



तरंग

OFFICIAL E- BULLETIN OF SCCM DELHI-NOIDA



A stylized, flowing logo for 'Tarang' in green and pink colors, set against a background of concentric circles containing various musical notes in green, pink, and white.

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Quarterly Newsletter of
SOCIETY OF CRITICAL CARE MEDICINE (SCCM)
Delhi-Noida

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CHAIRMAN'S MESSAGE



Dear Members colleagues & fellow countrymen,

It gives me immense pleasure to welcome you all to the fourth issue of “**Tarang**” !

My congratulations, gratitude and indebtedness to each one of you for making SCCM Delhi NOIDA “**The Best Metro Branch**” for the second time in succession! This has been one of the most appreciated accomplishments in recent times. Thank you all once again for attaining this feat.

I am equally delighted to share that during our current tenure, besides organising Clinical Meets, workshops & Walkathons. We introduced many new adventures in DCCS, the most cherished one was “**Ap ki Adalat**” ; Which give us an insight in the personal life of stalwarts in intensive care, with fun & grace. Some of legends who participated were Dr. Yatin Mehta, Dr. Rajesh Chawla, Dr. Rajesh Pandey, Dr. Depak Govil. Another value addition was a masterly quiz, which was very well priced. This was possible only through gigantic support of all members of SCCM Delhi, Noida. I must admit that I was unnerved with a Team of amazing members that comprised of Executive and Academic Council Members, who worked hard to make these events a splendid success.

Friends, this is going to be my last message as Chairman, and at the same moment, as promised at the time of taking this chair, I am glad to have made an imprint at the national level and bring accolades to SCCM Delhi NOIDA Branch

Merci Gracias Danke ;

Until Next time !

Dr. Anil Gurnani

Chairman

SCCM Delhi, NOIDA

SECRETARY'S MESSAGE



Respected SCCM Delhi - Noida Members,

Greetings of peace and love.

I extend my deep gratitude for giving me the chance to serve our society as secretary. SCCM, Delhi-Noida is one of the oldest & most prestigious branch of ISCCM. It's my pleasure to bring to you the fourth issue of the official e-newsletter (तरंग) of SCCM, Delhi-Noida.

This will serve as a mouthpiece for effective communication amongst the society members. The need to communicate remains integral to our purposeful existence in the society.

"(तरंग)" will positively tickle you every quarterly.

As our commitment is quite evident from back-to-back, uninterrupted Monthly Academic Meets which provides an opportunity for interaction, but the members who miss out the monthly meets for some reason can find "(तरंग)" a good tool to be informed & included.

"(तरंग)" will have many topics beneficial to those members who are pursuing different courses in critical care medicine.

"(तरंग)" will feature regular sections on pearls of wisdom, cases to remember, quiz, journal event, student's café, different announcements,

We would welcome your suggestions & look forward for you valuable feedbacks @ iscmdelhichapter@gmail.com to make ever better & interesting.

I wish to thank the editorial board & contributors for their untiring efforts to make this possible.

Happy Reading!
Long Live SCCM (Delhi-Noida)

Dr. Ashutosh Bhardwaj
Secretary
SCCM, Delhi-Noida

TREASURER'S MESSAGE



Dear friends

Today, I am honoured and delighted to be a part of "**Tarang**" our e-bulletin for SCCM Delhi-Noida members. Our goal in designing this bulletin was not only to share updates on branch activities but also to create a valuable educational resource for our students and trainees. The content of the bulletin is tailored to keep us informed about the challenges of managing critically ill patients, featuring expert opinions, journal scans, challenging case scenarios, a student's café, quizzes, and a touch of humor.

This achievement was made possible by the dedicated efforts and visionary leadership of our president, Dr. Anil Gurnani, and Secretary, Dr. Ashutosh, along with the editorial board members. Moving forward, we plan to publish this bulletin quarterly and encourage members to contribute interesting cases and articles.

I extend my heartfelt thanks to all branch members and senior colleagues who have contributed to the development of our e-bulletin.

Wishing everyone happy and enjoyable learning!

Thanks

Dr. Prashant Saxena

WE WELCOME OUR NEW MEMBERS

Membership No.	Name	Email	Mobile No.	State	City	Membership Type
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PEARLS OF WISDOM

Drowning – An Aquatic Emergency

Anil Gurnani¹, Suman Jha²

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Introduction:

Drowning is an unexpected tragedy, an aquatic emergency, a form of asphyxial death. We present a series of 14 cases of fresh water drowning in the age group of 12-16 years accidental in nature occurring in summer months presenting to our hospital.

Our objectives were:

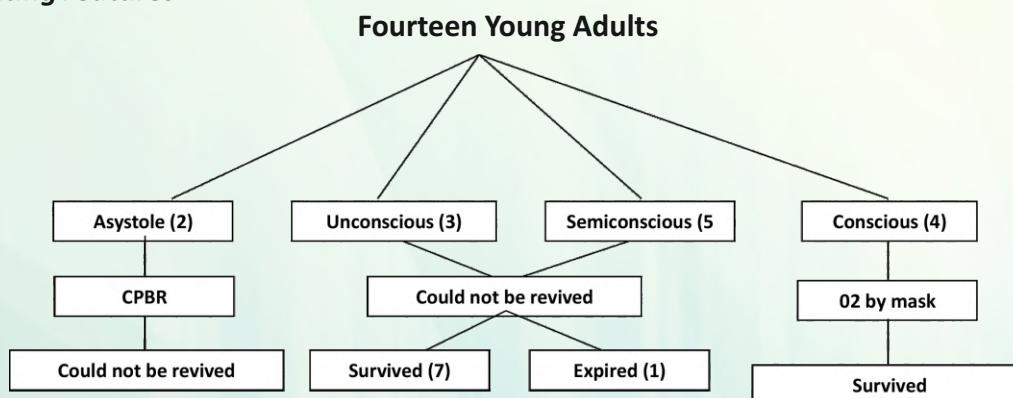
- Study the demographic profile
- Evaluate social problems
- Outline protocol for management
- Identify prognostic factors
- Predict the outcome

Mode of Retrieval

All these cases were cases of fresh water drowning 6 occurring in late, 6 in pond and the most unfortunate accident happened in a sewer manhole was recovered by fire brigade (1)

Outcome of drowning depends on submersion time. Victims who had submersion time of < 10 min had a better prognosis.

