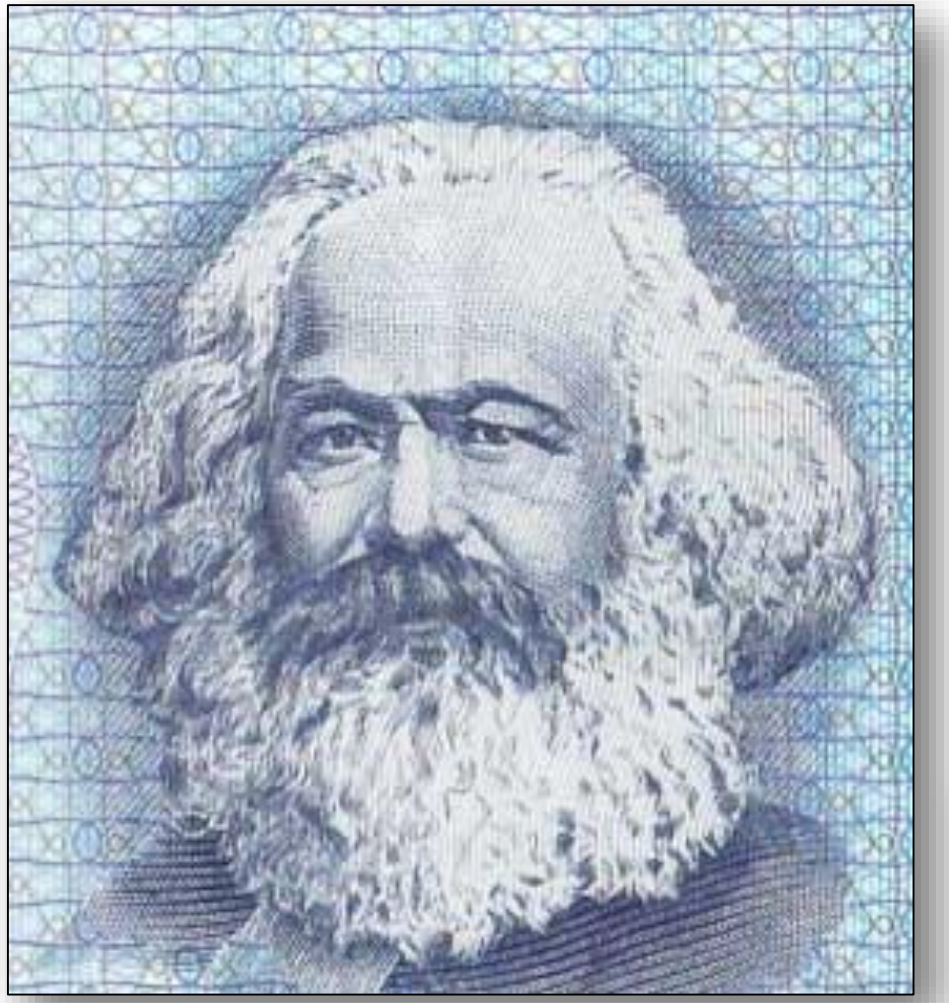
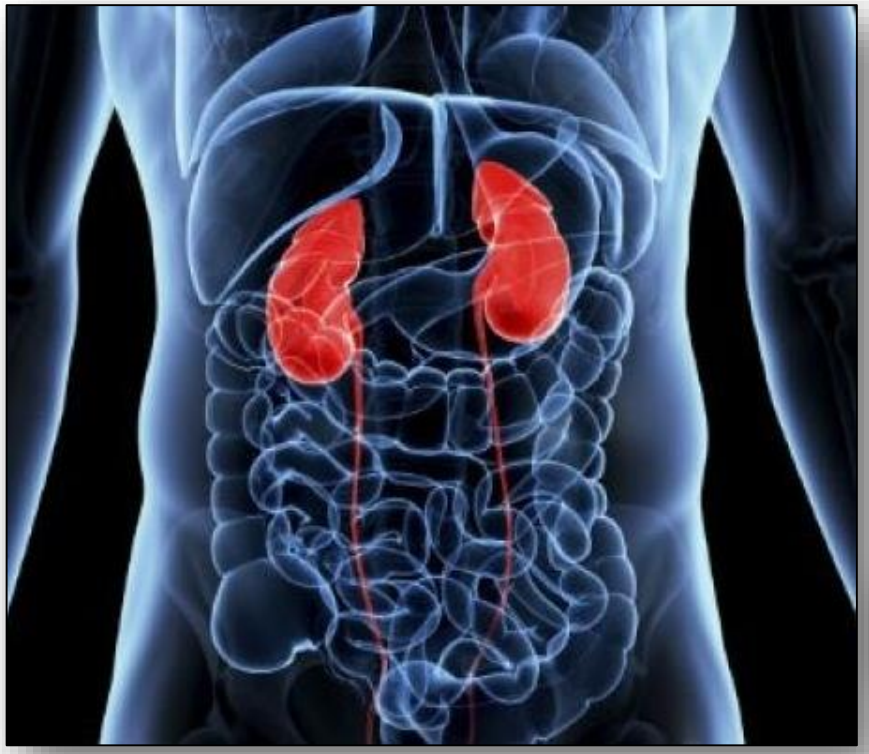
Contrast Nephropathy – a medical myth getting busted?





Jan T Kielstein

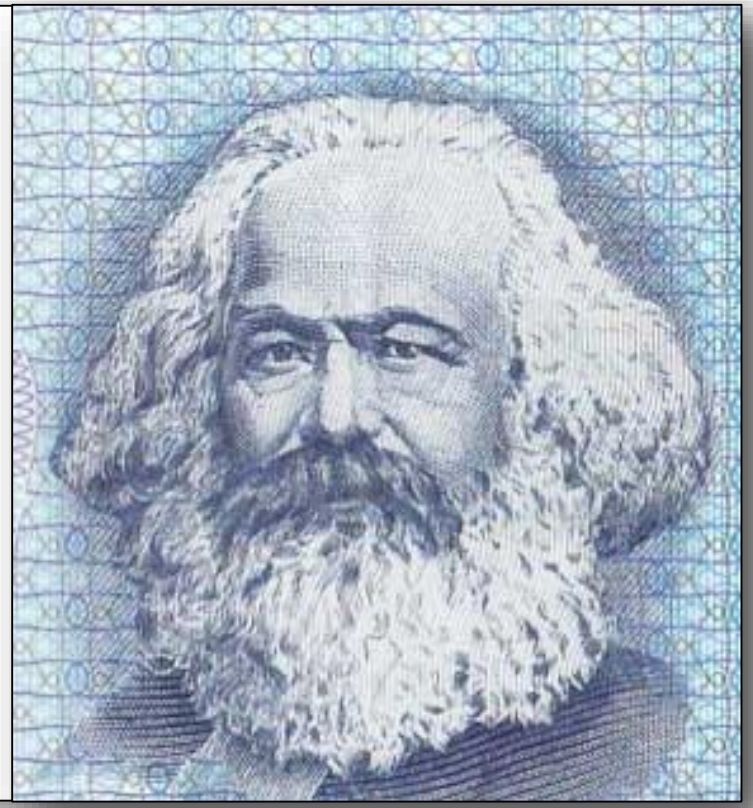
Nephrology | Rheumatology | Blood Purification

Academic Teaching Hospital Braunschweig GERMANY

Karl Marx and post contrast AKI

From Thesis and Anti-thesis to Synthesis in a medical debate

1) **Karl Marx and contrast nephropathy**
2) **Decades of deception**
3) **The inconvenient truth**
4) **What to do when you go back home?**



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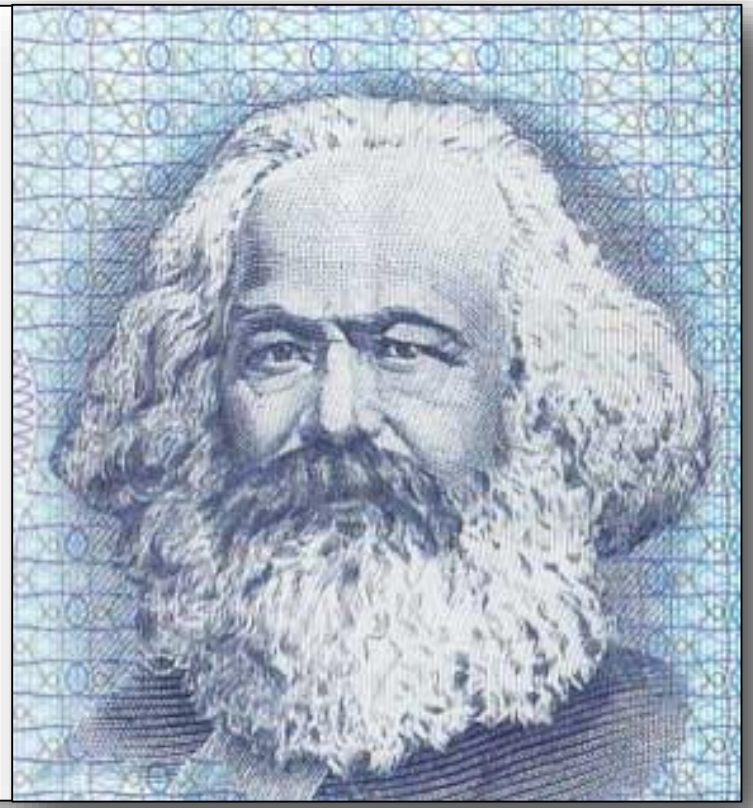

Karl Marx 05.05.1818 - 14.03.1883

“A spectre is haunting Europe – the spectre of communism. All the powers of old Europe have entered into a holy alliance to exorcise this spectre: Pope and Tsar, Metternich and Guizot, French Radicals and German police-spies.”

Karl Marx and CI-AKI

All powers of Medical subspecialties, from have entered into a holy alliance to exorcise the spectre of CI-AKI: measuring preexsiting renal function at best, progressing to the use of acetylcystein and and low osmolar contrast agents up to escalating to preventive hemofiltration, the equivalent of bulding the Berlin wall to "protect" people in East Germany from the evil of the west.

Wim van Biesen & Jan T Kielstein, NDT Rejected

Klinikum Braunschweig

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Fatal acute renal failure following intravenous pyelography in a patient with multiple myeloma

For correct evaluation of the risks attendant upon various diagnostic procedures it is important that all serious and especially all fatal complications attributed to diagnostic aids are published. This paper deals with a case of fatal acute renal failure due to intravenous pyelography.

Case History.

The patient was a man, aged 36 (Record no. 9967/56). He had been in good health until 1 year prior to admission when he noted acute back pain after having carried a heavy load. The pain subsided within a couple of days. During the following months he had several attacks of back pain, but he was able to continue his job until 2 months prior to admission, when he received a severe indirect trauma to the spine. Physiotherapeutic treatment was ineffective, his condition deteriorated with exacerbation of pain on coughing and sneezing becoming rapidly incapacitating. He was admitted to another hospital October 5th, 1956. X-ray examination of the vertebral column disclosed fractures of 9th and 11th dorsal vertebrae and of the 1st lumbar vertebra. A mottled osteoporosis was noted. The skull, both hands, right arm, both femora, left crus and both ankle joints appeared normal. Roentgenogram of chest was normal.

