The Critical Care Communications
A monthly newsletter of
Indian Society of Critical Care Medicine

Volume 4.2 • March-April, 2009

# CRITICARE- 2009
# 15th Annual Conference of Indian Society of Critical Care Medicine
## 18th -22nd February, 2009 (Agra, India)

**A brief summary**

International Critical Care Congress and 15th Annual Conference of the Indian Society of Critical Care Medicine has established new mile stone in the field of Critical Care Medicine and updates. A Total of 2200 delegates took part in the 11 workshops, CME's and main conference this year from18th Feb.,09 to 22nd Feb., 09 at Hotel Jaypee Palace, Agra. The theme of the conference this year was" Optimizing ICU Resources".

**A. CME**

The CME was especially designed on the curriculum of fellowship examination. The renowned teachers in the field of Critical Care Medicine shared their knowledge in this 2-day Continuing Medical Education programme. The topics were selected in a way that they covered almost all the important aspects in the field of Critical Care Medicine.

**B. ACLS provider course**

This course has been designed by the American Heart Association USA to train healthcare provider to deal with cardiac arrest or other cardiopulmonary emergencies ACLS course emphasizes the importance of basic life support CPR to patient survival, the integration of effective basic life support with advanced cardiovascular life support interventions; and the importance of effective team interaction and communication during resuscitation.

**C. FCCS Course**

This course was conducted under the auspices of Society Critical Care Medicine, USA. The FCCS is a standardized 2 days course being conducted in 25 countries all over the world. This is an ideal training course for Primary care physicians, Emergency medicine physicians, Critical Care Fellows, Resident and Physicians etc. The course consists of standardized 16 lectures referenced to syllabus material plus 7 interactive skill stations and pre & post assessment test.

The broad outline of topics covered during the course were : Assessment of Seriously Ill patient, Airway Management ,Cardiopulmonary Resuscitation ,Diagnosis and Management of Acute Respiratory Failure, Mechanical Ventilation Basic and Advanced, Life Threatening infections - Diagnosis and Antibiotic Selection, Diagnosis and Management of Shock, Acute Coronary Syndrome, Basic Hemodynamic Monitoring, Neurologic support, Electrolyte and Metabolic Disturbances Critical Care in Pregnancy, General Pediatric versus Adult Patient considerations and others.

**D. Basic& Advanced Mechanical Ventilation**

Most noted experts in field of Mechanical ventilation had conducted this workshop.

**E. Basic assessment & support in Intensive Care (BASIC) Course**

The Course was designed by University of Hong Kong and was suitable for doctors with little experience of Intensive Care.This consists of a series of lectures and skill stations covering many aspects of the care of critically ill patients with an emphasis on supportive management, particularly mechanical ventilation. Topics covered were airway management, CPR, acute respiratory failure, Mechanical ventilation,

Haemodynamic monitoring, diagnosis and management of shock, trauma, severe sepsis, transport of the critically ill, metabolic and electrolyte disturbances and acid based disturbances.

**F. Basic Pediatric Critical care workshop**

Some of the salient contents of this course were: how to recognize a sick child in emergency room, to manage a child with Neurologic Injury, principles of Analgesia and Sedation in PICU, Antibiotics and antifungals management in PICU procedures, Airway management and adjuncts, how to setup a ventilator, shock & its management, Fluid therapy and electrolyte, Nutrition issues in PICU, Blood component therapy in PICU and Transport of sick children and many more.

**G. Haemodynamic Monitoring & Echo-Cardiography & Doppler in Critically lll Patients**

Haemdynamic monitorinig is an invaluable tool used routinely in management of critically ill patients. During this 2 day workshop, various technique of haemodynamic monitoring like CVP, Arterial pressure, PA pressure monitoring and various techniques of Cardiac output measurement were discussed.

**H. CRRT & ABG Analysis**

This one day workshop was conducted in two parts:

I. Indications for renal replacement therapy, types of RRT and how one would start and maintain CRRT.

II. Blood gas interpretation, acid base disorders and complex electrolyte problems.

**I. Critical Care Nursing Update**

The world of critical Care Nursing practice is rapidly changing and evolving. This workshop was programmed to challenge all the practicing nurses into new ways of thinking and delivering health care in the field of Critical care Medicine. This was held at Puspanjali Hospital.