Presenting Features



- 4 patients were conscious and recovered well required only oxygen by face mask.
- 2 patient were brought in asystole and they could not be revived
- 8 patient were in variable levels of consciousness hence required mechanical ventilation

Problems Encountered

- The main problem faced was lack of basic life support at the accident site others were.
- Hypoxemia
- Pulmonary edema
- Hyponatremia
- Metabolic acidosis
- Hypothermia

Monitoring

- Level of consciousness
- ECG/ NIBP/ SpO₂/ ETCO₂/ CVP/ IBP/ ventilated
- S. Electrolyte/ ABG
- Skiagram chest
- Core temperature

We took the approach of Goal Directed Therapy:

- One patient required mechanical ventilation for < 9 hrs,
- Three patients required mechanical ventilation for around 72 hrs
- Four patients required mechanical ventilation > 72 hrs.

We managed complications in following way when we encountered them

- Sodabicarb (for acidosis) - 7.5%
- NaCL (for hyponatremia) - 5% (R/T)
- Diuretics (for cerebral oedema) - Frusemide , Mannitol

Predictability of outcome –

This depended on clinical laboratory and environmental factors which predict the outcome

- Hyponatremia : 13 (92.8%)
Na < 120 mmol/L
- Arterial pH < 7.1 : 3 (21.4%)
- pO₂ < 60 mmHg. : 4 (28.5%)
- GCS Score < 5 : 5 (35.7%)
- Prolonged submersion (2 hours) : 1
- Delayed CBPR : 7
- Asystole : 2

Survival Rate

	Reported	Our series
• Survival	- 44 %	77%
• Brain damage	- 17 %	NIL

We got better results than reported because, maybe some of the victim's submersion time was low and they were rescued earlier

Discussion

Drowning is defined as primary respiratory impairment submersion or immersion in a liquid medium (World Health Congress, Amsterdam 2002)

Liquid to air interface at the entrance to victims airway prevents the individual from breathing.

Downing is the sixth leading causes of accidental death all over the world. Annually around 3,72000 people die. It mostly occurs on weekends in summer months under the influence of alcohol and drugs other contributory factors are swimming in rip current, boating and water supports and fishing industry disaster in rough weather.

In India it is the most common form of suicide only 0.5 % population in India can swim. Vigilance is lacking in swimming pool facilities. Accidents also happens due to breath holding or swimming under water.

There are two types of drowning dry and wet.

- Dry drowning : No aspirate fluid in lungs
- Wet drowning : Aspirate fluid in lungs

Stages of Drowning

- Struggle to keep airway clear of water
- Initial submersion and breath holding
- Aspiration of water
- Unconsciousness
- Cardiorespiratory arrest
- Death

Mode of Death

- Main modes of death asphyxia, shock, concussion, syncope and apoplexy.
- Asphyxia occurs 2 minutes after complete submersion. Death is inevitable after submersion for 5 minutes.
- In exceptional cases if water does not enter lungs, they can be resuscitated even after 10-20 minutes

Pathophysiology - Asphyxia produces hypoxia, hypercarbia and acidosis (high anion gap)

FRESH WATER	SEA WATER
<ul style="list-style-type: none">• Alters the surface tension properties of pulmonary surfactant• Alveoli become unstable, increase in intra-pulmonary shunt	<ul style="list-style-type: none">• Does not change the surface tension of pulmonary surfactant• Pulls fluid from the circulation into alveoli

There is a difference in pathophysiology of fresh water and sea water drowning. Fresh water alters the surface tension and properties of pulmonary surfactant whereas sea water does not change the properties of pulmonary surfactant. Alveoli becomes unstable because of increase intrapulmonary shunt in fresh water. Whereas sea water pulls fluid from the circulation into the alveoli.

Prevention

To prevent these accidents constant vigilance is required in swimming facilities. Rescue attempts should be initiated by life guards if a person is not making purposeful movement for 10 sec. and rescue should be completed within 20 seconds. Person should be carried horizontally and neck should be protected suspecting cervical trauma. Water sports should require use of flotation devices.

Conclusion

- Our observation showed that following goal directed therapy, point of care technology, our predictors matched with outcome.
- We have learnt that to avoid these unfortunate accidents swimming should be taught to everyone especially in schools during formative years. No one should try to help a drowning victim if one cannot swim.
- We as a medical community can definitely teach Cardio Pulmonary Resuscitation to everybody on this planet earth for better outcome of these aquatic accidents.
- ***“Respect the water, it is like a fire ; if you see a burning fire you will not walk towards it !”***