It was thought that the patient suffered from a systemic disease and further examinations were carried out: hemoglobin 12.2 g per cent, WBC 4,040 per cmm, differential count normal, ESR 6—10 mm/1 hour (Westergren). Urine analysis showed marked proteinuria (10—15 grams per day). Microscopy of urine showed a few leukocytes but no red cells. The 24 hour urine volume ranged from 640—1,000 ml, the urine was spontaneously concentrated to 1,027 (corrected for proteinuria). Urea clearance was 40—48 ml per minute (75—89 per cent). Serum calcium 9.7 mg per 100 ml, serum phosphorus 3.9—3.8 mg per 100 ml, acid and alkaline phosphatases normal. Wassermann reaction in blood was negative. Sternal marrow smear: highly cellular, dominated by deeply basophilic stained, primitive plasma cells (71 per cent of nucleated cells).

Fatal Acute Renal Failure Following Intravenous Pyelography in a Patient with Multiple Myeloma.

By

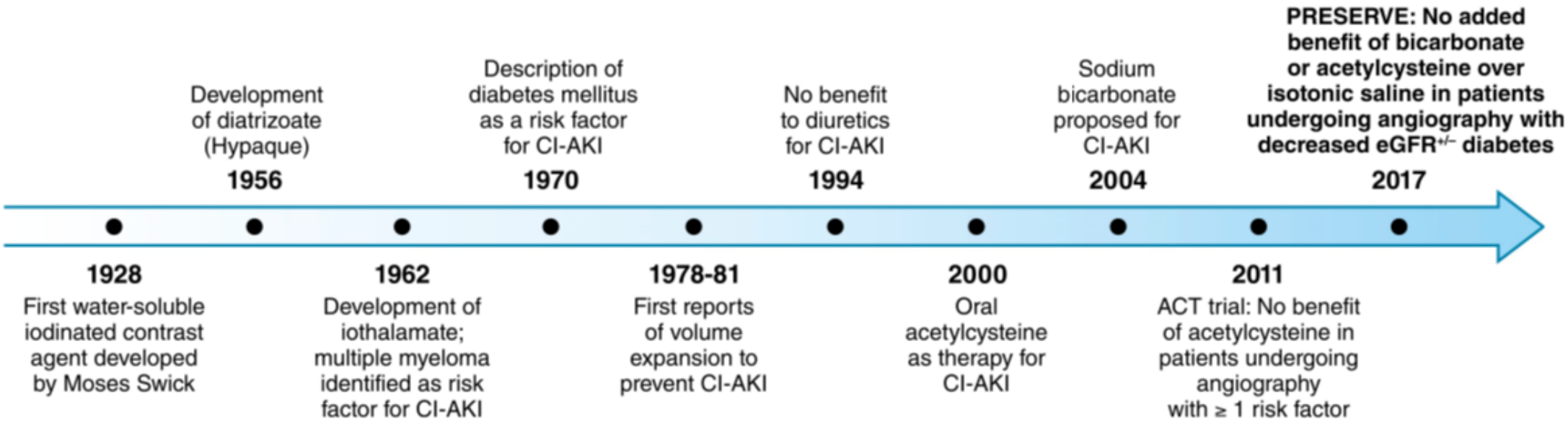
SVEN-ÅGE KILLMANN, STEFFEN GJØRUP and JØRN HESS THAYSEN.

(Submitted for publication March 1, 1957.)

KILLMANN et al. *Acta Medica Scandinavia*, 1957

Klinikum Braunschweig

Contrast-Induced Acute Kidney Injury in the PRESERVE Trial Lessons Learned



SIEW & LIU . *CJASN 2018*

Klinikum Braunschweig

Vascular pathology of homocysteinemia: implications for the pathogenesis of arteriosclerosis.

Vascular Pathology of Homocysteinemia: Implications for the Pathogenesis of Arteriosclerosis

Kilmer S. McCully, MD

INDIVIDUALS with homocystinuria [1,2] have been found to lack normal activity of the enzyme cystathionine synthetase.[3] In many of the patients progressive arterial disease develops in childhood, frequently resulting in death from thrombosis in a vital organ.[4,5] In addition, congenital dislocation of the lenses, mental retardation, and skeletal abnormalities—eg, osteoporosis, arachnodactyly, and pectus excavatum or pectus carinatum—usually are found.[5,6] The vascular changes and other abnormalities encountered in homocystinuria have been attributed either to the metabolic effects of the elevated tissue concentrations of methionine, homocysteine, or homocystine, or to the metabolic consequences of decreased tissue concentrations of cystathionine found in the disease.[7]

McKULLY *Am J Pathol* 56(1):111-28, 1969

Homocysteine Lowering and Cardiovascular Events after Acute Myocardial Infarction (n=3749)

Table 2. Plasma Levels of Total Homocysteine and B Vitamins at Baseline, after Two Months, and at the End of the Intervention.*

Variable	Folic Acid, B_{12}, and B_6 (N=937)†	Folic Acid and B_{12} (N=935)‡	B_6 (N=934)§	Placebo (N=943)¶
Total homocysteine (μmol/liter)				
Baseline	13.1±5.0	12.9±4.3	13.3±6.1	13.2±5.2
2 Mo	9.4±3.0	9.5±2.8	13.7±5.7	13.7±5.6
End of intervention	9.5±3.6	9.8±4.0	13.3±5.4	13.6±6.2

BOONA *N Engl J Med* 354:1578-88, 2006

Klinikum Braunschweig

Homocysteine Lowering and Cardiovascular Events after Acute Myocardial Infarction (n=3749)