**J. Ultrasound in Emergency and critical care Unit**

The course covered general principles of ultrasound, the anatomy and pathology of the thorax, abdomen, pelvis, limbs and vasculature for emergency and critical care ultrasonography, how to acquire and interpretate the ultrasound patterns of the-major severely acute syndromes, and the guidance technique of the major invasive procedures and how to effectively integrate such information into the clinical assessment and management of the critically ill or injured patient in various clinical settings.

The delegates had hands on practice sessions to be comfortable using ultrasound in critical care services.

**K.** Antibiotic Stewardship & Infection Control

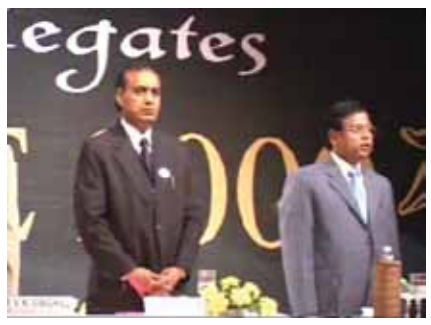
The goal of this workshop was to raise awareness about the escalating problem of antimicrobial resistance.

All workshops were coordinated by experts.The local coordinators provided their assistance to make workshop successful. To provide maximum benefit to attendees, seats were kept limited.

Main conference programme consisted of plenary sessions, theme session, orations and Guest lectures.

Around 20 foreign faculties and 160 national faculties presented their lectures during this conference. Scientific programme was designed by Dr. N. Rungta and locally it was co-ordinatored by Dr. Diptimala Agarwal, 80 medical companies took part in exhibition.

Conference was inaugurated on 19th Feb. evening by Hon'ble Dr. Venugopal, Former Dean, AIIMS, New Delhi. Organizing Secretary Dr. B.K Singh, Org. Chairman Dr. Vijay Singhal, Co-Org. Sec. Dr. Ranvir Tyagi along with their team members, did not left any stone unturned to make this conference a great success.


# MESSAGE FROM THE EDITOR

**Dr. Rajesh Chawla**
PRESIDENT ELECT

Dear Friends,

I am very happy that the Indian Society of Critical Care Medicine is conducting its first multi-centre study, the **INDICAPS STUDY**. It will not be possible to conduct and conclude this pioneer study without your support. With your cooperation, on completion of this study we will finally be able to generate some Indian Data. If you want to be part of this path breaking effort there is still time. You can register at the official website of the ISCCM www.isccm.org. I request all of you to please register and be part of this effort.

Dr. Subash Todi, Senior consultant and Head, Dept. of Critical Care Medicine, AMRI Hospitals, Dhakuria, Kolkata attended the 29th International Symposium on Intensive Care and Emergency Medicine at Brussels. He has been kind enough to give a brief review of recent concepts discussed in the conference in this issue.

Glucose control is once again in the news. The VISEP study and now the NICE SUGAR study have recently been published in The New England Journal of Medicine. There seems to be some evidence to believe that intensive Insulin therapy increases the risk of serious adverse events related to hypoglycemia. The NICE study demonstrates that intensive glucose control increases mortality among adults in the ICU. Critical Care Clinicians like us who use intensive insulin therapy all the time might be somewhat confused. In my opinion, till further evidence becomes available, it would seem reasonable to continue to optimize the management of blood glucose but avoid extreme numbers. You can read about the NICE sugar study in this issue.

