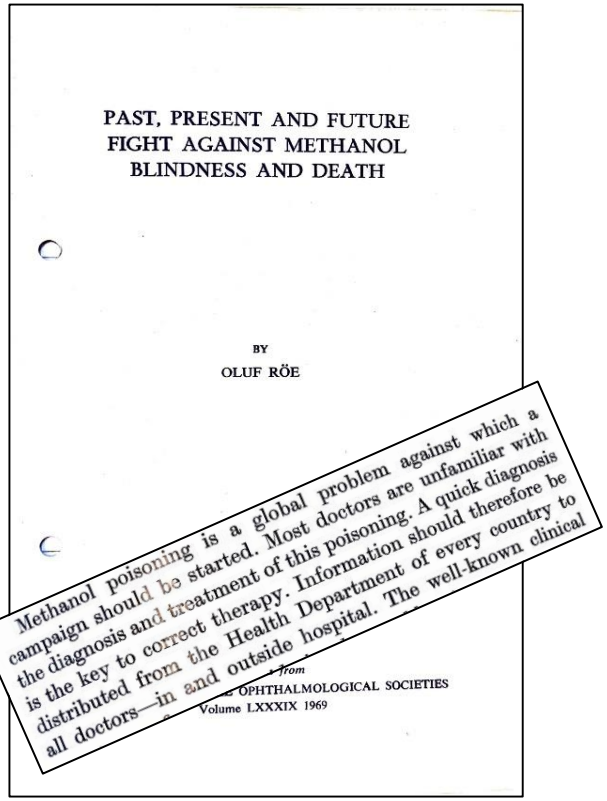
Methanol Poisoning condensed – a simplified approach to theory and clinical practice


PAST, PRESENT AND FUTURE
FIGHT AGAINST METHANOL
BLINDNESS AND DEATH

BY
OLUF RÖE

Methanol poisoning is a global problem against which a campaign should be started. Most doctors are unfamiliar with the diagnosis and treatment of this poisoning. A quick diagnosis is the key to correct therapy. Information should therefore be distributed from the Health Department of every country to all doctors—in and outside hospital. The well-known clinical

from
OPHTHALMOLOGICAL SOCIETIES
Volume LXXXIX 1969

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Asia Pacific Association of Medical Toxicology

(WWW.APAMT.ORG)

جمهوری اسلامی ایران
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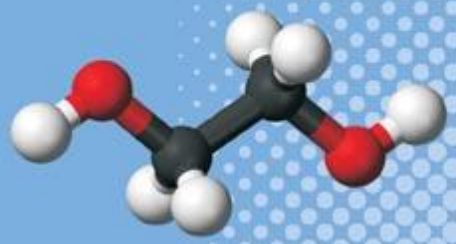
راهنمای بالینی درمان مسمومیت با متانول



اداره پیشگیری و درمان سوء مصرف مواد
دفتر سلامت روانی، اجتماعی و اعتیاد
تابستان ۱۳۸۸

راهنمای بالینی درمان مسمومیت با متانول

جمهوری اسلامی ایران
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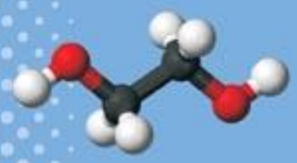
Ministry of Health and Medical Education

Clinical Guideline for

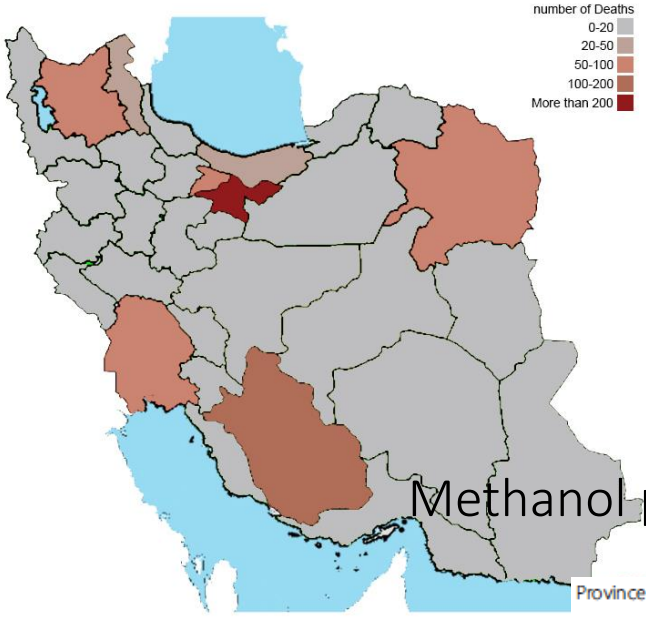
Treatment of Methanol Poisoning



Substance Abuse Prevention and Treatment Office
Bureau of Psychosocial Health and Addiction
Deputy for Health

Background: Methanol Poisonings

- Methanol = toxic alcohols
- Toxicity is not immediate: Several hours' time lag between ingestion and first symptoms of poisoning
- Alcoholic beverages that contain methanol do not have distinct alarming characteristi
- Limited time for treatment: impaired vision and blindness in non-fatal cases, high mortality rate
- Poisoning cases:
  - Mostly sporadic events
  - But: When people drink methanol-contained alcohol socially (e.g. weddings, holidays): Ability to cause a local outbreak



Importance for Nephrologists

Methanol poisoning cases and fatalities in Iran (23.02.-02.05.2020)

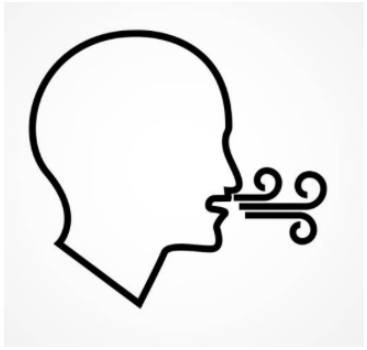
Province	Poisoning cases: hospital admissions (source: MOH)	Methanol deaths*	
		In hospital (source: MOH)	Total registered (source: LMO)
Tehran	173	87	205
Khuzestan	979	93	88
Fars	812	99	139
Razavi Khorasan	581	67	78
East Azerbaijan	483	50	75
Alborz	248	43	52
Ardebil	223	22	31
Isfahan	207	6	19
Kerman	139	0	2
Kermanshah	132	2	2
Mazandaran	100	10	28
Yazd	96	12	10
Markazi	87	4	4
Kurdestan	79	0	9
The other provinces	433	39	58
Total	**5876**	**534**	**800**

9.1% fatality rate

*Brain-dead cases considered dead
MOH Ministry of Health, *LMO* Legal Medicine Organization (data is available through https://bit.ly/2WUBfZo)

Mechanisms and patophysiology

Methanol and metabolism



Elimination through breath! (slow)

Ethanol

Methanol

Ethylene glycol

Fomepizole/ ethanol

Alcohol dehydrogenase

naldehyde

Aldehyd dehydrogenase

Formate

Folinic acid

10-formyl tetrahydrofolate synthetase

$CO_2 + H_2O$

Acetaldehyde

Glycol aldehyde

Aldehyde dehydrogenase

Glycolic acid

Glycolate oxidase

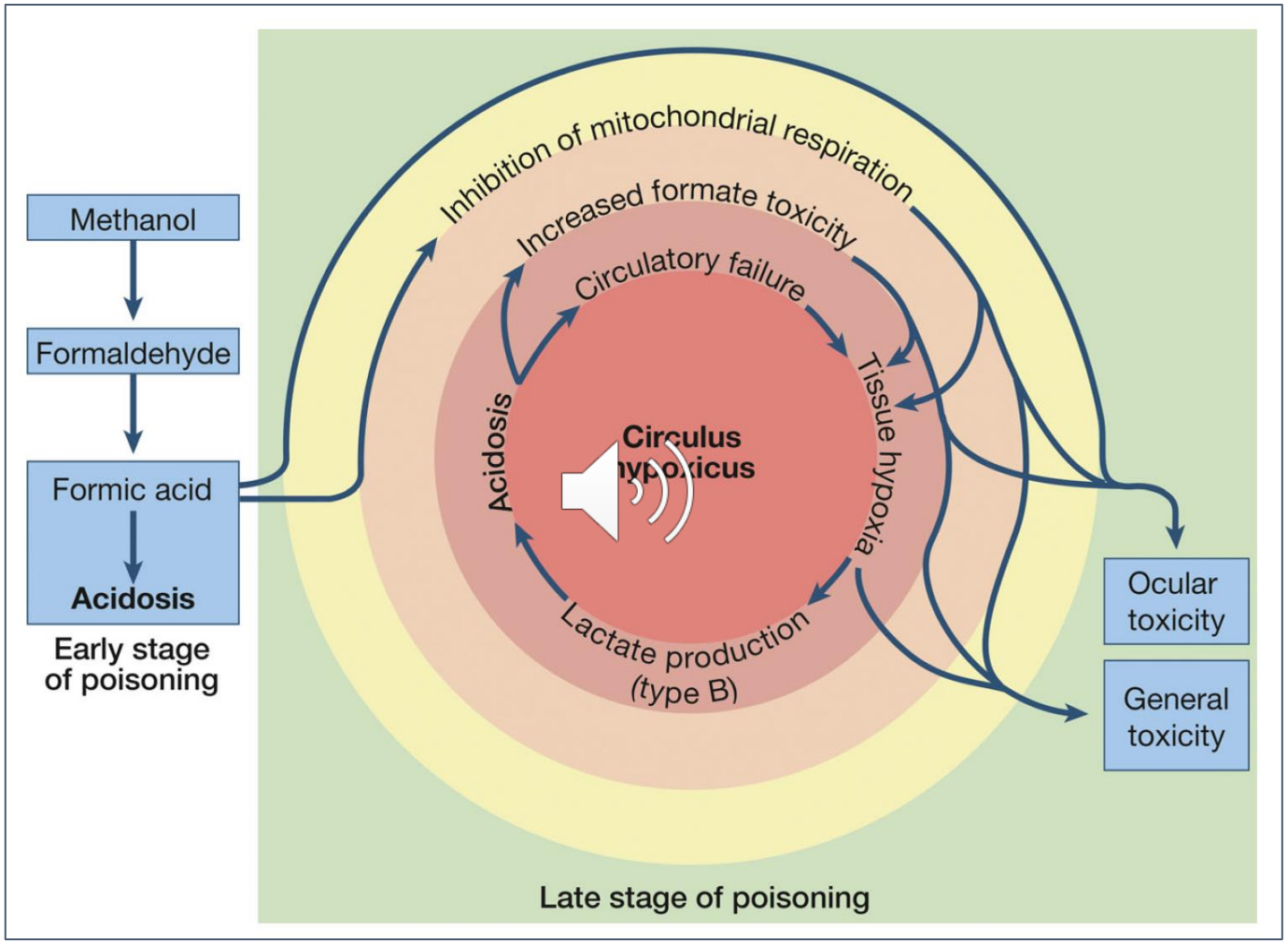
Oxalic acid

Calcium oxalate

Toxic!

