comment: It is unclear, whether this was primarily cyanide poisoning. The antidotes given (SN/STS) were not effective, which may well be due to a non-significant cyanide involvement in this case.*

Smith, 1977, 1978

Case 1: A 48 year old woman with lymphoma diagnosed in1965 began taking laetrile (6 mg i.v. each week and 500 mg tablets orally three times a day) in February 1977. On April 25 she was admitted to hospital with fever, malaise, headache and severe abdominal cramps. She had a diffuse macular erythematous rash, marked lymphadenopathy and hepatosplenomegaly, and abdominal tenderness without peritoneal signs. Laetril therapy was discontinued and after two days her symptoms cleared. Against advice she resumed the laetrile therapy on June 5 and was readmitted on June 20 with the same syndrome. Blood cyanide level was 1mg/dl; skin biopsy was consistent with drug eruption. Symptoms resolved within 48 hours of discontinuing laetrile therapy.

Case 2: A 46 year old man was found to have large cell anaplastic carcinoma of the lung metastatic to the left temporal-parietal area of the brain. In September 1976 he began taking laetrile, 500 mg orally each day. In March 1977, he was admitted with progressive neuromuscular weakness of both lower and upper extremities as well as bilateral ptosis. Laetrile therapy was discontinued; he has not recurrence of the above symptoms.

Comment: Both patients recovered without antidotal treatment.

Steffens et al, 1998; Steffens, 2002

A worker was splashed on the face, neck and chest. Assuming cyanide involvement he received STS (10 ml 10%) twice and was started on an N-acetylcysteine infusion, which caused an allergic reaction and had to be stopped. The maximum acetonitrile concentration in blood in a whole series of biomonitoring measurements was 6.9 mg/l, while cyanide could not be detected at any point in time. Either there had been no cyanide formation, or the cyanide formed with a latency had been efficiently captured by the applied STS. *Comment: No proven cyanide poisoning, so antidote efficacy cannot be assessed.*

A worker was steam cleaning a filter contaminated with propionitrile and thus inhaling it. One hour later he was feeling unwell and then developed nausea and dizziness, followed by somnolence and collapse. During the injection of 20 ml STS 10% he regained consciousness, after which he remained free of symptoms except for an anterograde amnesia for some time. The initial cyanide blood level had been 3.16 mg/l, and it dropped quickly (1.25 hours) by more than 50% after the STS injection, which was once repeated. In parallel the propionitrile concentration in blood dropped from an initial 12.74 mg/l by 90%. Nevertheless a second dose of 20 ml STS 10% was applied. *Comment: Moderate poisoning effectively treated with STS alone. The immediate positive reaction to STS (during the injection) indicates a rapid onset of antidotal effect.*

Steffens, 2003a,b

A 48 year old worker was exposed without protection equipment near a heat exchanger under repair, which was gassing out a.o. acrylonitrile. Approximately 30 minutes later he was vomiting and experiencing somnolence. Upon decontamination, he was given an i.v. application of N-acetylcysteine (150 mg/kg bw) during 15 minutes. Initially he showed increased nausea; then, he recovered. Biomonitoring later showed a low acrylonitrile concentration, but also possible life-threatening cyanide concentration in the whole blood (3.1 mg/l). There was a marked decrease of blood cyanide after application of N-acetylcysteine alone.

Three years later, the same worker accidentally inhaled acrylonitrile. There were few symptoms, only slight nausea. Being aware of the first incident, he was immediately given an i.v. application of the full N-acetylcysteine regimen (a total of 300 mg/kg bw as for paracetamol poisoning), and an additional i.v. application of 10 ml STS 10% approximately 2.5 hours later. The therapy was well tolerated except for skin itching and generalised erythema. Later biomonitoring showed again a potentially critical cyanide concentration in the whole blood (3.4 mg/l). The acrylonitrile level was low again. The marked decrease of the blood cyanide concentration was due to application of N-acetylcysteine alone, well before the injection of STS.

Steffens et al, 2003

The authors described two cases of life-threatening hydrocyanic acid (HCN) gas poisoning. In at least one case the uptake was strictly percutaneous, as a self-contained breathing apparatus had been used. A stream of gas containing 19% HCN was released from a leaking flange. One worker collapsed and was treated on site with 2 ml DMAP. At the same moment a fire fighter wearing a self-contained breathing apparatus collapsed, was unconscious and had foam on his mouth and nose. He also received 3 ml DMAP immediately. Upon arrival in the emergency room the cyanide levels were 5.3 and 6.75 mg/l respectively in the whole blood (after the application of DMAP). MetHb was 5.5 and 16.2% respectively. After decontamination both patients received 50 and 150 mg/kg bw N-acetylcysteine respectively, as parallel acrylonitrile poisoning had to be suspected. In addition they were given 30 and 40 ml STS 10% respectively. In both cases the cyanide levels dropped significantly to 0.035 and 0.036 mg/l on the following day, and both patients survived without sequelae. Conclusions: HCN gas can easily penetrate the skin and lead to possible fatal cyanide poisoning. If work in contaminated areas is necessary, a chemical protection suit has to be used, normal fire fighting equipment has been shown to be insufficient protection. The immediate application of DMAP at about 50% of the recommended dose has been shown to be life-saving, even though MetHb levels remained relatively low. The high cyanide levels after DMAP treatment show that there is no definitely lethal cyanide concentration. It might be possible, that the rapid increase of cyanide levels may play a greater role. *Comment: The application of N-acetylcysteine plus STS seems to be an effective antidote treatment. Gaseous HCN was absorbed through intact skin.*

Steffens, 2010

Two cases of life-threatening hydrocyanic acid (HCN) gas poisoning when a stream of gas containing 19% of HCN was released from a leaking flange.

Case 1 : A worker collapsed and was treated on site with 2ml 4-dimethylaminophenol (4-DMAP). Upon arrival in the emergency room the cyanide level was 5.3/l in whole blood (after the application of 4-DMAP) amd methemoglobin was 5.5%. After decontamination he received 50 and 150 mg/kg b.w. N-acetylcysteine (NAC) respectively, as parallel acrylonitrile poisoning had to be suspected. In addition 30 and 40mL sodium thiosulfate 10% respectively were given. Cyanide levels dropped significantly to 0.035 on the following day, and the patient survived without sequelae.