**EDITORIAL OFFICE**
**Dr. Rajesh Chawla**
Senior Consultant,
Respiratory & Critical Care Medicine,
Indraprastha Apollo Hospitals,
Room No. 4162, 1st Floor,
Gate No. 10, New Delhi - 110076
Telefax : 011-2682 5586
emails : drchawla@vsnl.net

We all know that Nosocomial infections are a very common cause of morbidity and mortality in mechanically ventilated patients. We talk a lot about VAP (Ventilator Associated Pneumonia) but do not generally consider Ventilator Associated Tracheobronchitis (VAT) which has similar clinical features as VAP except the absence of infiltrates on the chest skiagram. Recent data suggests that VAT is an important risk factor for VAP and targeted antibiotic therapy could be the new paradigm for prevention of VAP. An article published in Critical Care Medicine has focused on the effect of this aspect of therapy on patient outcomes.

All the major Critical Care Congresses of this year are over. The 15th Annual Conference of The Indian Society of Critical Care Medicine (CRITICARE- 2009) at The Jaypee Palace Convention Centre, Agra was a great success. Dr. B.K Singh and all the other members of the organizing committee put in lot of hard work to make it a truly memorable event. An overview of the Conference is presented in this issue.

In this issue, we have also published an updated list of Institutes with the names of teachers and other details, where the Indian Diploma in Critical Care Medicine (IDCC) and Indian Fellowship Critical Care (IFCC) courses are being conducted. You can also find the details of various branches of ISCCM in this issue.

Friends, I request you once again to please be part of the "INDICAPS STUDY" and register for it. It will go a long way towards better understanding of Critical Care issues in our country.

Published By : **INDIAN SOCIETY OF CRITICAL CARE MEDICINE,** Bldg. No.3, No.12, 5th Floor, Navjivan Commercial Premises Co-op. Society Ltd., Dr. D. Bhadkamkar Road, Mumbai Central, Mumbai 400 008. • Tel.: (022) 6526 8504 • Telefax: (022) 2305 4843

# MESSAGE FROM THE PRESIDENT

**Dr. J.V. Divatia**
Professor & In-Charge, Critical Care Service, Tata Memorial Hospital Mumbai

Dear Friends,

The 15th Annual conference of the ISCCM held at Agra from February 18-22, 2009 was a resounding success. The ISCCM Past President's Oration and the President's evening were introduced for the first time. The scientific and social programmes were well attended and appreciated. My congratulations to Drs. Vijay Singhal, SC Gupta, BK Singh, Ranvir Tyagi, Diptimala and the entire Agra team. My special and lasting gratitude to Dr. Narendra Rungta, Chairman Scientific Committee, along with his team of Deepak Govil and Manish Munjal for putting together a scientific feast.

At Agra, the ISCCM has launched its first multicentre, observational study to collect data on Indian intensive care: INDICAPS (**Ind**ian **I**CU **C**ase mix **A**nd **P**ractice patterns **S**tudy). The Investigators' meet was held and finer details of data collection discussed. Over 202 ICUs from across the country have registered for this project. The first day of data collection is planned in August 2009. The ISCCM will maintain contact with all investigators by email, SMS, and of course, this newsletter. The web designing and software is near completion. This is going to be a scientific and logistic challenge, but I am excited and optimistic. This study is vital both for our academic curiosity and national pride. Successful completion of the study will prove to all those who doubt our ability to generate authentic information that we have what it takes. Our initial enthusiasm must now be backed by disciplined and honest reporting of data. Investigators who have registered for this study should check the website for details; there will be SMS reminders everytime new content is hosted on the website. It is still not too late to register your ICU if you have not done so already. Visit the ISCCM website **(http://isccm.org)** and give information about their ICU in the relevant section **(http://isccm.org/ISCCM/Instituteregistration.aspx)**.

I had earlier written that the ISCCM must play a more meaningful social role, especially in the field of disaster management. We propose to run the Fundamentals of Disaster Medicine course (FDM). This is a 1-2 day course of the Society of Critical Care Medicine (USA). It aims to train health care professionals to organize an in-hospital response to epidemics, mass casualties, bioterrorism, etc. This will be invaluable for members who wish to know how to tackle the surge of patients, scale up the hospital and ICU facilities, and ensure optimum outcomes for patients, safety of hospital staff and co-ordinate with the overall disaster management system. It is a different kind of learning, but extremely relevant and useful in these troubled times. The first such course will be launched at the Delhi Critical Care Symposium, from September 4-6, 2009. Instructors from the SCCM will be present to kick-start this educational initiative. This is yet another example of the benefits of international co-operation between the ISCCM and the SCCM.