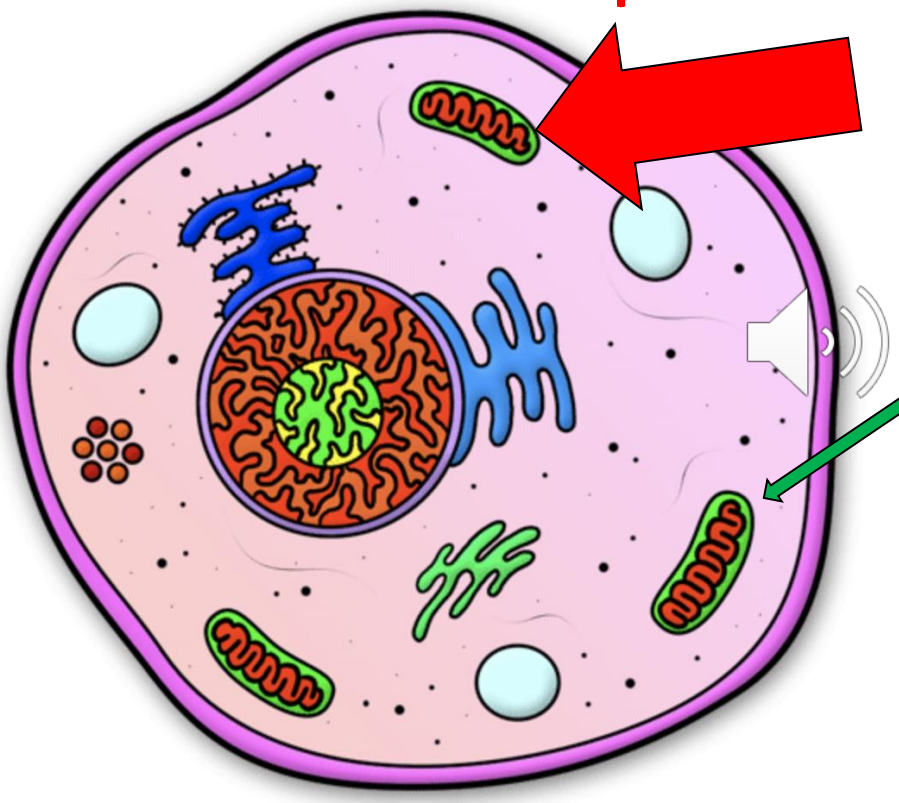
Hovda KE 2005 - Adjusted from PhD thesis

Circulus hypoxicus: The toxic mechanism of formic acid



Drangsholt E, Vangstad M, Zakharov S, Hovda KE, Jacobsen D. ***Basic Clin Pharmacol Toxicol*** 2018.

Toxicity of formic acid is much higher than formate



↑ inhibition

HCOOH ↔ H^+ + $HCOO^-$

Formic acid *formate*

↓ inhibition

pKa = 3.8 meaning ↓

pH 0.3 -> doubling of the undissociated acid!

Herken W & Rietbrock. *Naunyn Schmiedebergs Arch Exp Pathol Pharmakol 1968*

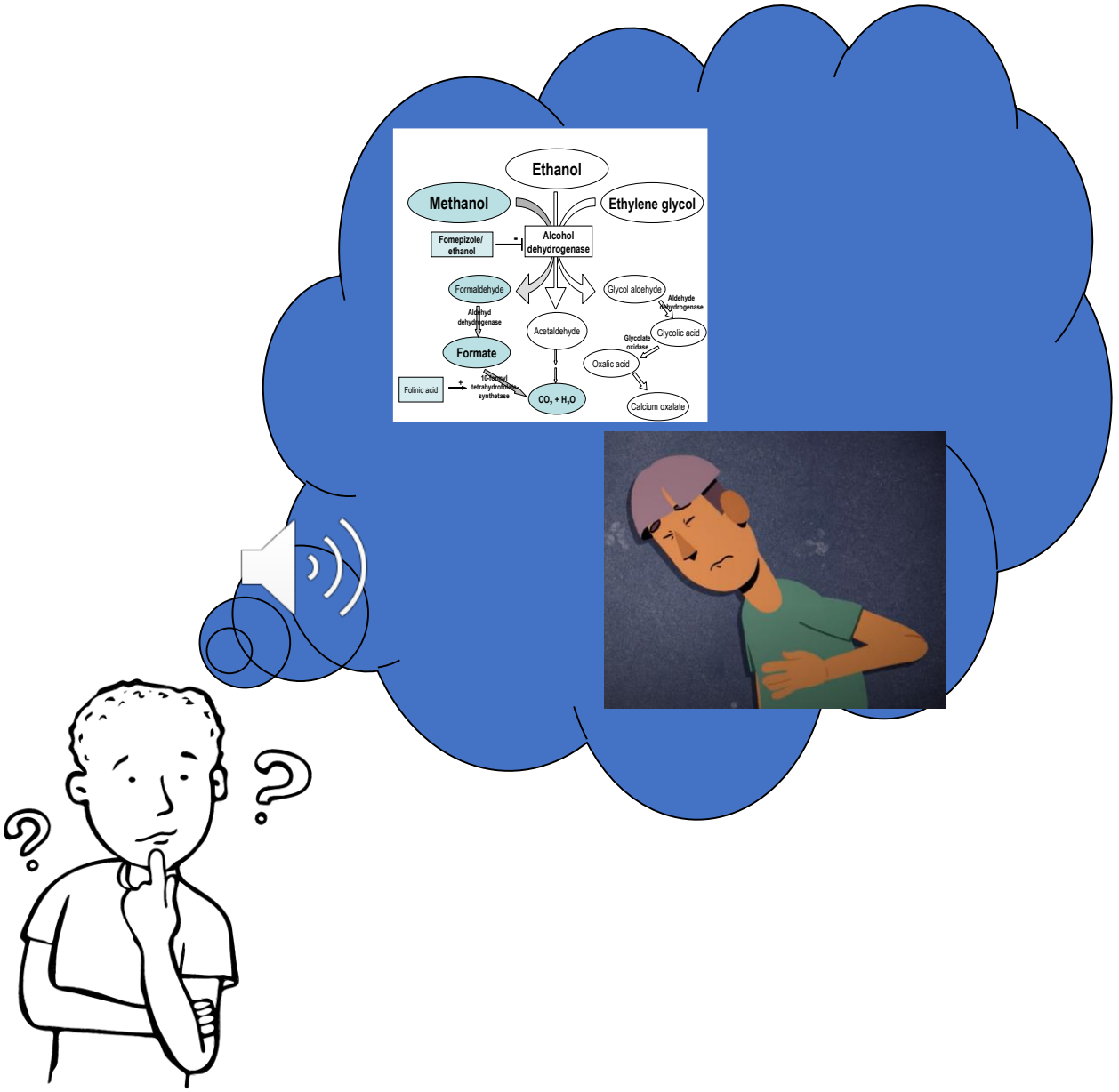
! More acidotic → more formic acid → more inhibition of the mitochondrium !

Diagnosis

Diagnosis

1. Patient history
2. Clinical features
3. Analytical options

➢**! Re-evaluate !**



Diagnostics – the basics

1. ***Patient history – be as precise as possible!***
   - Intake of illegal/bootleg/spurious alcohol?
   - Methanol itself does not make you drunk, but it is often (usually) mixed with ethanol (can make you drunk): but *symptoms only starts after ethanol is gone*
   - Others in surroundings with confirmed or suspect methanol poisoning (seriously ill, fatalities, blindness etc.)?
   - **Time from intake to symptoms >12 hours (often >24 hours)!**

Diagnostics – the basics

2. Clinical features

- **Hyperventilation (respiration >20-25/min)** / dyspnea
- Visual disturbances (ranging from blurred vision to blindness)
- Gastrointestinal symptoms (vomiting, abdominal pain etc.)
- Chest pain
- Severe/unusual “hang-over”: Feeling very sick the following day
- “Pseudopapillitis” -> blurred & hyperemic nervus opticus…

Diagnostics

- Patient history
- Clinical features
- Analytical options;
  - **Arterial blood gas** – *says nothing about the origin of the metabolic acidosis*
  - **The Osmolal* - (OG) & Anion gap (AG)** – *unspecific, often not available*
  - **Methanol analyses** – *(very) seldom available*
  - **Formate analysis**[1,2] – *until now less available*

**Never use osmolality from the ABG machine!!!*

[1]Hovda KE et al. J Anal Toxicol 2005; 29(6): 586-588
[2]Hovda KE et al.. Scand J Clin Lab Invest 2015; 75(7): 610-4

The formate analysis – a simple lab tes

Journal of Analytical Toxicology, Vol. 29, September 2005

Case Report

Increased Serum Formate in the Diagnosis of Methanol Poisoning

Knut Erik Hovda[1,*], **Petter Urdal**[2], and **Dag Jacobsen**[1]

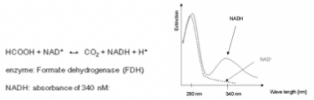
[1]*Department of Acute Medicine and* [2]*Department of Clinical Chemistry, Ullevaal University Hospital, NO-0407 Oslo, Norway*

- *Very sensitive (1.3 mmol/L or 4 mg/dL) and specific***
- *Upper reference range 0.4 mmol/L or 2 mg/dL***

Hovda KE et al. J Anal Toxicol 2005; 29(6): 586-588.