Case 2 : A fire fighter wearing SCBA in attendance collapsed, was unconscious and had foam in front of his mouth and nose. He also immediately received 3ml 4-DMPA. Upon arrival in the emergency room his blood cyanide level was 6.75 mg/l in whole blood (after the application of 4-DMAP) and Methemoglobin was 5.5%. After decontamination he received 50 and 150 mg/kg

b.w. N-acetylcysteine (NAC) respectively, as parallel acrylonitrile poisoning had to be suspected. In addition 30 and 40mL sodium thiosulfate 10% respectively were given. Cyanide levels dropped significantly to 0.036 mg/l on the following day, and the patient survived without sequelae.

Comment: HCN gas can easily penetrate the skin and give rise to possibly fatal cyanide poisonings as in at least one case the uptake was strictly percutaneously, as a self-contained breathing apparatus (SCBA) had been used. The immediate application of 4-DMAP at about 50% of the recommended dose has been shown to be life-saving, even though methemoglobin levels remained relatively low. The high cyanide levels after 4-DMAP treatment show, that there is no definitely lethal cyanide concentration. It might be possible, that the rapidity of increase of the cyanide levels may play a greater role. The application of NAC plus sodium thiosulfate seems to be an efficient treatment of cyanide poisonings and should be evaluated further.

Stickel, 2008 (Zilker and Eyer, 2005; Zilker and Schweizer, 1987)

Stickel describes eight moderate and severe intoxications with HCN. All were treated with DMAP. Five of the persons survived with DMAP therapy, three died despite being treated with the antidote. Zilker and Eyer (2005) reported the same cases in a historical overview.

Case 1: A 34 year old analytical chemist had taken KCN in a suicide attempt. Twenty minutes later he was found comatose with loss of peripheral muscle tonus. He was then intubated, ventilated and given sodium bicarbonate, Ringer's solution and, as specific cyanide antidotes, 0.25 g DMAP together with 10 g STS. Ten minutes later he was alert and responsive again. After gastric lavage the patient was taken to hospital, where ventilation was continued, the gastric lavage was repeated and a second dose of DMAP was administered. The blood cyanide level was 2.4 mg/l two hours after ingestion. The MetHb reached 37.7% within five minutes after the second antidotal treatment. The patient developed mild haemolysis (bilirubin 4.7 mg/dl and Hb decrease from 13.3 g/dl to 10.3 g/dl) as side effect of the DMAP-application. Altogether he made an uneventful recovery and could be discharged a few days later. This case is also published by Zilker and Schweizer (1987). *Comment: Moderate intoxication with efficacy of DMAP and STS applied. Side effects (haemolysis) were probably due to the second dose, the need for which is unclear.*

Case 3: A 28 year old female chemical laboratory assistant committed suicide with cyanide. After 10 minutes she was found in a coma, with tachycardia, tachypnoea, but satisfying circulation. She was intubated, ventilated and 500 mg DMAP together with 25 g STS were injected. Furthermore she was given sodium bicarbonate and sedatives. Two hours later she was admitted to hospital being in a non-reactive coma. Nevertheless she regained consciousness a

few days later (despite severe haemolysis with bilirubin of 9.3 mg/dl, Hb fall from 13.6 mg/dl to 9.0 mg/dl and LDH maximum of 648 U/l) and recovered completely. The blood cyanide levels were 25, 31 and 0.6 mg/l 20 minutes, 2 and 11 hours after poisoning, respectively. The MetHb-level was measured 110 minutes after the antidotal treatment and was still within the therapeutic level (33.4%). *Comment:* ***Severe poisoning*** *with very high cyanide blood level. Recovery took several days, so it is not fully clear, whether this was due to DMAP and STS. The high dose of DMAP given prompted adverse effects.*

Case 4: A 39 year old goldsmith ingested 5 g of KCN. Ten minutes later he lost consciousness and became cyanotic with gasping respiration and tachycardia. The circulation remained quite stable. The ambulance staff intubated and ventilated the patient and administered 250 mg DMAP in combination with 10 g STS (the latter was given twice). This treatment resulted in spontaneous respiration, sweating and hyper-salivation. One hour after poisoning the blood cyanide level reached its maximum value of 1.46 mg/l. The patient remained in a coma for one day and on the respirator for two more days. Although he developed severe haemolysis (Hb decreased from 15.8 mg/dl to 9.3 mg/dl) he recovered completely. *Comment:* ***Moderate intoxication****, the effectiveness of DMAP and STS is not clear, as recovery took relatively long. Significant adverse effect of DMAP at normal dose.*

Case 5: A 56 year old goldsmith had inhaled NaCN gas while at work. Only a few minutes later he collapsed and exhibited convulsions. His circulation was stable and he breathed spontaneously, but with difficulties. The patient was intubated and 1,000 mg of DMAP (i.e. 4 times the recommended dose) were administered, together with 25 g of STS. This resulted in severe cyanosis with a MetHb-level of 73% within 90 minutes after the antidotal treatment. Therefore blood was transfused and the amount of MetHb could be reduced to 46.3% within two hours and 20 minutes after the overuse of DMAP (33.8% after three hours and 40 minutes). The MetHb level further decreased to 32.6, 21.6 and 2%, after 3, 4 and 5 days post exposure, respectively. The cyanide level in blood was determined once (at admission) with 37 mg/l. The overdosage of DMAP caused massive haemolysis (bilirubin 8.9 mg/dl, Hb decrease from 13.9 to 7.3 g/dl, LDH 6600 U/l), and intermittent renal failure. In the end the patient recovered completely. *Comment:* ***Severe cyanide intoxication*** *could be rescued with DMAP (and STS), which thus were effective. The overdosage of this antidote resulted in serious side effects (e.g. changes in the blood count, and renal failure). The blood cyanide level was extremely high (37 mg/dl), perhaps because of the cyanide bound to MetHb. No explanation is given for the administration of 1,000 mg of antidote.*

Case 6: A 17 year old schoolboy ingested KCN in a suicide attempt. Within 20 minutes he developed cardio-respiratory arrest (the cyanide level in the blood sample counted 34 mg/l but it was available after a few hours only). The ambulance staff did not have any cyanide antidote with them so 750 mg DMAP and 17 g STS could only be administered 1.5 hours later