Talking of international collaboration, the ISCCM is likely to join the Asia Pacific Association of Critical Care Medicine in July this year. The ISCCM is participating in the MOSAICS study which deals with the adherence to the sepsis bundles in Asian ICUs. 18 selected ICUs are contributing data to this study. Several ICUs have volunteered to participate in the WELPICUS study, which is an international study of the views and attitudes of doctors towards end-of-life care across the world. The ISCCM has been invited by the European Society of Intensive Care Medicine (ESICM) to be a signatory to the Patient Safety Declaration at the ESICM Congress to be held at Vienna later this year. The ISCCM is being increasingly recognized at an international level. This is a positive development. However, we must strengthen our role by engaging in good research, social purpose and commitment to education, training and safe practice of intensive care.

This year promises to be a busy year. I will keep in touch with you from time to time. You can of course write in your comments and suggestions by post or by email to **isccm1@vsnl.net**

Good bye for now.

# Lessons learnt

## 29th International Symposium on Intensive Care and Emergency Medicine

**24-27March, 2009 • Brussels- Belgium- Exhibition & Convention Centre**

**Dr. Subash Todi**
Head -
Dept. of Critical Care Medicine
AMRI Hospitals,
Dhakuria, Kolkata

Four day symposium organized by Prof. Vincent, in Brussels, Belgium was attended by around 5,000 delegates around the globe. Very comprehensive with wide range of topics were covered in details.

1. Prof Vincent in his opening remarks gave an insight on the current limitations of deriving conclusions from randomized clinical trials (RCT). In a survey he conducted, only ten RCT's have shown beneficial effect in the entire field of critical care till date. Most of the studies have shown neutral effect. The reason for this plethora of negative trials could be that selection of patients at two extremes of severity. As an analogy, small inferior wall myocardial infarct may not benefit from thrombolysis. Corticus trial showed no benefit from steroid as opposed to Annane study as it included less severe patients. Activated protein C was not found to be useful in less severe sepsis. He commented that future trials should exclude patients who are not likely to benefit from the intervention.

   Secondly, heterogeneity of "critically ill" patients makes patient selection difficult. Patients with syndromes like ARDS, SEPSIS, AKI, SIRS etc are included in trials but these constitute a very heterogenous group and within these groups a more homogeneous population should be selected. For example High PEEP in ARDS will be beneficial only to patients with recruitable lung and prone position is beneficial in patients with ARDS with severe hypoxemia.

   Thirdly, results of RCTs are difficult to generalize as only about ten percent of patient screened are ultimately included due to various criterias and difficulties in getting informed consent. He highlighted importance of Observational studies and Metaanalyses. Harmful effects of Blood transfusion was noted first in observational studies. Recently leucodepleted RBC transfusion was found to be associated with less mortality especially in patients with ischemic heart disease. This has also been seen in observational studies.

   Lastly, he pointed out that mortality as an endpoint should not be the main criteria, because most of the studies are not powered enough, and control group mortality is decreasing. Other end points like morbidity, quality of life, and cost should be considered as they are equally important to the patient and family.

2. A round table meeting on "ICU acquired weakness" was held two days prior to the congress which will be published in "critical Care" as a supplement. It was highlighted that there is a very high incidence of muscular weakness in almost two third of ICU patients, which is underrecognised. It can happen very early in the course of ICU illness on third or fourth day and adds greatly to patient's morbidity. It is also recognized that this weakness can last for years after ICU discharge and many of the ICU survivors never return to normal work. Multiple risk factors like immobility, mechanical ventilation, sepsis, drugs like steroids, neuromuscular blockers contribute to this weakness Bedside diagnosis specially in drowsy and sedated patients is very difficult and more aggressive use of electrophysiological studies need to be done to diagnose this entity. Early mobility even in intubated ventilated patient, and on inotropes is being practiced more widely and till now is the only way to prevent this form of weakness.