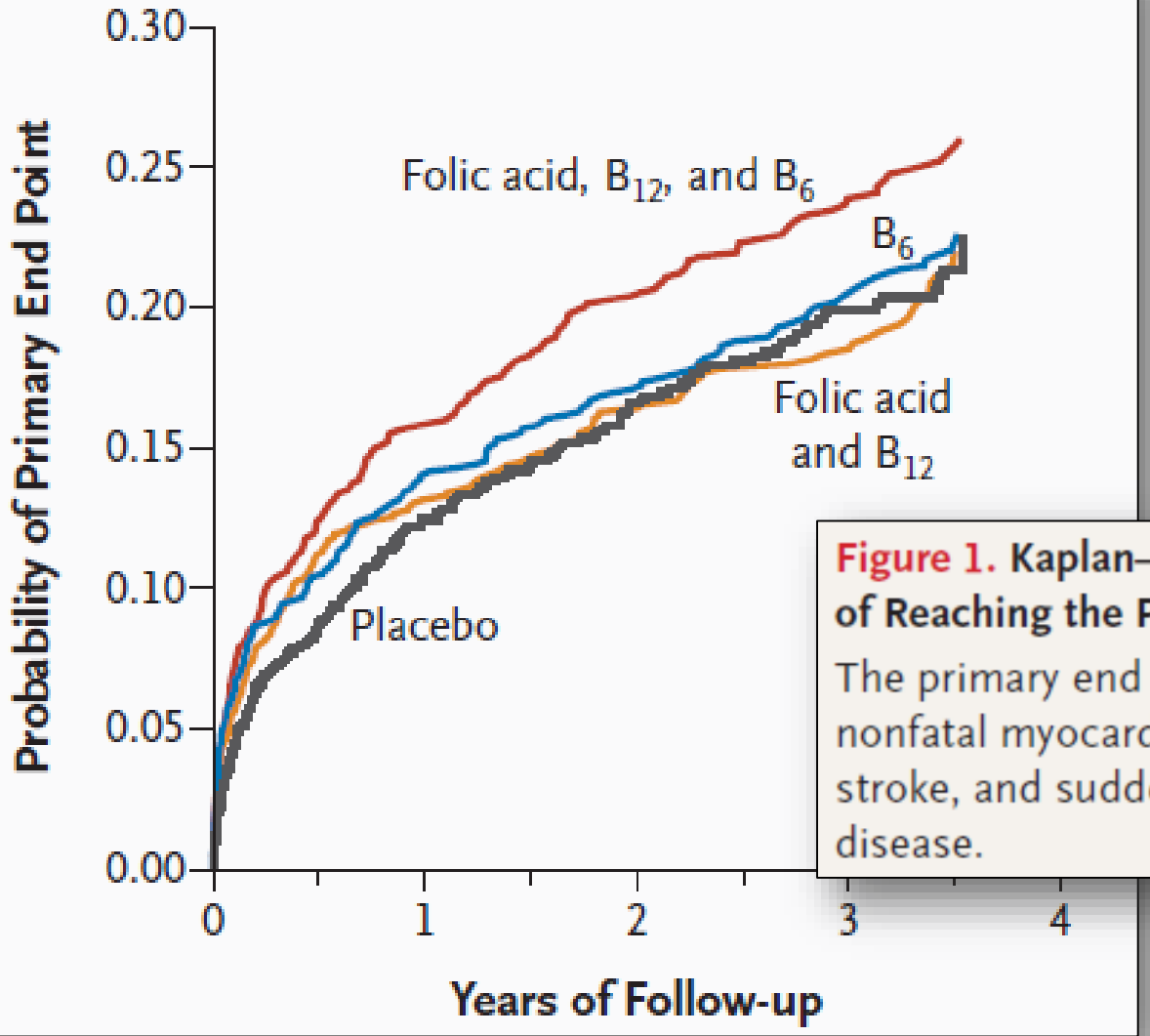


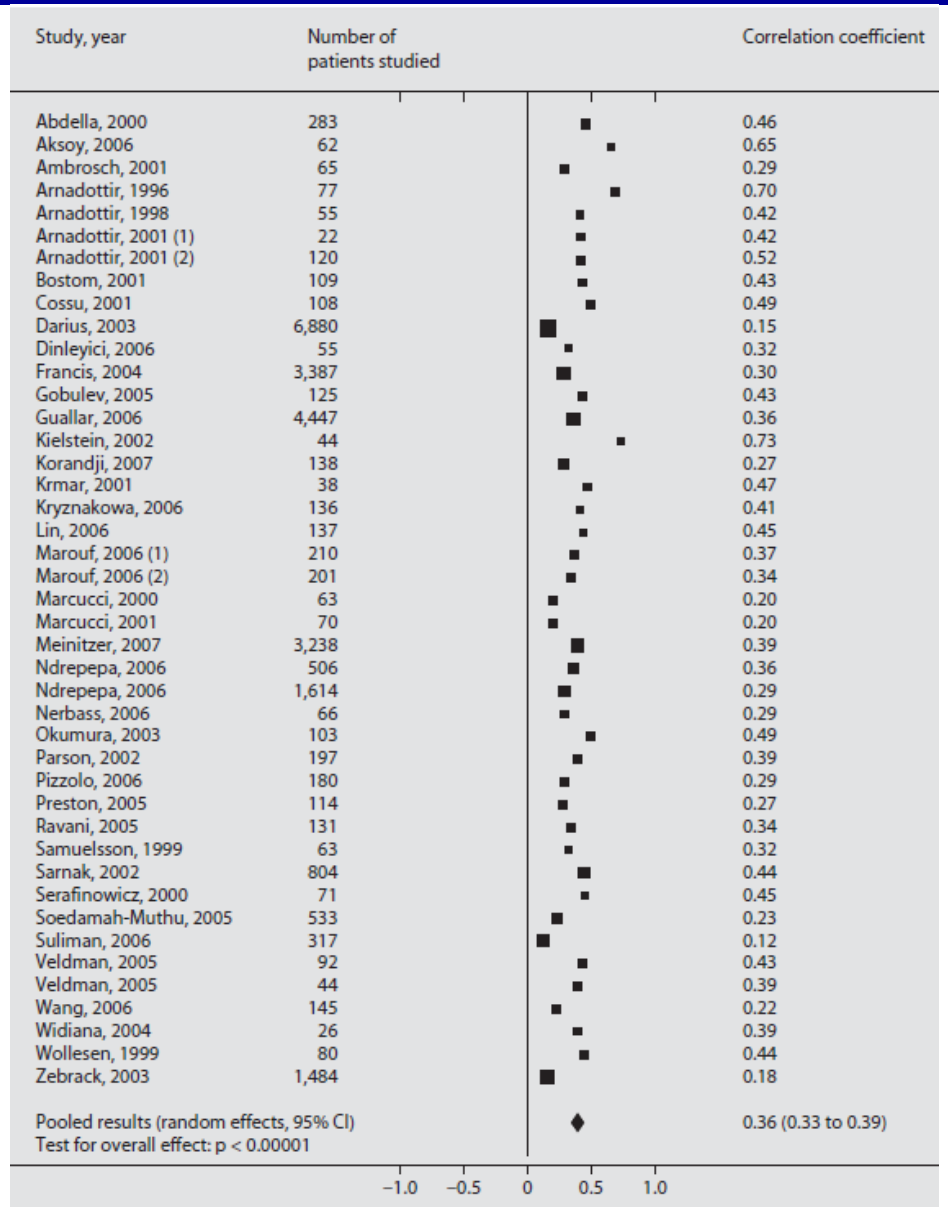
Figure 1. Kaplan–Meier Estimates of the Probability of Reaching the Primary End Point during Follow-up.

The primary end point was a composite of fatal and nonfatal myocardial infarction, fatal and nonfatal stroke, and sudden death attributed to coronary heart disease.

BOONA *N Engl J Med* 354:1578-88, 2006

Klinikum Braunschweig

Two Cardiovascular Risk Factors in One? Homocysteine and Its Relation to GFR
A Meta-Analysis of 41 Studies with 27,000 Participants



Study, year	Number of patients studied	Correlation coefficient
Abdella, 2000	283	0.46
Aksoy, 2006	62	0.65
Ambrosch, 2001	65	0.29
Arnadottir, 1996	77	0.70
Arnadottir, 1998	55	0.42
Arnadottir, 2001 (1)	22	0.42
Arnadottir, 2001 (2)	120	0.52
Bostom, 2001	109	0.43
Cossu, 2001	108	0.49
Darius, 2003	6,880	0.15
Dinleyici, 2006	55	0.32
Francis, 2004	3,387	0.30
Gobulev, 2005	125	0.43
Guallar, 2006	4,447	0.36
Kielstein, 2002	44	0.73
Korandji, 2007	138	0.27
Krmar, 2001	38	0.47
Kryznakowa, 2006	136	0.41
Lin, 2006	137	0.45
Marouf, 2006 (1)	210	0.37
Marouf, 2006 (2)	201	0.34
Marcucci, 2000	63	0.20
Marcucci, 2001	70	0.20
Meinitzer, 2007	3,238	0.39
Ndrepepa, 2006	506	0.36
Ndrepepa, 2006	1,614	0.29
Nerbass, 2006	66	0.29
Okumura, 2003	103	0.49
Parson, 2002	197	0.39
Pizzolo, 2006	180	0.29
Preston, 2005	114	0.27
Ravani, 2005	131	0.34
Samuelsson, 1999	63	0.32
Sarnak, 2002	804	0.44
Serafinowicz, 2000	71	0.45
Soedamah-Muthu, 2005	533	0.23
Suliman, 2006	317	0.12
Veldman, 2005	92	0.43
Veldman, 2005	44	0.39
Wang, 2006	145	0.22
Widiana, 2004	26	0.39
Wollesen, 1999	80	0.44
Zebrack, 2003	1,484	0.18
Pooled results (random effects, 95% CI)		0.36 (0.33 to 0.39)

Test for overall effect: $p < 0.00001$

KIELSTEIN et al.
Kidney Blood Press Res 31:259–267, 2008

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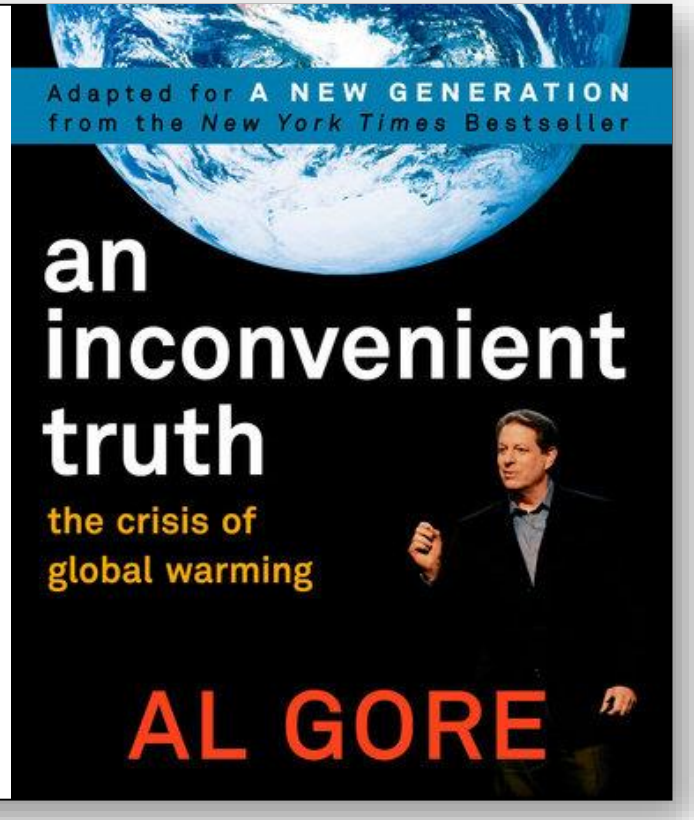
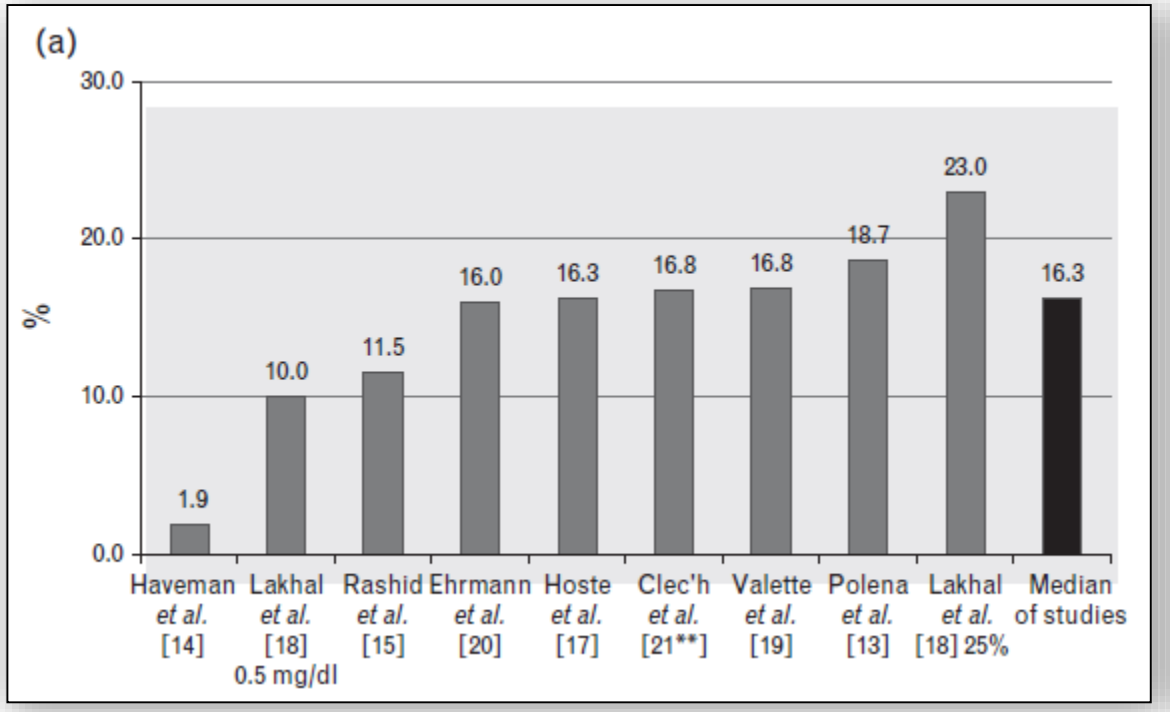
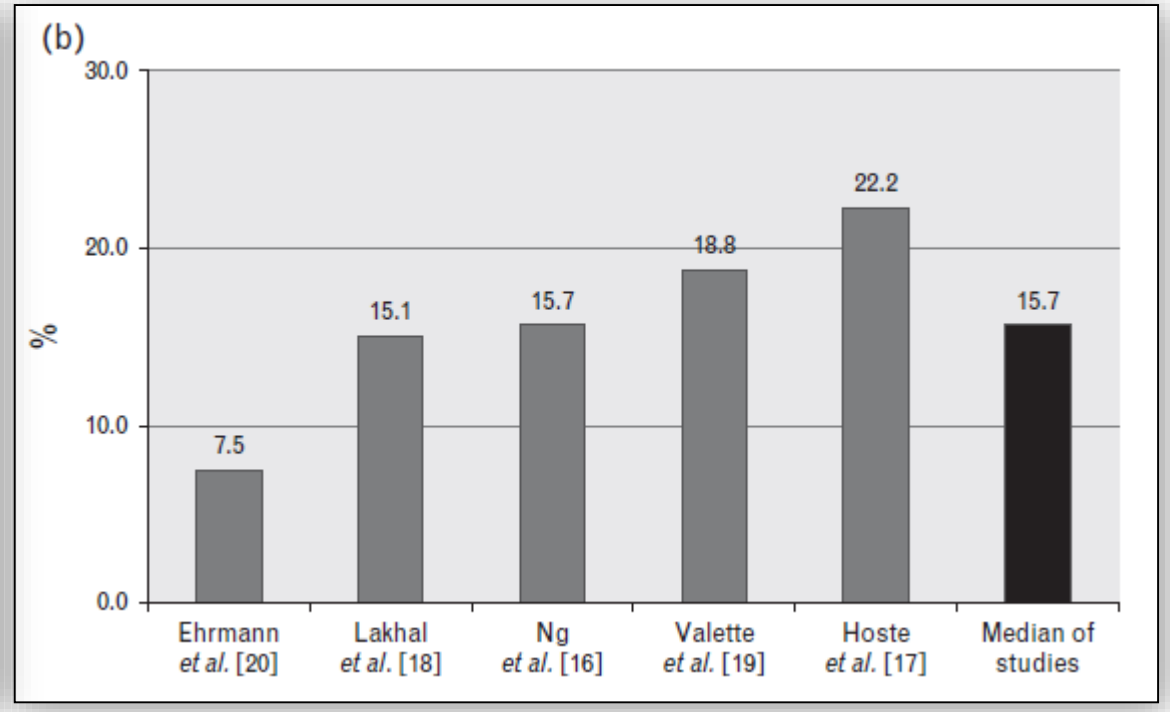


