Journal reading

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EMJ

Impact of a modified Valsalva manoeuvre in the termination of paroxysmal supraventricular tachycardia

S Walker and P Cutting

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Introduction

- There is no standardised, universally accepted 'gold-standard' Valsalva manoeuvre used in clinical practice.
- A recent paper by Smith et al does propose a gold standard Valsalva manoeuvre ：
  (40 mm Hg, 15 s, supine position) for use in the prehospital setting *based on an electronic database literature review.*

RATIONALE FOR THE MODIFICATION TO THE VALSALVA TECHNIQUE

- Oxford Handbook series and Advanced Life Support Group manuals give little or no information as to how exactly to perform the manoeuvre.
- First, as Taylor and Wong observe, doctors frequently incorrectly instruct patients on how to perform the most effective Valsalva manoeuvre.
- Second, there is a general underuse of Valsalva and an over-reliance on adenosine (despite high reversion rates) has a high side effect & cost implications.

Data Analysis

- **Retrospective case review**
- **Cross-sectional study/questionnaire**
- **Prospective observational study**

Study Design

- The patient was in a Trendelenberg position.
- A new 20-cm precut length of suction tubing was attached to a designated aneroid pressure gauge (figure 1), through which the patient blow as hard as possible, aiming > 40 mm Hg for at least 15 seconds.
- Following pressure release, the patient was instructed to relax.
- If the Valsalva was not initially successful, the manoeuvre was repeated 3 times with a 1-min rest between attempts.
- If Valsalva did not result in reversion, patients received adenosine as guidelines.

Figure 1 Aneroid pressure gauge and attached length of suction tubing.

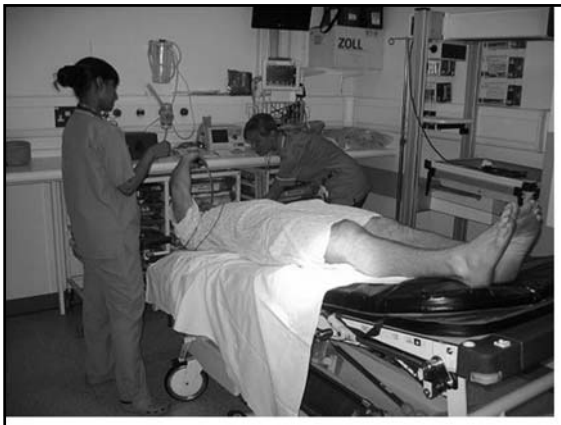

Figure 2 Demonstration of modified Valsalva manoeuvre.

Table 1 Summary of research findings on cardiovascular response to patient position

Study finding	Author (Reference)
A significant reduction (20% vs 54%) in the efficacy of Valsalva at reverting paroxysmal SVT when standing compared with supine in a group of 35 patients with inducible and sustained SVT.	Mehta *et al*[8]
Sixty-five healthy subjects in sinus rhythm performing Valsalva in a series of positions studied established a significant increase in mean postmanoeuvre R-R interval (and therefore decrease in heart rate) when supine compared with sitting or semirecumbent.	Wong *et al*[11]
Studied the effects of respiration and posture on 11 patients with known paroxysmal SVT and demonstrated that increasing body dependency to a head-down position alone increased blood pressure sufficiently to revert 8 out of 11 patients (albeit in a highly controlled laboratory environment).	Waxman *et al*[12]
Compared baroreflex sensitivity with postural changes in 14 healthy subjects. A significant decrease in heart rate during the fourth phase of Valsalva, a significant increase in cardiac vagal activity and a decrease in sympathetic activation when comparing head-up to a head-down position were observed.	Kardos *et al*[13]
Examined the effects of head-down tilt on 17 patients with paroxysmal SVT and concluded that it conferred no additional benefit (Valsalva method used was asking the patients to 'bear-down' for 5 s).	Ornato *et al*[10]

Table 2 Summary of responses to departmental Valsalva questionnaire

Summary of responses Valsalva questionnaire		
Self-rated success rate at reversion	20% mean (range 0%–100%)	(Mean for Consultant respondents—12%)
Position for Valsalva manoeuvre	Sitting Sitting forward Semi-recumbent Supine Mixed positions	14 1 4 8 3
Duration of attempt	Mean—10 s	(4 respondents—as long as possible)
Adjunct used	Syringe Tubing None (closed mouth)	28 1 1