CASES TO REMEMBER

GASTROENTERITIS TRIGGERED BRASH SYNDROME IN A PATIENT WITH CHRONIC KIDNEY DISEASE-A CASE REPORT

Dr Amit Goel

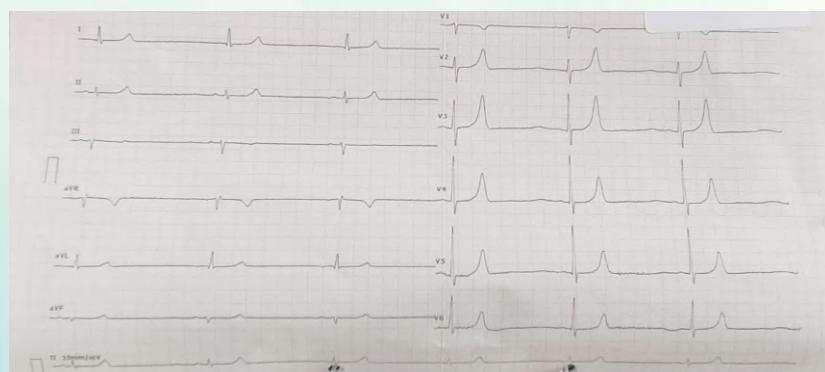
Director & Head

Department of Critical Care Medicine

Yatharth Super Speciality Hospitals, Noida Extension, UP

A 59-year, previously known hypertensive, and stage 4 CKD (not on dialysis) male, presented to the hospital with restlessness, uneasiness, mild to moderate grade fever, multiple episodes of loose motions for 1-2 days. History of toothache, hiccups decreased appetite was also noted. He was on β blocker-(Bisoprolol-2.5 mg once a day), calcium channel blockers (amlodipine 5 mg once a day), oral nonsteroidal anti-inflammatory drugs (NSAID), and oral antibiotics for his medical condition. On arrival he was drowsy yet arousable (Glasgow Coma Scale-12-13), restless, required 2 litres supplemental oxygen to maintain normal oxygen saturations. He had severe bradycardia HR-32-36/min with hypotension BP-80/50 mmHg. Immediately Intravenous (IV) line was secured, and 1 mg adrenaline and 0.6 mg atropine IV bolus given. Followed by stat 1 litre IV normal saline fluid. Vasopressor noradrenaline was started in view of hypotension. Arterial blood gas was suggestive of severe metabolic acidosis (pH-7.16, bicarb-12) with hyperkalaemia (5.8 meq/L). Serum potassium level was 6.1 meq/L. ECG was suggestive of sinus bradycardia with tented T-waves (Figure 1). Immediately Antihyperkalemic measures were initiated with 20 ml injection Calcium Gluconate, and 100 ml sodium bicarbonate. Salbutamol nebulisation for 20-30 minutes and 50% Dextrose 100 ml with 20 units insulin was also initiated. Over next one hour his heart rate started to improve to 55-60/min and repeat ABG was suggestive of improvement in acidosis and hyperkalaemia. His basic lab investigations were sent and with ongoing noradrenaline, antihyperkalemic measures, he was shifted to intensive care unit for further stabilisation.

One session of hemodialysis was done in view of azotaemia (blood urea-193 mg/dl, Serum Creatinine-9.7 mg/dl) and persistent hyperkalaemia. He improved hemodynamically, started accepting orally well with no loose motions over next 2 days. He was shifted to step down care unit after thorough cardiac evaluation. Later, on day 5, he was discharged in stable condition with necessary instructions for care and follow up.

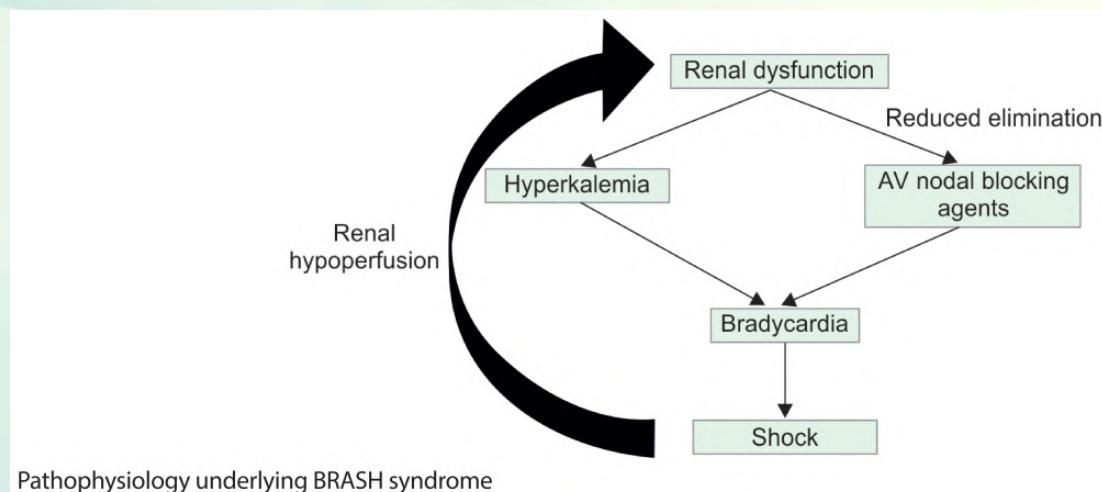


Discussion

BRASH syndrome represents a vicious cycle involving bradycardia, renal failure, AV nodal block, shock, and hyperkalaemia. It is frequently observed in elderly with underlying comorbidities, such as CKD, coronary artery disease, and hypertension. This term was first used and reported by Farkas et al. in 2016.(1) However it was under reported earlier but it has been widely recognised and published in last couple of years.(2–8) Most observed contributing factors leading to the development of BRASH syndrome are old age, kidney dysfunction, requirement of AV node blocking agents, hypovolemia, renal hypoperfusion.(2) The proposed mechanism in BRASH syndrome is the synergistic effect of AV-nodal blocking agents and hyperkalaemia causing refractory bradycardia and hypotension.(1) This leads to renal hypoperfusion and further deranged renal function leading to hyperkalaemia and reduced drug elimination initiating a vicious cycle.