**Urdal P. Clin Chem 1984; 30: 911-913

Measurement of serum formate



Principles of the test
In the presence of the enzyme formate dehydrogenase (FDH) and nicotinamide-adenine dinucleotide (NAD) formate is converted to carbon dioxide giving rise to equivalent amounts of NADH. The enzyme FDH is specific for formate.

Equipment required
The absorbance is measured by a photometer or an automated biochemistry analyser at 340 nm and at +20°C or +37°C. It is preferable to use the automated biochemistry analyser that analyses at +37°C.

Reagents
Formate dehydrogenase (FDH) 250 U
NAD (lithium salt, coded "p" [pure] or higher category, sodium salt or free acid. Please notice that pH may be affected if free acid is used.)
Phosphate buffer (0.1M, pH 7.5)

Preparation of working reagents
Reagent 1 (R1, NAD 9.4 mmol/L): Dissolve 350 mg NAD in 56 mL buffer. Stable 5 hours at +20°C and 7 days at +2-8°C.
Reagent 2 (R2, FDH 16 U/mL): Dissolve the contents of 1 bottle of formate dehydrogenase (FDH, 250 U) in 16 mL buffer. Stable 5 hours at +20°C and 7 days at +2-8°C.

Analysis
Mix 1 part sample and 10 parts R1. Incubate 1-2 minutes (+20°C or +37°C). Read A1 absorbance at 340 nm. Add 5 parts of R2. Incubate at +37°C. (8-10 minutes) or at +20°C (25-30 minutes). Read A2 absorbance at 340 nm.
In each analysis include a reagent blank (water as sample).

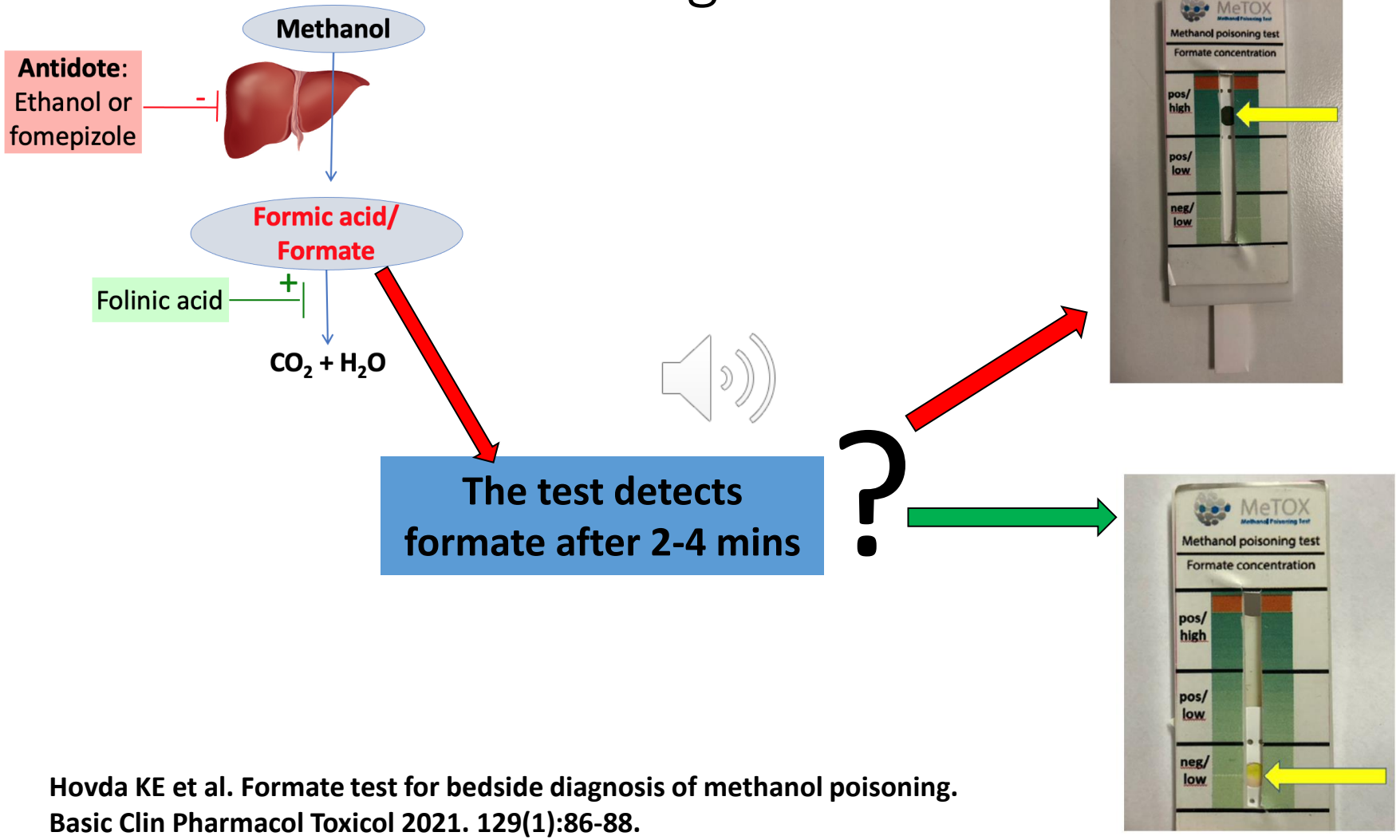
Comments:
1. The final concentrations, in the cuvette when reagents and sample are all added, are FDH 5 U/mL and NAD 6 mmol/L. The serum fraction is approx 0.06 (serum constitutes 6% of the total volume).
2. It is acceptable to prepare R1 and R2 with other concentrations of FDH and NAD at the condition that the final ratio of the reagent mixture and the serum is proportional.
3. It is preferable to have a 2-reagent method, firstly incubating a mixture of the reagent containing R1 and serum before R2 is added. Incubation of the sample and the reagent 1 allows NADH-producing side reactions to run to end before FDH is added and the specific reaction started.
4. If the automated biochemistry analyzer dilutes the reagents and sample with water by less than 20%, this will not affect the measurement result. Otherwise, one should increase the initial concentration of reagents, maintaining a necessary ratio.
5. In some centers suspicion of methanol poisoning occurs only sporadically, and most of the expensive reagents may go to waste because of limited stability. Freezing at -80 degrees Celsius in small portions has given acceptable stability. The reagents should always be tested with a standard after thawing. If -80 degrees is not available, enzymes should be stored and made from dry reagents every time. There are insufficient data to support storage of solution in -20 degrees (-18 degrees), and this should thus be avoided until better data exists.

Calculation of result
Calculate the difference between the two optical density readings (ΔA=A2 - A1).

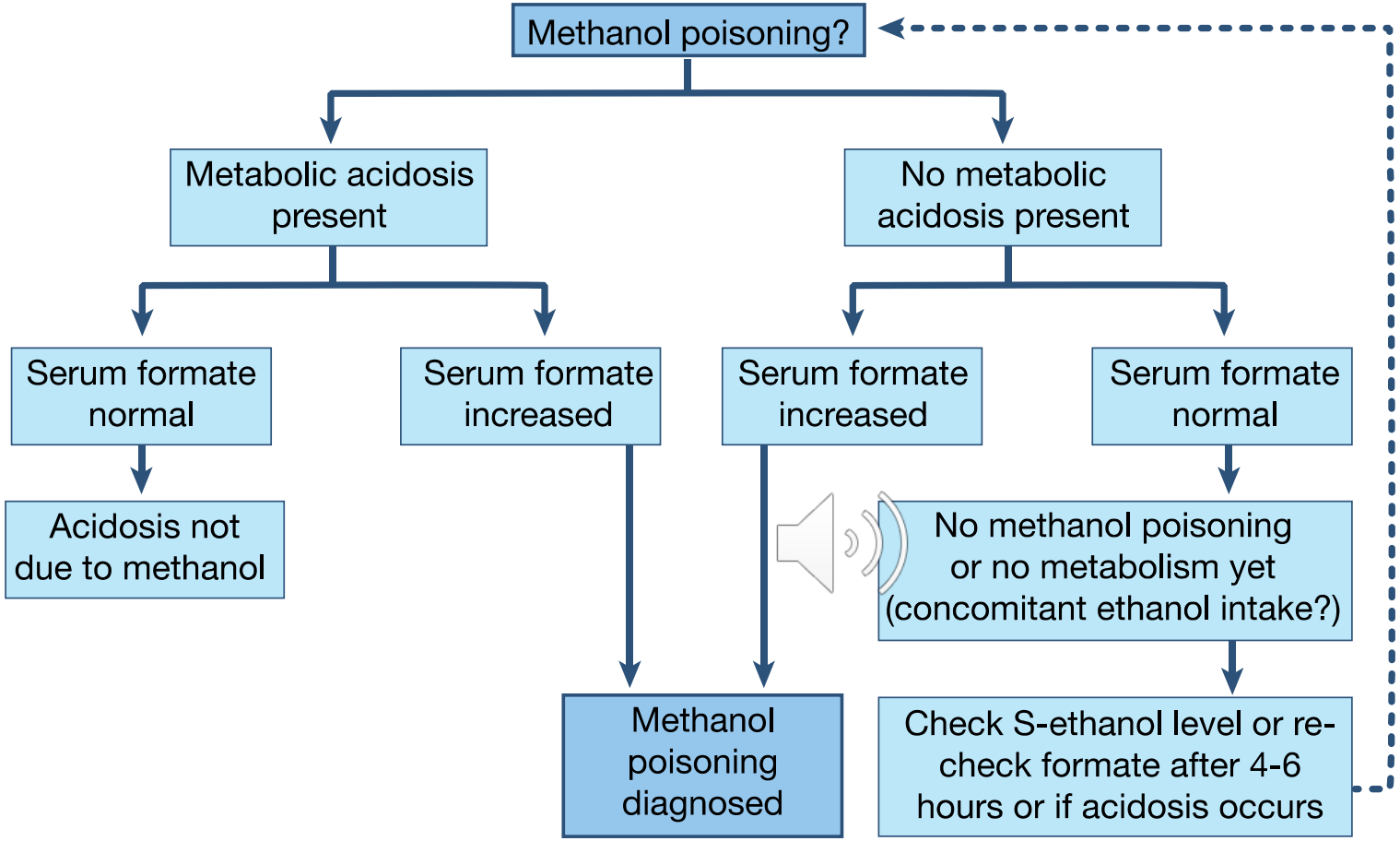
To calculate concentration: Formate = (ΔA_{sample} / $\Delta A_{calibr.}$) x ($Conc._{calibr.}$)

If the results are above 6 mmol/L, dilute the sample 1+9 with saline (0.9% NaCl) or 0.1 M phosphate buffer (pH=7.0 – 7.5) and reanalyse. Multiply the obtained reading by 10 (dilution factor).