3. NICE SUGAR study was presented for the first time, which has now been published in NEJM, by Simon Finfer of ANZICS group, who showed that 90 day mortality was significantly less in patients with blood sugar 140-180 mg/dl than tight sugar control of 80-110 mg/dl. The algorithm they followed for sugar control has been described in their website.

4. Gastrointestinal system-

   Dr. Malbrain highlighted the present limitations of definitions of "GUT failure". He emphasized that though gut is the "motor for multiorgan failure", it is not included in severity scoring systems like SOFA score. He presented a scoring system for gut failure (GIF score).

   0- Normal g.i. function

   1- < 50 % of feed tolerated

   2- No feed tolerated or Intra abdominal hypertension

   3- No feed tolerated AND Intraabdominal hypertension

   4- Abdominal compartment syndrome

   He presented data on validation of this scoring system with outcome. Details of this scoring system and other information can be obtained from the website of World society of abdominal Compartment syndrome.

   He also emphasized the concept of abdominal compliance and plotting a pressure volume curve for abdominal pressure similar to in lung and heart. At a critical point any further volume will cause a sharp rise in intraabdominal pressure at which time further fluid loading will cause IAH, and in order to detect this continuous IAP monitoring with various devices was highlighted.

   He also emphasized that to prevent post operative IAH, IAP should be measured in the operation theater while closing the abdomen and two hours post operatively.

   In a symposium on pancreatitis, early enteral feeding was emphasized and if patient can tolerate gastric route was equally useful as nasojejunal feed. Use of prophylactic antibiotic was discouraged. Role of surgery was deemphasized and if necessary should only be performed after 3 to four weeks. CT guided drainage, minimally invasive laparoscopic assisted surgery or endoscopic drainage was discussed.

   In a symposium on feeding, early enteral feeding even in patients with shock, on vasopressor was not found to be harmful and can safely be practiced. The importance of cumulative energy and protein debt over ICU stay and their relation to bad outcome was emphasized. It was noted that there is much discrepancy in calculated and real energy and protein requirement in ICU patients and direct measurements like, indirect calorimetry and 24 hour urine for UUN should be more liberally used. Data from recent international survey, in which five Indian hospitals also participated conducted by Prof Heyland from Canadian critical group, was presented. Details of this

survey and further information can be obtained from their website www.criticalcarenutrition.org .

5. Fluid responsiveness.

   The limitations of static measures like CVP, PAOP and usefulness of dynamic parameters like SPV, PPV and SVV in assessing fluid responsiveness were discussed. A value of 10-12% was found to be a useful cut off in ventilated and sedated patient for assesing this response. Prof Teboul who has done maximum work in this field discussed fluid responsiveness in spontaneously breathing patient and found that passive leg raising and seeing changes in SVV or PPV is useful specially if patient is upright at 45 degree to start with and then put supine and leg raised. He also introduced a relatively new concept of assesing fluid responsiveness after an end expiratory pause and observing rise of PPV or SVV.

   A note of caution was also given by one of the speakers regarding fluid challenge. He emphasized that mere presence of volume responsiveness should not automatically lead to a volume challenge especially in patients with acute lung injury and one needs to measure extravascular lung water and be very cautious in fluid challenge if EVLW was rising.

   A further note of caution was given regarding interpretation of vascular pressures in patients with increased intraabdominal pressure. Roughly fifty percent of IAP is transmitted so this should be taken off all static pressure measurement to get a true vascular pressure. Moreover PPV is a better marker of fluid responsiveness in patients with increased IAP than SPV._

   Detrimental effects of saline resuscitation and risks of hyperchloremic acidosis was discussed in one session. Renal friendly profile of newer hydroxyethyl starch solution was emphasized. A novel concept of "total balanced volume replacement' was presented using physiologically balanced crystalloids and colloids.

6. Infectious disease.

   In a PRO-CON debate on descalation Prof Niederman emphasized the usefulness of de-escalation and cited data that showed that de-escalation can be safely practiced, even in culture negative patients. Descalated patients have less mortality in observational studies. Main emphasis of de-escalation is to take away carbapenem load on third day of broad spectrum if feasible.