Contrast-associated AKI in the critically ill: relevant or irrelevant?

increase of serum creatinine of 25% or 0.5 mg/dl or greater within a 48–96 h

AKIN definition





van den BERGHE et al. *Curr Opin Crit Care* 20:596–605, 2014

Klinikum Braunschweig

Risk of contrast induced nephropathy in the critically ill: a prospective, case matched study

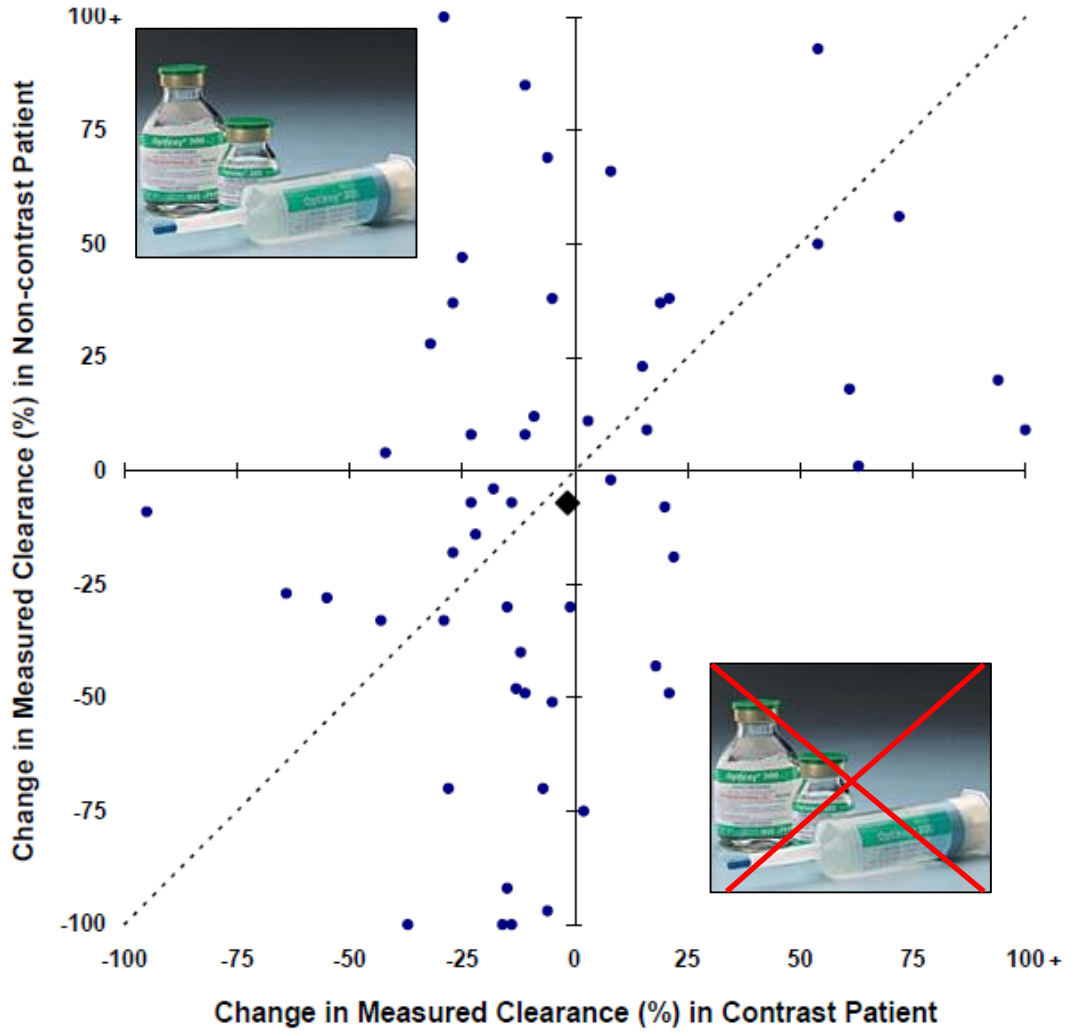


Figure 4 mCr_{Cl} three days after scanning, relative to pre-scan values. Filled circles represent individual matched patient pairs, and the filled diamond mean values for contrast and non-contrast patients. Points above the diagonal line imply greater loss of renal function in the patient who received contrast, and below the line greater loss in the patient who did not receive contrast.

CELY et al. *Crit Care* 12;16(2):R67 2012

Klinikum Braunschweig

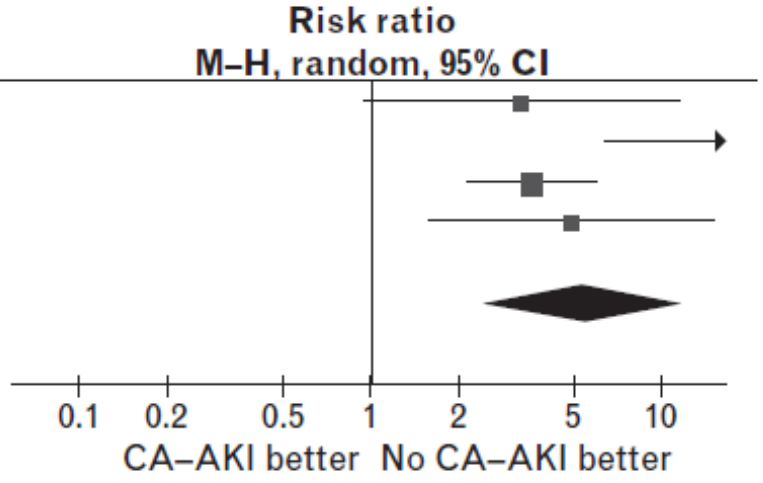
Contrast-associated AKI in the critically ill: relevant or irrelevant?

(a)

Study or subgroup	CA-AKI Events	CA-AKI Total	no CA-AKI Events	no CA-AKI Total	Weight	Risk ratio M–H, Random, 95% CI	Year
Rashid *et al.* [15]	3	16	7	123	21.2%	3.29 [0.95, 11.48]	2009
Lakhal *et al.* [18]	10	45	2	254	17.4%	28.22 [6.39, 124.56]	2011
Hoste *et al.* [17]	21	128	30	659	37.8%	3.60 [2.13, 6.09]	2011
Valette *et al.* [19]	5	17	5	84	23.6%	4.94 [1.60, 15.21]	2012
Total (95% CI)		**206**		**1120**	**100.0%**	**5.45 [2.48, 11.99]**	
Total events	39		44				

Heterogeneity: Tau² = 0.36, Chi² = 6.98, df = 3 (P 0.07); I² = 57%

Test for overall effect: Z = 4.21 (*P* < 0.0001)

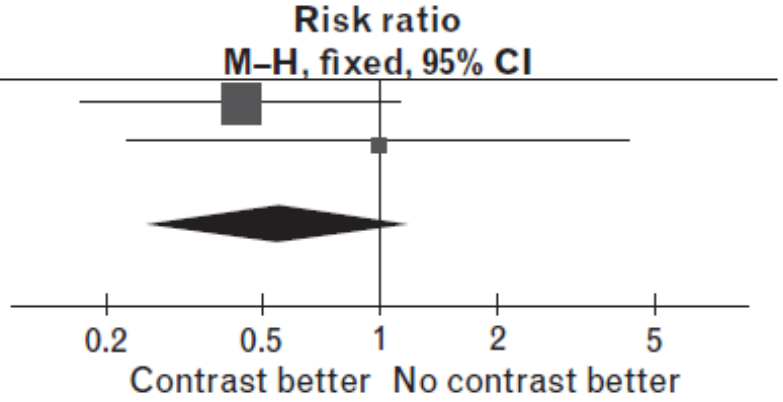


(b)

Study or subgroup	Contrast Events	Contrast Total	No contrast Events	No contrast Total	Weight	Risk ratio M–H, fixed, 95% CI	Year
Ng *et al.* [16]	6	185	14	192	79.9%	0.44 [0.17, 1.13]	2010
Ehrmann *et al.* [20]	3	146	4	192	20.1%	0.99 [0.22, 4.34]	2013
Total (95% CI)		**331**		**384**	**100.0%**	**0.55 [0.25, 1.21]**	
Total events	9		18				

Heterogeneity: Chi² = 0.79, df = 1 (P 0.37); I² = 0%

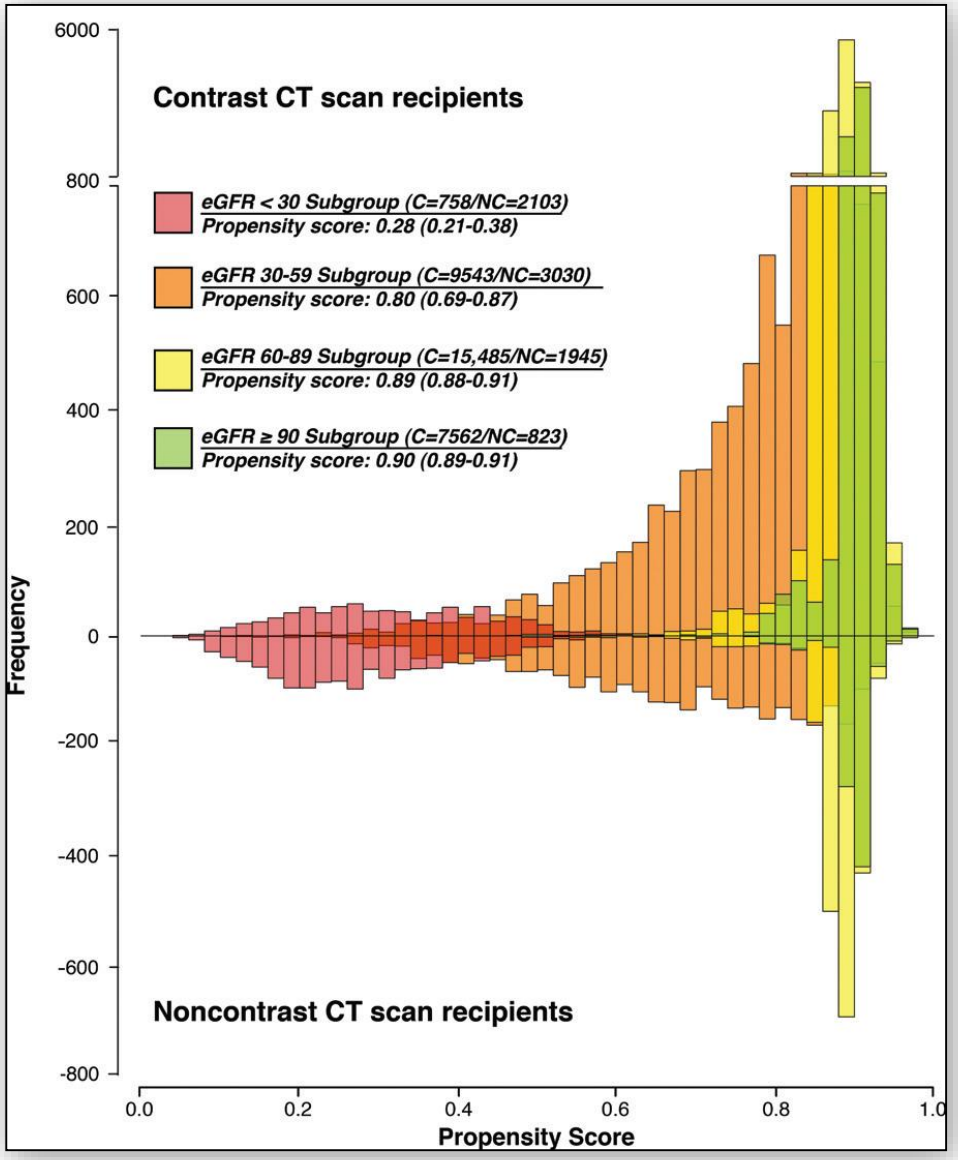
Test for overall effect: Z = 1.49 (*P* = 0.14)