Ongoing AV nodal blocking medications puts these group of patients at very high risk of persistent life threatening bradycardia even with moderate hyperkalaemia.(1-3) β blockers and CCBs are the most frequently incriminated medications in this category for BRASH Syndrome.(4,5) Other triggering agents are angiotensin-converting enzyme inhibitors, potassium-sparing diuretics and nephrotoxic drugs like aminoglycoside antibiotics and NSAIDS.(1,8) Combination of one or more of these precipitating factors may be present, as in our case-AV-nodal blocking agents, NSAIDS, sepsis and hypovolemia (loose motion).

The clinical presentation is variable, ranging from asymptomatic bradycardia, cardiogenic shock , multisystem organ failure and even death in some cases.(2,8) Eliciting thorough history and a strong clinical suspicion is required to diagnose BRASH syndrome especially in patients with renal dysfunction presenting with bradycardia, with ongoing AV-nodal blocking agents.(2,6) ECG may show constellation of findings varying from normal pattern to sinus bradycardia to arrhythmias due to AV node block.(3,6–8) Junctional rhythm present occasionally, can prove life threatening.



Management of BRASH syndrome includes early recognition and rectification of underlying

triggering factors. Standard management as per advanced cardiac life support (ACLS) including atropine & cardiac pacing would not be sufficient for bradycardia. (2,3) Prompt measures should be taken to break vicious cycle of bradycardia, decreased cardiac output, renal hypoperfusion, hyperkalaemia. However intravenous fluid (IV), vasopressors should be started aggressively, they have been shown to respond better with agents with positive chronotropic effect on β -1 receptors. viz- isoproterenol, adrenaline, dopamine, and dobutamine. Isoproterenol has rather shown to have favourable outcome in these scenarios. (2) Antihyperkalemic measures must be started immediately for management of hyperkalaemia- i) IV calcium for the cardiac membrane stabilisation. ii) IV insulin with glucose and continuous nebulisation with β 2 agonist for intracellular potassium shifting. iii) IV soda bicarbonate, if severe metabolic acidosis is present. iv) Kaliuresis with loop diuretics-furosemide should be tried once hypovolemia is corrected. v) Calcium polystyrene sulfonate powder (potassium-binding resin) may be tried enterally once airway is secured. vi) Urgent dialysis in refractory cases. (8)

Cases with refractory AV nodal blockade due to β blocker may need to be treated specifically with high-dose insulin euglycemic therapy (HIET) or intravenous glucagon. Intralipid emulsion-lipid sink and extracorporeal therapies may also be tried as rescue measure. (3,8)

Conclusion

History, thorough clinical examination, strong clinical suspicion, and prompt treatment is the key to management of BRASH syndrome. AV nodal blocking drugs should be avoided in patients having renal dysfunction. This may unfold life threatening refractory bradycardia and shock with moderate hyperkalaemia. Clear understanding and recognizing the pathophysiology of BRASH syndrome can facilitate a more comprehensive and organized management strategy for these patients and a better outcome.

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JOURNAL QUEST

EARLY RESTRICTIVE OR LIBERAL FLUID MANAGEMENT FOR SEPSIS-INDUCED HYPOTENSION

The National Heart, Lung, and Blood Institute Prevention and Early Treatment of Acute Lung Injury Clinical Trials Network. NEJM 2023. DOI: 10.1056/NEJMoa2212663.

CLOVERS TRIAL

Clinical Question

In patients with sepsis-induced hypotension does a restrictive fluid strategy (with early vasopressor usage) compared to a liberal fluid strategy result in lower mortality before discharge by day 90?

Background

- The [2021 SSC Guidelines](#) recommend the use of large volumes of fluid (30ml/kg) during the initial resuscitative phase of septic shock
- This however is based on low quality evidence – with only a weak, low quality recommendation in the above guidelines
- Practice varies widely, with [an Australian observational study](#) showing a median of 4.2L of fluid given to septic patients within the first 24 hours
- The recently published [CLASSIC trial](#) showed no difference in 90-day mortality when comparing a restrictive fluid strategy to standard care; however randomisation for this trial occurred in ICU after initial fluid resuscitation

Design

- Multi-centre, randomized, unblinded superiority trial
- Patients randomly assigned in 1:1 manner to restrictive or liberal strategies
- Randomised via central web based system
 - Stratified by trial site
- Protocol amended in October 2019
 - Limited initial infusion to 1000ml if vitals signs stabilised and felt to be volume replete
- Pre-specified stopping boundaries (for both futility or efficacy in either group) at 1/3 or 2/3rds recruitment
 - Study was stopped at the 2nd interim analysis due to futility, there were no concerns of harm from the DSMB
- Informed consent required from patients or legal representatives
- Protocol adherence monitored for first 300 patients and in 10% (random sample) during remainder of trial
- Power calculation:
 - Assuming baseline mortality of 15% and an absolute difference of 4.5% in the

- restrictive group
- 2320 patients needed to have 90% power at a significance level of 0.05

Setting

- 60 US centres
- March 2018 to January 2022

Population

- **Inclusion:**
 - Adult patients with suspected / confirmed infection
 - Sepsis induced hypotension (SBP < 100 mmHg or MAP < 65mmHg following > 1000ml IV fluid)
- **Exclusion:**
 - >4 hours since meeting inclusion criteria for sepsis induced hypotension
 - >24 hours since hospital presentation
 - >3L IVT (including pre-hospital)
 - Inability to obtain informed consent
 - Pregnancy
 - Fluid overload
 - Blood pressure is at known or reported baseline level
 - Hypotension suspected to be from non-sepsis cause
 - Severe volume depletion from non-sepsis causes
- 12276 met inclusion criteria → 4868 met eligibility criteria (i.e. no exclusion criteria) →
1563 randomised
 - 782 restrictive group
 - 781 to liberal group
- Comparing baseline characteristics of restrictive vs. liberal group
 - Generally well matched
 - Age: 59.1 vs 59.9
 - Female: 47 vs 47%
 - Chronic heart failure: 13 vs 10%
 - ESRF with haemodialysis: 4 vs 5%
 - SOFA score: 3.4 vs 3.5
 - SBP: 93 vs 94 mmHg
 - Lactate: 2.9 vs 2.9
 - Median time from eligibility to randomisation: 61 vs 60 mins
 - Median volume of fluid administered before randomisation: 2050 mls vs 2050 mls