   On the contrary, it was argued that descalation might be dangerous in an era of multidrug resistant infection and in culture negative patients and polymicrobial infection. It was suggested that decreasing duration of broad spectrum antibiotic therapy to five days guided by clinical features and biomarkers like procalcitonin, should be practiced.

   A plethora of new and fast diagnostic testing for bacteria and fungi which are PCR based and mainly applicable to Blood specimen was discussed. This methodology has a promising future for application of early and appropriate antibiotic therapy. It is also able to detect resistance genes in the bacterias. Studies comparing PCR with conventional blood cultures have shown PCR be particularly useful in patients who have been exposed to antibiotics prior to culture, in culture negative and in fungal sepsis. Prof. Reinhard cautioned regarding oversensitivity of these tests and need for more clinical research before utilizing them in daily practice.

   There was a full session on MDR bacterias and it was sobering to know that Europe is increasingly facing this problem , with Greece being affected worse , where rates of MDR acinetobacter is exceedingly high, Colistin is being used very commonly. Bad news is that and they have started seeing Colistin resistance. All the speakers emphasized that there are no new antibiotics for gram negative organism in the pipeline. Use of rifampicin along with colistin for MDR acinetobacter was advocated by some. Terminologies like Pan drug resistant (PDR), Multi drug resistant (MDR) and XDR need to be defined better.

   Dr Antonelli described epidemiology of acinetobacter and in a Q & A session mentioned that he was unable to find a common source for acinetobacter and most of the acquisition was from outside the ICU and it was very difficult to eradicate

   Use of aerosolized colistin and tobramycin was discussed and was found to be heplpful in Ventilator associated tracheobronchitis and as an adjunct to systemic therapy in VAP. Drug delivery system needs to be carefully chosen while delivering aerosol therapy.

   Use of Daptomycin in Vanc resistant enterococci and MRSA was highlighted.

   Selective decontamination of digestive tract was revisited and was found to be an excellent preventive mechanism in selected hospitals in Netherlands. Use of only oral decontamination with chlorhexidine was discussed. Concerns were raised regarding its role in ICUs with high background of resistant infection.

   Antibiotic stewardship was emphasized. Prospective audit and bed side discussion with primary consultant while the antibiotic was being prescribed was found to be the best solution to curb overuse of antibiotics .

   Role of "immunoparesis" in critically ill which makes them prone to nosocomial infection was discussed in details. Markers like HLA DR4 has been linked to immunosupression and patients expressing this are more likely to die of nosocomial sepsis. Use of Interferon as an immune adjuvant was suggested in these patients.

   A new technology using "Microfibres" which consists of nano size fibres was described and was found to be much more efficient in cleaning the environment and make it germ free than conventional washing.

   Benefits of hand washing with alcohol rubs was emphasized except in contact with c. diff, where spores are resistant to alcohol and washing with soap is manadatory. . Computer based surveillance program results in better implementation of antibiotic stewardship.

   Role of microbial surveillance in ICU was discussed. Routine surveillance of health care workers for MRSA and ESBL was discouraged. Focussed surveillance for intubated patient to predict microbiology of subsequent nosocomial sepsis was discussed. It was noted that further clinical studies are needed to clarify role of such approach.

7. Renal

   Use of new urinary biomarker like NGAL which is available as a bedside kit was highlighted for early detection of acute tubular necrosis. Dr carlos presented the results of a recently completed Australasian multicenter study in which a higher dose strategy (40 ml/kg) of renal replacement was compared to a loer dose (20 ml/kg) in 1508 patients with acute kidney injury. There was no difference between the groups in 90 day mortality rates, icu

length of stay, duration of mechanical ventilation, or development of organ dysfunction.

8. Pulmonary

Dr gattinoni presented outcome of a recent study on prolonged prone ventilation which was negative, but a subgroup analysis showed it to be useful in severely hypoxemic patient.

Dr Brochard pointed that an Upright position with legs dangling might be a more feasible and user friendly approach of positioning in ARDS.