(intermediate actions not described). Circulation stabilised straight after this treatment, but no EEG activity could be detected. The patient developed cerebral oedema, diffuse cerebral bleedings and acute renal failure. He died five days after the intoxication because of circulatory failure. *Comment:* ***Severe intoxication with antidotal treatment being established too late*** *and thus remaining ineffective. Multi-organ failure had already been initiated. Also the DMAP dose was very high.*

Case 7: A woman (aged 36) had ingested KCN. It took 90 minutes before the diagnosis of cyanide intoxication was established. During this time the treatment consisted of supportive actions like intubation, ventilation and application of catecholamines, as the patient was in a coma, apnoeic and showed circulatory failure. After being diagnosed correctly the woman received 250 mg DMAP and 10 g STS. This resulted in sufficient circulation (blood pressure 150/100 mm Hg, tachycardia), but no EEG activity could be reconstituted and she died five days later. The cyanide levels were 10.96 mg/l two hours later, and approximately 1 mg/l 11 and 24 hours post exposure. The MetHb-level reached 19% within 15 minutes after the antidotal treatment. *Comment:* ***Severe poisoning, late initiation of antidotal treatment****, which improved circulatory, but not neurological status and overall was only partially effective – probably due to treatment delay.*

Case 8: A 40 year old woman collapsed after cyanide ingestion. The ambulance arrived 45 minutes later and found a comatose patient in cardiac arrest. She was intubated, ventilated and received 250 mg DMAP and 10 g STS. This led to satisfying circulation, but a few hours later irreversible ventricular fibrillations occurred and the patient died. The cyanide level in blood was 14 mg/l and MetHb 14.8%, both 45 minutes after the antidotal treatment. *Comment:* ***Severe intoxication*** *with very high cyanide level. DMAP and STS were only temporarily effective to improve circulation.*

In conclusion, Stickel reports on eight cases of cyanide poisoning, all treated with DMAP. The antidote worked well for five patients (moderately and severely poisoned ones). But three patients with severe intoxications died as the antidote was not effective at all. Mild haemolysis was regularly seen, even under normal dosage; nevertheless this adverse effect could have been easily managed. The MetHb level became life threatening for one patient who had been given an antidote dose that was too high. However this could be treated and the patient survived without sequelae. The case reports show that DMAP is an effective antidote even in severe cases. It must be admitted that after cardiac arrest had taken place, DMAP was not as effective as in patients whose circulation was stable. Under the recommended dose the adverse effects are mild and can be managed without any problems. Severe side effects normally occur only if DMAP is overdosed. All patients should receive STS to support the cyanide elimination.

Suchard et al, 1998

A 41 year old woman ingested about 30 chewed apricot kernels as an alleged health food. Twenty minutes later weakness developed, followed by dyspnoea; the paramedics found her in a deep coma and unresponsive to painful stimuli. Cyanide poisoning was assumed and later confirmed, the application of AN, SN and STS (Eli Lilly Kit) was successful and led to immediate improvement. As four hours later cyanide in blood was still or again about 1 mg/l, a second half dose of the antidote kit (SN and STS) was given. After the first dose MetHb was 7.3%, and 10.5% after the second dose. *Comment: In this severe case of poisoning AN/SN/STS were fully effective.*

Sutter et al, 2010

A 34 year old female, suspected to have cyanide poisoning because of decreased oxygen extraction from arterial to venous side and profound metabolic acidosis, had a cardiac arrest and was resuscitated. Under the suspicion of cyanide poisoning HOCO 5 g followed by STS 12.5 g were given. This led to rapid improvement, although a dramatically elevated lactic acid level persisted. Haemodialysis was attempted, but could not be performed due to false 'blood leak' alarm of the machine, which was caused by the plasma discoloration from HOCO. *Comment: HOCO and STS were effective in this severe case.*

Tartière et al, 1992

After ingestion of KCN, a 32 year old man was found sweating and with convulsion-like movements, though awake. Oxygen was given; then 5 g HOCO as infusion for over three hours, in spite of circulatory stability and only slight somnolence. He fully recovered after the treatment. *Comment: In this mild poisoning case, HOCO led to recovery and was effective.*

Tassan et al, 1990

A 15 year old girl tried to kill herself by intake of 2.5 g KCN. One and a half hour later she presented comatose, with hemodynamic instability and severe metabolic acidosis. In the ED gastric lavage was performed, mechanical ventilation initiated and sodium bicarbonate and beta-mimetics administered. She received 4 g of HOCO as specific cyanide antidote and 8 g of STS. Upon admission, the cyanide level was 494 µmol/l (13.3 mg/l, high). The girl recovered completely, despite of the delay in the therapeutic intervention and the severity of the intoxication. *Comment: Very high cyanide level, yet full recovery after HOCO/STS in this severe case of poisoning.*

Thiess and Hey, 1969

A further case of acetone cyanohydrin poisoning has been reported in a 23 year old worker filling acetone cyanohydrin from a rail tanker into steel drums. One of his cotton gloves was drenched, which he did not notice. He put the glove into his pocket. Five minutes later nausea set in, vomiting occurred, and ten minutes afterwards, he lost consciousness and collapsed. AN and artificial respiration led to short improvement, but then coma and convulsions set in. The use of SN and STS also improved the status only for minutes. After the glove had been found, clothing was changed, and a decontamination bath was performed. *Comment: SN and STS led to an immediate improvement and were effective. Cyanide was not measured.*

Thomas and Brooks, 1970

A 19 year old boy who was working in a photography shop had his face contaminated with KCN powder. A short time later, he complained of stomach cramps, was pale and lost consciousness. Twenty minutes after the incident he arrived at the ED. Once there his blood pressure went down to only 90/70 mm Hg. His stomach was washed and he was given the nitrite antidote kit (AN pearls, SN 300 mg and STS 12.5 g). After the antidotes had been administered, he quickly regained consciousness. As pain in his stomach, agitation and a weakness of the upper extremities persisted, the therapy was repeated two hours later (half the initial dose) and again ten hours afterwards (whole dose because of a new opisthotonos). The patient recovered completely. *Comment: This was moderate cyanide poisoning effectively treated with AN/SN/STS. Whether the third dose had really been necessary might be discussed.*