Intervention

- Restrictive Group
 - If SBP < 100mmHg or MAP < 65 mmHg after receipt of 1–3L crystalloid:
 - All bolus and maintenance fluids ceased
 - Up to 2L fluid boluses allowed (including pre-randomisation) at discretion of clinician
 - Following this if, MAP < 65 mmHg or SBP < 90 mmHg
 - Titration of norepinephrine +/- second vasopressor aiming MAP > 65

- Once MAP in target then fluids limited to KVO, medications and nutrition
- Rescue fluids recommended for (500ml bolus):
 - Severe hypotension (SBP < 70, MAP < 50 mmHg)
 - Refractory hypotension (SBP < 90 or MAP < 65 on Norepinephrine > 20 mcg min or equivalent)
 - Lactate > 4 mmol/L and increasing after 2 hours of therapy
 - Sinus HR > 130 for 15 mins
 - Echocardiographic evidence or haemodynamic evidence of extreme hypovolaemia
 - Felt to be in best interests by treating team

Control

- Liberal Group
- SBP < 100mmHg or MAP < 65 mmHg after receipt of 1–3L crystalloid
 - Halt all maintenance
 - Prescribe 2L at randomisation (to be completed within 180 mins)
 - 2nd litre can be withheld if volume replete following clinical assessment following first litre
 - If any of the following present then further 500ml bolus:
 - MAP < 65 or SBP < 90 mmHg
 - Lactate > 4 mmol/L and increasing
 - UOP < 30mls/hr
 - HR > 110 bpm (sinus)
 - Requirement for vasopressors
 - Measured or clinical assessment
- Rescue vasopressors allowed if severe hypotension (defined as per restrictive group), lactate > 4 mmol/L and increasing after 2 hours, > 5L total IV fluid given, clinical manifestations of fluid overload or treating team felt to be in best interests

Management common to both groups

- Assigned protocol followed for 24 hours
- Hourly reassessments or after any intervention
- Protocol could be over-ridden at any time if felt to be in best interests of patient
- Vasopressors could be administered peripherally
- 40% in restrictive group and ~25% in liberal group had peripheral vasopressor infusion

Outcome

- Primary outcome:**
 - Death before discharge home by day 90: 14.0% in restrictive group vs 14.9% in liberal group
 - Estimated difference -0.9% (95% CI -4.4 to 2.6), p = 0.61
- Efficacy and Safety outcomes:**
 - No significant difference in
 - days free from organ support therapy by day 28:
 - days free from ventilator use at 28 days

- days out of ICU from day 1 to 28
- new intubation with mechanical ventilation by day 28
- ARDS onset between day 1 and day 7
- Serious Adverse Events:
 - 3 vs 0 counts of extravasation of vasopressors in restrictive group (all managed conservatively)
- **Subgroup Analysis:**
 - No subgroup favoured with liberal or restrictive fluid use
 - These included: age, sex, race, location at randomisation, pneumonia, baseline SBP < 90 or vasopressor use
- **Post-hoc Analysis:**
 - Higher rates of ICU admission in restrictive group (Table S8):
 - 0–24 hours: 67 vs 59%
 - 0–7 days: 70% vs 62%

Primary Outcome		
	Restrictive (n=782)	Liberal (n = 781)
Death before discharge home by day 90	14.0%	14.9%
Absolute percentage difference -0.9% (95% CI -4.4 to 2.6)		
Selected Secondary Outcomes		
<i>None statistically significant</i>		
No. of days free from organ support therapy at 28 days	24.0%	23.6%
No. of days free from ventilator use at 28 days	23.4%	22.8%
Number of days out of the ICU from day 1 to day 28	22.8	22.7
New intubation with invasive mechanical ventilation by 28 days	11.0%	12.7%
ARDS onset between day 1 and day 7	2.5%	2.6%
KDIGO score on day 3	0.35	0.34
Serious Adverse Event - number of events	21	19
Therapies administered during intervention period		
IV fluid administered over 6-hr period (mls)	500	2300
Difference -1800 mls (95% CI -1889 to -1711)		
IV fluid administered over 24-hr period (mls)	1267	3400
Difference -2134 mls (95% CI -2318 to -1949)		
Vasopressor administration during first 24-hr period	59.0%	37.2%
Duration of vasopressor use during first 24-hr period amongst those who received vasopressor therapy (hrs)	9.6	5.4

Authors' Conclusions

A restrictive fluid strategy (with earlier vasopressor use) did not result in significantly lower (or higher) mortality before discharge home by day 90 than a liberal fluid strategy

Strengths

- Randomised
- Multi-centre trial with broad inclusion criteria means results applicable to ED and early ICU practice where patients present in undifferentiated manner
 - The caveat to this is that sepsis is a heterogenous process and different causes of sepsis may respond differently to fluid
- Excellent design with high internal validity that helps answer an important and frequently occurring question with prior limited evidence base
- High level of adherence to protocol
 - 97% adherence in restrictive group and 96% in liberal group (within audited sample)
 - This is impressive given time constraints on ED management
- Achieved separation in fluid administered in first 24 hours
- Almost identical amounts of fluid administered in both groups following the intervention to day 7 (Table S3)
- Excellent safety outcome reporting including on peripheral vasopressor administration