New point-of-care diagnostic



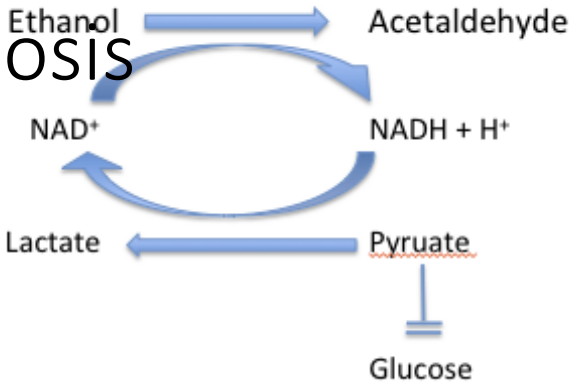
Hovda KE et al. Formate test for bedside diagnosis of methanol poisoning. Basic Clin Pharmacol Toxicol 2021. 129(1):86-88.



Hovda KE, McMartin KE, Jacobsen D. **Methanol and formaldehyde poisoning**.
In: Brent J et al (eds). Critical Care Toxicology, 2nd Edition. Springer Publishing. 2017.

Differential diagnosis

Most important - Alcoholic ketoacidosis



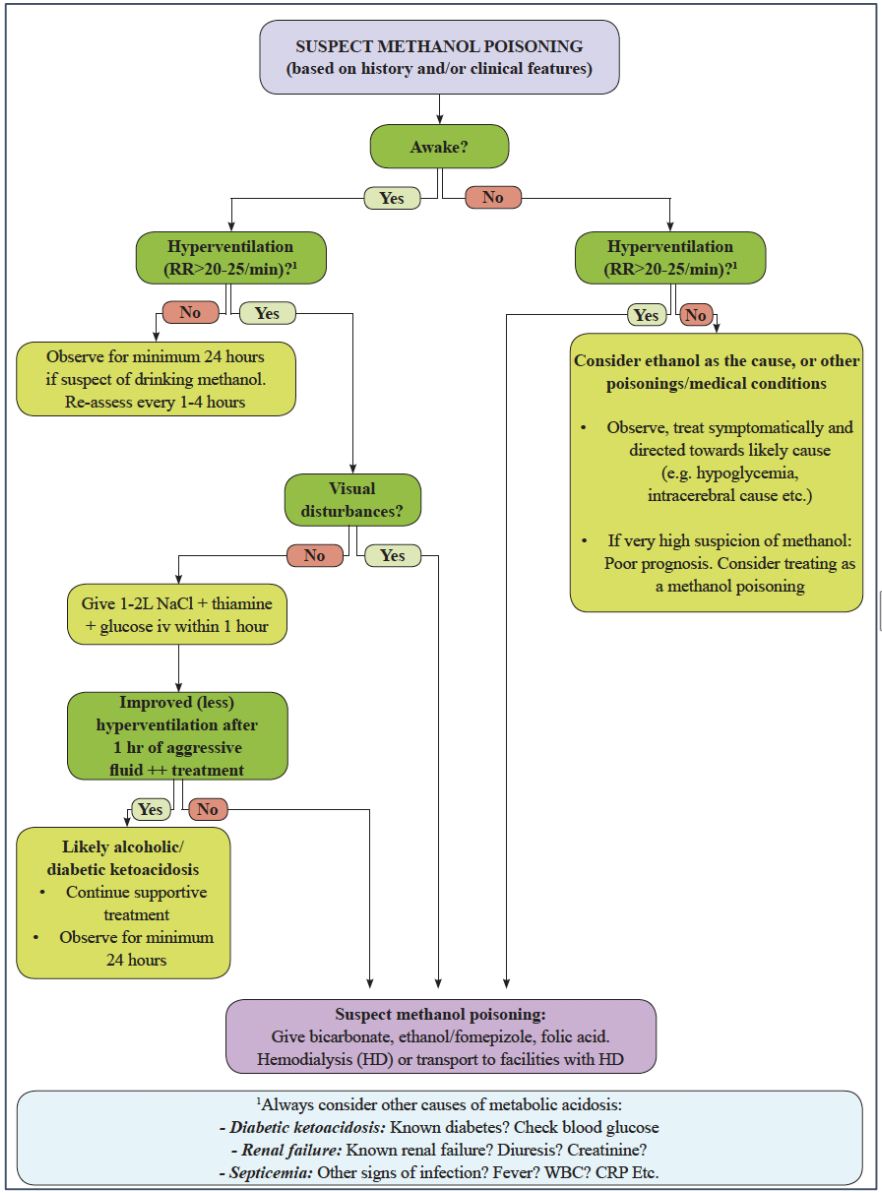
- Alcohol can among other things give:
  - ***Low extracellular volume (ECV):***
    Low ECV → α-adrenergic stim → ↓ insulin release
    => the liver oxidizes alcohol → ***ketones***

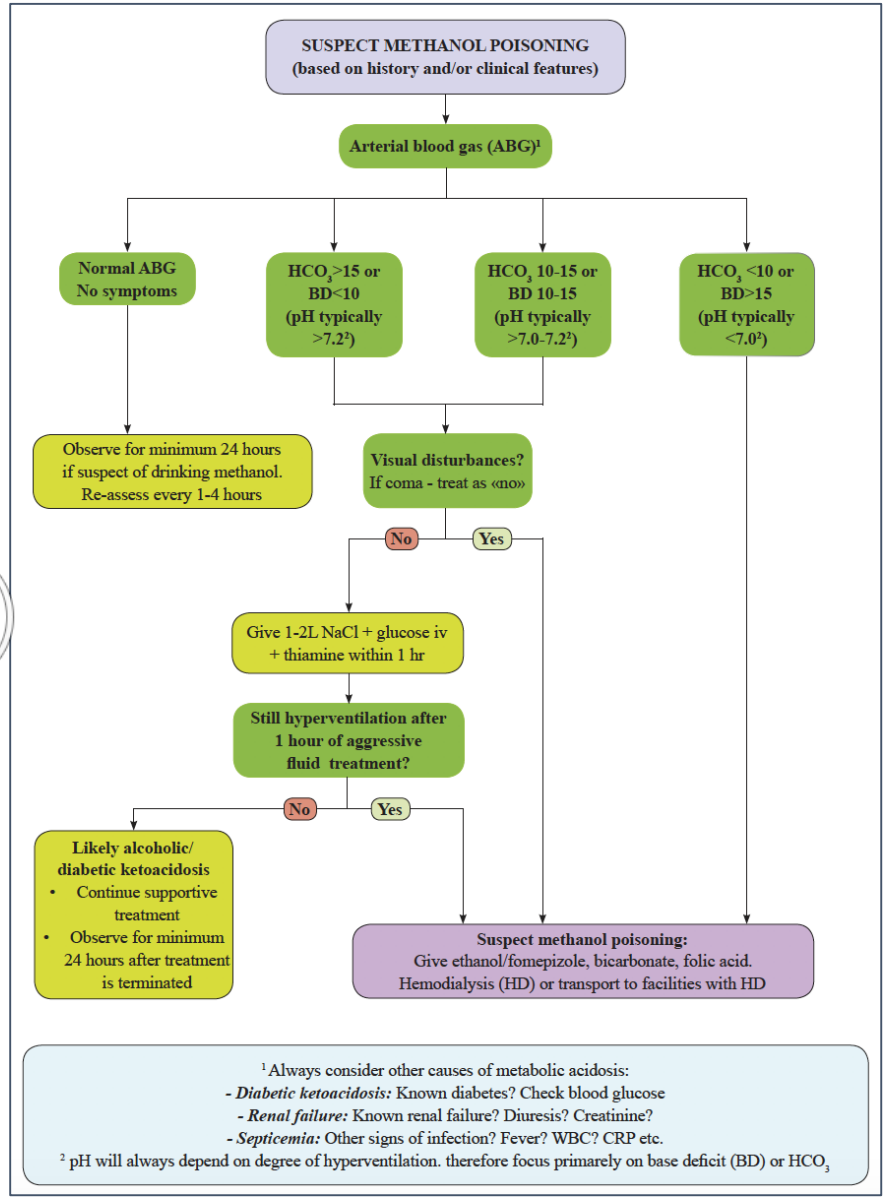
  - ***↑ L-lactate*** (**because**: ↑ NADH/NAD+, [illegible] failure, convulsions, lack of thiamine)
    Thiamine = cofactor when pyruate enters Krebs´ cyclus (inhibits formation of lactate)