van den BERGHE et al. *Curr Opin Crit Care* 20:596–605, 2014

Klinikum Braunschweig

Risk of Intravenous Contrast Material–mediated Acute Kidney Injury: A Propensity Score–matched Study Stratified by Baseline-estimated GFR (n=12 508)



McDONALD RADIOLOGY 271: 1, 2014

Klinikum Braunschweig

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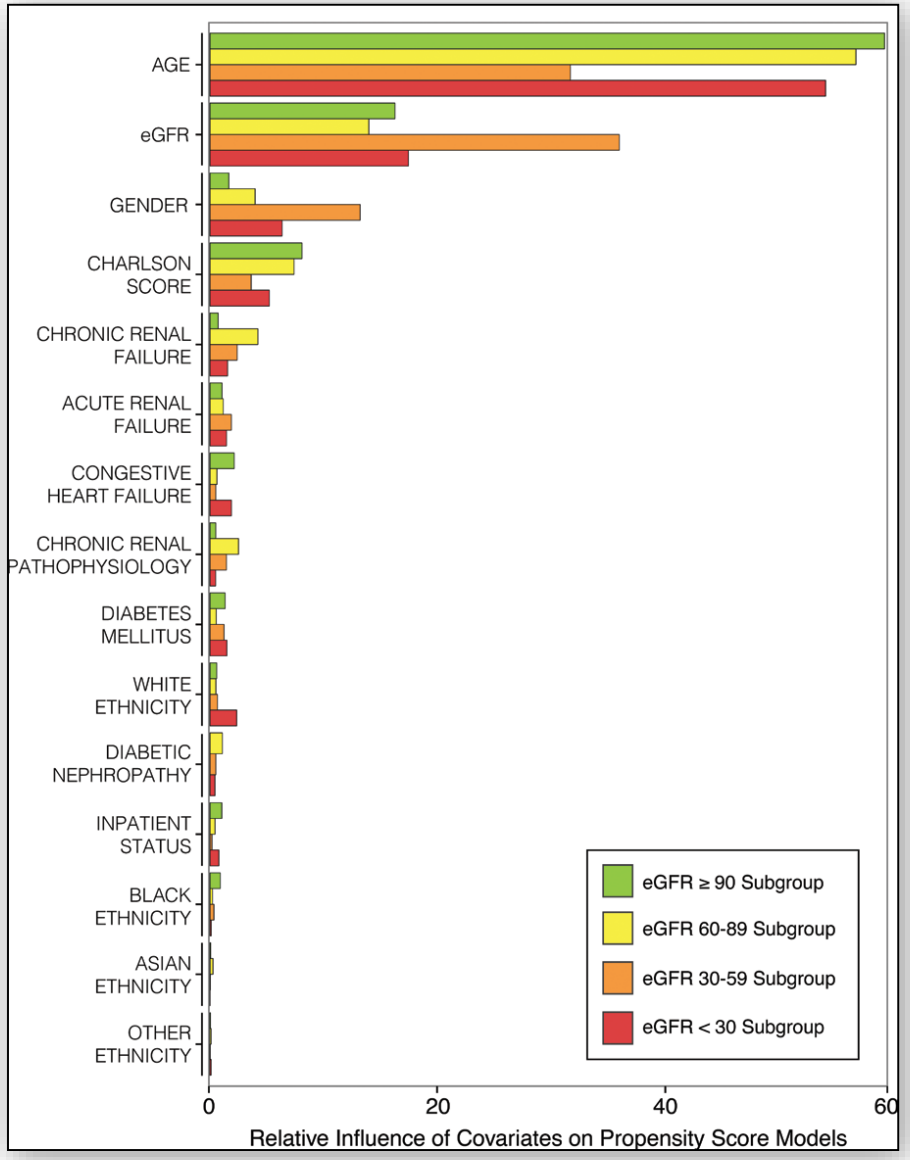


McDONALD RADIOLOGY 271: 1, 2014

Klinikum Braunschweig

Risk of Intravenous Contrast Material–mediated Acute Kidney Injury: A Propensity Score–matched Study Stratified by Baseline-estimated GFR (n=12 508)

Propensity Score–adjusted Risk of AKI Following Contrast-enhanced or Unenhanced CT

eGFR Subgroup (mL/min/1.73 m²)	AKI Following Contrast-enhanced Scanning*	AKI Following Unenhanced Scanning*	OR†	*P* Value‡
≥ 90	10/821 (1.2)	11/821 (1.3)	0.91 (0.38, 2.15)	.82
60–89	40/1935 (2.1)	39/1935 (2.0)	1.03 (0.66, 1.60)	.99
30–59	161/2755 (5.8)	170/2755 (6.2)	0.94 (0.76, 1.18)	.65
< 30	102/743 (14)	105/743 (14)	0.97 (0.72, 1.30)	.89

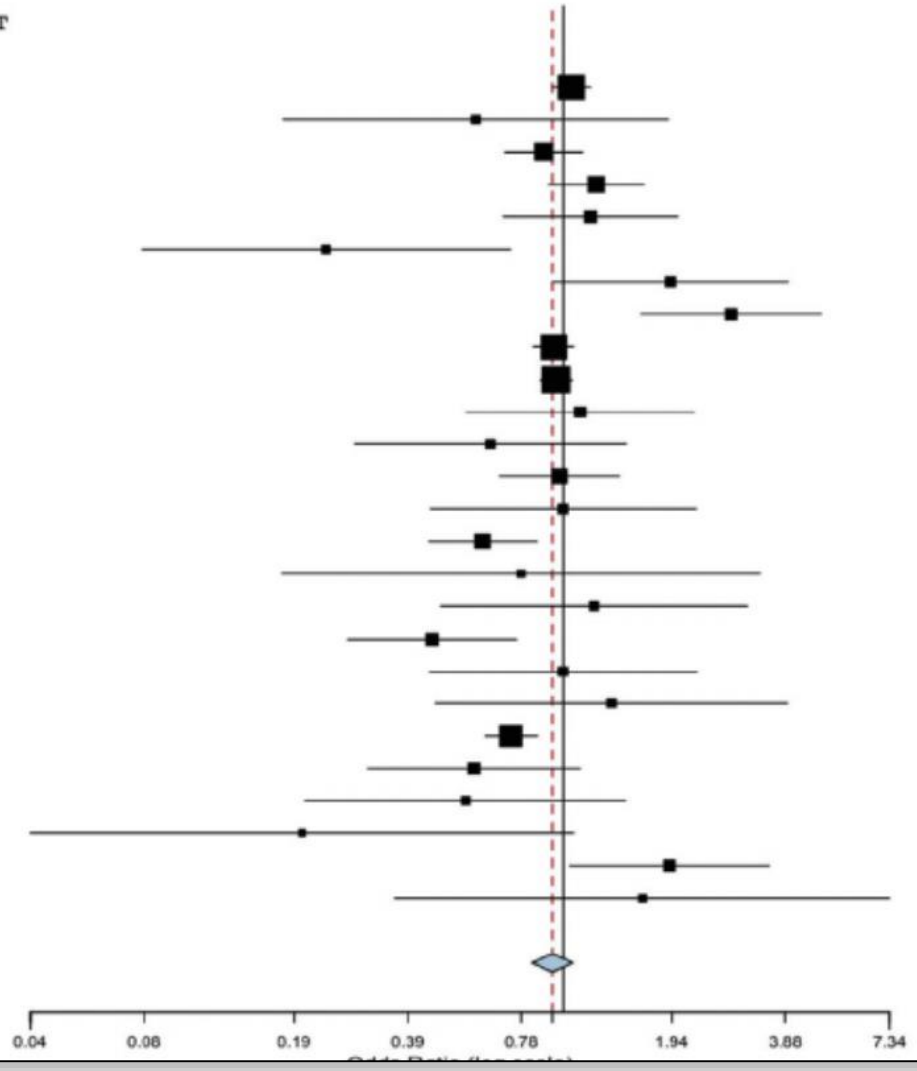
"In conclusion, our findings provide additional evidence that the administration of intravenous contrast material does not increase the risk of AKI, even in patients with substantially compromised renal function."

McDONALD RADIOLOGY 271: 1, 2014

Acute kidney injury after computed tomography: A meta-analysis.