Townsend and Boni, 1975

A 34 year old man ingested 48 apricot kernels, both raw and in a milk shake, and developed vomiting, headache, flushing, perspiration, dizziness and faintness. After induced vomiting he recovered quickly. *Comment: No cyanide measurements were done, no antidotes were applied.*

Trapp, 1970

Trapp reported the case of a 30 year old male who crouched in a tank full of silver cyanide to escape police after a robbery. He fell and immediately lost consciousness with seizures despite AN and i.v. STS. He received one session of hyperbaric oxygen at 3 ATA which resulted in prompt woke up with a good urine flow and correction of metabolic acidosis with IV sodium bicarbonate. Therefore, the pressure was slowly reduced to 2 ATA. The pH was measured during the hyperbaric oxygen therapy at 7.2. He remained alert, volunteered his name, and said he had

swallowed ‘gallons’ of tank solution. He was discharged without sequelae but with burning sensation of his right eye and scrotum. *Comment: A combination of oral-dermal-inhalation intoxication can be assumed. Cyanide levels in the blood were not measured, clinical data are incomplete. AN/STS were not effective in this case, but the doses were not given.*

Turchen and Manoguerra, 1989; Turchen et al, 1991

A 39 year old woman ingested 59 ml of Super Nail Off (containing 99% acetonitrile) in a suicide attempt. Admission vital signs were blood pressure, 108/60 mm Hg; 124 beats/min; respiration 28 breaths/min; compensated metabolic acidosis with a normal arterial pH and a base deficit of 17. The whole blood cyanide level was 313 µg/dl and the ethanol level 150 mg/dl. The patient underwent gastric lavage. Following a latent period of approximately 12 hours, the patient developed cyanide poisoning with severe metabolic acidosis, seizures, and shallow respirations. She responded to the administration of SN (0.3 g) and STS (12.5 g), although the administration of nitrite produced bradycardia and hypotension. She developed several relapses over the course of her hospitalisation and each time responded to STS administration (at 26.5 hours 12.5 g STS, at 34.5 hours 0.3 g SN plus 12.5 g STS were given i.v.). Nine hours later she was given another 12.5 g STS with a rapid response. Her cyanide level peaked 59 hours post-ingestion at 1.281 mg/dl. Laboratory tests showed a continuing metabolic acidosis and mild hypernatraemia. She was tachypnoeic with a respiration rate of 28 breaths/min. Sixty-five hours after ingestion, her cyanide level was 1049 µg/dl and thiocyanate was 12 µg/dl. Despite persistently high cyanide levels her vital signs improved on the fourth day. She was discharged the morning of her sixth hospital day. *Comment: The delayed onset of symptoms is due to delayed cyanide formation from acetonitrile. The administration of STS was more effective than SN which exacerbated the patient’s hypotension as side effect.*

Uitti, 1985

An 18 year old man ingested 975 to 1,300 mg of KCN in a suicide attempt. In the ED he was comatose, not responding to painful stimuli. Blood pressure was 160/70 mmm Hg, and pulse was 108/min. He was treated with gastric lavage, endotracheal oxygen, and i.v. SN and STS. He regained consciousness in 7 hours, but he had severe Parkinson syndrome, characterised. He died 18 months later after he had taken an overdose of imipramine and alcohol. *Comment: In this severe poisoning SN/STS were effective with some delay. The reason for the Parkinson syndrome is not clear.*

Valenzuela et al, 1992

A 16 year old man ingested 1 g KCN. On admission he was comatos, his blood pressure was undetectable, and he had deep respiratory depression. His heart rate was 70 beats/min. Besides cardiopulmonary resuscitation, he received i.v. STS. Three hours later he was fully conscious. After 24 hours he was neurologically intact and the results of general physical examination were normal. A few days later he developed a severe dystonia syndrome. He had a positive response to an apomorphine test and showed improvement with levodopa treatment. A 21-year follow-up showed minimal neurological sequelae; CT showed bilateral putaminal lucencies. *Comment: The administration of STS was successful, but a delayed severe dystonia syndrome occurred.*

Zaknum (2005) + Rachinger (2002), 2005

A 35 year old female laboratory assistant attempted suicide by taking an unknown amount of cyanide. 10 minutes later she was found comatose with stable cardiovascular function and was immediately given emergency treatment. Under cotrolled respiration she was administered DMAP and STS, which resulted in a linkeage with cyanide, and anto oedema treatment was administered. After withdrawal of sedatives and respirator she exhibited agitation and akinetic mutism. A dopamine agonist (amantadine sulphate) was administered i.v. and later orally. She received extensive phsyio-, logo- and ergotherapy but CSN necrosis and sequelae persisted.

van Dijk et al, 1987 / van Heijst et al, 1987

A 21 year old male was found deeply comatose. His sister found NaCN pellets nearby and called for help. He arrived at hospital about 1 hour after ingestion. He was cyanotic and deeply comatose with bradycardia and first-degree AV block, which descended into cardiac arrest. He was resuscitated, but remained cyanotic. He received STS 20 g, then about 1 hour later received 300 mg SN. Fifty minutes after dosing, his MetHb was ‘only’ 3.6%, so he received 250 mg 4-dimethylaminophenol (DMAP). Twenty-five minutes later he was so deeply brown-blue cyanosed that the physician administered empirically 160 mg toluidine blue. A MetHb obtained shortly thereafter revealed 69% methaemobloglobinaemia. Toluidine blue 250 mg was given and the MetHb dropped to 46%, then later to 4.4%. Because this MetHb level was considered too low, DMAP was injected again, the physician this time giving only 80 mg, which resulted in a MetHb of 52%. Over the next 10 hours, DMAP was administered 5 times with close monitoring of MetHb. During the whole treatment, the patient remained deeply comatose. Thirty hours after admission, ventricular fibrillation occurred and he died.

van Heijst et al, 1987 / Douze et al, 1987

All the patients reported by van Heijst received combinations of different antidotes together with HOCO in a sub-therapeutic dose. The low doses of HOCO applied in these cases are not considered to be usable for assessment.