Table 3 Exclusion criteria for modified Valsalva manoeuvre study

Exclusion criteria
Glasgow Coma Score <15/15
Evidence of new heart failure
Cardiac-sounding chest pain
Systolic blood pressure <90 mm Hg
Inability, through any reason, for the patient (or parent in the case of paediatric cases) to provide informed consent

Table 4 Breakdown of results of modified Valsalva manoeuvre

	Success with modified Valsalva	No success with modified Valsalva	Significance level (p Value)
Number patients	6	13	Not analysed
Male/female	4:2	5:8	0.35
Mean age in years (SD)	42.5 (12.61)	49.7 (17.35)	0.378
Median duration SVT in hours (IQR)	2.25 (5.5)	2 (5.5)	0.77
Median pressure sustained in mm Hg (IQR)	60 (30)	30 (40)	Not analysed
Previous SVT?	4	9	Not analysed
Previous adenosine?	2	6	Not analysed

Significance level for male/female analysed with Fisher's exact test.
Two-tailed t test used for age and exact Mann–Whitney test for duration.

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DISCUSSION

- age ↗ has been described in many studies to be associated with response ↘ to Valsalva manoeuvre.
- In our study, the mean age for the group who reverted < non-reversion group (42.5 vs 49.7 years), although this was not statistically significant.
- It is anticipated that ageing is associated with attenuated autonomic responsiveness.

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DISCUSSION

- In comparison, adenosine has a relatively high side effect profile such as nausea / vomiting, chest pain, headache, flushing and light-headedness, with studies reporting between 22% adverse events in children and 9% - 27% in adults.

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LIMITATIONS

- The principal limitation for a study of this nature is size. It is difficult to draw statistically significant conclusions from the findings.
- Furthermore, this study age range was 27-77 years despite plans to recruit children and adolescents in SVT.
- Anecdotally, our department have reported success with the modified Valsalva manoeuvre in children on occasions since the study has finished.

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CONCLUSIONS

- we would recommend modified Valsalva maneuver to be attempted as the first-line treatment for SVT.
  Head down tilt
  Expiration for ≧15 seconds
  Blow as hard as possible to achieve ≧40 mm Hg
  Repeated up to 3 times if necessary)
- This appears to be safe, simple and more effective as it can be adapted by patients for use at home, has the potential to reduce presentations to hospital, prevent the need for cannulation and decrease drug costs.

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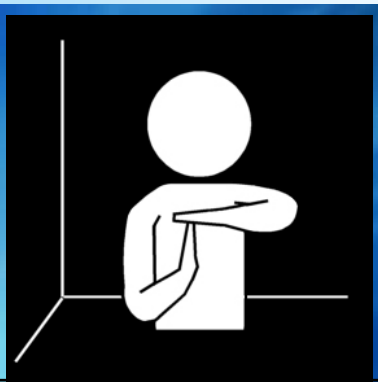
Time Out !



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CLINICAL PRACTICE

Disease Progression in Hemodynamically Stable Patients Presenting to the Emergency Department With Sepsis

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Introduction

- The initial evaluation of patients with suspected infection in the ED is complicated by

(1) the lack of specificity of systemic inflammatory response syndrome (SIRS) criteria for infection.

(2) the heterogeneity of clinical manifestations, including clinical signs and symptoms, site of infection, comorbid conditions, and etiologic microorganisms.

(3) the challenge in rapidly identifying patients most likely to progress to severe illness or death, especially not severely ill at initial evaluation.

Introduction

- The objective of this prospective, multicenter cohort study of patients presenting to the ED with sepsis ：

(1) describe the clinical presentation of sepsis, including types of infection and causative microorganisms

(2) determine the incidence and mortality associated with early progression to septic shock.

(3) evaluate patient characteristics associated with early progression to septic shock.

Adjudication of Infections and Patient Outcomes

- Infection status was categorized ：

(1) infection and causative organism identified

(2) infection, but causative organism not identified.

(3) infection unlikely.

- Causative organisms were classified as ：

(1) ≧1 blood culture(+) for SA, Gram(-) bacteria, Candida albicans, or S. pneumoniae;

(2) ≧2 blood cultures(+) for another single organism

(3) culture(+) from another sterile source (e.g., CSF, joint)

(4) a urinary pneumococcal antigen (+) with clinical compatible with pneumonia.