  - **Low intake of calories** → the liver uses alcohol as energy → ***ketones***

- ***Treatment: IV fluids, thiamine and glucose (+insulin?)***

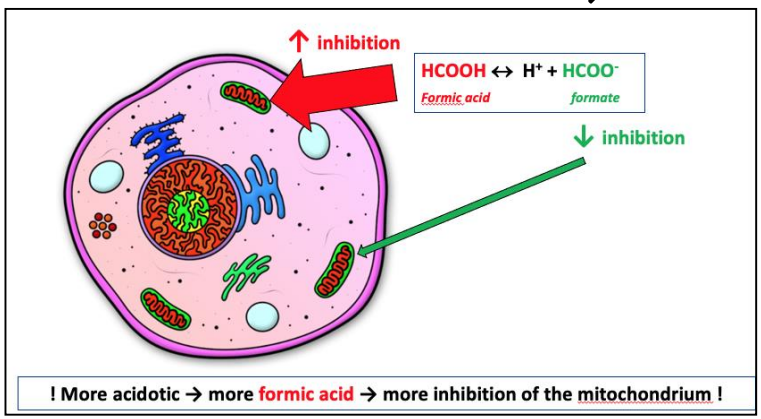
New flow charts – for use without or with blood gas machine

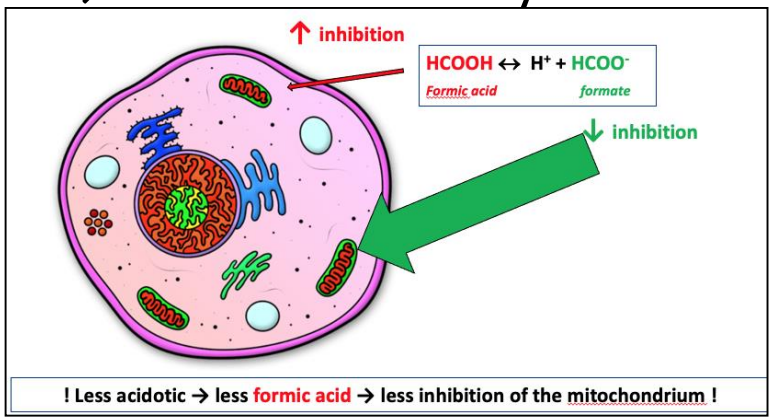




Treatment

Bicarbonate → less acidosis → less toxicity





Bicarbonate

HCOOH ↔ H⁺ + HCOO⁻

formic acid ***formate***

pKa = 3.8 meaning ↑

pH 0.3 -> *reduces formic acid amount to the half!*

HCOOH ➡ H⁺ + HCOO⁻

formic acid ***formate***

pH dependent reabsorption in th

Clinical Toxicology, 43:221–227, 2005
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ISSN: 0731-3810 print / 1097-9875 online
DOI: 10.1081/CLT-200058936

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ARTICLE

Methanol and Formate Kinetics During Treatment with Fomepizole

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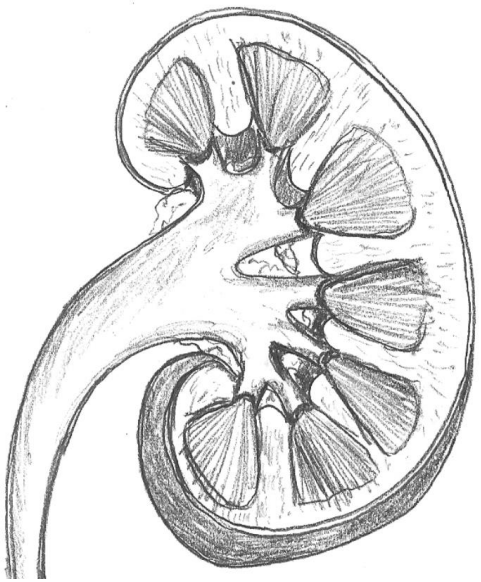


↓ excretion

↑ excretion

$HCOOH \leftrightarrow H^+ + HCOO^-$

Formic acid ***formate***

- **<u>Simplified</u>**:
  - Formate ($HCOO^-$) is excreted through the kidneys
  - Formic acid (HCOOH) is reabsorbed in the kidneys

➢ **<u>More acidotic, more reabsorption</u>**
<u>=> more bicarbonate, more excretion</u>!

Dosing of bicarbonate

1. **“Full correction”**

   Base deficit x patient weight x 0.3 **(= mmol bicarb)**

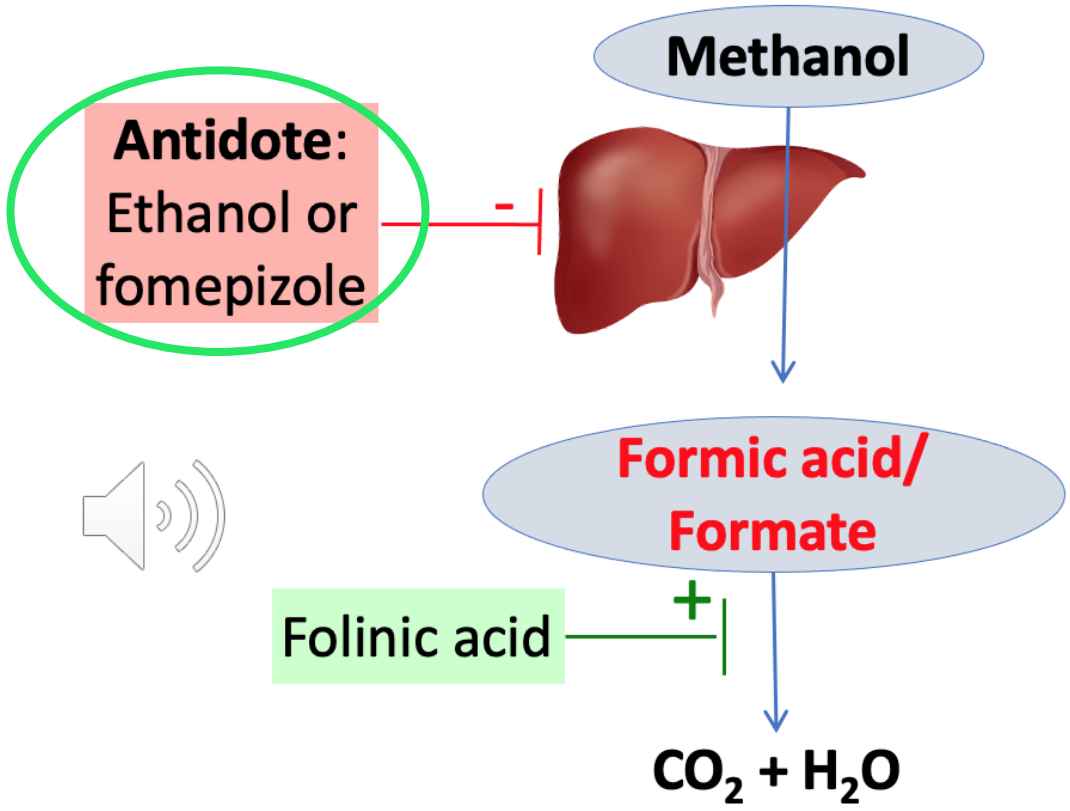
2. **If no ABG available:**

   Give bicarbonate until no more hyperventilation is apparent

Antidote

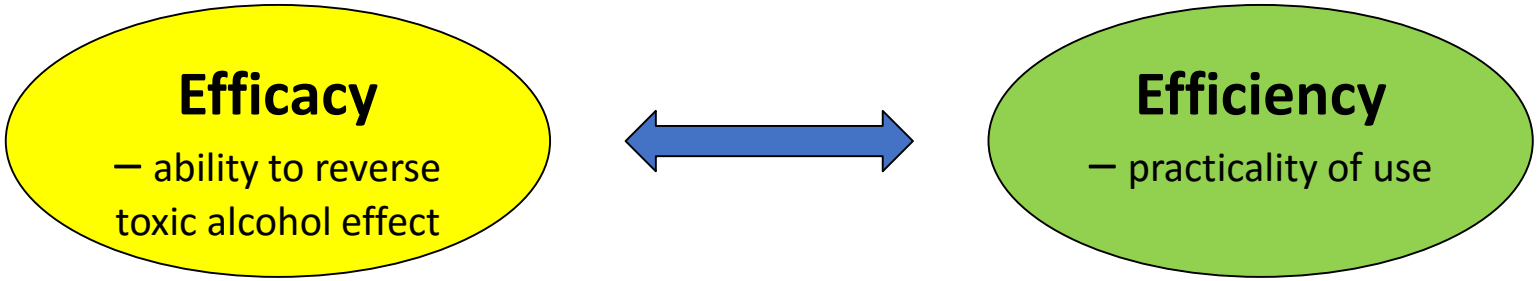
Rationale:

Stop the metabolism of the parent alcohol to the toxic metabolite (***the alcohols are not toxic themselves***)



Two different antidotes.
What are the criteria for choosing one or the other?

- Availability
- Outcome
- Safety of use
- Cost (antidote + overall cost)
- Simplicity of use
- Availability of supplementary treatment (e.g. ICU, dialysis)
- Other (religious issues etc.)