Case 1: A 32 year old male who attempted suicide by ingesting 3 g of KCN. He was found unconscious with inadequate respirations, receiving mouth to mouth respiration. He was extremely bradypnoeic on admission (respiration rate = 2), with heart rate 30 beats/min and blood pressure 110/80 mm Hg. A smell of bitter almonds led clinicians to administer 10 ml SN and 50 ml STS. Convulsions were treated with diazepam and acidosis with sodium bicarbonate. Because he remained unconscious, the treatment with SN and STS was repeated. One hour after admission, he awoke, complaining of shortness of breath. His skin colour was brownish blue. The MetHb was 58%. He received methylene blue 30 ml of a 1% solution. He was transferred the next day from the ICU to psychiatry in good physical condition. *Comment: No blood levels determined, but serious clinical presentation this seems to have been moderate poisoning. SN/STS at double dose was effective, but caused high MetHb levels.*

Case 2: A 23 year old male student took KCN. The dose could not exactly be determined. In addition he tried to inhale some HCN-gas by heating KCN. He was brought to hospital approximately 20 minutes after the event. On admission the patient was semi-comatose and in convulsions. The heart rate was 100/min, the blood pressure 120/60 mm Hg, the respiratory frequency was normal. He was given 20 mg of HOCO i.v. followed by intubation and artificial ventilation with 100% oxygen. 100 ml of 25% STS were administered i.v. As the dose of HOCO was insufficient DMAP was administered i.v. By mistake 1,000 mg were given. The MetHb rose to 70%. This was treated by toluidine blue. Three hours later MetHb came down to 25%. The patient developed jaundice caused by massive haemolysis. The highest bilirubin was 7.2 mg/dl, the lowest Hb was 7.7 g/dl. The anaemia was treated with blood transfusions. The patient survived. *Comment: Patient treated with DMAP as main antidote (highest dose).*

Vogel et al, 1984

A 24 year old officer on a cargo ship was sprayed with acrylonitrile, when a valve burst. He had dizziness, flushing, nausea and vomiting and soon began to hallucinate. The cyanide level was 0.13 mg/l upon admission and reached a maximum of 0.184 mg/l one day later. The patient was treated with 15 (!) doses of the “cyanide kit”, each containing 10 ml 3% SN and 50 ml 25% STS solutions, over 72 hours. Even though the highest MetHb level reached was 14.5%. When the level fell below 10%, the patient started hallucinating again, convulsions occurred once. Additionally oxygen and HOCO were given at an unspecified dose. Extra-corporal dialysis was attempted influencing the hallucination ascribed to cyanide. Later the laboratory values indicated

rhabdomyolysis, which was treated by hydration. The patient made a full recovery. *Comment: The measured cyanide levels were insignificant and do not explain even the mild to moderate course. This is in line with the inefficacy of the cyanide antidotes. It can be disputed, whether this was acrylonitrile, and not cyanide poisoning.*

Vogel, 1991

A chemist, 30 years old, tried to commit suicide with KCN which he had taken from his lab. He was seen in the emergency department in a comatose state, with a tachycardia of 120/min and with gasping respiration (20/min). He was unresponsive to painful stimuli and the corneal reflex could not be triggered. The anion gap was 30 mmol/l and the base excess at 17.8 mmol/l. The remaining biochemical parameters were within the normal range. He was intubated, ventilated and gastric lavage was performed. Moreover he got activated charcoal and forced diuresis was induced. No specific antidotal treatment was applied. Nevertheless the patient recovered with supportive care only. He became alert three hours later and could be extubated. Six hours after the suicide attempt he admitted the intake of cyanide and the blood cyanide level was then measured (0.23 mg/l but that is not valuable because of the long delay). *Comment: Obviously a severe poisoning, survival with supportive treatment only.*

Wananukul and Kaojarern, 1992

A 19 year old male accidently ingested KCN at an electroplating shop. He vomited shortly after, turned red, had a generalised seizure and became unconscious. In the ED he was stuporous, with shallow respirations. Initial blood cyanide concentration was 12 mg/l. He received 10 ml of SN some 3.5 hours after admission then, STS. He was described as having fully recovered six hours after treatment. *Comment: This moderate poisoning was effectively treated with SN/STS.*

Weng et al, 2004

A man, aged 51, took an uncertain amount of KCN solved in a milk-emulsion (3 g of KCN per litre milk) in a suicide attempt. Twenty minutes later he had become non-reactive, collapsed and lost consciousness. He developed convulsions and stayed in coma. The blood pressure was 77/60 mm Hg one and a half hour after the ingestion of the poison. He was tachycardic (147/min) and exhibited 7 points on the GCS. The blood gases showed a metabolic acidosis (pH 7.22) with a lactic acid of 12 mmol/l. The cyanide level in the blood was 2.9 mg/l. The patient was intubated, ventilated and was given activated charcoal. It took 2.5 hours before 5 g of HOCO were administered i.v. The blood cyanide level increased to 4.2 mg/l one hour after the antidote application. The patient's clinical conditions improved step by step and three hours later

he could be extubated and he could be discharged after five days in complete remission. *Comment: This is a severe poisoning case effectively treated with HOCO, even though this was applied late, and though the cyanide level initially increased after antidote application.*

Werner, 1979

This is a dissertation on the treatment of poisoning with hydrocyanic acid, its salts and derivatives with 4 DMAP. All cases included in the dissertation are included in the publication by Zilker and Eyer, 2005.

