Nadciśnienie śródbrzuszne/podatność jamy brzusznej jako problem intensywnej terapii

Intra-abdominal hypertension/abdominal compliance in critically ill patients – it is a problem?

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WSACS Ambassadors

WSACS came to realize that expanding our network is essential to achieve our goals, and we have been looking for new ways to connect to all healthcare workers around the world. For this reason we appointed WSACS Ambassadors in different parts of the world. WSACS Ambassadors are IAH and ACS experienced and will assist WSACS to improve IAH and ACS knowledge in their country and region. We expect WSACS Ambassadors to

- Promote the WSACS locally
- Liaise with local surgical and critical care societies
- Assist the WSACS in translating the latest WSACS guidelines
- Stimulate participation in WSACS studies
- Participate in WSACS activities

Please feel free to contact the WSACS Ambassador in your area if you have any question for them – use the email address provided.

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Rys historyczny

Short history

- ✓ XIX wiek Marley podkreślił ścisłą zależność pomiędzy uniesieniem przepony a ciśnieniem panującym wewnątrz jamy brzusznej *(in the XIX century Marley described strict correlation between diaphragm elevation and intra-abdominal pressure – IAP)*
- ✓ Bradley i *Bradley stwierdzili, że wzrost IAP powoduje spadek przepływu nerkowego (Bradley and Bradley noted, that an increase in IAP resulted in reduced renal blood flow)*
- ✓ Heinricus zauważył, że wzrost IAP do wartości powyżej 27 cmH_2O powoduje śmiertelne w skutkach upośledzenie oddychania *(Heinricus showed that an increase in IAP > 27 cmH_2O resulted in fatal respiratory insufficiency)*
- ✓ Emerson stwierdził, że wzrost ciśnienia w jamie brzusznej powoduje spadek powrotu żylnego i upośledzenia krążenia wieńcowego *(Emerson presented strong significant correlation between IAP and cardiac function)*

Rys historyczny

Short history

- ✓ **1951 Baggot – pierwszy opis przypadku śmierci noworodka, u którego dokonano jednoetapowej operacyjnej korekcji wrodzonego wytrzewienia. On też po raz pierwszy wprowadził określenie „ostry ciśnieniowy pneumo – peritonit" twierdząc, że wzrost ciśnienia w jamie brzusznej może doprowadzić do niewydolności wielonarządowej oraz śmierci**

- ✓ 1951 Baggot – first description of infant death following rapid increase in IAP after single-step correction of total eventeration. He defined this patology as „a acute pressure pneumo-peritonit".

Ciśnienie śródbrzuszne

(IAP-Intra Abdominal Pressure) to ciśnienie w jamie brzusznej determinowane przez elementy sprężyste i sztywne.

Intra-abdominal pressure (IAP) is a pressure measured in the abdominal cavity determined by static and flexible part of abdominal wall.

0 - 7 mmHg

Kilka ważnych definicji *(some important deffinitions)*

- ✓ nadciśnienie śródbrzuszne (IAH - Intra-Abdominal Hypertension) – IAP > 12 mmHg
- ✓ zespół ciasnoty śródbrzusznej (ACS - Abdominal Compartment Syndrome) – patologia kliniczna będąca następstwem IAH *(each pathologies resulted from IAH)*:

  - pierwotny ACS *(primary ACS),*
  - wtórny ACS *(secondary ACS).*

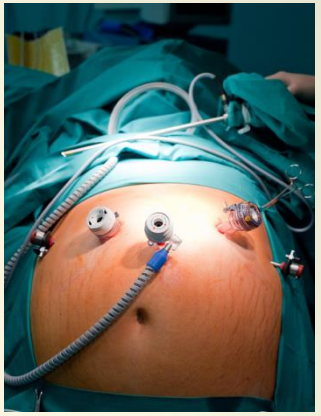
- ✓ APP – abdominal perfusion pressure = MAP – IAP.

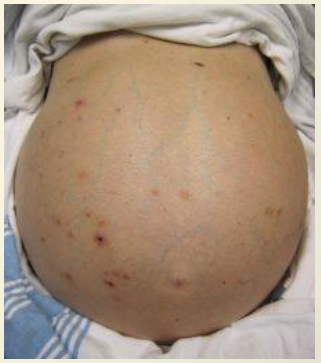
I° 12 – 15 mmHg;

II° 16 - 20 mmHg;

III° 21 – 25 mmHg;

IV° > 25 mmHg.

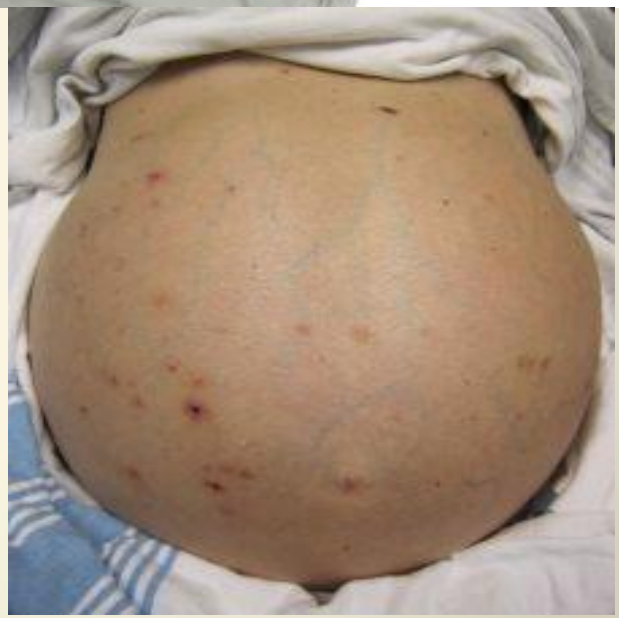




Pierwotny ACS

primary ACS

- ✓ otyłość *(obesity),*
- ✓ ciąża *(pregnancy),*
- ✓ uraz jamy brzusznej *(abdominal trauma),*
- ✓ krwawienie do przestrzeni zaotrzewnowej *(intra-peritoneal bleeding),*
- ✓ zapalenie otrzewnej *(peritonitis)*
- ✓ zakażenia wewnątrzbrzuszne *(intra-abdominal infections)*
- ✓ ostre zapalenie trzustki *(pancreatitis)*
- ✓ sepsa i wstrząs septyczny *(sepsis and septic shock)*
- ✓ szybko narastające wodobrzusze *(ascites)*



Wtórny ACS

secondary ACS

- ✓ chirurgiczny „packing" (surgical packing)
- ✓ r
- ✓ k

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RESEARCH ARTICLE

Intra-Abdominal Pressure Correlates with Extracellular Water Content

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Abstract

Background

Secondary increase in intra-abdominal pressure (IAP) may result from extra-abdominal pathology, such as massive fluid resuscitation, capillary leak or sepsis. All these conditions increase the extravascular water content. The aim of this study was to analyze the relationship between IAP and body water volume.

Material and Methods

Adult patients treated for sepsis or septic shock with acute kidney injury (AKI) and patients undergoing elective pharyngolaryngeal or orthopedic surgery were enrolled. IAP was measured in the urinary bladder. Total body water (TBW), extracellular water content (ECW) and volume excess (VE) were measured by whole body bioimpedance. Among critically ill patients, all parameters were analyzed over three consecutive days, and parameters were evaluated perioperatively in surgical patients.

Results

One hundred twenty patients were studied. Taken together, the correlations between IAP and VE, TBW, and ECW were measured at 408 time points. In all participants, IAP strongly

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Witters[1], Coli
[3, 4], Niels Van

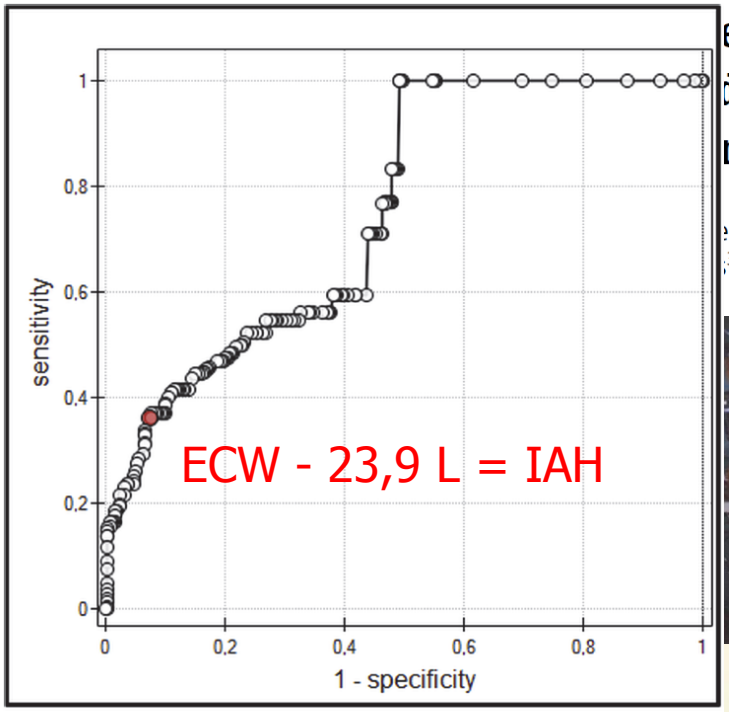


Fig 2. ROC curve for extracellular water content (ECW) as a diagnostic test of intra-abdominal hypertension (IAH). Cut-off point of ECW for IAH = 23.9 L. Area under the curve (AUC) = 0.87.

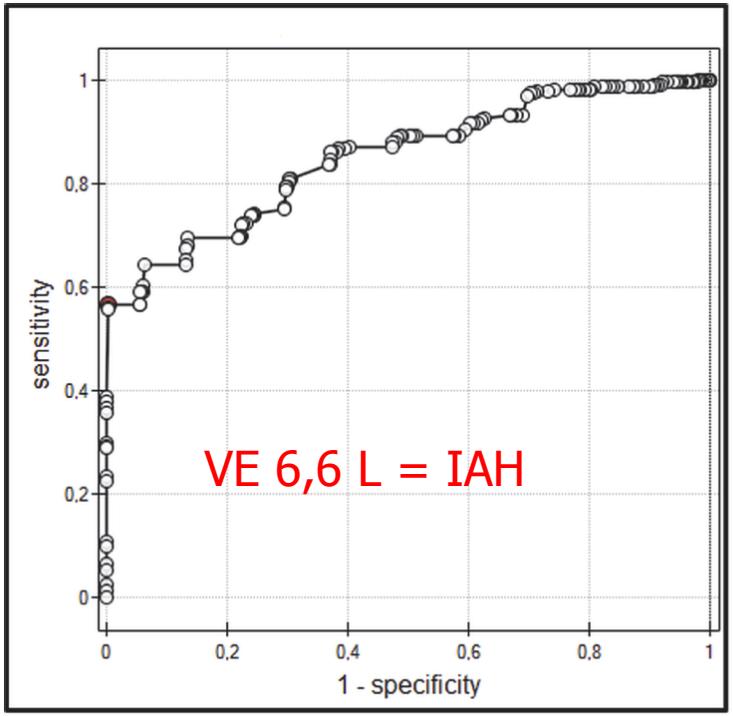
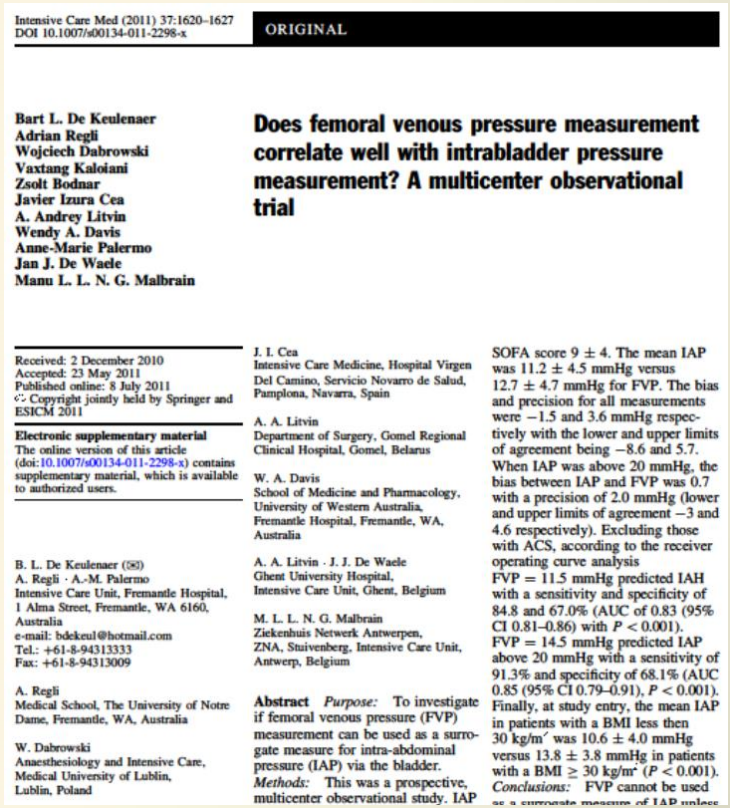


Fig 3. ROC curve for volume excess (VE) as a diagnostic test of intra-abdominal hypertension (IAH). Cut-off point of VE for IAH = 6.6 L. Area under the curve (AUC) = 0.85.