Dr. Marco Ranieri presented the result of an Italian study comparing early (days 3-5) and late (days 10-12) tracheostomy, noting that early tracheostomy was not associated with any beneficial effect on development of VAP or mortality. Although the incidence of successful weaning was greater, ICU length of stay shorter, and sedative use less in the early group. Dr Duncan young reported similar results on equivalency of early vs late trach in TracMan study in U.K.

An Airway management workshop was conducted which displayed increasing use of Video assisted laryngoscopy in all intubation which has become standard of care in airway management.

An entire session was dedicated to patient ventilator synchrony and role of NAVA (Neurally adjusted ventilator assist).

9. Neurocritical care

Use of prognostic biomarker Copeptin which is derived from vasopressin has been studied extensively. It helps in triaging patients with TIA who are likely to get a stroke, prognosticates short and long term outcome in stroke patients.

In a tutorial on head injury use of cerebral tissue oxygen monitoring with minimally invasive device and monitoring metabolites of hypoperfusion like lactate with microdialysis technique was discussed. It was emphasized that ICP and CPP targeted therapies may not result in a better outcome unless coupled with improvement in tissue oxygen and meeting oxygen demand of cerebral tissue. Therapeutic moderate hypothermia in head injury and maintaining normothermia in stroke was discussed

Participation was invited for an ongoing study RescueICP on decompressive craniectomy vs conventional measures to decrease ICP

10. Quality control- New severity scoring systems like SAPS II, MPM III, and APACHE IV were deiscussed. It was highlighted that the previous scoring system overestimated ICU performance. Analysis of new scoring system have shown that contribution towards mortality due to acute physiology derangements has decreased substantially and at present it is the co morbidities which is determining patient outcome predominantly.

Having a daily checklist have been found to improve ICU performance

Dr.Kollef mentioned the SMART approach (specific, measurable,achievable, reliable, and time bound) for quality control.

**POSTER PRESENTATION**

Around 500 posters were displayed during the congresses which are available (free access) at the website www.ccforum.com. The following posters got Best Poster Prize

1. Adaptive Support ventilation prevents ventilator induced diaphragmatic dysfunction ; an in vivo piglet study- from france
2. Blunt abdominal trauma in children : a score to predict absence of organ injury – from Canada
3. Cox-2 and e selectin expression evaluation after acute normovolemic hemodilution- from brazil
4. Multicenter randomized trial of sedation using daily wake-up calls, bispectral index or clinical sedation scores in a mixed medical surgical ICU population – from Netherlands
5. The selective nicotine acetylcholine receptor agonist attenuates ventilator induced inflammation and lung injury – from Netherlands.

**EXHIBITS:**

State of the art exhibits were displayed during the congress details of which are available from www.intensive.org.

Airway Management- Video assisted laryngoscopy devices, Newer LMAs, Aerosol delivery devices, new humidification devices, newer endotracheal tube designs with micro cuffs, closed loop systems for maintaining cuff pressure, Nutrition- Enteral immunonutrition with Omega 3 fatty acid (OXEPA),

Urinary NGAL-biomarker for ATN-

Cooling devices- external and internal

Extracorporeal devices with polymyxin adsorbent, LPS adsorbent, CO2 removal

PCR based technology for bacterial and fungal detection. A stall offered MRSA screening in two minutes for the delegates.

Balanced Intravenous resuscitation fluids

Minimally invasive cardiac output monitoring with finger pressure measurement, bioreactance technology,esophageal Doppler

Tissue oxygen monitoring with NIRS (Near Infrared Spectroscopy) , microcirculation visualistion equipments, newer pulse oximeters with multiple wavelength and extra filters to provide accurate reading during low perfusion state and during transport, display of continuous non invasive hemoglobin, displaying Carboxy and methemoglobin, and plethysmographic variability for volume responsiveness.

Computerised clinical information system equipments

Pulmotrack- a novel wheeze monitor to track pulmonary sounds electronically through acoustics.