Acute Kidney Injury

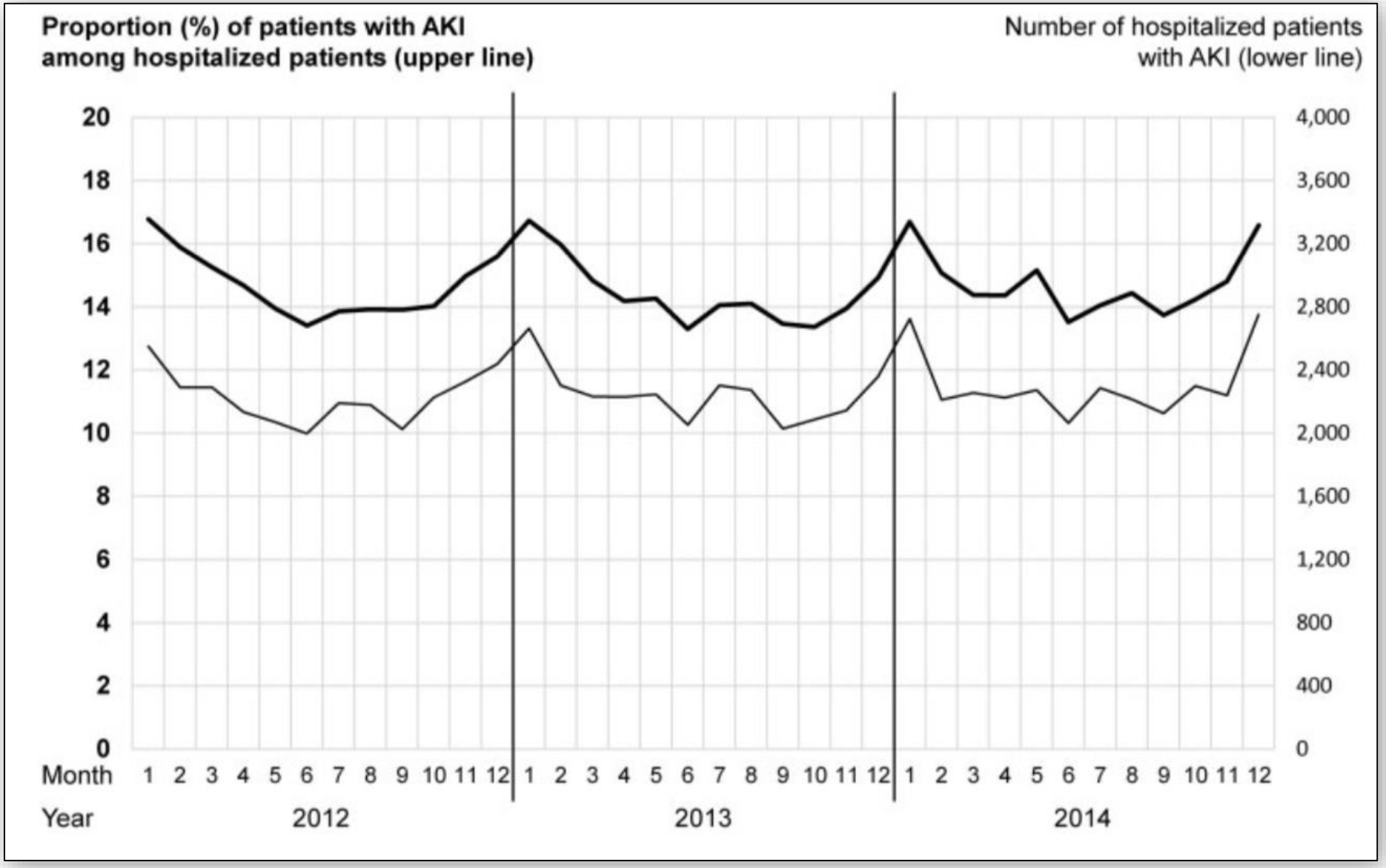
Studies	Estimate (95% C.I.)	#AKI/Total CECT	#AKI/Total non-CECT
Hinson 2017	1.052 (0.938, 1.180)	766/7201	559/5499
Ehrlich 2016	0.587 (0.182, 1.896)	5/157	7/132
Heller 2016	0.889 (0.702, 1.126)	598/6954	87/909
Gao 2015	1.225 (0.918, 1.634)	70/474	235/1896
Hemmet 2015	1.184 (0.695, 2.016)	36/326	26/274
Paliwal 2015	0.236 (0.077, 0.724)	5/203	9/93
Sonhaye 2015	1.928 (0.941, 3.953)	21/620	12/672
Alsafi 2014	2.787 (1.608, 4.830)	62/677	17/487
McDonald 2014	0.944 (0.834, 1.068)	515/10673	544/10673
Davenport 2013	0.960 (0.869, 1.060)	835/10121	867/10121
Kidoh 2013	1.111 (0.556, 2.222)	13/143	27/327
Cely 2012	0.642 (0.280, 1.472)	14/53	19/53
Murakami 2012	0.978 (0.680, 1.407)	57/938	68/1096
Silcock 2012	1.000 (0.445, 2.247)	13/132	13/132
Sinert 2012	0.613 (0.441, 0.852)	44/773	265/2956
Aulicky 2010	0.776 (0.181, 3.332)	5/164	3/77
McGillicuddy 2010	1.209 (0.476, 3.073)	18/822	6/330
Lima 2010	0.450 (0.269, 0.755)	28/575	35/343
Ng 2010	1.000 (0.443, 2.258)	14/81	14/81
Bansal 2009	1.343 (0.459, 3.932)	8/65	7/74
Bruce 2009	0.728 (0.622, 0.854)	252/5790	440/7484
Oleinik 2009	0.581 (0.304, 1.111)	21/348	19/191
Langner 2008	0.552 (0.208, 1.466)	7/100	12/100
Tremblay 2005	0.204 (0.039, 1.069)	2/56	6/39
Heller 1991	1.917 (1.045, 3.516)	35/479	16/405
Cramer 1985	1.623 (0.359, 7.340)	4/193	3/233
Overall (I^2=6513 % , P< 0.001)	**0.938 (0.825, 1.065)**	**3448/48118**	**3316/44677**



AYCOCK et al.
Ann Emerg Med. 2018;71:44-53, 2018

Klinikum Braunschweig

Seasonality of acute kidney injury incidence and mortality among hospitalized patients



IWAGAMI *Nephrol Dial Transplant* 33: 1354–1362, 2018

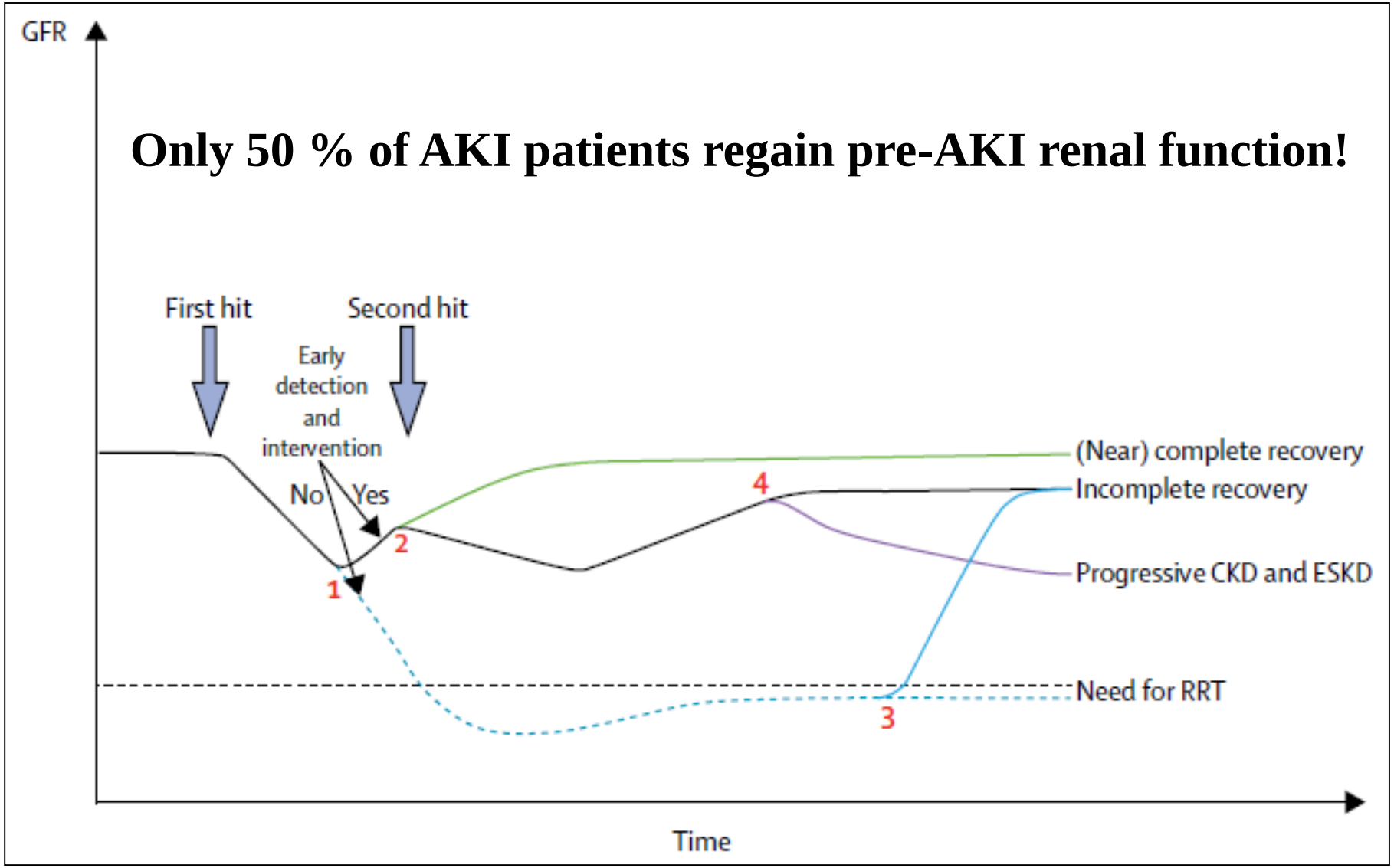
Klinikum Braunschweig

Seasonality of acute kidney injury incidence and mortality among hospitalized patients

	Overall, $N=81\,279$ n (%)	Spring (Mar–May), $N=19\,947$ n (%)	Summer (Jun–Aug), $N=19\,555$ n (%)	Autumn (Sep–Nov), $N=19\,494$ n (%)	Winter (Dec–Feb), $N=22\,283$ n (%)	P-value
Age category (years)						<0.001
<65	11 764 (14.5)	2830 (14.2)	3037 (15.5)	2947 (15.1)	2950 (13.2)	
65–74	14 724 (18.1)	3640 (18.3)	3580 (18.3)	3542 (18.2)	3962 (17.8)	
75–84	26 519 (32.6)	6444 (32.3)	6272 (32.1)	6570 (33.7)	7233 (32.5)	
≥85	28 272 (34.8)	7033 (35.3)	6666 (34.1)	6435 (33.0)	8138 (36.5)	
Sex (male)	41 272 (50.8)	10 106 (50.7)	9831 (50.3)	9928 (50.9)	11 407 (51.2)	0.284
Comorbidities						
Hypertension	37 601 (46.3)	9196 (46.1)	8973 (45.9)	9127 (46.8)	10 305 (46.3)	0.289
Diabetes mellitus	18 657 (23.0)	4566 (22.9)	4299 (22.0)	4522 (23.2)	5270 (23.7)	0.001
CKD	20 069 (24.7)	4986 (25.0)	4811 (24.6)	4759 (24.4)	5513 (24.7)	0.588
Liver disease	3213 (4.0)	840 (4.2)	782 (4.0)	767 (3.9)	824 (3.7)	0.059
Cancer	14 523 (17.9)	3475 (17.4)	3695 (18.9)	3675 (18.9)	3678 (16.5)	<0.001
Admission diagnosis category						<0.001
Cardiovascular disease	24 248 (29.8)	6178 (31.0)	5224 (26.7)	5707 (29.3)	7139 (32.0)	
Pulmonary disease	11 265 (13.9)	2906 (14.6)	2461 (12.6)	2384 (12.2)	3514 (15.8)	
Abdominal disease	10 408 (12.8)	2626 (13.2)	2579 (13.2)	2700 (13.9)	2503 (11.2)	
Genitourinary disease	6213 (7.6)	1454 (7.3)	1741 (8.9)	1559 (8.0)	1459 (6.6)	
Injury	5877 (7.2)	1384 (6.9)	1347 (6.9)	1520 (7.8)	1626 (7.3)	
Others	23 268 (28.6)	5399 (27.1)	6203 (31.7)	5624 (28.9)	6042 (27.1)	
Sepsis (yes)	21 439 (26.4)	5052 (25.3)	5317 (27.2)	5283 (27.1)	5787 (26.0)	<0.001
Nephrotoxic substances						
Contrast agents for CT scans	7192 (8.9)	1760 (8.8)	1745 (8.9)	1807 (9.3)	1880 (8.4)	0.028
ACEIs/ARBs[a]	5882 (7.2)	1421 (7.1)	1423 (7.3)	1455 (7.5)	1583 (7.1)	0.474
Diuretics[a]	3647 (4.5)	880 (4.4)	933 (4.8)	882 (4.5)	952 (4.3)	0.093
NSAIDs[a]	6085 (7.5)	1448 (7.3)	1489 (7.6)	1525 (7.8)	1623 (7.3)	0.091
Aminoglycosides	2972 (3.7)	762 (3.8)	693 (3.5)	741 (3.8)	776 (3.5)	0.151
Vancomycin	1975 (2.4)	493 (2.5)	488 (2.5)	447 (2.3)	547 (2.5)	0.552
Amphotericin B	44 (0.1)	8 (<0.1)	8 (<0.1)	13 (0.1)	15 (0.1)	0.450
Cisplatin	156 (0.2)	37 (0.2)	34 (0.2)	50 (0.3)	35 (0.2)	0.110