Zasady pomiaru ciśnienia śródbrzusznego

- ✓ pomiar bezpośredni (umieszczenie czujnika wewnątrz jamy brzusznej) *(direct measurement in abdominal cavity)*

- ✓ pomiar pośredni *(indirect measurement)*
  - ✓ żyła udowa *(femoral vein)*
  - ✓ cewnik dożołądkowy *(stomach catheter)*
  - ✓ sonda doodbytnicza *(rectal catheter)*
  - ✓ cewnik Foley'a (medota Krona) *(Foley's catheter)*



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ORIGINAL

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Does femoral venous pressure measurement correlate well with intrabladder pressure measurement? A multicenter observational trial

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Abstract *Purpose:* To investigate if femoral venous pressure (FVP) measurement can be used as a surrogate measure for intra-abdominal pressure (IAP) via the bladder. *Methods:* This was a prospective, multicenter observational study. IAP SOFA score 9 ± 4. The mean IAP was 11.2 ± 4.5 mmHg versus 12.7 ± 4.7 mmHg for FVP. The bias and precision for all measurements were −1.5 and 3.6 mmHg respectively with the lower and upper limits of agreement being −8.6 and 5.7. When IAP was above 20 mmHg, the bias between IAP and FVP was 0.7 with a precision of 2.0 mmHg (lower and upper limits of agreement −3 and 4.6 respectively). Excluding those with ACS, according to the receiver operating curve analysis FVP = 11.5 mmHg predicted IAH with a sensitivity and specificity of 84.8 and 67.0% (AUC of 0.83 (95% CI 0.81–0.86) with $P < 0.001$). FVP = 14.5 mmHg predicted IAP above 20 mmHg with a sensitivity of 91.3% and specificity of 68.1% (AUC 0.85 (95% CI 0.79–0.91), $P < 0.001$). Finally, at study entry, the mean IAP in patients with a BMI less then 30 kg/m² was 10.6 ± 4.0 mmHg versus 13.8 ± 3.8 mmHg in patients with a BMI ≥ 30 kg/m² ($P < 0.001$). *Conclusions:* FVP cannot be used

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...fect of IAH on cardiac

Intra-abdominal hypertension increases spatial QRS-T angle and elevates ST-segment J-point in healthy women undergoing laparoscopic surgery

Wojciech Dabrowski, MD, PhD,[a,*] Andrzej Jaroszynski, MD, PhD,[b,c]

Effect of intra-abdominal hypertension (IAH) on voltage of ST segment J point (STJ). The powers of all analyses were very high ($1 - \beta > 0.9$). Time points: 1) BL-1, before anesthesia and surgery, while patients were lying in the supine position (baseline value); 2) IAH-S, just after the induction of pneumoperitoneum in the supine position; 3) IAH-T, 2 min after induction of Trendelenburg position; 4) BL-2, just after surgery completion; and 5) POD-1, on the morning of postoperative day 1.

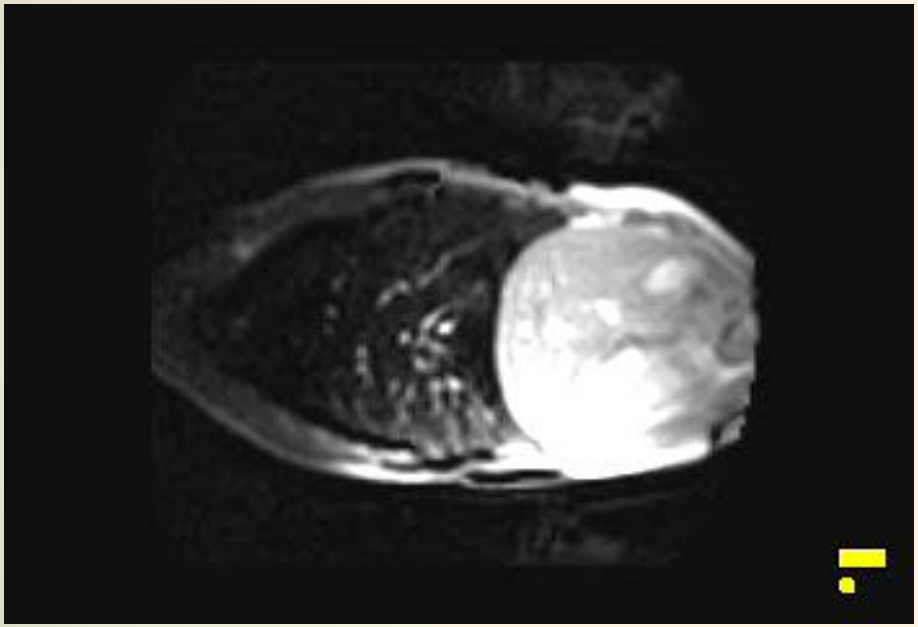
		Time points				
		BL-1	*IAH-S*	*IAH-T*	*BL-2*	*POD-1*
STJ (mV)	I	0 [0; 0.02]	0.01 [0; 0.03]	0.01 [0; 0.02]	0.01 [0; 0.02]	0.01 [0; 0.02]
	II	0 [−0.01; 0.02]	0.02 [−0.01; 0.04]	0.03** [0.01; 0.04]	0.02*** [0.01; 0.04]	0.01* [0; 0.03]
	III	0 [−0.02; 0.01]	0.02** [−0.01; 0.04]	0.02*** [0.01; 0.03]	0.01*** [0; 0.03]	0.01** [0; 0.02]
	aVR	0 [−0.01; 0.01]	0 [−0.02; 0.02]	−0.01† [−0.03; 0]	−0.01 [−0.01; 0]	−0.01 [−0.02; 0]
	aVL	0.01 [−0.01; 0.01]	0 [−0.01; 0.01]	0** [−0.02; 0]	0* [−0.01; 0.01]	0 [−0.01; 0.01]
	aVF	0 [−0.02; 0.02]	0.01* [−0.01; 0.04]	0.02*** [0.01; 0.04]	0.02*** [0; 0.03]	0.01** [0; 0.02]
	V_1	0.01 [0; 0.03]	0.02 [0; 0.02]	0.01 [0; 0.03]	0.01 [−0.01; 0.03]	0.02 [0; 0.03]
	V_2	0.02 [0.01; 0.03]	0.03** [0.02; 0.05]	0.03** [0.03; 0.04]	0.03** [0.01; 0.04]	0.03* [0.01; 0.03]
	V_3	0 [0; 0.03]	0.02* [0; 0.06]	0.03** [0; 0.06]	0.04** [0.02; 0.05]	0.02 [0; 0.03]
	V_4	0.01 [−0.02; 0.03]	0.01 [−0.03; 0.04]	0† [−0.02. 0.03]	0.05** [0.01; 0.06]	0.01 [0; 0.03]
	V_5	0 [−0.03; 0.02]	−0.01 [−0.03; 0.02]	0 [−0.02; 0.02]	0.02***†† [0.01; 0.04]	0.01**† [0; 0.03]
	V_6	−0.01 [−0.02; 0.01]	0 [−0.01; 0.02]	0 [−0.02; 0.03]	0.02** [0; 0.03]	0.01* [−0.01; 0.02]

Wpływ IAH na układ oddechowy *(an effect of IAH on respiratory system)*

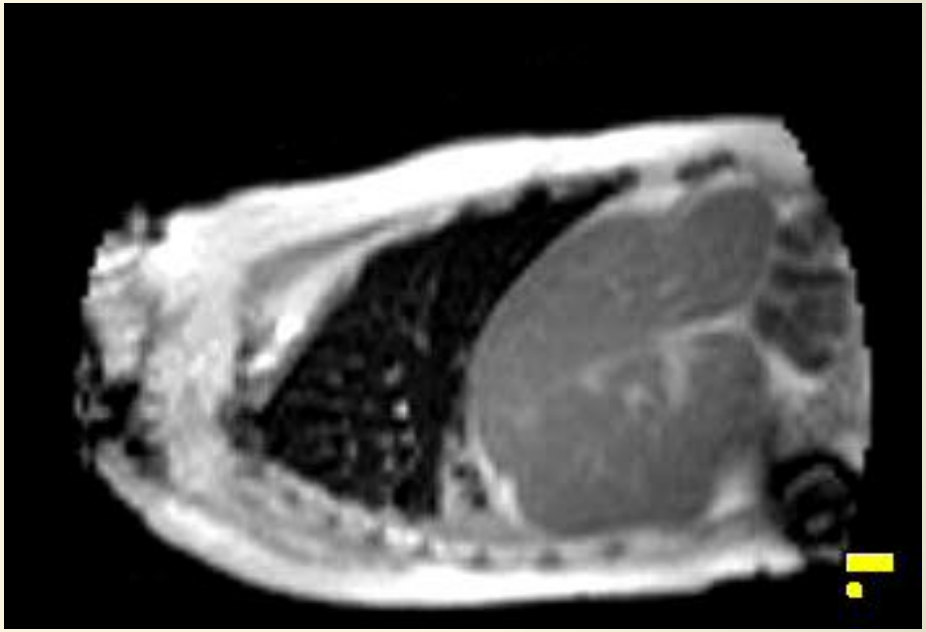
IAP > 15mmHg:

- ✓ spadku statycznych i dynamicznych wartości spirometrycznych *(decrease in static and dynamic spirometry variables),*
- ✓ spadku pojemności życiowej *(decrease in vital capacity)*
- ✓ spadku czynnościowej pojemności zalęgającej *(decrease in residual capacity),*
- ✓ wzrostu ciśnienia opłucnowego *(increase in pleural pressure),*
- ✓ wzrostu szczytowego ciśnienia wdechowego *(increase in Peak pressure),*
- ✓ wzrostu ciśnienia wdechowego *(increase in inspiratory pressure),*
- ✓ rozwoju ognisk niedodmy, której efektem jest hipoksja i hiperkapnia tętnicza *(increase in collapsed lung resulted in hypoxia and hypercapnia).*