A 30 year old woman ate 35 pieces of bitter almonds as she wanted to commit suicide. She felt sick and dizzy. The vital parameters were stable as was the consciousness. She was administered 250 mg DMAP and 10g STS and recovered completely. *Comment: A very mild poisoning not requiring treatment. So the antidote efficacy/effectiveness cannot be assessed.*

The second case reports on another woman, aged 38, who had ingested bitter almonds. She complained about dizziness, nausea and a sore throat. She smelled like bitter almonds. The blood pressure was 115/70 mm Hg, HF 100/min. She was administered 500 mg DMAP together with 15 g STS. The treatment caused severe haemolysis and the patient reported a headache. The side effects could be treated within a few days. *Comment: Mild poisoning, no antidote indication, so that efficacy/effectivenes cannot be evaluated. Instead significant adverse effects occurred at double dose.*

Wesseon, 1985

A 59 year old scientist was found comatose with a suicide note thereby. He was given fluids i.v. and naloxone. When coming to the emergency department he showed spontaneous respiration, the pupils were reactive to light but reflexes could not be triggered. The blood pressure was 140/80 mm Hg, the heart rate tachycardic (136/min) and the respiration fast (32/min). The pH decreased form 7.13 at admission to 6.99 after half an hour, despite bicarbonate infusion. The lactate was very high with 29.2 mmol/l (but it seemed to be relatively well compensated). He was intubated, the stomach was washed and activated charcoal was administered. At this time the poison was not known so a haemodialysis was performed (also for treating the acidosis). After some time a friend of the patient's suspected cyanide intoxication and the nitrite antidote kit was then applied (SN 300 mg and STS 12.5 g). The man became alert after about five hours and made an uneventful recovery. The cyanide level was determined at the beginning (0.4 mg/l), shortly before the antidotes were given (2.1 mg/l) and at the end of the dialysis (0.1 mg/l). *Comment: In this (probably) moderate poisoning SN/STS were effective in spite of late application.*

Wolfsie, 1951

Wolfsie reports on 12 cases that had been accidentally poisoned by cyanide either via the respiratory or via the dermal route. All were male workers between 29 and 48 years. Two patients had suffered from mild intoxications without loss of consciousness and ten from moderate poisonings with coma but sufficient circulation. The mild and four of the moderate intoxications were treated with AN, the other six with SN (0.3 g) and STS (12.5 g). Three of these patients received the antidote only after spontaneous clinical recovery. All patients were ventilated and decontaminated when the exposure route had been the dermal one. The men recovered completely. *Comment: 12 mild or moderate intoxications were treated with nitrites, which showed good efficacy. Diagnosis by the patients' colleagues might not always have been reliable. In at least in 3 cases antidote (given after recovery) were not required.*

Wright and Vesey, 1986

A 42 year old woman drank 100 ml of a potassium-gold-cyanide-solution ($KAu(CN)_2$, corresponding to 540 mg cyanide and 2.0 g gold) in a suicide attempt. She vomited at once, but did not go to the hospital before two hours had elapsed. Once there she was responsive, with a heart rate of 110/min. Gastric lavage was performed as first line therapy. Two hours later the patient became drowsy, tachypnoeic and hypotensive (blood pressure 90/60 mm Hg), oxygen and 2 ampoules of AN were given per inhalation. As specific antidote 300 mg of Co-EDTA together with 4 g of glucose in 20 ml were infused. She reacted anaphylactic to this treatment in the form of a flush and peri-orbital oedema. In the further course the patient developed more and more hypotonia and tachycardia as well as she became anuric. Steroids, adrenaline, antihistaminics, i.v. fluids and oxygen were administered, however, her clinical conditions deteriorated. She had to be intubated 9.5 hours after the intake of the poison (five hours after antidotal treatment). She died 3.5 hours later because of cardiac arrest. The blood gases were determined as well as the cyanide levels in the plasma and in the erythrocytes (Table below).

Blood measurements by Wright and Vesey, 1986

Time (h)	Event/parameter	Remark				
16.30	Ingestion of $KAu(CN)_2$					
19.00	Admission to hospital					
21.00	Administration of antidote					
	pH	**Base excess (mmol/l)**	**Blood CN (µmol/l)**		**Blood CN (mg/l)**	
			Plasma	Erythrocytes	Plasma	Erythrocytes
21.35			2.1	188.5	0.056	5.1
22.05	7.30	–6.7				
23.50			0.5	42.9	0.013	1.2 [a]
1.00.	7.30	–11.2				
2.30	7.15	–17.0				
2.40			0.14	5.5	0.004	0.15
3.10	7.20	–10.5				
5.15	7.18	–8.7				

[a] The gold level was 14.2 mg/l

Comment: Initially the intoxication did not seem to be very serious, but in spite of antidote application (AN and Co-EDTA) it had a fatal outcome. This could have been because of the delayed treatment, or a combination of gold and cyanide intoxication and the patient died most likely due to the gold poisoning for which there is no real antidote. The cyanide antidote Co-EDTA caused severe side effects in the woman, although it had been administered in the recommended dose.

Wu et al, 2001

A 27 year old attempted to commit suicide by ingesting 5 ml gold KCN solution ($KAu(CN)_2$). He developed vomiting and abdominal pain three hours later. At the hospital gastric lavage and activated charcoal was administered. He was referred to another hospital (based on the suspicion of cyanide intoxication). On arrival, he was conscious and complained of vomiting and showed hyperamylasaemia, and hepatic dysfunction. The blood cyanide test was reported as undetectable (<0.1 mg/dl). A cyanide antidote was not given. The authors mentioned that aurocyanide is extremely stable; so that very little cyanide should be liberated (formation constant is 10 exp. 38). *Comment: Not a significant cyanide poisoning and no antidotes required.*

Würzburg, 1996

Thirty-seven employees were exposed to cyanide between 1956 and 1985. One was found dead. Thirty-six employees were treated; most were given nitrite and oxygen. Some received oxygen alone. All recovered completely. One-third of these employees were unconscious. One was convulsing. Most were discharged home at 6 h post-exposure. Some employees remained at the plant to work an additional shift. AN and/or oxygen were the only agents used with 33 employees. Forced oxygen was administered to the unconscious, apnoeic employees. Three employees were given SN and STS i.v. Treatment generally began within 3 min. In 5 to 20 minutes all of the unconscious employees reacted positively to the use of forced oxygen and forced AN or SN. There were no residual effects except headache and transient loss of appetite. AN and oxygen have been effective tools in the treatment of cyanide intoxication at this plant site. There have been no intercurrent or residual drug effects that outweigh the life-saving capacity of these agents. SN should be employed if the use of oxygen and AN fails to improve the cardiovascular status/level of consciousness in 5-10 min.