Dosing of ethanol - a suggestion

Suggested dosing regimen for ethanol
(***be aware of individual differences*** and frequent under-dosing):

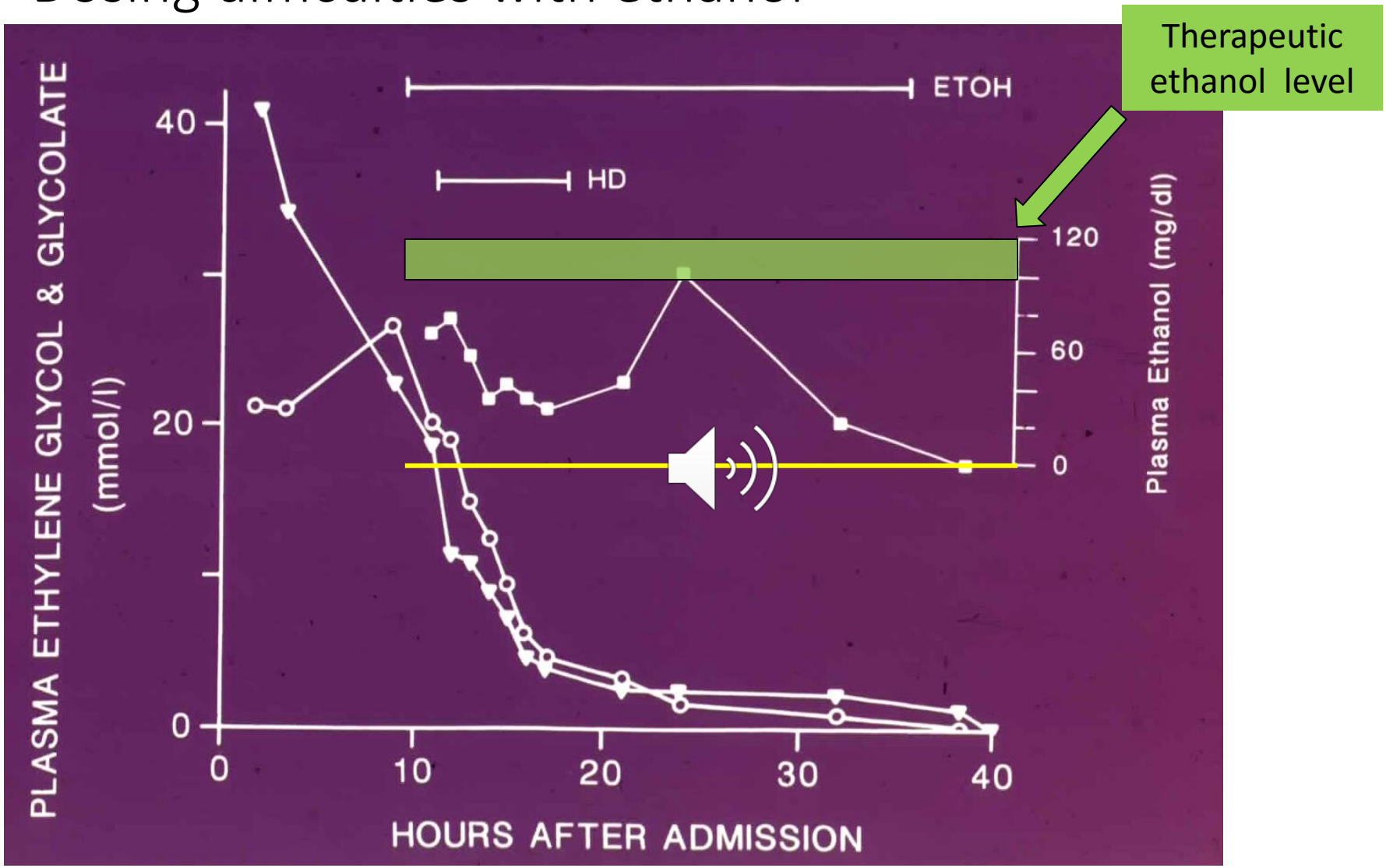
	5% ethanol	10% ethanol	20% ethanol	40% ethanol
Loading dose	15mL/kg	7.5mL/kg	4mL/kg	2mL/kg
Infusion rate (not regular drinker)	2mL/kg/hr	1mL/kg/hr	0.5mL/kg/hr	0.25mL/kg/hr
Infusion rate (regular drinker)	4mL/kg/hr	2mL/kg/hr	1mL/kg/hr	0.5mL/kg/hr
Infusion rate during HD (not regular drinker)	4mL/kg/hr	2mL/kg/hr	1mL/kg/hr	0.5mL/kg/hr
Infusion rate during HD (regular drinker)	6mL/kg/hr	3mL/kg/hr	1.5mL/kg/hr	0.8mL/kg/hr

Rule of thumb: Beer contains 5%, wine 12-14% and spirits 40-45% ethanol.

https://msf.no/mpi

Hovda KE et al. Methanol and formaldehyde poisoning. Critical Care Toxicology, 2nd Edition 2017. 1769-86.

Dosing difficulties with ethanol



Jacobsen D et al. Am J Med 1988;84:145-52

Annals of Emergency Medicine

TOXICOLOGY/ORIGINAL RESEARCH

Use of Out-of-Hospital Ethanol Administration to Improve Outcome in Mass Methanol Outbreaks

Sergey Zakharov, MD, PhD*; Daniela Pelclova, PhD; Pavel Urban, PhD; Tomas Navratil, PhD; Olga Nurieva, MD; Katerina Kotikova, MD; Pavel Diblik, MD; Ivana Kurcova, MD; Jaromir Belacek, RNDr, PhD; Martin Komarc, MA; Michael Eddleston, MD, PhD; Knut Erik Hovda, MD, PhD

*Corresponding Author. E-mail: sergey.zakharov@vfn.cz.

Editor's Capsule Summary

What is already known on this topic

Delayed treatment with an antidote is known to worsen the outcome of methanol poisoning.

What question this study addressed

Does the out-of-hospital administration of ethanol decrease mortality and morbidity of methanol poisoning?

What this study adds to our knowledge

In this case series of 100 methanol overdoses, the 30 patients who received out-of-hospital ethanol had improved survival and fewer visual and central nervous system deficits than those who did not.

How this is relevant to clinical practice

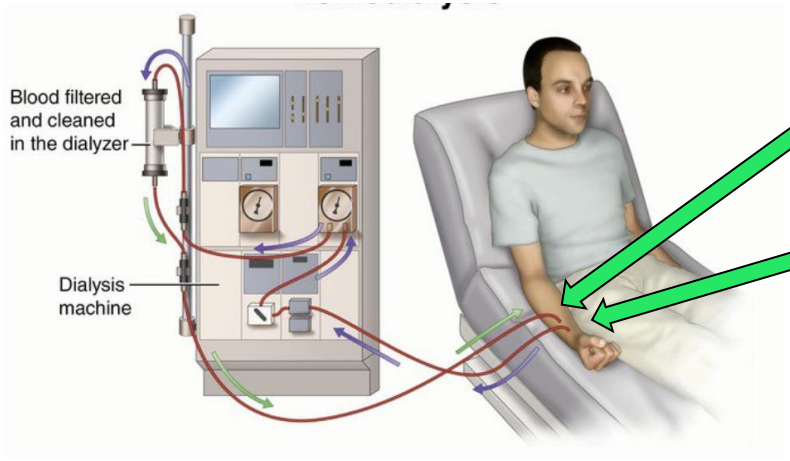
Although this study was uncontrolled, it provides support for the out-of-hospital administration of ethanol in mass-casualty methanol overdose events.

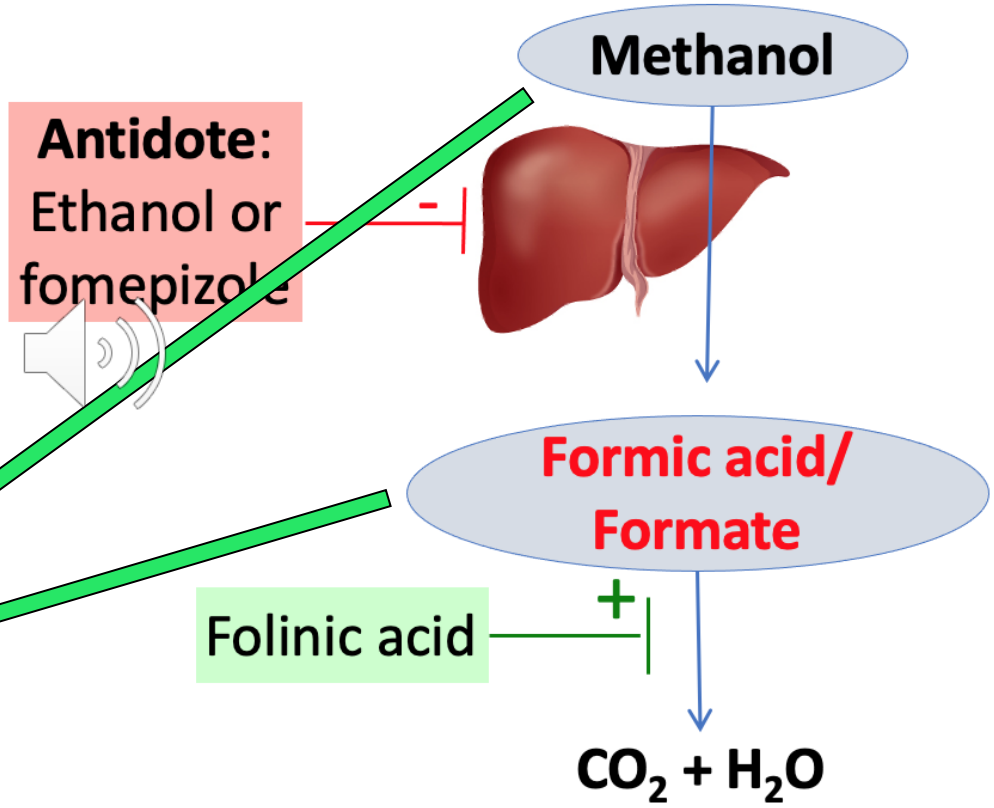
-> i.e.: Start the antidotal treatment as early as possible – prehospitally if strong suspicion!!!

Dialysis

Rationale:

Eliminate the toxic alcohol (methanol) + the toxic metabolite (formate) + correct the acidosis





IHD vs. CRRT – elimination of toxic

http://www.kidney-international.org clinical investigation

OPEN

Intermittent hemodialysis is superior to continuous veno-venous hemodialysis/hemodiafiltration to eliminate methanol and formate during treatment for methanol poisoning

Sergey Zakharov[1], Daniela Pelclova[1], Tomas Navratil[2], Jaromir Belacek[3], Ivana Kurcova[4], Ondrej Komzak[4], Tomas Salek[5], Jiri Latta[6], Radovan Turek[7], Robert Bocek[8], Cyril Kucera[9], Jaroslav A. Hubacek[10], Zdenka Fenclova[1], Vit Petrik[1], Martin Cermak[11] and Knut Erik Hovda[12]

ISN kidney INTERNATIONAL
OFFICIAL JOURNAL OF THE INTERNATIONAL SOCIETY OF NEPHROLOGY

6.9 vs. 7.1 IHD). The mean elimination half-life of methanol was 3.7 and formate 1.6 h with IHD, versus 8.1 and 3.6 h, respectively, with CVVHD/HDF (both significant). The 54% greater reduction in methanol and 56% reduction in formate elimination half-life during IHD resulted from the higher blood and dialysate flow rates. Increased blood and dialysate flow on the CVVHD/HDF also increased elimination significantly. Thus, IHD is superior to CVVHD/HDF for more rapid methanol and formate elimination, and if CVVHD/HDF is the only treatment available then elimination is greater with greater blood and dialysate flow rates.