Weaknesses

- Unblinded
- Single country may limit external validity
- Early cessation
- Large numbers eligible but not enrolled ($n = 3303$, ~25% of all those screened) – this may introduce a selection bias. This includes:
 - 900 as study team unable to obtain informed consent
 - 887 patient or surrogate refused consent
 - 873 MD refusal
 - 346 not excluded but not enrolled
- Patients may not have been that unwell at baseline (median SBP > 90, low lactate levels, an ICU admission rate of ~63% and only ~60% patients in restrictive group required vasopressors)
- No dynamic methods used to assess fluid responsiveness
- The fluid regimes don't account for patient weight / size – 2L in a 50kg patient is likely to have a different physiological response to that in a 120kg patient
- Significant amounts of the study were left to individual clinician preference, introducing bias
- This trial compares to protocolised approaches – other approaches may have different outcomes
 - [ARISE-FLUIDS](#) and [ANDROMEDA-SHOCK 2](#) will add more information

STUDENT'S CAFÉ

WEARABLES AND EARLY WARNING SIGNS IN HOSPITALS

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Wearables are here to stay. And the amount of information they steal from our body is far too much to quantify. What use it will be put into is a mystery which time will unfold. We the healthcare workers are a bit slow in putting to use the newer devices and inventions in patient care. We have recently started talking and discussing a lot about AI in healthcare. What it can do? How can it shape or change patient care in the future? I am sure most of it would be the database from the vitals that we measure with devices. The common man however does not appreciate the advances/capability of a multi-slice imaging machine or a smart MRI machine, but does a Doctor care about them? I have yet to hear doctors going gaga on smarter imaging or smarter patient care ventilators in the intensive care units.

Focusing more on the wearable it is prudent to mention that they have come discreetly and are here to stay. Imagine a simple watch that came first to measure my steps and heart rate, and went on to do so on a 24-hour basis for weeks, with the advent of longer battery life. These tiny devices even started interpreting Sleep and the sleep-wake cycle. Adding more to life, more parameters were put into the watch such as respiratory rate, Spo2, Blood pressure monitoring, and even heart rhythm monitoring- to look for atrial fibrillation and inform the buddy/next of kin to close the loop/chain of survival.

There have been reports of smart watches detecting fatal arrhythmias. We are not done yet with the watches, but we have started seeing the soft entry of wearable rings. How can these devices be of help to us in the hospitals and healthcare settings? A lot, yes a lot can be gained by using these and incorporating them into the healthcare systems. The sad part of the story is the timely synergism, yes the engineers who design these watches do not meet doctors and do not hear what we need to say and how we want the information to reach us. It seems like they have a very secret and protected laboratory where they work. And I have never understood in the past 3 decades how and in which way these companies choose the Doctors who would help them design these products. Not that I miss meeting these techies or designers, but I feel the most basic things like remote home care monitoring should have been in place by now. We have learned strong lessons in the COVID pandemic and must use them to the best of our ability.

Coming back to the wearable, my 2 cents of suggestions are the following, which is also a loud shout to the engineers, techies, and manufacturers who design these wearables –such as watch rings, smart watches, and patient alarm systems.

In the hospital scenario, we should have watches or rings that can be given to the patient at the time of admission. These are like patient identification tags which a patient gets at the time of admission. The patient wears them all the time even during surgeries, during the procedures, and even if one is admitted into the intensive care unit. At the time of discharge, this patient submits the wearable to the hospital.

While the patient is admitted to the hospital the information received from the wearable can be streamed to the Nurse's station / central monitoring console which can be monitored in real-time "Early warning signs" and attended to immediately. The alert for the same would also go to the floor resident doctor and consultant in charge of the patient, who would also act promptly. This early pick up of **Early warning signs** and prompting the cascade of healthcare workers to attend to it would save many lives. And the good thing is we have all that is required –all that we need to do it integrate these.

What the hospital gets in the end is also loads of databases about the patient's vital parameters such as Heart rate, pulse rate, arrhythmias, saturation, blood pressure, sleep-wake cycle, and patient health. A lot of information for the insurance companies as to know what was happening and when. It would open up a Pandora's box of information. Hope we would be able to use this information diligently.

But I am sure it is worth the effort to integrate the current wearable into the healthcare systems in predicting and acting on "Early warning signs in the hospital". Helping the RRT and code blue teams to reach fast act fast and save more lives.

The colossal data which can be used to analyze the causes and factors that lead to the change in vital parameters.

I am sure these are the shape of things to come in the future.

Officially, in terms of emergency care, the [Apple Watch](#) keeps an eye out for early warnings of atrial fibrillation (AF), unusual heart rates, and serious falls. You don't have to look very far to find [examples](#) of buyers claiming the Apple Watch has [saved their lives](#), usually diagnosing an unknown heart condition before symptoms became obvious. Apple Watch has shown it might have more medical functionality than even Apple is aware of. The Apple Watch has managed to save another life — and this time via a condition that the watch doesn't officially detect.

SALUTE TO THE ACHIEVERS

SCCM Delhi-Noida, Best Metro branch (Consecutive 2nd year) by
ISCCM at CRITICARE KOLKATTA 2024





CRITICARE 2024

28th Feb - 3rd March 2024 | KOLKATA

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Prof. Sheila Nainan Myatra
President, ISCCM

Dr. Y. P. Singh
General Secretary, ISCCM





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PREVIOUS CLINICAL MEETINGS

SOCIETY OF CRITICAL CARE MEDICINE, (SCCM) DELHI-NOIDA

At Marigold Hall, India Habitat Centre, New Delhi
Friday, 19th January 2024, 07:00 pm - 09:30 pm