Study Definitions

- Uncomplicated sepsis was defined as sepsis without evidence of shock or end-organ dysfunction.

- Severe sepsis was defined as two or more SIRS criteria with evidence of end-organ dysfunction.

- Septic shock was defined as tissue hypoperfusion, including hypotension (sBP < 90 mm Hg or MAP < 65 mm Hg) persisting despite initial fluid challenge or a blood lactate concentration ≧4 mmol／L.

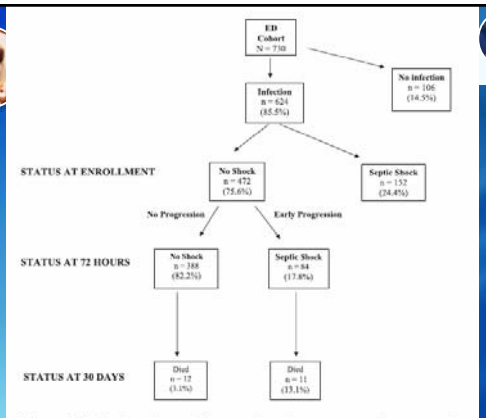

Figure 1. Study cohort. The study cohort at enrollment and subsequent patient outcomes at 72 hours and 30 days.

METHODS

- Data from ：

Community Acquired Pneumonia &Sepsis Outcome Diagnostics (CAPSOD) study, which was a prospective, multicenter National Institutes of Health–sponsored study.

- Study Setting and Population：

A total of 730 patients over 18 years of age were enrolled.

The final data for analysis contained 472 patients with confirmed infection who were not in shock at the time of enrollment in the ED.

Data Analysis

- A multiple variable logistic regression model was performed.
- A Kaplan-Meier survival analysis was performed to evaluate the temporal association of early progression to septic shock with 30-day mortality.
- All analyses were performed using SAS, Version 9.1.2 (SAS Institute, Cary, NC).

RESULTS

Variable	
Age (yr), median (IQR)	52 (44-66)
Sex, *n* (%)	
Male	248 (52.5)
Female	224 (47.5)
Race, *n* (%)	
African American	264 (55.9)
White	186 (39.4)
Other	22 (4.7)
Site, *n* (%)	
Duke, NC	246 (52.1)
Henry Ford, MI	195 (41.3)
Durham, NC	31 (6.6)
Apache II score, median (IQR)	9.0 (5.0–13.5)
Laboratory values, median (IQR)	
Creatinine (mg/dL)	1.1 (0.9–1.6)
Hematocrit (%)	37 (33–41)
Platelet count (×10³/μL)	246 (179–318)
White blood cell count (×10³/μL)	13.2 (9.1–16.7)

IQR = interquartile range.

Comorbidities, *n* (%)	
Alcohol abuse	41 (8.7)
Cancer	37 (7.8)
Chronic renal failure	60 (12.7)
Chronic lung disease	108 (22.9)
Cirrhotic liver disease	7 (1.5)
Diabetes mellitus	132 (28.0)
Drug use	55 (11.7)
Heart failure	41 (8.7)
Hemodialysis	47 (10.0)
Human immunodeficiency virus	7 (1.5)
Smoker	85 (18.0)
Clinical variables, median (IQR)	
Heart rate (beats/min)	109 (96–122)
Respiratory rate (breaths/min)	20 (20–25)
Temperature (°C)	38.3 (37.4–39.0)
Blood pressure, mean arterial, mm Hg (IQR)	87 (78–98)

Table 1
Characteristics of 472 Patients With Sepsis but No Evidence of Shock at the Time of ED Presentation

Table 2
Infection Sites, Causative Microorganisms, and Outcomes for 472 Patients With Sepsis but No Shock at the Time of ED Presentation