Wpływ IAH na układ oddechowy *(an effect of IAH on respiratory system)*



IAP = 6 mmHg



IAP = 20 mmHg

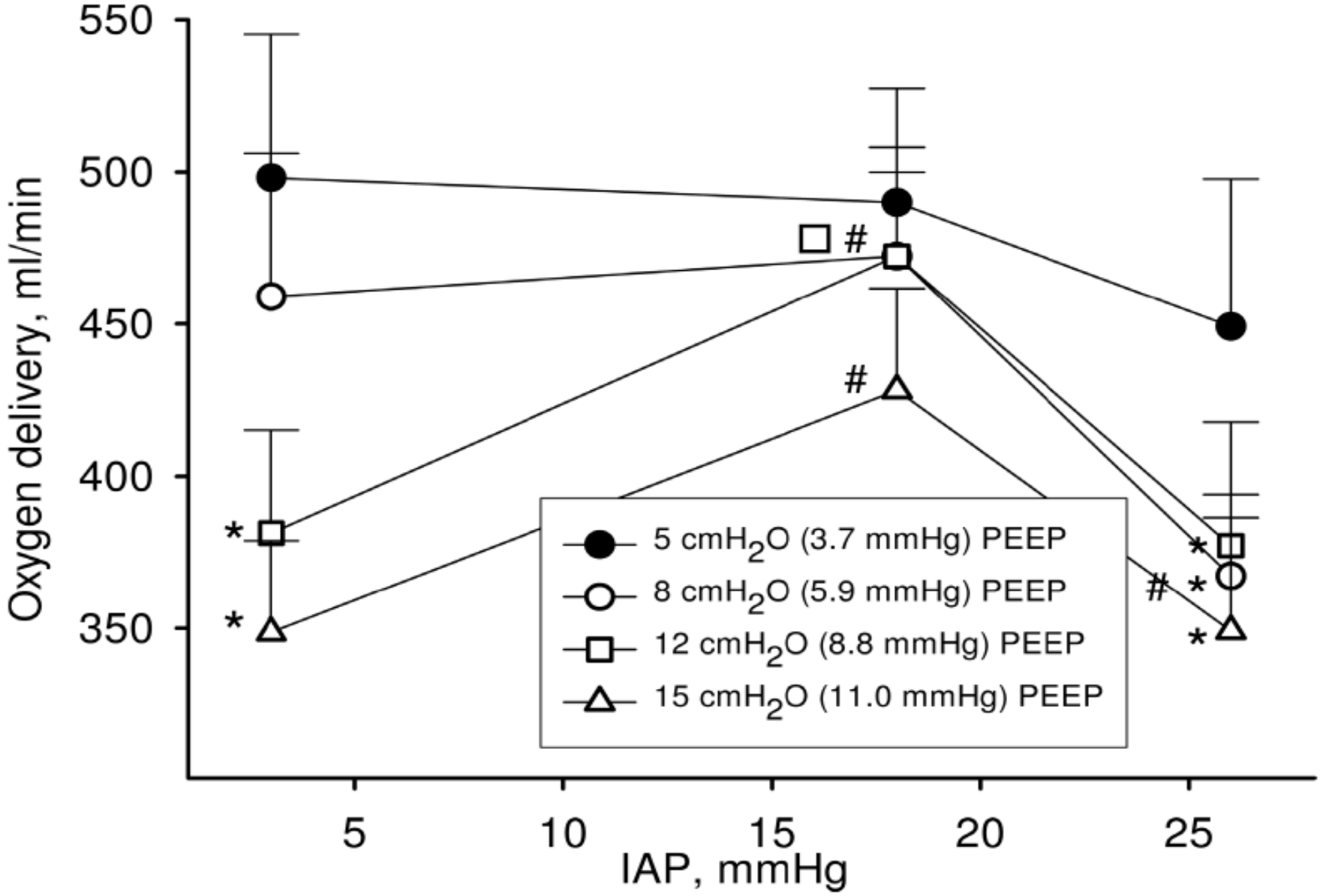


Figure 3 Influence of intra-abdominal pressure and positive end-expiratory pressure on oxygen delivery. Oxygen delivery in ml/min in function of different levels of intra-abdominal pressures (IAP) (3 mmHg (baseline), 18 mmHg (grade II intra-abdominal hypertension), and 26 mmHg (grade IV intra-abdominal hypertension)) at different positive end-expiratory pressures (PEEP). Mean and SE are shown. ANOVA and *post hoc* Student-Newman-Keuls were used for statistical testing *, $P < 0.05$ within an IAP setting vs. the corresponding value at 5 cmH_2O PEEP. #, $P < 0.05$ within a PEEP setting vs. the corresponding value at baseline IAP. For clarification additional symbol is added where necessary.

Wpływ IAH na nerki (an effect of IAH on kidney)

IAH 15 – 20 mmHg:

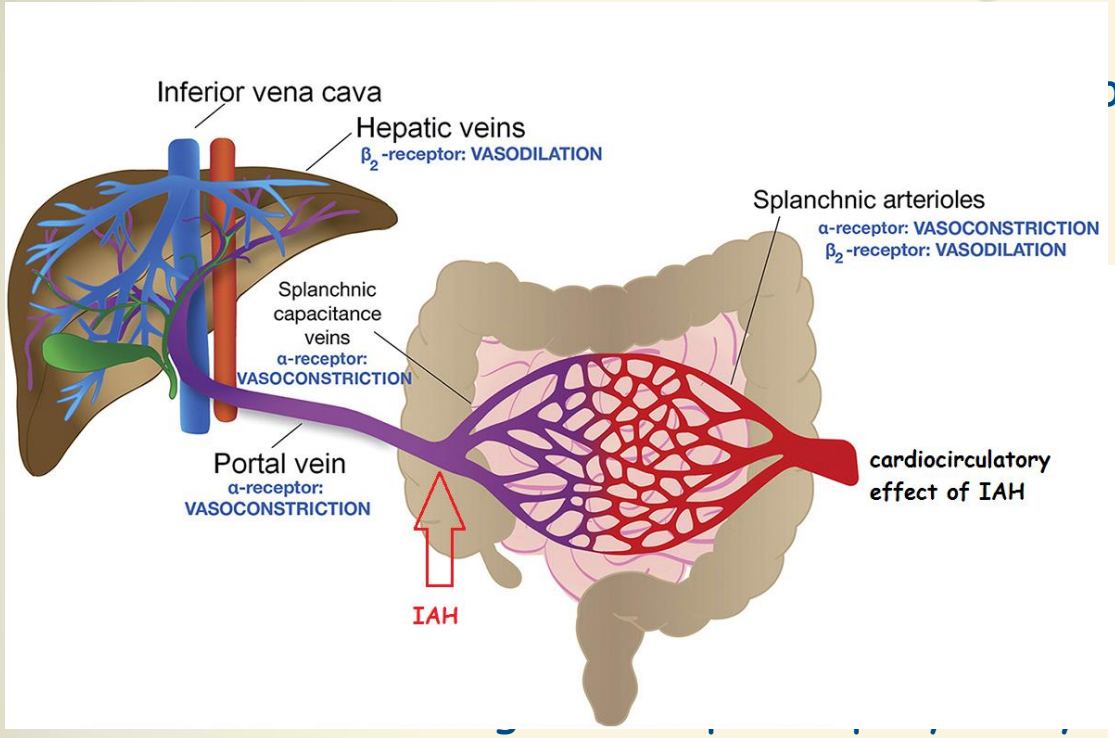
- ✓ redukcji przepływu nerkowego oraz filtracji kłębuszkowej, a co za tym idzie spadku diurezy do wartości 02 – 05ml/kg mc/godz., a następnie anuriii *(reduction in renal filtration pressure (2 – 5 ml/kg bw), which leads to oliguria followed by anuria),*

- ✓ wzrostu naczyniowych oporów w tętnicach nerkowych *(an increase in renal vascular resistance),*

    - ✓ IAH = 20mmHg - 500%
    - ✓ IAH = 40mmHg – 1500%
    - ✓ IAP = 30 mmHg całkowite zahamowanie diurezy *(anuria),*

- ✓ martwicy kłębuszków nerkowych
  (glomerular necrosis which leads to renal insufficiency).

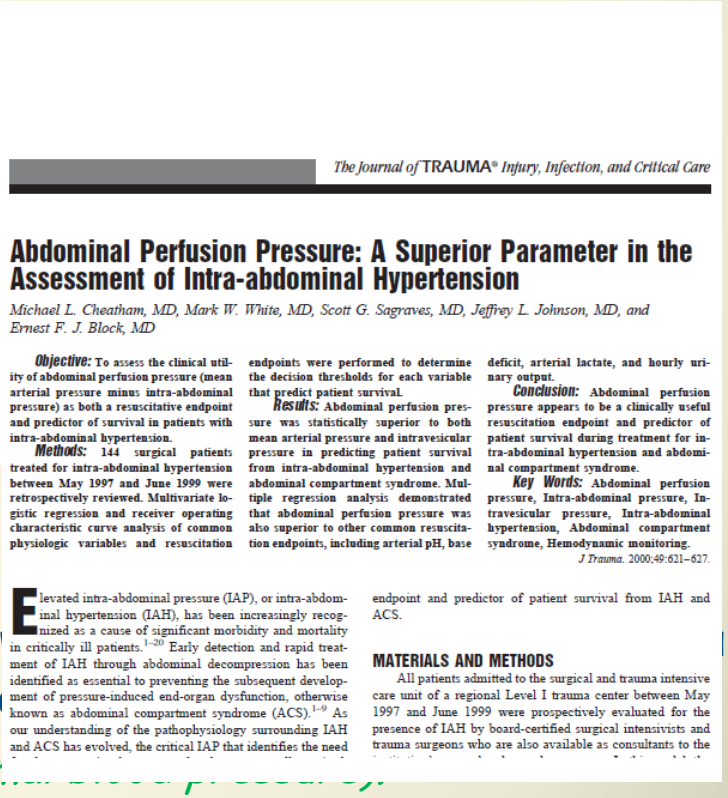
Wpływ IAH na narządy jamy brzusznej
(an effect of IAH on abdominal organs)



owej

(reduction in intestine motility),

- ✓ IAH > 15 mmHg - 40% spadek perfuzji
  prawidłowych wartości ciśnienia tętniczeg
  (reduction in hepatic blood flow, even with nor



The Journal of TRAUMA® Injury, Infection, and Critical Care

Abdominal Perfusion Pressure: A Superior Parameter in the Assessment of Intra-abdominal Hypertension

Michael L. Cheatham, MD, Mark W. White, MD, Scott G. Sagraves, MD, Jeffrey L. Johnson, MD, and Ernest F. J. Block, MD

Objective: To assess the clinical utility of abdominal perfusion pressure (mean arterial pressure minus intra-abdominal pressure) as both a resuscitative endpoint and predictor of survival in patients with intra-abdominal hypertension.

Methods: 144 surgical patients treated for intra-abdominal hypertension between May 1997 and June 1999 were retrospectively reviewed. Multivariate logistic regression and receiver operating characteristic curve analysis of common physiologic variables and resuscitation endpoints were performed to determine the decision thresholds for each variable that predict patient survival.

Results: Abdominal perfusion pressure was statistically superior to both mean arterial pressure and intravesicular pressure in predicting patient survival from intra-abdominal hypertension and abdominal compartment syndrome. Multiple regression analysis demonstrated that abdominal perfusion pressure was also superior to other common resuscitation endpoints, including arterial pH, base deficit, arterial lactate, and hourly urinary output.

Conclusion: Abdominal perfusion pressure appears to be a clinically useful resuscitation endpoint and predictor of patient survival during treatment for intra-abdominal hypertension and abdominal compartment syndrome.