IHD vs. CRRT – efficiency of acidemia correction

CLINICAL TOXICOLOGY, 2016
http://dx.doi.org/10.1080/15563650.2016.1250901

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CLINICAL RESEARCH

OPEN ACCESS

Efficiency of acidemia correction on intermittent versus continuous hemodialysis in acute methanol poisoning

Sergey Zakharov[a], Daniela Pelclova[a], Tomas Navratil[a,b,c], Jaromir Belacek[d], Jiri Latta[e], Michal Pisar[e], Jan Rulisek[f], Jiri Leps[g], Pavel Zidek[h], Cyril Kucera[i], Robert Bocek[j], Miroslav Mazur[j], Zdenek Belik[k], Josef Chalupa[l], Viktor Talafa[m], Kamil Kodras[n], Daniel Nalos[o], Ctirad Sedlak[p], Michal Senkyrik[q], Jan Smid[r], Tomas Salek[s], Darren M. Roberts[t] and Knut Erik Hovda[u]

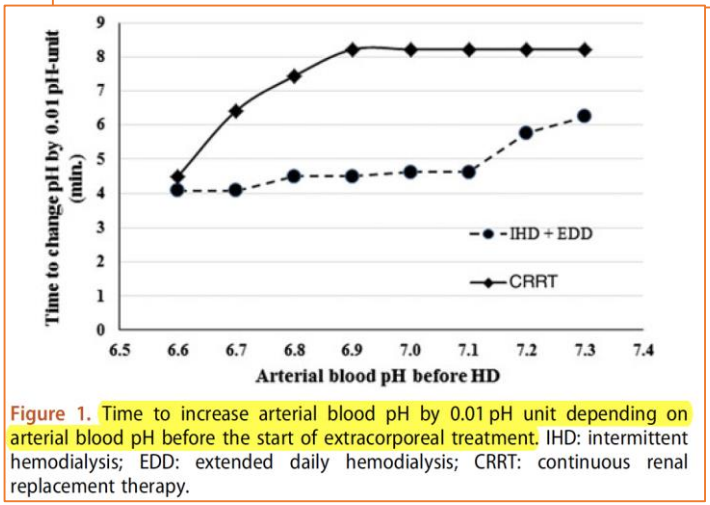


Figure 1. Time to increase arterial blood pH by 0.01 pH unit depending on arterial blood pH before the start of extracorporeal treatment. IHD: intermittent hemodialysis; EDD: extended daily hemodialysis; CRRT: continuous renal replacement therapy.

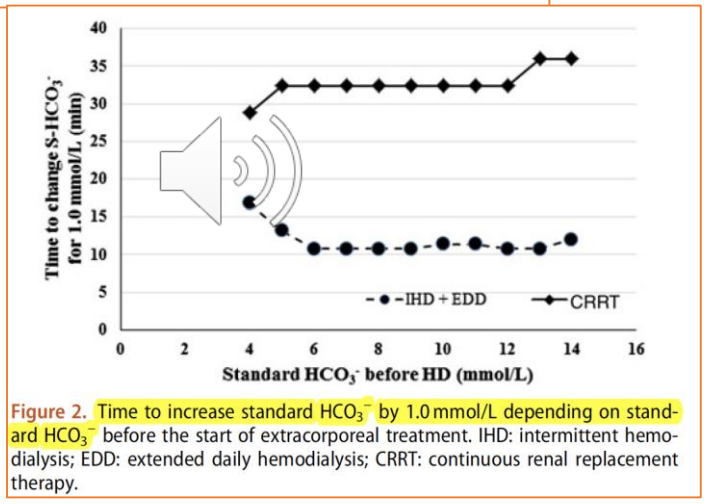
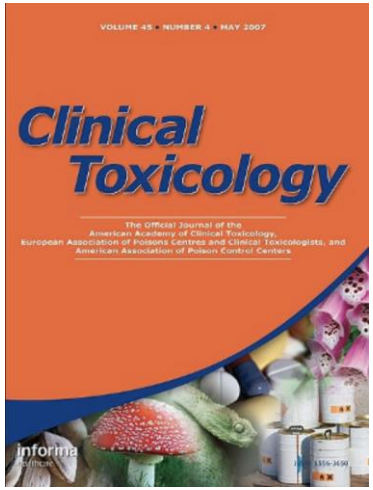


Figure 2. Time to increase standard HCO_3^- by 1.0 mmol/L depending on standard HCO_3^- before the start of extracorporeal treatment. IHD: intermittent hemodialysis; EDD: extended daily hemodialysis; CRRT: continuous renal replacement therapy.



($r = -0.758$, $p < 0.001$ and $r = -0.602$, $p < 0.001$, correspondingly).
The mean time for HCO_3^- to increase by 1 mmol/L was 12 ± 2 min for IHD versus 34 ± 8 min for CRRT ($p < 0.001$), and the mean time for arterial blood pH to increase 0.01 was 7 ± 1 mins for IHD versus 11 ± 4 min for CRRT ($p = 0.024$). The mean increase in HCO_3^- was 5.67 ± 0.90 mmol/L/h for IHD versus 2.17 ± 0.74 mmol/L/h for CRRT ($p < 0.001$).
Conclusions: Our study supports the superiority of IHD over CRRT in terms of the rate of acidemia correction.

IHD vs. CRRT - outcome

Zakharov *et al. Ann. Intensive Care (2017) 7:77*
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Annals of Intensive Care

RESEARCH Open Access

Intermittent versus continuous renal replacement therapy in acute methanol poisoning: comparison of clinical effectiveness in mass poisoning outbreaks

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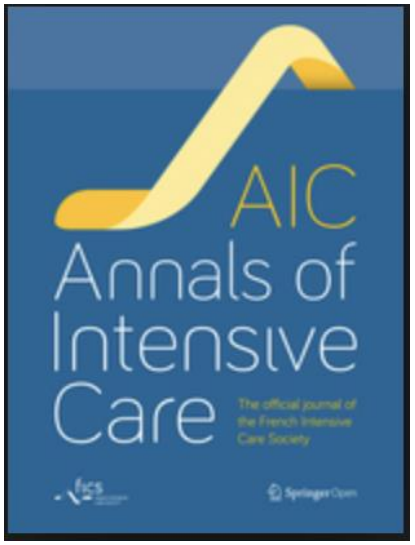


ciation of ECTR modality with both mortality and the number of survivors with visual and CNS sequelae of poisoning, but this association was not present after adjustment for arterial blood pH and GCS on admission (all $p > 0.05$).

Conclusions: In spite of the faster correction of the acidosis and the quicker removal of the toxic metabolite in intermittent dialysis, we did not find significant differences in the treatment outcomes between the two groups after adjusting for the degree of acidemia and the severity of poisoning on admission. These findings support the strategy of "use what you have" in situations with large outbreaks and limited dialysis capacity.

Keywords: Methanol poisoning, Mass poisoning outbreak, Continuous renal replacement therapy, Intermittent

Peritoneal dialysis

- Significantly slower elimination (not well documented) on elimination of methanol as well as correction of acidosis

- Probably only indicated if nothing else available, transport to dialysis facilities is not feasible AND the procedure is well known to the treating doctors