	Total	Shock Progression (Within First 72 Hours)	Death (Within 30 Days)
Infection category			
Infection, organism identified	177 (37.5)	38 (21.5)	8 (4.5)
Infection, organism not identified	295 (62.5)	46 (15.6)	15 (5.1)
Total	472 (100)	84 (17.8)	23 (4.9)
Infection source			
Bone	13 (2.8)	4 (30.8)	1 (7.7)
Cardiac	1 (0.2)	0 (0)	0 (0)
Catheter	20 (4.2)	11 (55)	0 (0)
Central nervous system	3 (0.6)	0 (0)	0 (0)
Ear, nose, and throat	16 (3.4)	1 (6.3)	0 (0)
Gynecologic	4 (0.8)	0 (0)	1 (25)
Intraabdominal	47 (10)	5 (10.6)	1 (2.1)
Pulmonary	162 (34.3)	23 (14.2)	13 (8.0) (11.1)
Skin	65 (13.8)	8 (12.3)	1 (1.5)
Urinary tract	66 (14)	11 (16.7)	1 (1.5)
Unknown	75 (15.9)	21 (28)	5 (6.7)
Total	472 (100)	84 (17.8)	23 (4.9) (15.5)
Infection causative organism*			
S. aureus	46 (26)	8 (17.4)	0 (0)
S. pneumoniae	30 (16.9)	4 (13.3)	3 (10)
Other Gram-positive cocci	12 (6.8)	4 (33.3)	2 (16.7)
E. coli	30 (16.9)	3 (10)	0 (0)
Aerobic Gram-negative bacilli	36 (20.3)	11 (30.6)	1 (2.8)
Polymicrobial	4 (2.3)	3 (75)	0 (0)
Fungi and *Candida*	3 (1.7)	1 (33.3)	0 (0)
Anaerobes	9 (5.1)	1 (11.1)	0 (0)
Viral	4 (2.3)	1 (25)	0 (0)
Other	3 (1.7)	2 (66.7)	1 (33.3)
Total	177 (100)	38 (21.5)	7 (4.0)

Values are *n* (%).
*Includes only patients with infection where a causative organism was definitely identified ($N = 177$).

Table 3
Risk Factors for Progression to Septic Shock Within 72 Hours Among 472 ED Patients With Sepsis

	Univariate Model			Multivariate Model		
	OR	95% CI	p-value	OR	95% CI	p-value
Demographics						
Age (decade of life)	1.16	1.01–1.32	0.039	1.22	1.05–1.42	0.008
Sex, female	2.67	1.59–4.50	<0.001	2.57	1.50–4.40	<0.001
Race, white	0.92	0.61–1.40	0.699			
Vital signs						
Temperature (°C)	1.26	1.02–1.55	0.029	1.34	1.06–1.68	0.013
Respiratory rate (breaths/min)	1.01	0.98–1.05	0.445			
Heart rate (beats/min)	1.01	1.00–1.02	0.045			
Comorbidities						
Alcohol abuse	0.78	0.32–1.91	0.581			
Cancer	0.70	0.27–1.86	0.480			
Cirrhotic liver disease	1.87	0.36–9.80	0.459			
Diabetes mellitus	1.11	0.66–1.87	0.686			
Drug abuse	0.64	0.29–1.45	0.288			
Heart failure	2.67	1.34–5.35	0.005			
Hemodialysis	1.47	0.72–3.03	0.291			
Human immunodeficiency virus	0.77	0.09–6.46	0.807			
Lung disease	2.21	1.32–3.68	0.002	2.30	1.29–4.10	0.005
Smoker	0.72	0.37–1.39	0.329			
Laboratory values						
White blood cell count (×10³/μL)	1.00	0.98–1.03	0.698			
Hematocrit (%)	0.94	0.90–0.97	0.001	0.96	0.92–1.00	0.046
Platelet count (×10³/μL)	1.00	1.00–1.00	0.668			
Serum lactate (mmol/L)	1.13	0.78–1.70	0.671			
Organ dysfunction						
Pulmonary	1.07	0.50–2.31	0.855			
Metabolic	0.77	0.09–6.46	0.807			
Renal	0.59	0.20–1.69	0.312			
Infection site			0.003			<0.001
Pulmonary	0.78	0.45–1.37		0.57	0.31–1.05	
Urine	0.55	0.46–1.88		0.88	0.41–1.89	
Vascular catheter	5.80	2.25–14.9		5.66	1.81–14.1	
Other/unknown	Reference					
Causative microorganism			0.705			
S. aureus	0.99	0.44–2.23				
S. pneumoniae	0.72	0.24–2.15				
Gram-negative aerobes	1.26	0.66–2.43				
Other/unknown	Reference					