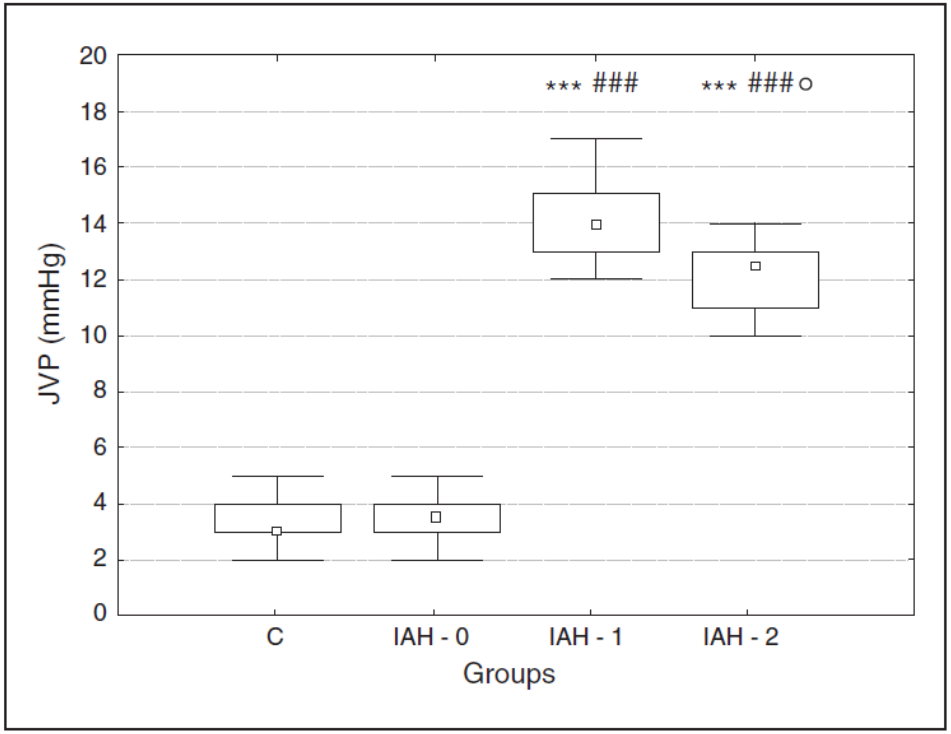
Key Words: Abdominal perfusion pressure, Intra-abdominal pressure, Intravesicular pressure, Intra-abdominal hypertension, Abdominal compartment syndrome, Hemodynamic monitoring.

J Trauma. 2000;49:621–627.

Elevated intra-abdominal pressure (IAP), or intra-abdominal hypertension (IAH), has been increasingly recognized as a cause of significant morbidity and mortality in critically ill patients.[1–20] Early detection and rapid treatment of IAH through abdominal decompression has been identified as essential to preventing the subsequent development of pressure-induced end-organ dysfunction, otherwise known as abdominal compartment syndrome (ACS).[1–9] As our understanding of the pathophysiology surrounding IAH and ACS has evolved, the critical IAP that identifies the need endpoint and predictor of patient survival from IAH and ACS.

MATERIALS AND METHODS

All patients admitted to the surgical and trauma intensive care unit of a regional Level I trauma center between May 1997 and June 1999 were prospectively evaluated for the presence of IAH by board-certified surgical intensivists and trauma surgeons who are also available as consultants to the



Magnesium Research 2012; **25** (2): 89-98 ORIGINAL ARTICLE

Increase in intra-abdominal pressure raises brain venous pressure, leads to brain ischaemia and decreases brain magnesium content

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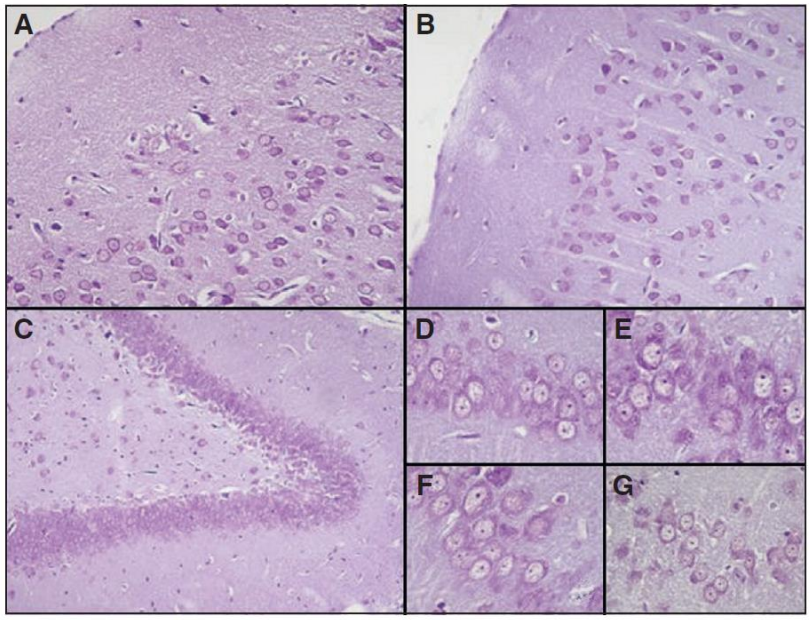
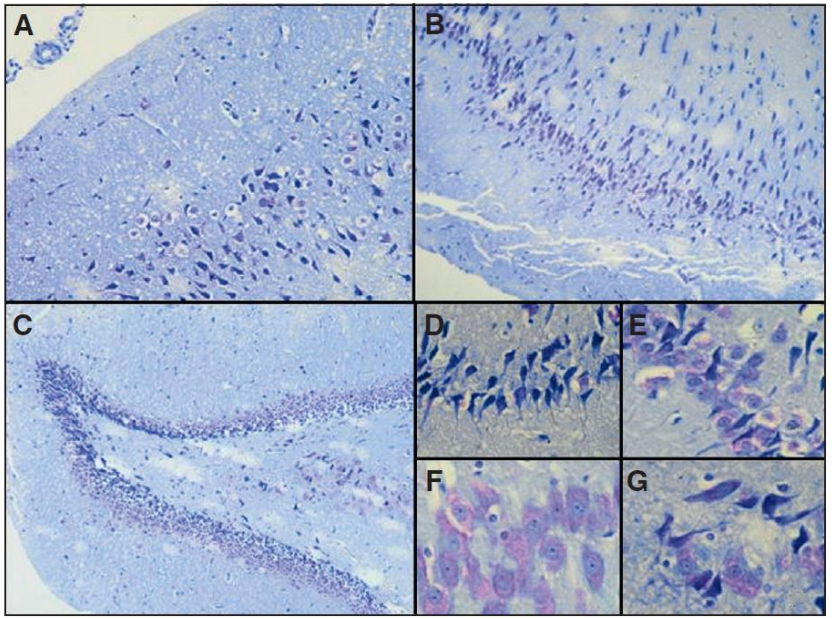
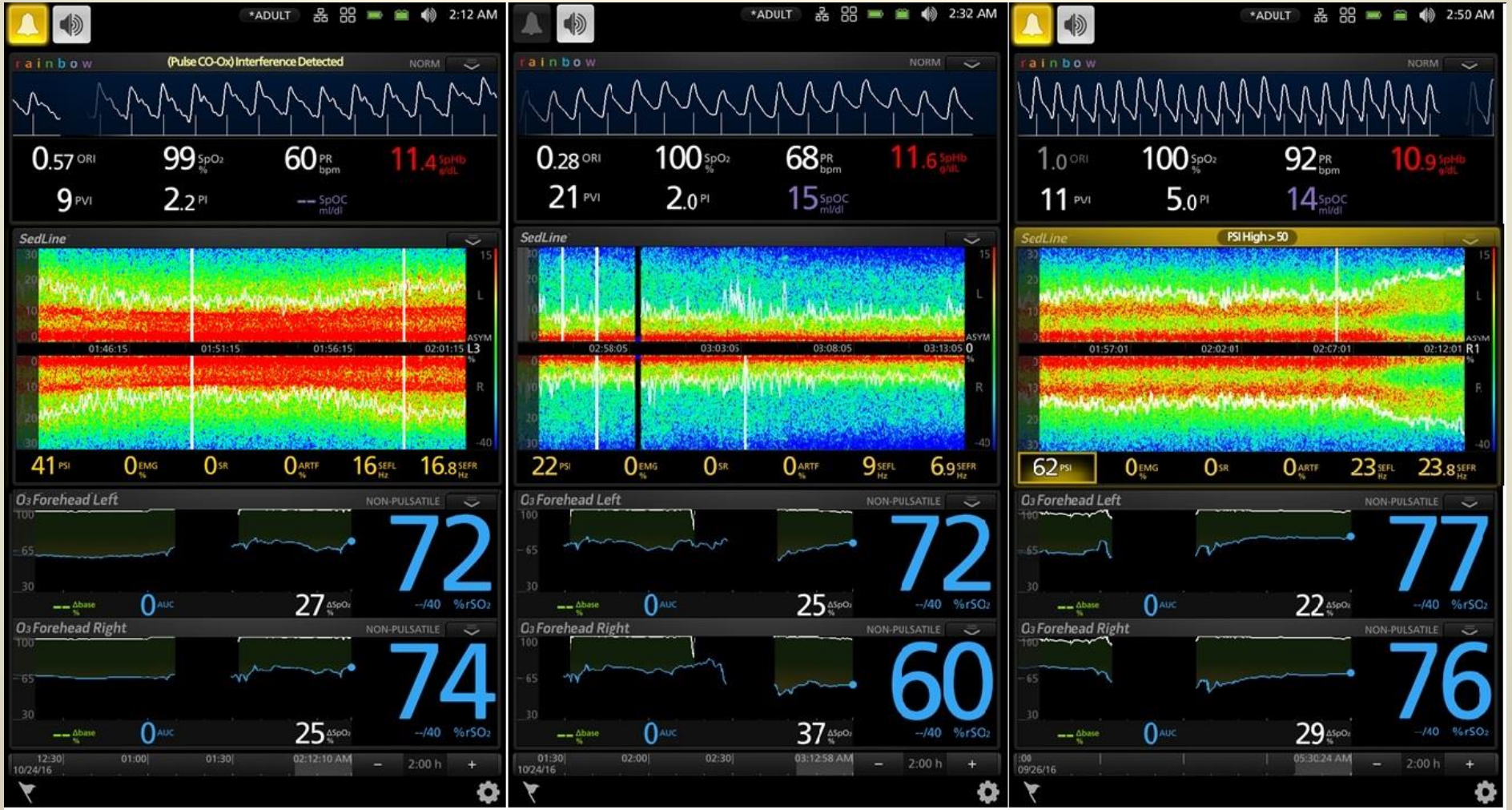
Abstract. *Background.* Intra-abdominal hypertension (IAH) may increase brain venous pressure, which may lead to brain injury. The aim of the present study was to analyse the effect of IAH on brain venous pressure and brain total and ionised magnesium (tMg and iMg), calcium (Ca) and zinc (Zn) contents in rats. *Material and methods.* Forty four adult Wistar rats were examined. Animals were divided into two groups: control, and IAH: rats with intra-abdominal pressure (IAP) elevated to 25 mmHg. IAP was measured directly in the abdominal cavity. After retrograde cannulation of the jugular vein, the jugular venous pressure (JVP) was measured as the brain venous pressure. JVP and IAP were noted after induction of anaesthesia, immediately following induction of IAH and 90 min after induction of IAH. In all rats, brains were removed for biochemical and histological analysis. *Results.* Biochemical analysis was performed in 30 rats, histological visualisation in 14. IAP elevated to 25 mmHg increased JVP in the IAH group. After 90 min, JVP decreased; however, its value was still higher compared with pre-IAH. In the IAH group, tMg and iMg were significantly lower than in the control group. Moreover, Ca and Zn levels were higher in the IAH group compared with the control group. The histological examination showed changes indicative of ischaemic neuronal cell stress. *Conclusions.* Firstly, increase in IAP elevates JVP. Secondly, raised JVP decreases tMg and iMg. Thirdly, raised JVP increases the Ca and Zn content in the rat brain. Fourthly, IAH leads to changed characteristics of brain ischaemia.