Yacoub et al, 1974

Case 2: A chemical analyst (50 years old) who was working in an electro-plating company tried to commit suicide by taking approximately 1 g of KCN. He complained about stomach pain immediately and he smelt of bitter almonds. His sons took him to the ED. There he presented somnolent and cyanotic about 30 minutes after the ingestion. He had vomited more than once, was shivering and showed a both-sided miosis (*in contrast to the mydriasis generally seen in cyanide poisoning*). The blood pressure was 90/60 mm Hg and the heart rate 140/min. He was acidotic (pH 7.25) and the cyanide level in the blood was 15 mg/l for the free ion and 42 mg/l for the whole blood cyanide (very high for comparatively mild symptoms). After gastric lavage 50 ml of a 10% STS solution was instilled, parallel he was treated with 2 pearls of AN per inhalation, glucose and sodium bicarbonate i.v. As cyanide antidotes he received 0.3 g of Co-EDTA and 2 g of STS, followed by 4 g of HOCO. Within a few minutes he developed an allergic reaction (urticaria, Quinke's oedema) that could be treated with steroids and antihistaminics. He showed a pink coloured skin because of the HOCO-infusion. Five hours and a half after the incident the patient was conscious, a bit drowsy and tachypnoeic (respiration frequency 24/min). He could be discharged at the fifth day in complete remission. The free blood cyanide level before treatment with antidotes were 15 mg/l (total 42 mg/l) and after treatment (second day) 0.18 mg/l (total 50.8 mg/l). *Comment: The blood-cyanide level was extremely high, maybe erroneously high in comparison to the relatively mild clinical presentation. The need for combined antidotal treatment with AN, Co-EDTA and HOCO is not obvious. Significant side effects (allergy) were seen, which can have been due both to CO-EDTA and especially HOCO.*

Case 1: M. S. Lorenzo, aged 27, working in a factory using cyanide, attempted to commit suicide by ingesting an unknown quantity of potassium cyanide on 14 June 1973. He was driven to hospital and taken to the intensive care unit. Upon arrival the patient was in cardiac arrest with bilateral mydriasis, sweating and cyanosis. Resuscitation attempts, with external cardiac massage, intubation and assisted ventilation, administration of isoprenaline and THAM proved ineffective. Electrocardiogram confirmed the existence of a cardiac arrest that remained irreversible. An anatomical examination performed 48 hours later shows the existence of a strong odor of cyanide at the opening of the body, a massive congestion of all organs, "buff" looking liver, the presence of liquid in the bronchi and hemorrhagic staining of the gastric mucosa.

Yatziv and Simcha, 1969

In Israel a two year old girl ingested a large amount of apricot pits. A few minutes later the first symptoms appeared, and she quickly became unconscious. This coma lasted for 45 minutes. Gastric lavage and oxygen led to rapid improvement, and the girl recovered fully within eight hours. *Comment: No antidote was given.*

Yeh et al, 1992

A 75-year old non-smoking monk was admitted to the hospital with altered mental status and apperent seizure activity. He had no significant past history, but he did posess Laetrile. Prior to admission, the patient had been alert and weak, with abdominal discomfort. An episode of emesis was followed one hour later by coma. On admission the patient had a tremor and was unresponsive to painful stimuli. Vital signs were pulse 114/min with atrial fibrillation, respirations 28/min, blood pressure 157/67 mm Hg. The pupils were equal with diminished reactivity to light. Both gag and deep tendon reflexes were decreased. The admission arterial blood gas value on 6 litres nasal oxygen/min was pH 7.31, pCO2 16 mm Hg, pO2 154 mm Hg, oxygen saturation 99%. Initial therapy was lorazepam, later a Swan-Ganz catheter was placed and administration of dopamine and dobutamine was started. Seven hours later, the patient became alert and revealed that he had ingested 10 Laetrile tablets. He received 25g of sodium thiosulphate and gradually improved, and was discharged within 5 days. Subsequently, his cyanide level on admission was found to have been markedly elevated at 180 micromol/L. *Comment: The administration of thiosulphate was successful.*

Yen et al (1995)

21 cases of cyanide poisoning were identified at the Taipei Veterans General Hospital from 1985 to 1992. No individual case details are provided. This group of 21 was selected using specific

criteria from among 138 cases of cyanide poisoning seen in the hospital. Fifty-three of the 138 received the cyanide antidote kit, of whom 38 (72%) survived. Among the 21 selected cases, 5 were identified as having no or mild effect. The remaining 16 were identified as having severe effects. Two of 5 in group 1 received the cyanide kit. Apparently, all but two in Group 2 received the kit. Six patients died in that group in spite of the kit for an overall survival in selected patients of 71%. Two groups are differentiated. 5 exhibited mild effects two of which got antidote treatment with the nitrite antidote kit. All survived and would have probably survived without any antidotal treatment. 16 cases were graded as severe. 15 of them had lost consciousness, showed respiratory suppression, 4 apnoea, 5 bradycardia, 8 had hypotension, 4 a cardiovascular collapse, 13 metabolic acidosis. 8 of the survivors had antidotal treatment, 6 of the none-survivors had antidote treatment, but two had no such therapy. The survival rate in the treatment group was 57%. The survival rate in the group which did not get the antidote was zero if only the severe cases are taken into consideration. *Comment: The study points to a positive effect of the treatment with the nitrite antidote, but the number of severe cases which had no treatment and died was very small (2).*

Zaknum, 1905

A 35 year old female technican ingested a lethal dose of KCN in a suicide attempt at a hospital`s laboratory. DMAP and STS were promptly administered. On admission to the ICU 15 minutes after intoxication she was in coma but pupils were not dilated, blood pressure was 120/70, heart rate of 110/min, mixed crepitate rales on the base of the lungs were reported. Following artificial coma she presented with an agitative state for several days followed by akinetic mutism, buccofacial and ideomotoric aphasia. Severe rigid-akinetic syndrome, dysarthria, dysphagia and generalized dystonia developed weeks later. MRI revealed lesions in the caudate and lentiform nuclei, precentral cortex, and cerebellum. SPECT by (123-I)2ß-carbomethoxy-3-ß-(4-iodophenyl)-Tropan on two occasions revealed progressive loss of dopamine transporter suggestive of nigral neuronal aptosis. Striatal and frontal hypometabolism and hypoperfusion were found by FDG-PET and HMPAO-SPECT. *Comment: Successful administration of DMAP and STS (doses were not reported) after ingestion of a lethal amount of KCN, but significant neurological sequelae.*