PHOTOGRAPHS	WRITEUP
    	<p><u>19th January 2024</u></p> <p>19th Monthly Clinical Meet Total Number of attendees- 54</p> <p>Dr Anil Gurnani (Chairman) welcomed all the members for 19th Clinical Academic Meet. Scientific meeting was organised on behalf of SCCM, Delhi-Noida by Dept of critical care medicine, Swami Dayanand Hospital</p> <p>3rd Edition of e-bulletin “तरंग” was also launched.</p> <p>The 1st presentation was Comparison of early continuous hemodiafiltration vs delayed continuous hemodiafiltration in patients of AKI with septic shock the case was presented by Dr Ranajit Chatterjee.</p> <p>The 2nd case was presented by Dr Shivani Kasana, on Comparison of Helmet vs face mask for NIV in patients of ACPE.</p> <p>The 3rd was IV fluids beyond 30ml/kg -how much is too much presented by Dr Ramesh Subedi.</p> <p>The 4th was Anesthetic conserving device(AnaConDa)-an overview presented by Dr Shivangi Gupta</p> <p>Dr Ashutosh Bhardwaj (Secretary) delivered vote of thanks to everyone in the meeting and for participating in the meeting and for a wonderful clinical discussion that ensued after each session, and felicitated all speakers with certificates of participation.</p> <p>Meeting was followed by dinner.</p> <p>Regards.</p> <p><i>Ashutosh</i></p> <p>Dr Ashutosh Bhardwaj Secretary SCCM Delhi Noida</p>

At Magnolia Hall, India Habitat Centre, New Delhi
Friday, 22nd March 2024, 07:30 pm

PHOTOGRAPHS	WRITEUP															
	<p>22nd March 2024</p> <p>A grand celebration for getting best branch award for two consecutive years.</p>															
	<p>Total Number of attendees- 64</p> <p>Dr Anil Gurnani (Chairman) welcomed all the members for a grand celebration for getting best branch award for two consecutive years.</p>															
	<p>Dr Ashutosh Bhardwaj (EC member) delivered vote of thanks to everyone present in the celebrations.</p> <p>Celebration party was followed by dinner.</p>															
	<p>26th April 2024</p> <div style="background-color: #e0f2e0; padding: 10px; text-align: center;">  SCCM, DELHI-NOIDA <i>Invites You to</i> 20th Monthly Clinical Meet <i>Presented By:</i> Sri Balaji Action Medical Institute Date: 26th April 2024, Friday Time: 7:30 PM onwards Venue: Maple Hall India Habitat Centre <p style="color: red;">*Meeting followed by Dinner.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <thead> <tr> <th style="width: 10%;">Sr. No.</th> <th style="width: 60%;">TOPICS</th> <th style="width: 30%;">SPEAKERS</th> </tr> </thead> <tbody> <tr> <td>1.</td> <td>Management of Refractory septic shock in ICU</td> <td>Dr. Rajat Gupta</td> </tr> <tr> <td>2.</td> <td>Management of ACLF in critically ill patients</td> <td>Dr. Ashish Sikarwar</td> </tr> <tr> <td>3.</td> <td>Difficult intubation in ICU, Anticipation, Risk and outcomes</td> <td>Dr. Abhimanyu</td> </tr> <tr> <td>4.</td> <td>Role of interventional radiology in ICU settings</td> <td>Dr. Abhishek Bansal</td> </tr> </tbody> </table> <p style="text-align: center;">Moderator : Dr Rajat Gupta</p> <div style="display: flex; justify-content: space-around; margin-top: 10px;"> <div style="text-align: center;">  Dr. Anil Gurnani Chairman </div> <div style="text-align: center;">  Dr. Ashutosh Bhardwaj EC Member </div> </div> <div style="display: flex; justify-content: space-around; margin-top: 10px;"> <div style="text-align: center;">  Dr. Akhil Taneja EC Member </div> <div style="text-align: center;">  Dr. Amit Patel EC Member </div> <div style="text-align: center;">  Dr. Anubhav Kumar Singh EC Member </div> <div style="text-align: center;">  Dr. Piyush Sood Treasurer </div> <div style="text-align: center;">  Dr. Nitin Jain EC Member </div> <div style="text-align: center;">  Dr. Brijesh Agarwal EC Member </div> <div style="text-align: center;">  Dr. Baljeet Singh EC Member </div> </div> </div>	Sr. No.	TOPICS	SPEAKERS	1.	Management of Refractory septic shock in ICU	Dr. Rajat Gupta	2.	Management of ACLF in critically ill patients	Dr. Ashish Sikarwar	3.	Difficult intubation in ICU, Anticipation, Risk and outcomes	Dr. Abhimanyu	4.	Role of interventional radiology in ICU settings	Dr. Abhishek Bansal
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QUIZ

Q-1-Which of the following is True:

- ◆ Phased array probe has better penetration compared to curvilinear probe
- ◆ Phased array probe has better resolution compared to curvilinear probe
- ◆ Linear probe provides the best balance of resolution and penetration
- ◆ Phased array probe provides the best resolution

Q-2-This profile nearly rules out all of the following Except:



- ◆ Pleural Effusion
- ◆ COPD
- ◆ Pulmonary embolism
- ◆ Pneumothorax

Q-3- "Curtain Sign" on Lung USG signifies

- ◆ Subcutaneous Emphysema
- ◆ Expanded and aerated Lung
- ◆ Overlapping pleural and pericardial effusion
- ◆ Pulmonary pathology at lateral lung bases and costophrenic recesses.

Q-4- What is indicated by the presence of a lung pulse?