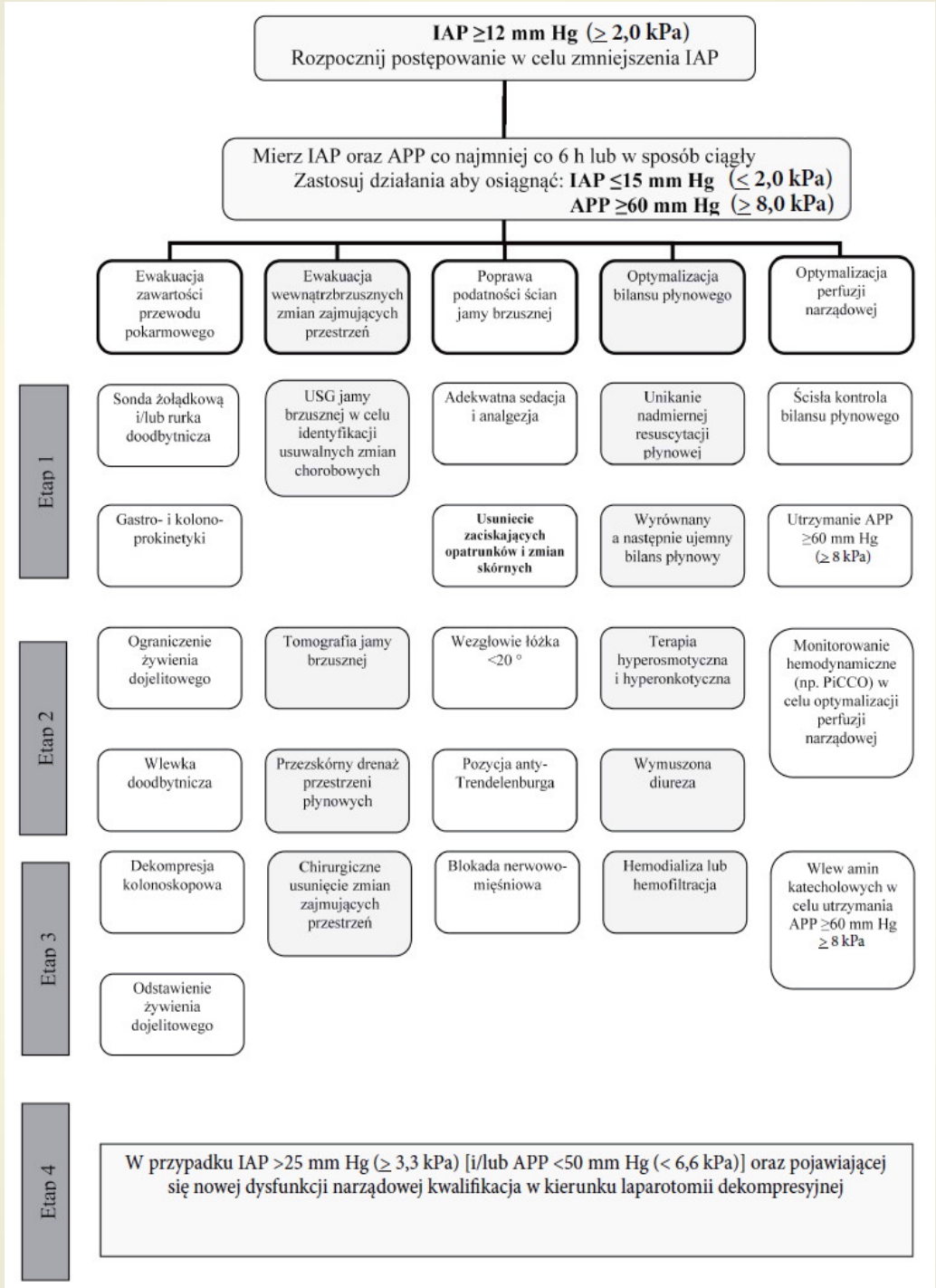
Key words: intra-abdominal pressure, intra-abdominal hypertension, brain ischaemia, brain venous hypertension, magnesium, calcium, zinc





Wpływ IAH na ośrodkowy układ nerwowy w praktyce klinicznej *(effect of IAH on central nervous system in clinical practice)*




IAP ≥12 mm Hg (≥ 2,0 kPa)
Rozpocznij postępowanie w celu zmniejszenia IAP
Mierz IAP oraz APP co najmniej co 6 h lub w sposób ciągły
Zastosuj działania aby osiągnąć: IAP ≤15 mm Hg (≤ 2,0 kPa)
APP ≥60 mm Hg (≥ 8,0 kPa)
Ewakuacja zawartości przewodu pokarmowego
Ewakuacja wewnątrzbrzusznych zmian zajmujących przestrzeń
Poprawa podatności ścian jamy brzusznej
Optymalizacja bilansu płynowego
Optymalizacja perfuzji narządowej
Etap 1
Sonda żołądkową i/lub rurka doodbytnicza
USG jamy brzusznej w celu identyfikacji usuwalnych zmian chorobowych
Adekwatna sedacja i analgezja
Unikanie nadmiernej resuscytacji płynowej
Ścisła kontrola bilansu płynowego
Gastro- i kolono-prokinetyki
Usunięcie zaciskających opatrunków i zmian skórnych
Wyrównany a następnie ujemny bilans płynowy
Utrzymanie APP ≥60 mm Hg (≥ 8 kPa)
Etap 2
Ograniczenie żywienia dojelitowego
Tomografia jamy brzusznej
Wezgłowie łóżka <20 °
Terapia hyperosmotyczna i hyperonkotyczna
Monitorowanie hemodynamiczne (np. PiCCO) w celu optymalizacji perfuzji narządowej
Wlewka doodbytnicza
Przezskórny drenaż przestrzeni płynowych
Pozycja anty-Trendelenburga
Wymuszona diureza
Etap 3
Dekompresja kolonoskopowa
Chirurgiczne usunięcie zmian zajmujących przestrzeń
Blokada nerwowo-mięśniowa
Hemodializa lub hemofiltracja
Wlew amin katecholowych w celu utrzymania APP ≥60 mm Hg ≥ 8 kPa
Odstawienie żywienia dojelitowego
Etap 4
W przypadku IAP >25 mm Hg (≥ 3,3 kPa) [i/lub APP <50 mm Hg (< 6,6 kPa)] oraz pojawiającej się nowej dysfunkcji narządowej kwalifikacja w kierunku laparotomii dekompresyjnej

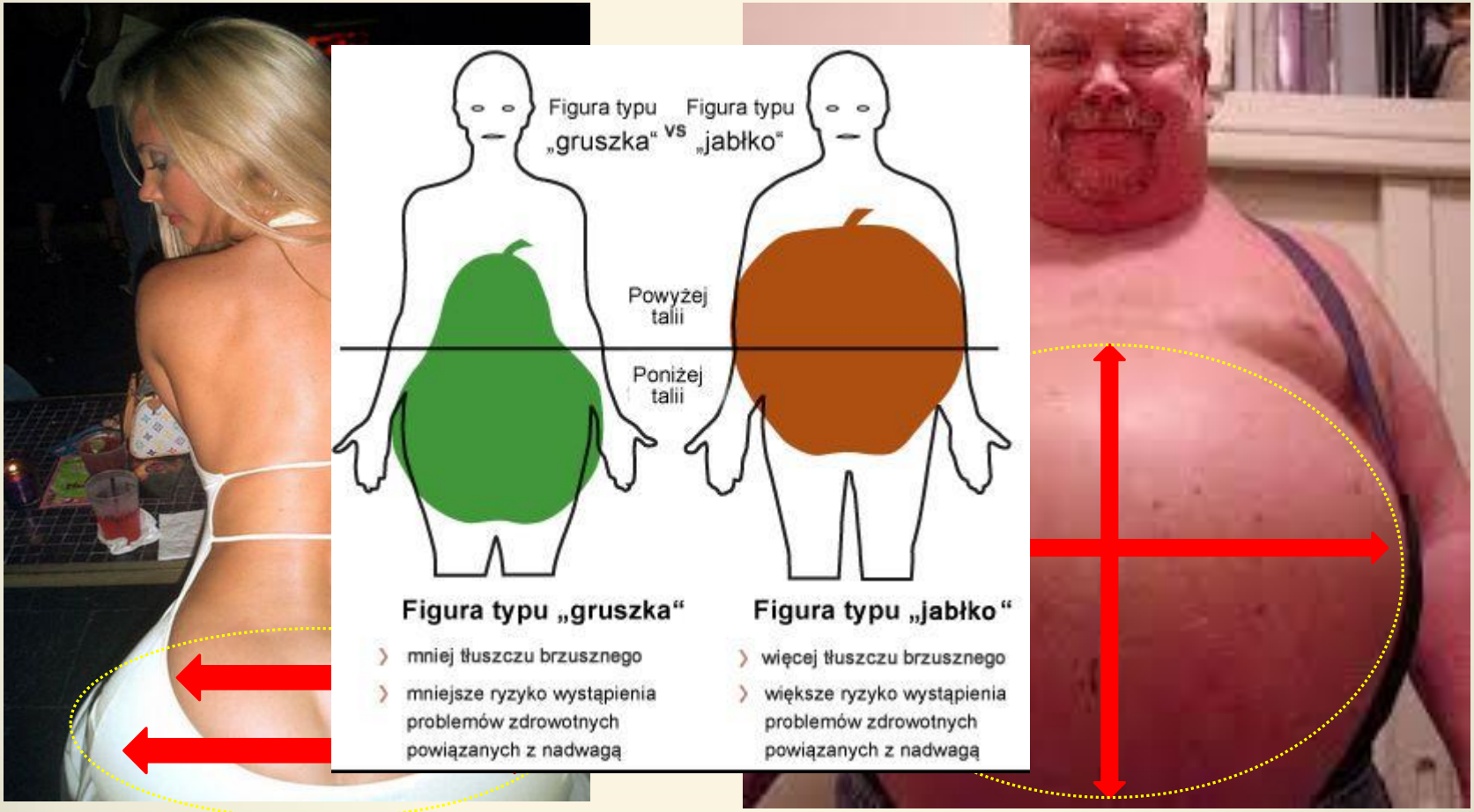
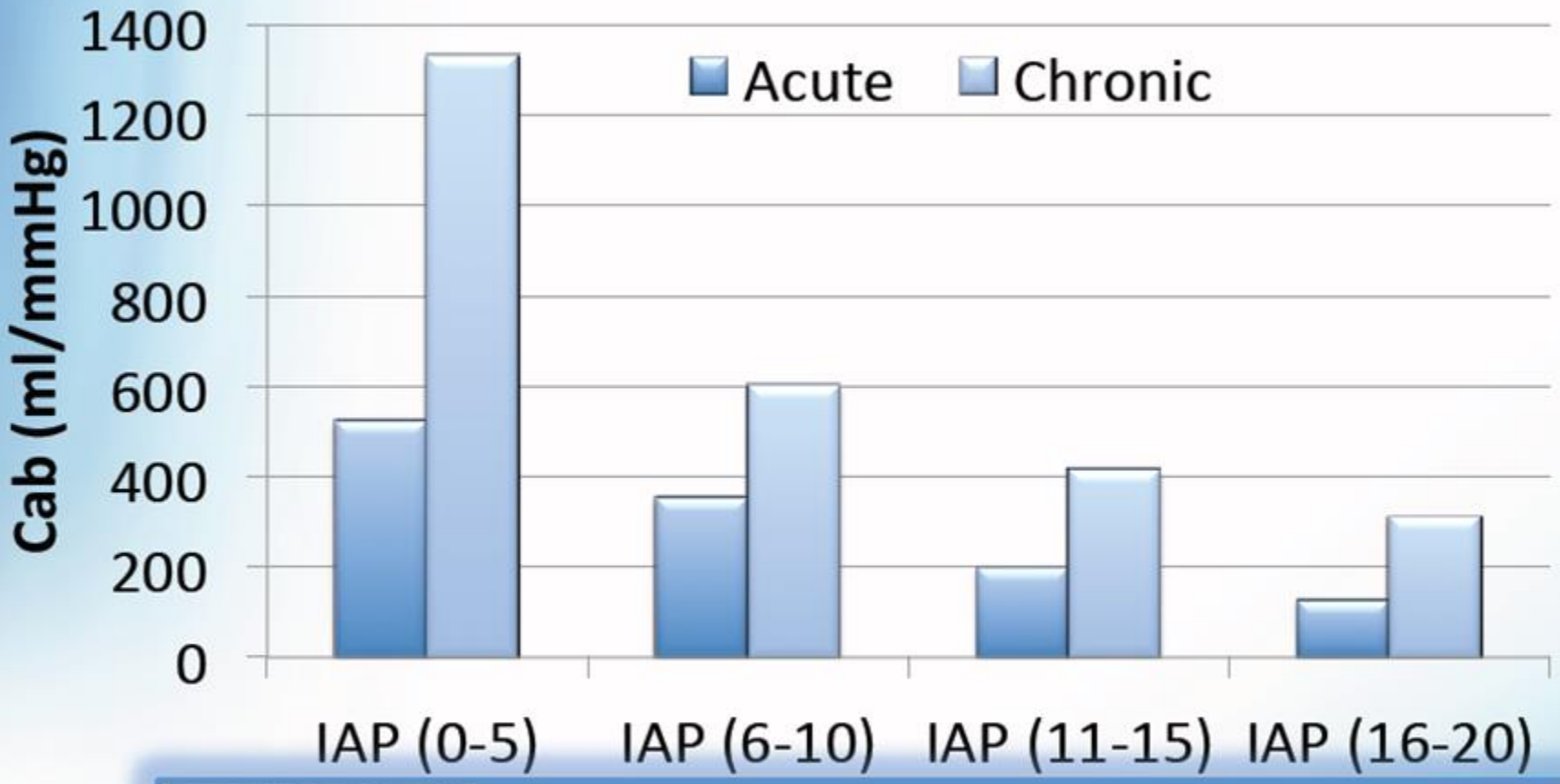