Zavotsky et al, 2004

A 52-years-old man came in contact with a liquid chemical believed to be 2,4,5 trichhloro-6-carbopentoxyphenyl oxylate. Approximately two to four hours after exposure, he felt nauseous and began vomiting. Upon arriving at the emergency department, the staff decontaminated the patient. The vital signs were 109 heart beats/min, 20 breaths/min, blood pressure 110/38 mm Hg and SaO_2, 98%. He was still vomiting and complained of mild upper abdominal pain. Based on

laboratory findings and the patient's presentation, he was evaluated and treated for acute biliary disease and possible pancreatitis. After 18-20 hours after exposure the patient became acutely confused with BP of 80/30 mm Hg, and increasing heart and respiratory rates to 120 beats/min and 40 breaths/min. The patient's agitation became unmanageable, so he was sedated and intubated. BUN and creatinine were now 6 mg/dl, and 2.3 mg/dl, respectively, with associated oliguria, and a lactate level of 10.06 mmol/l indicating a profound metabolic acidosis. In a urine screen a thiocyanate level of >64 mg/l was found. After a further request to the employer a MSDS of acetonitrile was provided. 300 mg SN were administered intravenously over 10 minutes followed by 12.5 g STS intravenously over 10 minutes. Immediately the patient's condition began to improve. The patient was monitored for 72 hours. He had an acute episode of confusion shortly after beginning transferred to a medical-surgical floor and was treated again with SN and STS as a precaution. He was discharged home after six days.

Zilker, 2009

A female patient had ingested cyanide in a suicide attempt. She lost consciousness but was cardiorespiratory stable. 250 mg of DMAP were administered and she recovered completely. Cyanide was detected in the blood, quantity not given. *Comment: The case was reported on personally. DMAP showed good efficacy with no adverse effects. Although it was not explicitly cited it seems likely that the patient was given additional STS as this is recommended in cyanide poisoning.*

Zilker and Eyer (2002)

Seven intoxications with hydrogen cyanide through inhalation or inhalation together with skin contact are reported on. Five patients suffered mild intoxications (not even in coma). These persons were given 250 mg DMAP between 12 minutes and 5.5 hours after exposition and all recovered completely. The antidotal treatment did not lead to any adverse effects. The other two patients had been poisoned more seriously. They presented in deep coma. They also received 250 mg DMAP each and made an unremarkable recovery (one showed an elevated MetHb-level of 40% but without any symptoms of haemolysis). 5 mild intoxications, 2 moderate intoxications. All seven patients made a full recovery under DMAP treatment, but whether it was actually indicated in the mild poisoned patients could be discussed.

Zilker and Eyer, 2005

Zilker and Eyer reported 23 cases of intoxications with cyanide in a historical overview in 2005. Of the 23 cases 1 was an accidental administration of DMAP, 13 were also published by Werner (1979) in his thesis, 1 by Daunderer (1974) and a further 8 by Zilker and Stickel (2008).

Zilker, 2010

Personal communication. A 64-year female patient ingested an unknown amount of KCN showing signs and symptoms of moderate cyanide poisoning. After administration of 250 mg DMAP and 10 g STS she made a full recovery.

APPENDIX D: EFFECTS OF HOCO ON LABORATORY MEASUREMENTS AND PARAMETERS

Tissue / Parameter	Apparatus	HOCO (mg/l)	Interference (Change of concentration)	Remark	Reference
Plasma *in vitro*					
AST	Boehringer Mannheim/Hitachi 736 Analyzer	100, 500, 1,000	Decrease	At all three HOCO concentrations	Curry et al, 1994
GGT			Decrease		
Phosphorus			Increase		
Triglycerides *			Increase		
Human blood *in vitro*					
Total Hb	Co-oxymeter IL 482, CCD 270, AVL 912, OSM3, ABL 520	192.5 – 798.4 mg/l (143 - 593 µmol/l)	Increase	Overestimation of all three parameters, the magnitude of which depends on the concentration of HOCO	Gourlain et al, 1995
COHb			Increase		
MetHb			Increase		
LDH	Vitros® 750 XRC	31 - 1,125	Inaccuracy	At > 600 mg HOCO/l	Vest, 2002
Bilirubinaemia (total and conjugate), iron, phosphorus, creatinine, CPK, AST, protein, glucose, lactate			Inaccuracy	At all HOCO concentrations	
Bilirubinaemia (total and conjugate), creatinine, CPK	Cobas® Integra 700	31 - 1,125	Inaccuracy (≥ 10%)	At ≥ 124 mg HOCO/l	Vest, 2002
Total Hb	Co-oxymeter GEM Premier 4000s	0, 0.25, 0.5, 1, 2 g/l	Increase	Up to 2.6 g/dl	Pamidi et al, 2009
HbO_2	IL 682		Decrease	2 - 20% bias	
COHb	Radiometer ABL 735		Increase	0 - 6% bias	
MetHb	Siemens RapidPoint 405		Increase	0 - 15% bias	

Tissue / Parameter	Apparatus	HOCO (mg/l)	Interference (Change of concentration)	Remark	Reference
Rabbit blood *in vivo***					
Total Hb	Co-oxymeter	165 mg/kg i.v.	1.784 mM	Overestimation (2 - 3%)	Lee Ann, 2007
HbO_2		1.784 mM	0.892 mM	Underestimation (62 - 8%)	
COHb		0.892 mM	0.892 mM	Overestimation (2 - 16%)	
MetHb		0.382 mM	0.892 mM	Overestimation (1 - 2%)	
Swine blood *in vitro* (haemolysed, oxygenated, de-oxygenated)					
MetHb	Spectrophotometer Varian Cary 100	X = 317.46		Overestimation from 0.1X to 10X	Denninghoff et al, 2008
		0.1X 0.33X 2X 3.3X 6.66 X 10X 20X		The clinical relevance of this study is questionable	

* HOCO had no effect on the concentration of Albumin, Alkaline Phosphatase, ALT, Amylase, BUN, Calcium, Cholesterol, Cholinesterase, CPK, Creatinine, Glucose, Iron, LDH, Magnesium, Total bilirubin, Total protein, Uric acid

** New Zealand strain

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Responsible Editor:
Dr Alan Poole
ECETOC AISBL
Av. E. Van Nieuwenhuyse 2 (box. 8)
B-1160 Brussels, Belgium
VAT: BE 0418344469
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