IWAGAMI *Nephrol Dial Transplant* 33: 1354–1362, 2018

Management of patients at risk of acute kidney injury



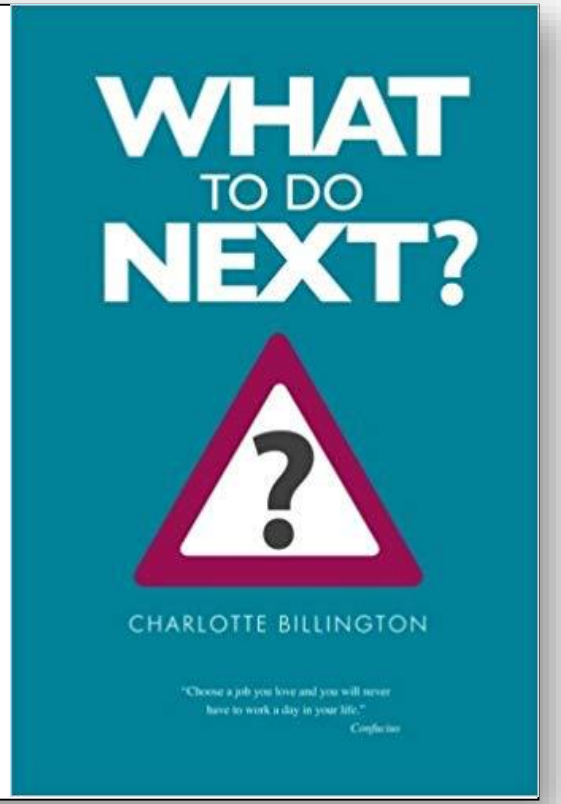
VANMASSENHOVE, KIELSTEIN, JÖRRES
Lancet 389: 2139–51, 2017

Klinikum Braunschweig

Karl Marx and post contrast AKI

From Thesis and Anti-thesis to Synthesis in a medical debate

1) Karl Marx and contrast nephropathy
2) Decades of deception
3) The inconvenient truth
4) **What to do when you go back home?**



KDIGO Clinical Practice Guideline for Acute Kidney Injury

4.1: Define and stage AKI after administration of intravascular contrast media as per Recommendations 2.1.1–2.1.2. (*Not Graded*)

4.1.1: In individuals who develop changes in kidney function after administration of intravascular contrast media, evaluate for CI-AKI as well as for other possible causes of AKI. (*Not Graded*)

4.2.1: Assess the risk for CI-AKI and, in particular, screen for pre-existing impairment of kidney function in all patients who are considered for a procedure that requires intravascular (i.v. or i.a.) administration of iodinated contrast medium. (*Not Graded*)

4.2.2: Consider alternative imaging methods in patients at increased risk for CI-AKI. (*Not Graded*)

4.3.1: Use the lowest possible dose of contrast medium in patients at risk for CI-AKI. (*Not Graded*)

4.3.2: We recommend using either iso-osmolar or low-osmolar iodinated contrast media, rather than high-osmolar iodinated contrast media in patients at increased risk of CI-AKI. (*1B*)

4.4.1: We recommend i.v. volume expansion with either isotonic sodium chloride or sodium bicarbonate solutions, rather than no i.v. volume expansion, in patients at increased risk for CI-AKI. (*1A*)

4.4.2: We recommend not using oral fluids alone in patients at increased risk of CI-AKI. (*1C*)

4.4.3: We suggest using oral NAC, together with i.v. isotonic crystalloids, in patients at increased risk of CI-AKI. (*2D*)

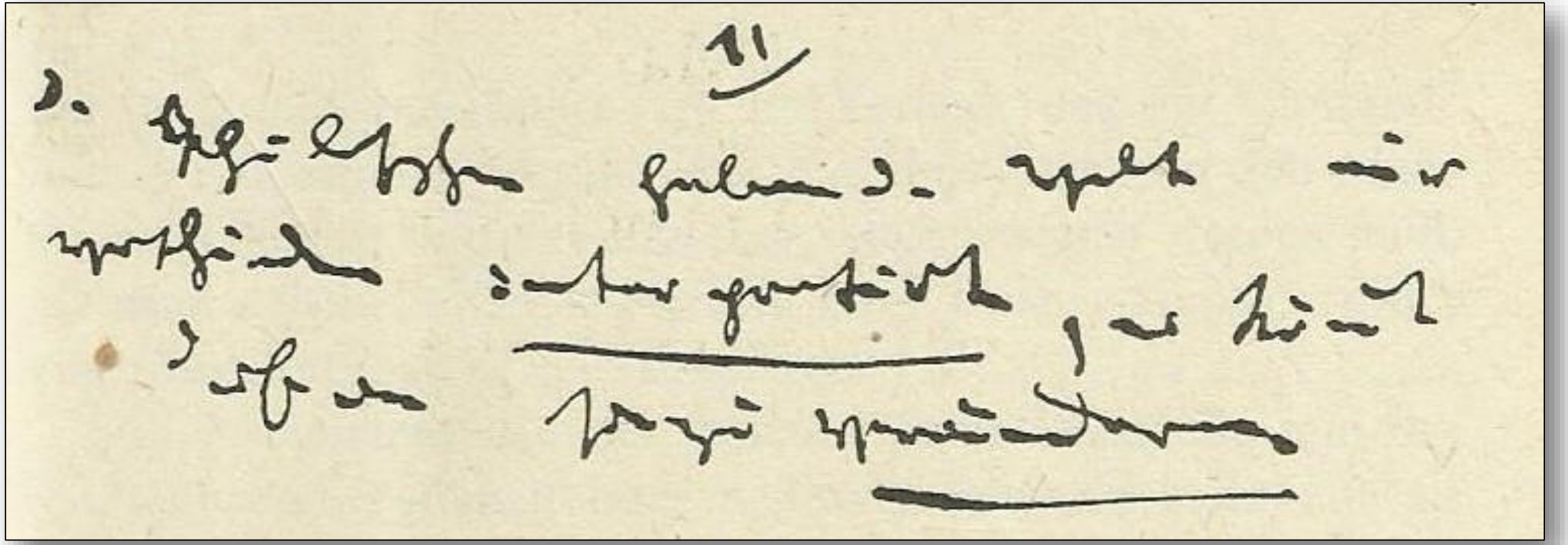
4.4.4: We suggest not using theophylline to prevent CI-AKI. (*2C*)

4.4.5: We recommend not using fenoldopam to prevent CI-AKI. (*1B*)

4.5.1: We suggest not using prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for c removal in patients at increased risk for CI-AKI. (*2C*)

Kidney International VOLUME 2 | ISSUE 1 | MARCH 2012

“Philosophers have only interpreted the world, in various ways. The point, however, is to change it.”



„Ludwig Feuerbach und der Ausgang der deutschen Philosophie"

Klinikum Braunschweig

Post-contrast acute kidney injury – Part 1: Definition, clinical features, incidence, role of contrast medium and risk factors

1) **PC-AKI is the preferred term for renal function deterioration after contrast.**

2) **PC-AKI has many possible causes.**

3) **The risk of AKI caused by intravascular contrast medium has been overstated.**

4) **Important patient risk factors for PC-AKI are CKD and dehydration.**

van der MOOEN al. *European Radiology*
https://doi.org/10.1007/s00330-017-5246-5

Comparative effect of contrast media type on the incidence of contrastinduced nephropathy: a systematic review and meta-analysis.

Type of contrast media

- There were no differences in risk for contrast-associated AKI associated with different types of low-osmolal contrast media
- Iodixanol was associated with a non–clinically significant reduction in risk for contrast-associated AKI as compared, in aggregate, with low-osmolal contrast media