Figura typu „gruszka“ vs Figura typu „jabłko“
Powyżej talii
Poniżej talii
Figura typu „gruszka“
mniej tłuszczu brzusznego
mniejsze ryzyko wystąpienia problemów zdrowotnych powiązanych z nadwagą
Figura typu „jabłko“
więcej tłuszczu brzusznego
większe ryzyko wystąpienia problemów zdrowotnych powiązanych z nadwagą

$$C_{ab} = \Delta IAV / \Delta IAP$$

Normal Cab is around 375 mL/mmHg (240-635)

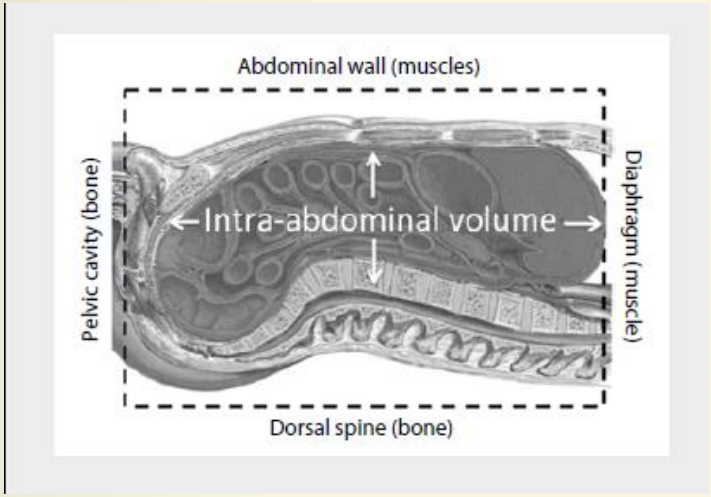
Cab in relation to baseline IAP

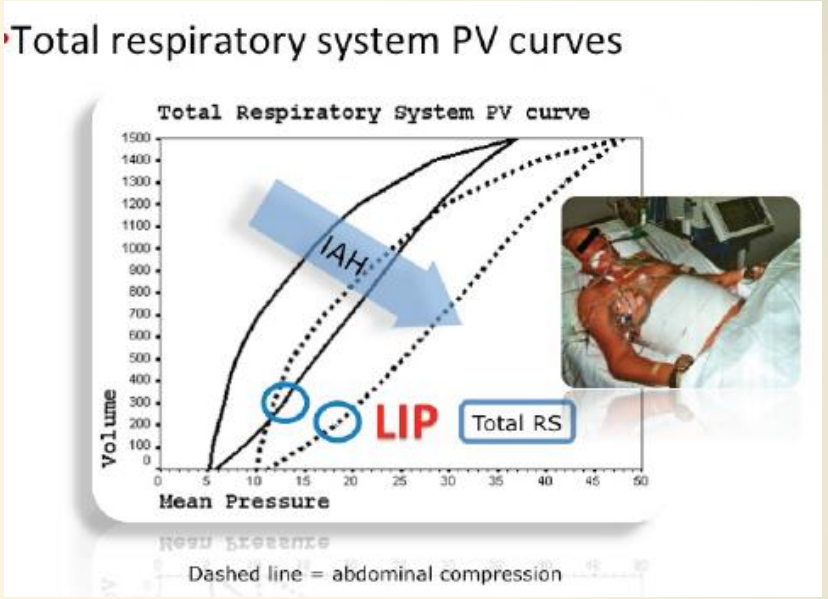


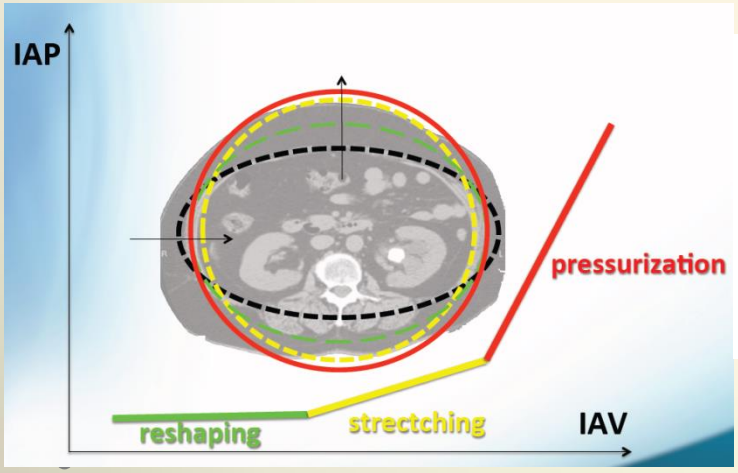
The higher the IAP, the lower the Cab

Podatności jamy brzusznej *(abdominal compliance)*

zespół ciasnoty śródbrzusznej *(ACS)*





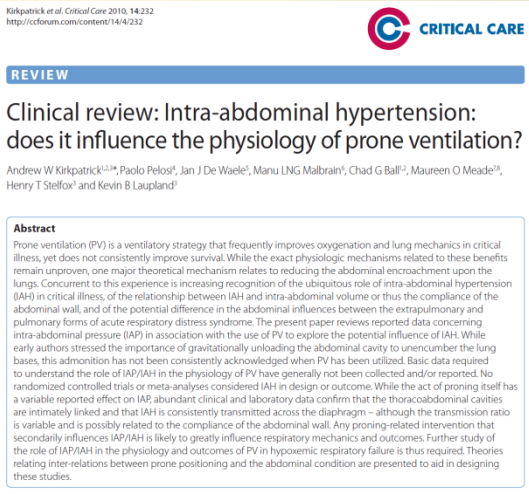


VIA MEDICA REVIEWS

Anaesthesiology Intensive Therapy 2014, vol. 46, no 5, 406–432 ISSN 1642–5758 DOI: 10.5603/AIT.2014.0063 www.ait.viamedica.pl

The role of abdominal compliance, the neglected parameter in critically ill patients — a consensus review of 16. Part 2: measurement techniques and management recommendations

Manu L.N.G. Malbrain[1], Inneke De laet[1,2], Jan J. De Waele[2], Michael Sugrue[3], Alexander Schachtrupp[4], Juan Duchesne[5], Gabrielle Van Ramshorst[6], Bart De Keulenaer[7], Andrew W. Kirkpatrick[8], Siavash Ahmadi-Noorbakhsh[9], Jan Mulier[10], Paolo Pelosi[11], Rao Ivatury[12], Francisco Pracca[13], Marcelo David[14], Derek J. Roberts[15]



Kirkpatrick *et al. Critical Care* 2010, **14**:232 http://ccforum.com/content/14/4/232

CRITICAL CARE

REVIEW

Clinical review: Intra-abdominal hypertension: does it influence the physiology of prone ventilation?

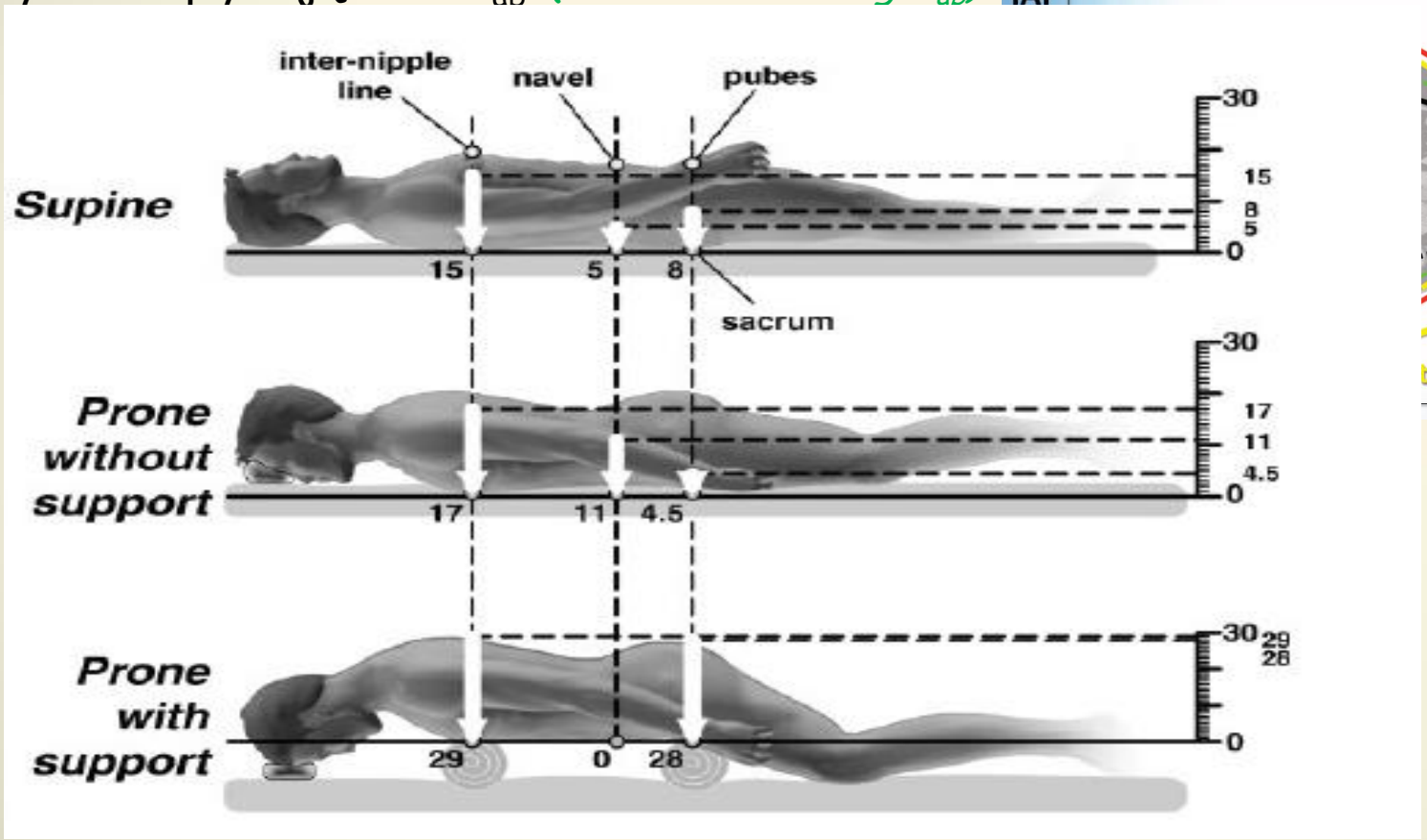
Andrew W Kirkpatrick[1,2,3*], Paolo Pelosi[4], Jan J De Waele[5], Manu LNG Malbrain[6], Chad G Ball[1,2], Maureen O Meade[7,8], Henry T Stelfox[3] and Kevin B Laupland[3]