- ◆ Pneumothorax at the location of the probe
- ◆ No pneumothorax at the location of the probe
- ◆ Pleural effusion
- ◆ Tamponade

Q-5- "Bat sign" is absent in following condition

- ◆ Pneumothorax
- ◆ Pleural Effusion
- ◆ Sub-cutaneous Emphysema
- ◆ Severe ARDS

Q-6- "Sinusoid sign" is

- ◆ Is not validated as a measure to determine Vti
- ◆ Helps to measure Vti in 2D-echo
- ◆ Depicts IVC variation
- ◆ M-mode equivalent of Quad Sign

Q-7-Identify this picture-



- ◆ Jelly Fish Sign
- ◆ Plankton sign
- ◆ Dynamic Air Bronchogram
- ◆ Spine Sign

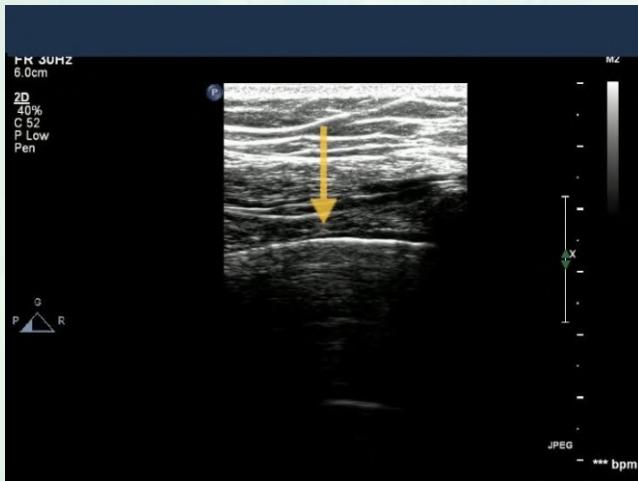
Q-8- All of the following can create the ABSENCE of lung sliding except

- ◆ Mainstem intubation
- ◆ History of pleurodesis
- ◆ Mucus plug
- ◆ Dense Consolidation

Q-9- Which is NOT true of B lines?

- ◆ They enhance A-lines
- ◆ They are vertical hyperechoic lines
- ◆ B lines move in sync with lung sliding
- ◆ They can exist with a pneumothorax

Q-10- What is indicated by the arrow in this picture?

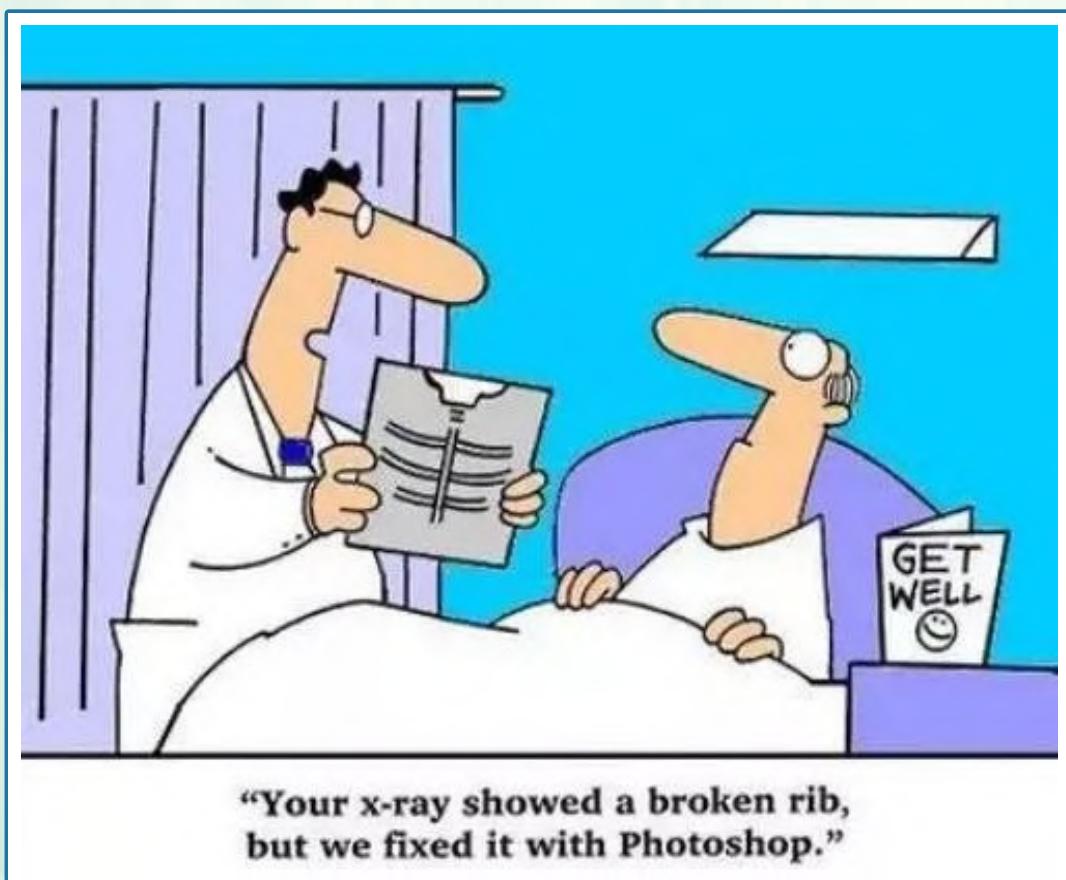


- ◆ Intercostal muscle
- ◆ Pleura
- ◆ Effusion
- ◆ Fatty tissue

QUIZ RULES

- ◆ Answers to be submitted at isccmdelhichapter@gmail.com
- ◆ Mention your name, mobile no. and ISCCM membership ID in the mail.
- ◆ Answer to these Question will be provided in monthly meet to be held in May 2024.
- ◆ Winner of quiz (only 1) will be announced and given a lucrative prize.
- ◆ In case of tie between candidates, winner will be chosen by lucky draw during monthly meet in May 2024.
- ◆ Answers as considered right by the organizing team will be final and no dispute will be entertained.

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**After
Vaccination
Can I Be
100% Safe**

**People Wish
You A Happy
Married Life At
The Time Of
Marriage. It Is
Similar**



We can approach this one
of two ways, Mr. Smith.



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GLASBERGEN

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"It's good that you're eating more fresh fruit and vegetables, but be careful to chew more thoroughly."