ENG al.
Ann Intern Med. 164(6):417-424. 2016

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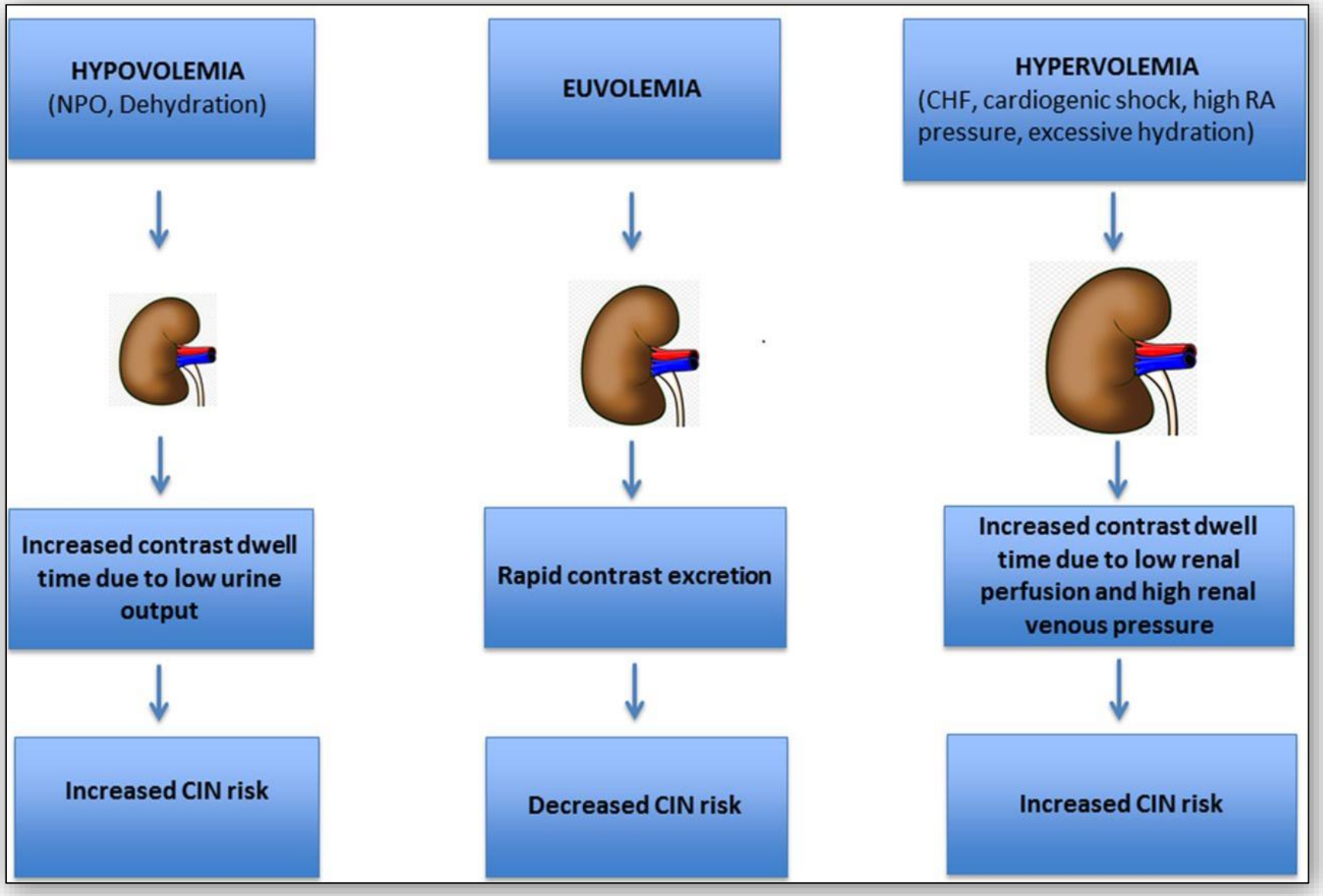
Intravenous Hydration and Contrast-Induced Acute Kidney Injury: Too Much of a Good Thing?



GUPTA al.
J Am Heart Assoc 5:e003777, 2016

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A randomized comparison of 1-h sodium bicarbonate hydration versus standard peri-procedural saline hydration in patients with chronic kidney disease undergoing intravenous contrastenhancedcomputerized tomography.

A. 250 mL 1.4% Na-bicarbonate 1 h before contrast CT

B. 2000 mL 0.9% NaCl

- 1000 mL before und 1000 mL after contrast CT

Table 2. Incidence of CI-AKI according to the AKI criteria

AKIN stage	Sodium bicarbonate (95% CI)		Saline (95% CI)		RR (95% CI)	
I (increase >26.5 μmol/L or 150–200% from baseline)	11/263, 4.2%	(2.3–7.4)	17/273, 6.2%	(3.9–9.8)	0.7	(0.3–1.4)
II (increase 200–300% from baseline)	0/263	(0.0–1.7)	0/273	(0.0–1.7)	1.0	(0.0–52.1)
III (increase >300% from baseline, or ≥354 μmol/L, or on RRT)	0/263	(0.0–1.7)	0/273	(0.0–1.7)	1.0	(0.0–52.1)

KOOIMAN et al. *Nephrol Dial Transplant* (2014) 29: 1029–1036

Klinikum Braunschweig

Post-contrast acute kidney injury – Part 1: Definition, clinical features, incidence, role of contrast medium and risk factors

Table 4 Risk of PC-AKI

(a) Levels of eGFR at which there is a risk

The risk of PC-AKI in patients with eGFR ≥ 30 ml/min/1.73m^2 after intravenous and intra-arterial CM administration with second-pass renal exposure is very low, but there is conflicting evidence on the risk for intra-arterial CM administration with first-pass renal exposure

Level of Evidence: B

Preventive measures are recommended for patients with eGFR < 30 ml/min/1.73m^2 before intravenous and intra-arterial CM administration with second-pass renal exposure

Level of Evidence: C

Preventive measures are recommended for patients with eGFR < 45 ml/min/1.73m^2 if they are in ICU or if they will receive intra-arterial CM administration with first-pass renal exposure

Level of Evidence: C

Recommendations for prevention of PC-AKI in adults may also be used in children and adolescents

Level of Evidence D

van der MOOEN al. *European Radiology*
https://doi.org/10.1007/s00330-017-5246-5

Post-contrast acute kidney injury – Part 1: Definition, clinical features, incidence, role of contrast medium and risk factors

(b) Risk factors

The principal risk factor for PC-AKI is impaired renal function. Most other published patient-related risk factors are risk factors for the presence of chronic kidney disease or AKI, and are not specific for PC-AKI

Level of Evidence B

There is no difference in PC-AKI risk between IOCM and LOCM. The use of ionic, high-osmolar CM and repeated CM injections in a short period (48–72 h) should be avoided

Level of Evidence C

When CM are injected intravenously, there is insufficient evidence that CM dose is a risk factor. When CM are injected intra-arterially, the ratio of CM dose (in gram Iodine) / *absolute* eGFR (in ml/min) should be kept below 1.1 or the ratio of CM volume (in ml) / eGFR (in ml/min/1.73m^2) should be kept below 3.0 when using a CM concentration of 350 mgl/ml

Level of Evidence C

van der MOOEN al. *European Radiology*
https://doi.org/10.1007/s00330-017-5246-5

Post-contrast acute kidney injury – Part 1: Definition, clinical features, incidence, role of contrast medium and risk factors

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van der MOOEN al. *European Radiology*
https://doi.org/10.1007/s00330-017-5246-5

Post-contrast acute kidney injury. Part 2: risk stratification, role of hydration and other prophylactic measures, patients taking metformin and CKD5D

1) **In CKD, hydration reduces the PC-AKI risk.**

2) **IV normal saline and IV sodium bicarbonate are equally effective.**

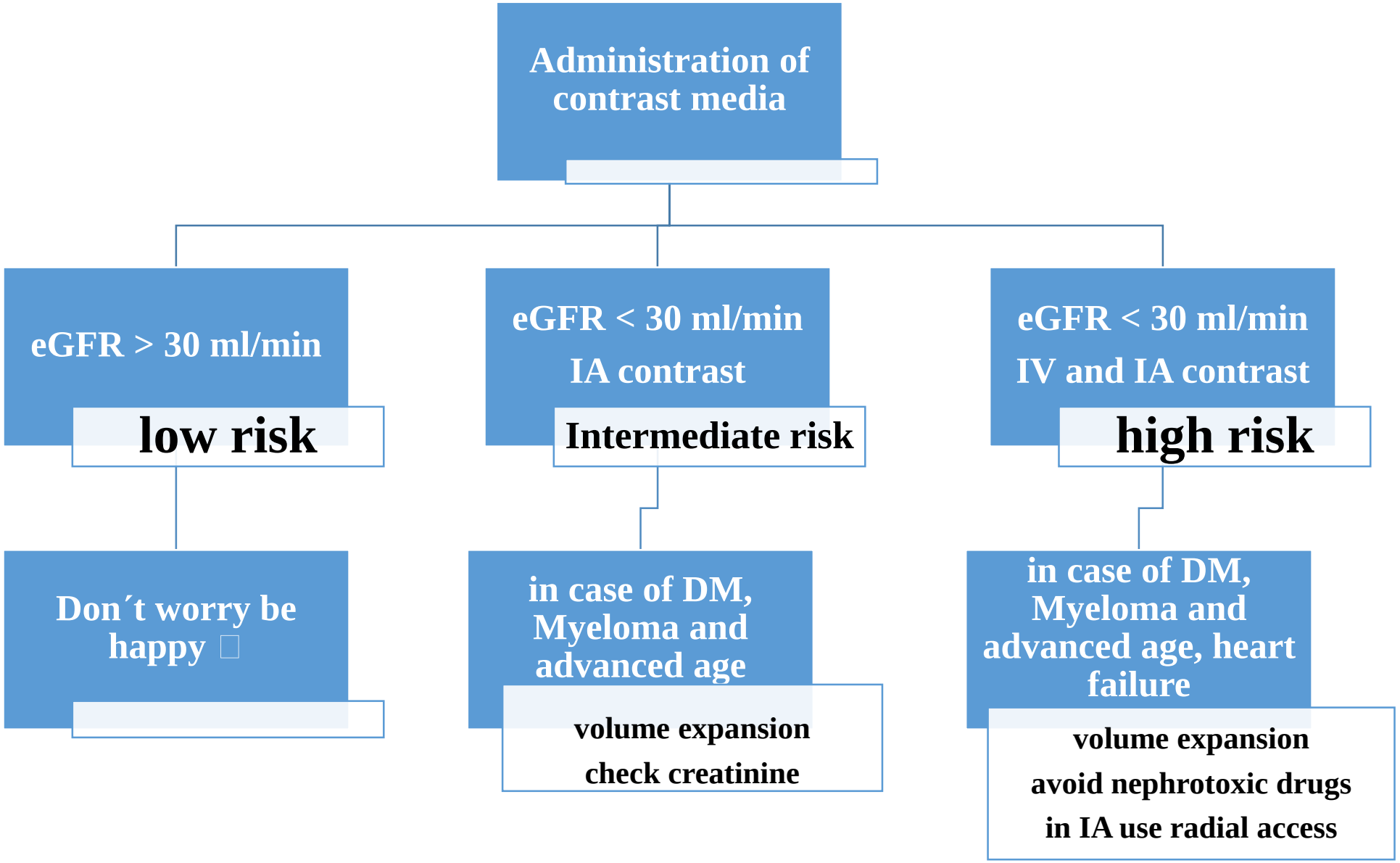
3) **No drugs have been consistently shown to reduce the risk of PC-AKI.**

4) **Stop metformin from the time of contrast medium administration if eGFR < 30.**

5) **Dialysis schedules need not change when intravascular contrast medium is given.**

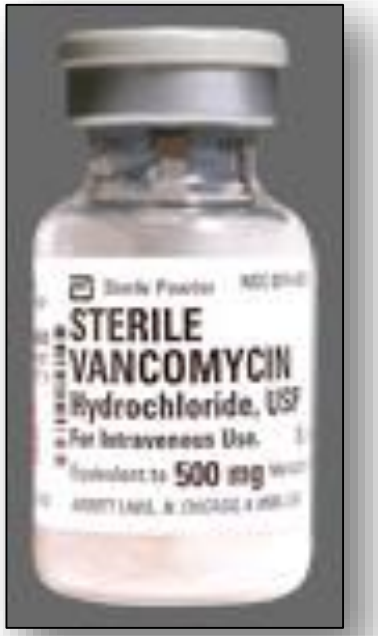
Van der MOLEN al. *European Radiology*
https://doi.org/10.1007/s00330-017-5247-4

A proposed flow chart



Klinikum Braunschweig

AKI is not a switch…..



JTK

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AKI is a dimmer

JTK

Klinikum Braunschweig

Temporas mutantur, nos et mutamur in illis.

Klinikum Braunschweig


Proper contrast users of all Countries, Unite!