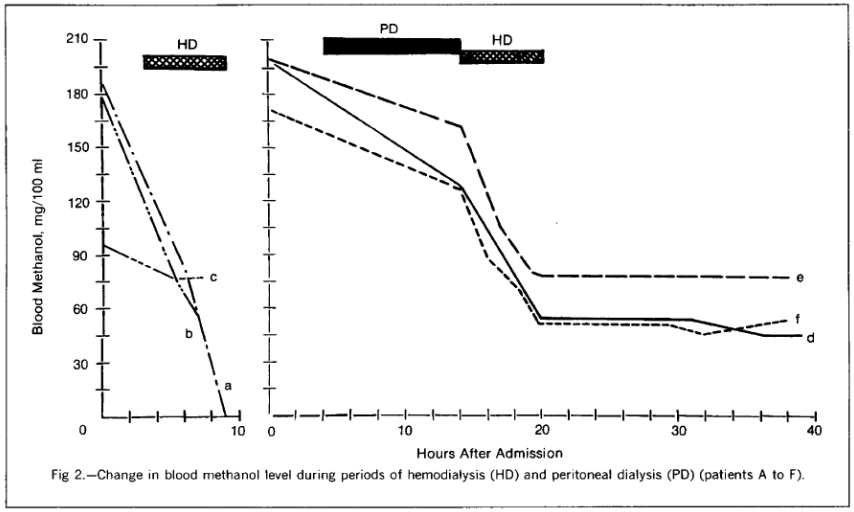


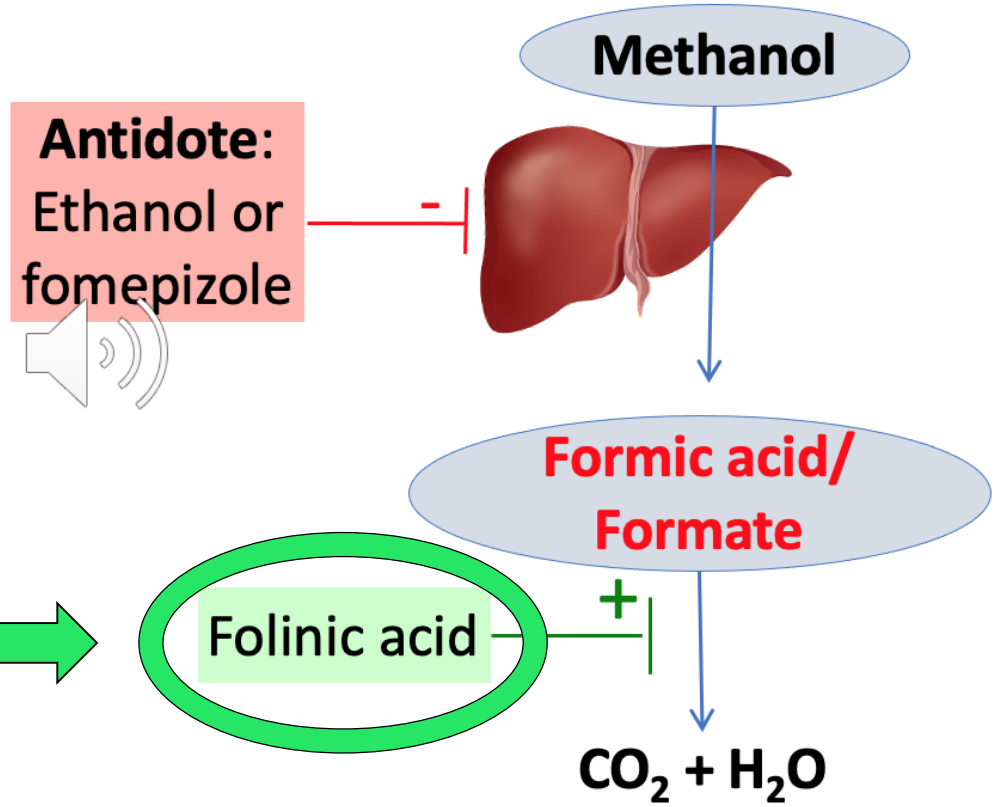
Fig 2.—Change in blood methanol level during periods of hemodialysis (HD) and peritoneal dialysis (PD) (patients A to F).

Keyvan-Larijarni H et al. *Arch Intern Med.* 1974;134(2):293-296.

Folic/folinic acid

Rationale:

Increase the endogenous (the body´s own) metabolism of formic acid/formate



Simple. No/little side effects (vitamin B). Cheap. Limited documentation on outcome.

Prognosis

Prognosis - methanol poisoning

- Coma on admission[1,2,3,4,5,6,7]
- Degree of metabolic acidosis (pH<6.9-7.0, BE -24-28) [1,2,3,4,5,6,7]
- Ability to hyperventilate (pH vs. pCO_2) [1,2,6,7]
- S-methanol NOT prognostic!

[1]Paasma R et al. Clin Toxicol *2007*; 45:152-158
[2]Hovda KE et al. J Intern Med *2005*; 258(2): 181-90
[3]Hassanian-Moghaddam H et al. Hum Exp Toxicol 2007; 26(7): 583-6
[4]Liu JJ et al. J Toxicol Clin Toxicol 1998; 36(3): 175-81
[5]Kute VB et al. Saudi J Kidney Dis Transpl 2012;23(1):37-43
[6]Paasma R et al. Clin Tox 2012; 50: 823-31
[7]Zakharov et al. Clin Tox 2014; 52: 1013-1024

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REVIEW

Consensus statements on the approach to patients in a methanol poisoning outbreak

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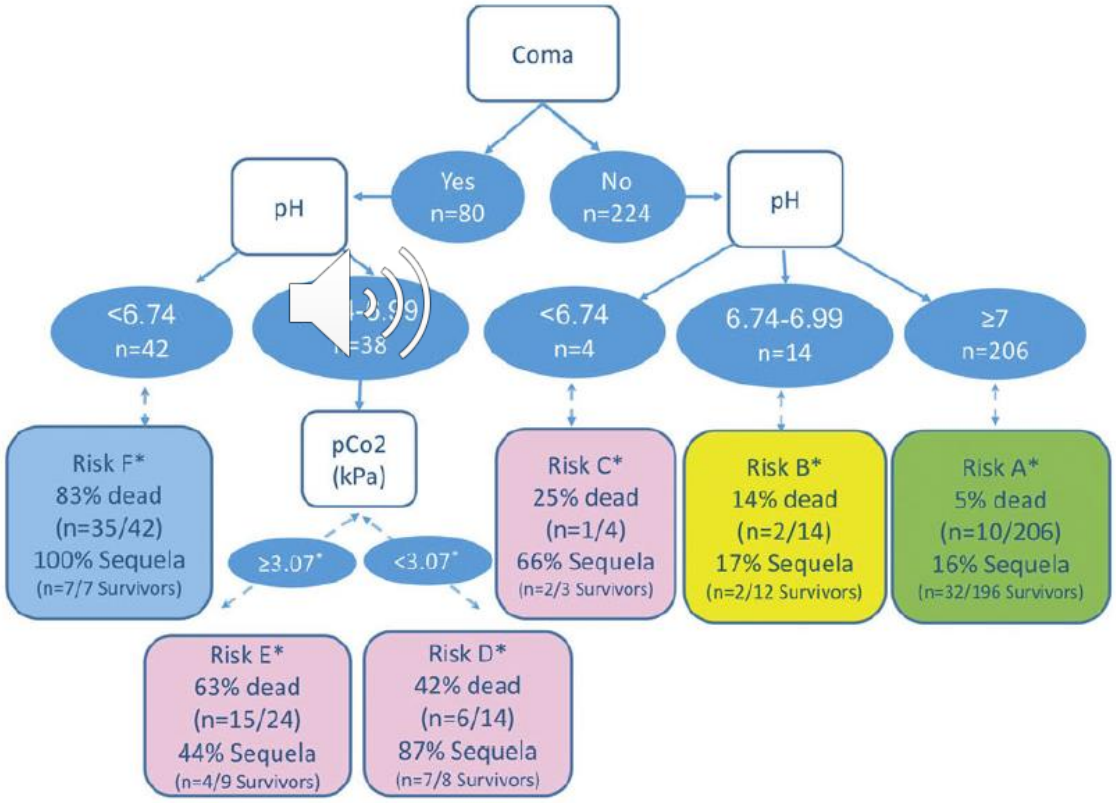


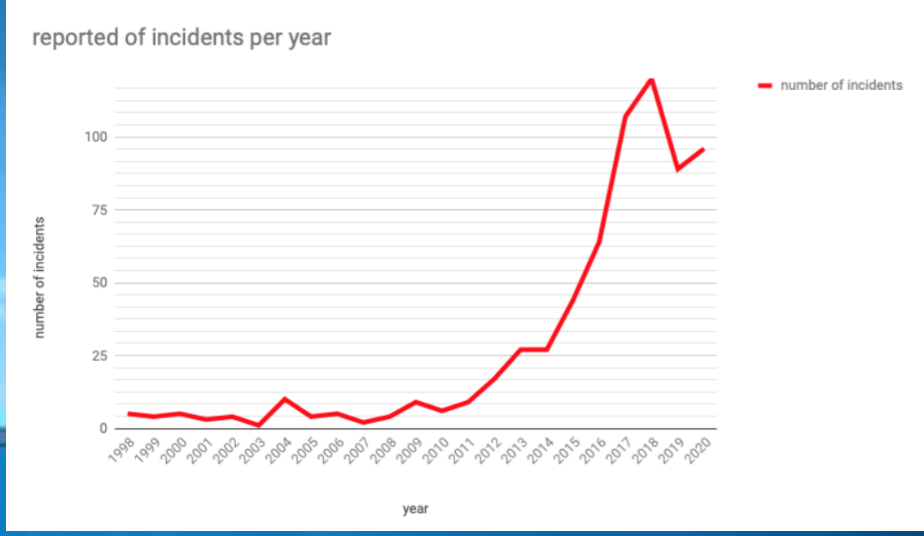
Figure 1. Overview of outcomes on the basis of admission conscious level, blood pH and pCO_2, based on aggregated data from Paasma et al. [16] and Zakharov et al. [2]*. Version 1 (color): * 3.07 kPa = 23 mmHg. Version 2: B&W: * 3.07 kPa = 23 mmHg.

What is the range of the problem?

The range of the problem…

- identified outbreaks of methanol poisoning

Reported methanol incidents – the tip of the iceberg



https://msf.no/mpi

Thousands are being poisoned, blinded or dying every year, but cases are rarely reported...

Summary

- Methanol causes thousands of fatalities, but is not toxic by itself
- Diagnosis is difficult, but can be approached from various sides
- Treatment is effective, but needs early initiation
- Overall handling requires emergency preparedness systems
- Handling can be done everywhere, but requires a basic set of knowledge.
- Adaptation to local circumstances is crucial to have success

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Methanol mass poisoning in Iran: role of case finding in outbreak management

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ABSTRACT

Background There are no guidelines addressing the public health aspects of methanol poisoning during larger outbreaks. The current study was done to discuss the role of active case finding and a national guideline that organizes all available resources according to a triage strategy in the successful management of a methanol mass poisoning in Rafsanjan, Iran, in May 2013.

Methods A retrospective cross-sectional study was performed reviewing the outbreak Emergency Operation Center files. The objectives were to describe the characteristics, management and outcome of a methanol outbreak using Active Case Finding to trace the victims.

Results A total of 694 patients presented to emergency departments in Rafsanjan after public announcement of the outbreak between 29th May and 3rd June 2013. The announcement was mainly performed via short message service (SMS) and local radio broadcasting. A total of 361 cases were observed and managed in Rafsanjan and 333 were transferred to other cities. Seventy-five and 100 patients underwent hemodialysis (HD), retrospectively. The main indication for HD was refractory metabolic acidosis. Eight patients expired due to the intoxication. Except for the deceased cases, no serum methanol level was available.

Conclusion In developing countries, where diagnostic resources are limited, use of active case finding and developing national guidelines can help in the management of large outbreaks of methanol poisonings.

Keywords methanol, poisoning, outbreak, epidemics, mortality, intoxication