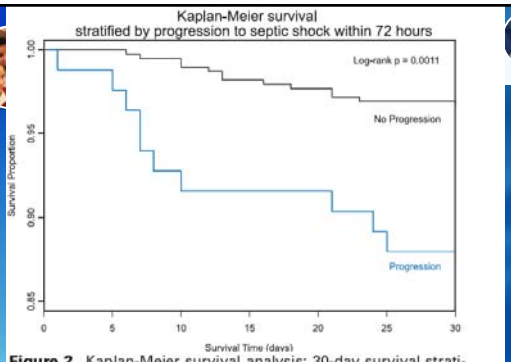

Figure 2. Kaplan-Meier survival analysis: 30-day survival stratified by early progression (72 hours) to septic shock. The figure plots 30-day survival for patients with confirmed infection who were not in shock at the time of initial ED evaluation stratified by whether they developed septic shock within 72 hours of initial ED evaluation.

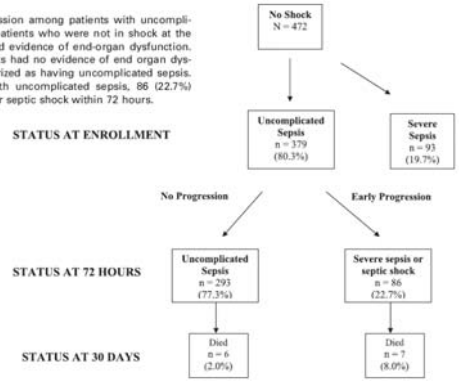

Figure 3. Disease progression among patients with uncomplicated sepsis. Of the 472 patients who were not in shock at the time of enrollment, 93 had evidence of end-organ dysfunction. The remaining 379 patients had no evidence of end organ dysfunction and were categorized as having uncomplicated sepsis. Of these 379 patients with uncomplicated sepsis, 86 (22.7%) developed severe sepsis or septic shock within 72 hours.

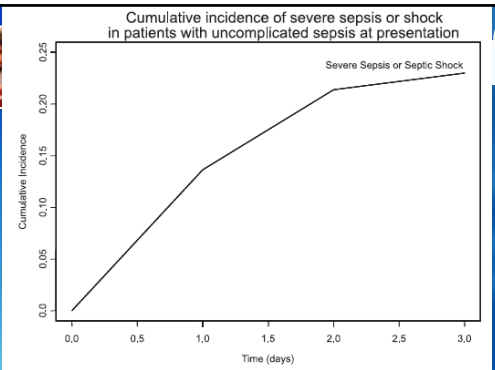

Figure 4. Cumulative incidence of severe sepsis or septic shock among ED patients with uncomplicated sepsis (i.e., no evidence of shock or end-organ dysfunction). The figure plots 72-hour cumulative incidence of severe sepsis or septic shock

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DISCUSSION

- This study found that nearly 1/5 patients with sepsis who were not in shock upon presentation to the ED developed septic shock within 72 hours.
- This progression was observed despite appropriate antibiotic therapy.
- Patient factors associated with sepsis progression included older age, female sex, anemia, comorbid lung disease, hyperthermia, and vascular access infection.
- Early progression to septic shock was associated with higher 30-day mortality.

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DISCUSSION

- This study ：pyrexia in patients without shock who present to the ED is similarly associated *with an increased risk of early progression to septic shock.*
- However, in our study, women 2x progress to septic shock ≦ 72 hrs of presentation.
- Patients with vascular access device infections had the highest rate of progression to septic shock ≦ 72 hours of admission (11 of 20, 55%).
- Catheter-related septicemia ： poor outcomes & nearly 30% mortality.

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DISCUSSION

- In our cohort of 730 patients, elevated lactate was an independent predictor of death.
- In addition, lactate may be elevated for a variety of reasons, *including impaired clearance, depressed cellular respiration secondary to insufficient oxygen tissue delivery, impaired microcirculation, and mitochondrial dysfunction.*
- Thus, additional metabolic biomarkers may be needed to identify high-risk sepsis patients earlier.

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CONCLUSIONS

- Near 1/4 patients with confirmed infection who presents to the ED with uncomplicated sepsis →severe sepsis or septic shock within 72 hours.
- Early progression to septic shock is associated with higher 30-day mortality.
- Better diagnostic tools are needed to identify ED patients with sepsis who are at high risk for disease progression to organ dysfunction or shock.

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Thanks for your attention!

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