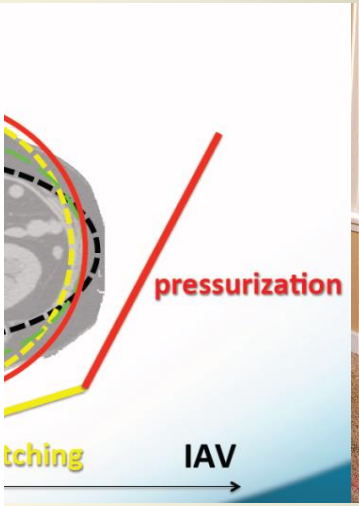
Abstract

Prone ventilation (PV) is a ventilatory strategy that frequently improves oxygenation and lung mechanics in critical illness, yet does not consistently improve survival. While the exact physiologic mechanisms related to these benefits remain unproven, one major theoretical mechanism relates to reducing the abdominal encroachment upon the lungs. Concurrent to this experience is increasing recognition of the ubiquitous role of intra-abdominal hypertension (IAH) in critical illness, of the relationship between IAH and intra-abdominal volume or thus the compliance of the abdominal wall, and of the potential difference in the abdominal influences between the extrapulmonary and pulmonary forms of acute respiratory distress syndrome. The present paper reviews reported data concerning intra-abdominal pressure (IAP) in association with the use of PV to explore the potential influence of IAH. While early authors stressed the importance of gravitationally unloading the abdominal cavity to unencumber the lung bases, this admonition has not been consistently acknowledged when PV has been utilized. Basic data required to understand the role of IAP/IAH in the physiology of PV have generally not been collected and/or reported. No randomized controlled trials or meta-analyses considered IAH in design or outcome. While the act of proning itself has a variable reported effect on IAP, abundant clinical and laboratory data confirm that the thoracoabdominal cavities are intimately linked and that IAH is consistently transmitted across the diaphragm – although the transmission ratio is variable and is possibly related to the compliance of the abdominal wall. Any proning-related intervention that secondarily influences IAP/IAH is likely to greatly influence respiratory mechanics and outcomes. Further study of the role of IAP/IAH in the physiology and outcomes of PV in hypoxemic respiratory failure is thus required. Theories relating inter-relations between prone positioning and the abdominal condition are presented to aid in designing these studies.

Spadek podatności jamy brzusznej *(decline in C_{ab})*

Czynniki wpływające na C_{ab} *(factors affecting C_{ab})*:

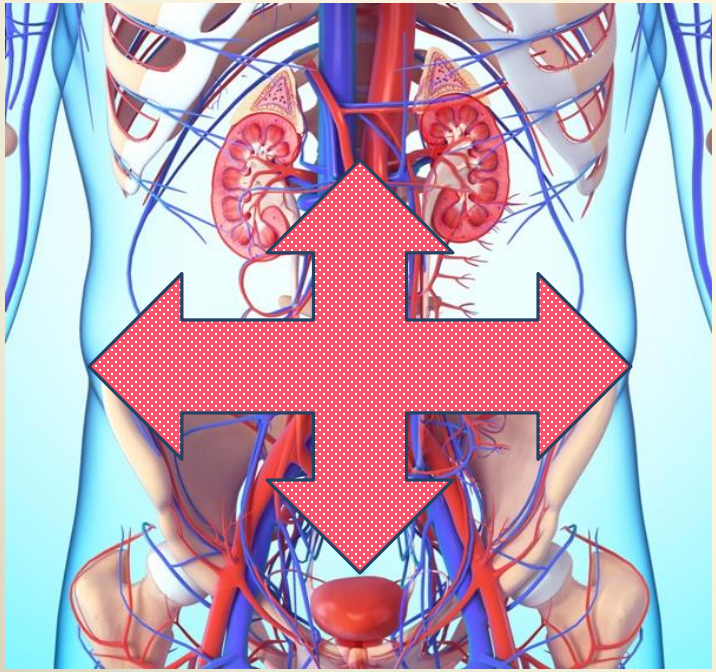


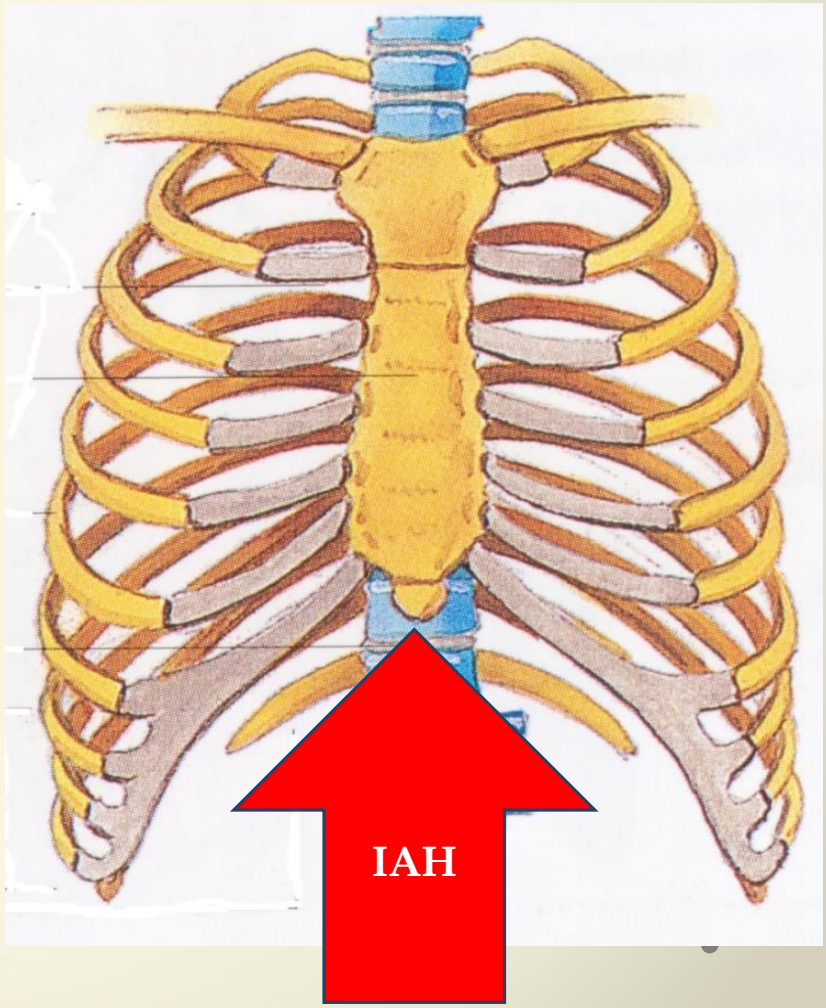


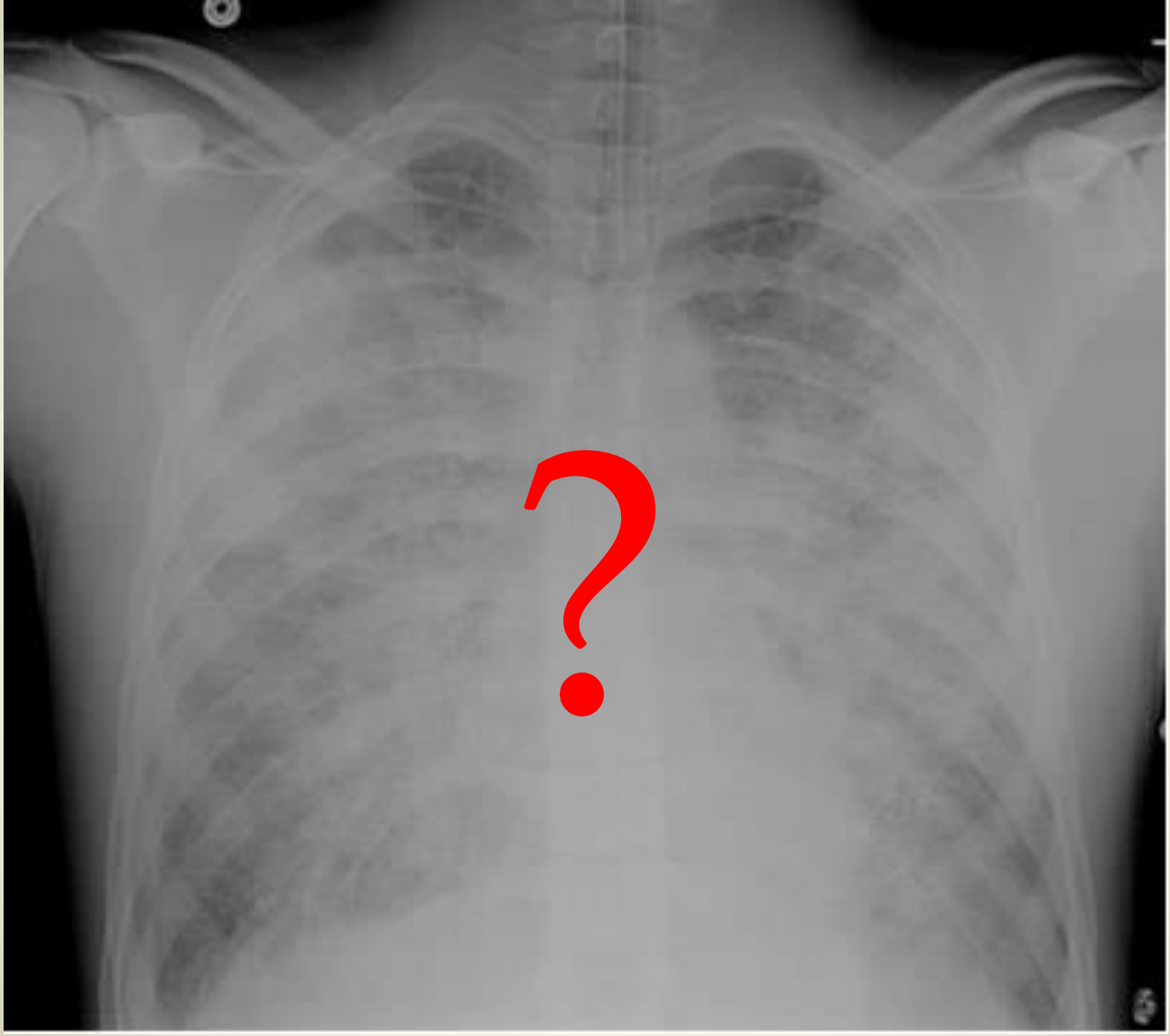
choroby narządów jamy brzusznej *(intra-abdominal diseases).*

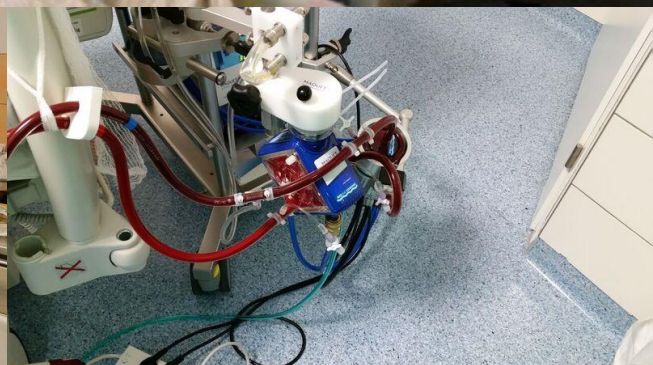
Spadek podatności jamy brzusznej

Oddychanie *(respiration)*:
wartości spirometryczne ↓*(spirometric variables)*,
PaO_2 ↓,
$PaCO_2$ ↑,
Qs/Qt ↑,
podatność płuc ↓ *(pulmonary compliance)*,
ciśnienie wewnątrz klatki piersiowej ↑
(intra-thoracic pressure).

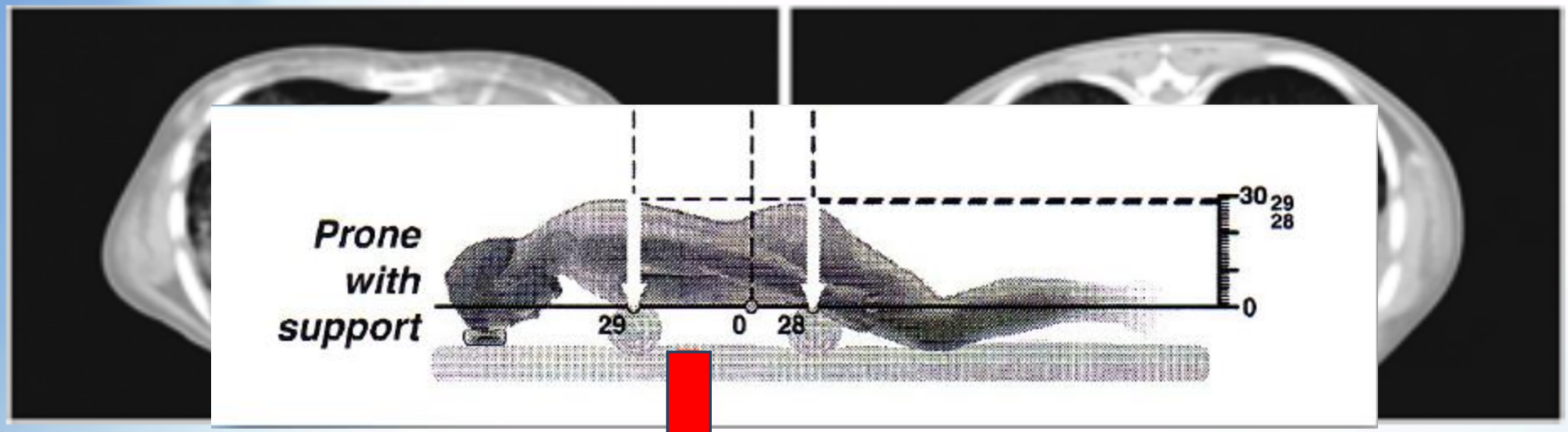








Prone ventilation seems more physiologic



supine

prone

- Gattinoni, CMAJ 2008;178:1174-1176
- Gattinoni, N Engl J Med 2001;345:568-573

Better V/Q matching
Greater dorsal lung recruitment compared to ventral
Overall increase in end-expiratory lung volumes
EELV due to diagrammatic release

Podatność jamy brzusznej a podatność płuc – alternatywa dla pozycji „prone"

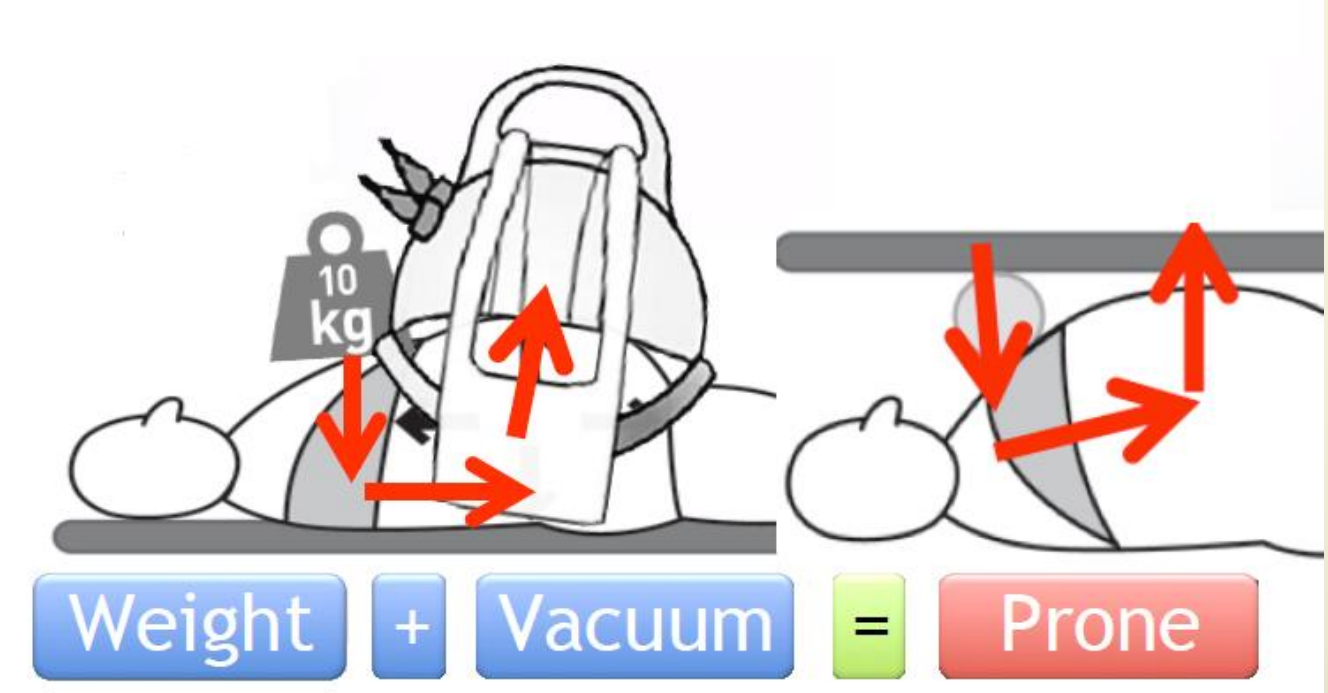
(Abdominal compliance and pulmonary compliance alternative for prone position)

zwiększenie Cab + zmniejszenie podatności klatki piersiowej

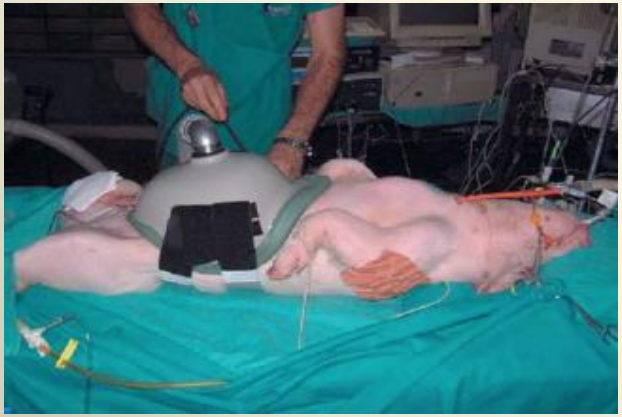
=

rekrutacja obszarów przypodstawnych płuc

(increase in Cab + reduction in thoracic compliance = pulmonary recruitment)



IAP = 14 mmHg



Intensive Care Med (2005) 31:105–111
DOI 10.1007/s00134-004-2483-2

ORIGINAL

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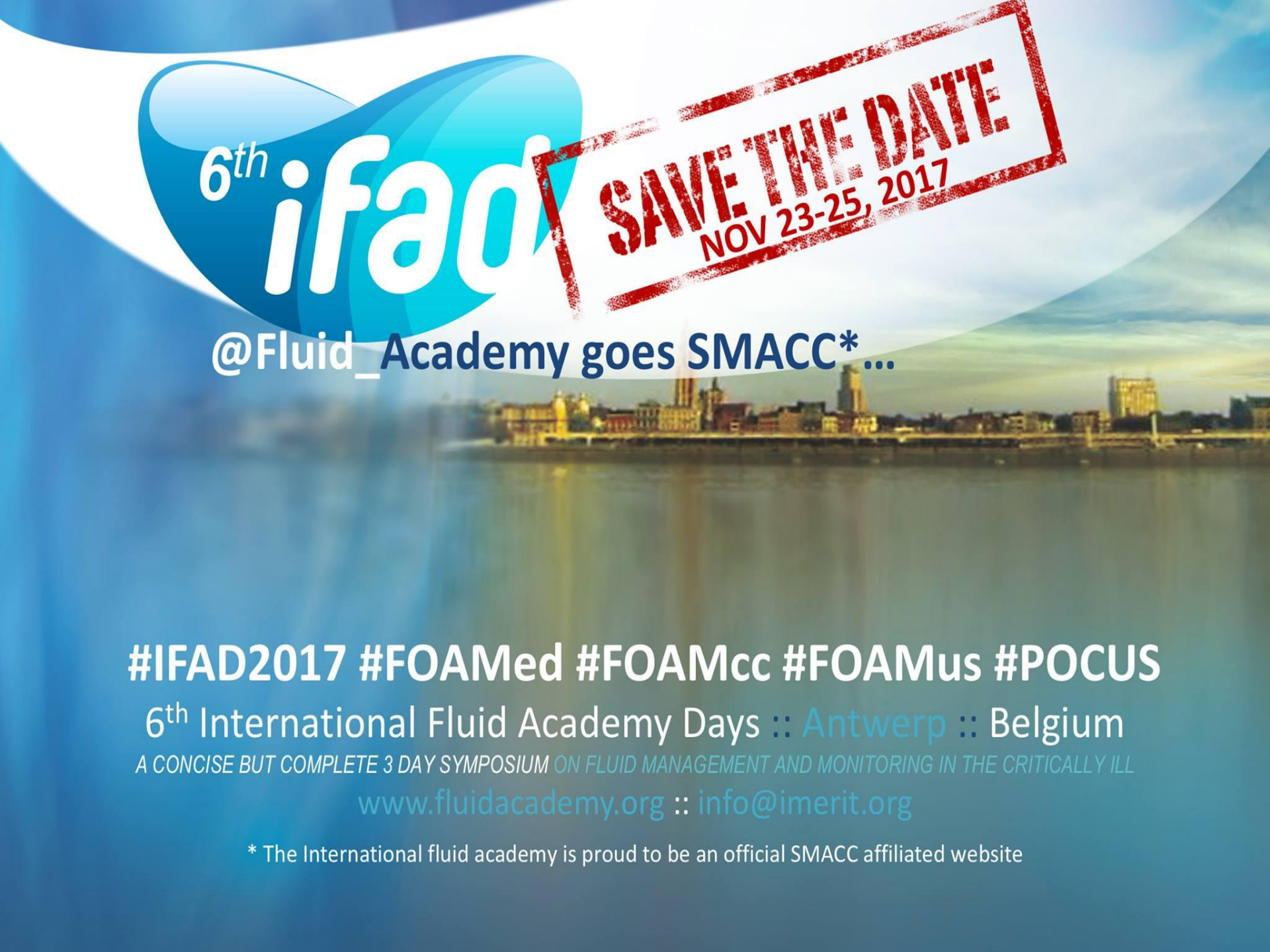
Effects of continuous negative extra-abdominal pressure on cardiorespiratory function during abdominal hypertension: an experimental study

IAP = 9 mmHg

Key words: Expiratory abdominal compression, Tidal volume, Prolonged mechanical ventilation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