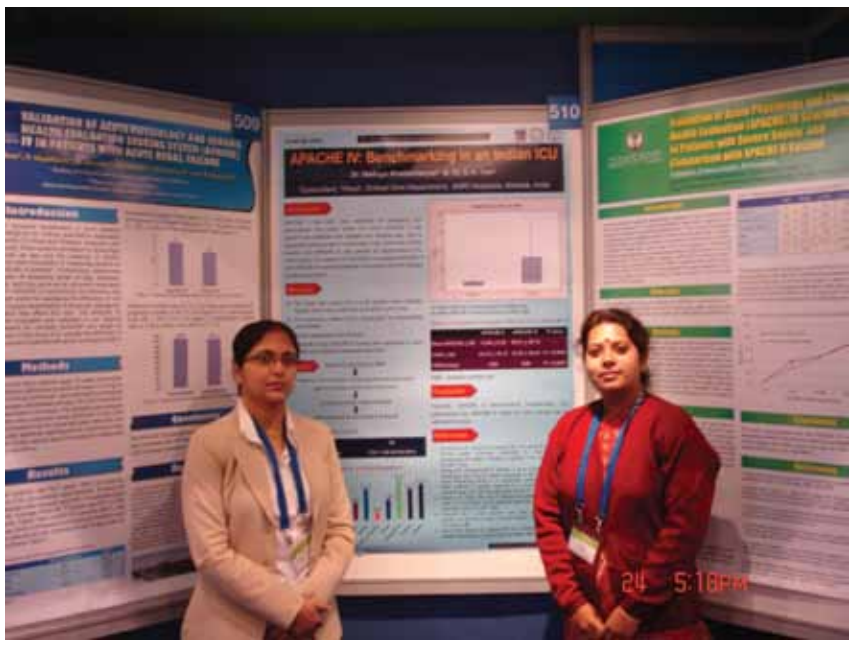
NAVA Adaptive support ventilation and high frequency ventilation devices.

Human prothrombin complex concentrates for massive bleeding

Intraabdominal pressure measurement equipment

Newer defibrillators with command prompt for inadequate CPR, alarm for hands off time, online display of adequacy of CPR, and filtered ECG to show background ecg rhytm while CPR is going on.

Final comments: Need more representation from India in the form of poster presentation and high quality scientific research.



# INDIAN SOCIETY OF CRITICAL CARE MEDICINE

## BRANCHES ALL OVER IN INDIA

### Committee Executive Members

**Agra**

| Chairman | Secretary | Treasurer | Exec. Members | Contact Person: Vijay Kumar Singhal |
|---|---|---|---|---|
| Dr. M.C.Gupta | Dr.V.K.Singhal | Dr.Atish Sharma | Dr. B.K.Singh, Dr.Ranvir Tyagi, Dr.Mod Yadav, Dr.M.S.Negi, Dr.Arvind Jain, Dr.K.K.Sharma | 15/376, Noori Gate, Agra, U.P - 282002<br>Telephone : 9837054578<br>E-Mail : drvksinghal@gmail.com |

**Ahemdabad**

| President | Secretary | Treasurer | Exec. Members | Contact Person : Raj Rawal |
|---|---|---|---|---|
| Dr.Pratibha Dileep | Dr.Raj Rawal | Dr.Bhavesh Patel | Dr.Parthiv Mehta, Dr.Harjit Dumbra, Dr.Jay Kothari, Dr.Jigar Mehta, Dr.Amit Patel | 307 K.K. Nagar, Sector 4, Ranna Park, Ahemdabad, Gujarat - 380061<br>Telephone : 9825327633<br>E-Mail : drrajrawal@hotmail.com |

**Aurangabad**

| President | Secretary | Treasurer | Exec. Members | Contact Person : Sadhna Kulkarni |
|---|---|---|---|---|
| Dr.J.S.Kandi | Dr. Sadhana Kulkarni | Dr. M.Khandewal | Dr.S.Gadappa, Dr.R.Z Gandhi, Dr. R.C Naik, Dr. V.P Magarkar, Dr.Menon | E-Mail : sudhir@agd2.dot.net.in |

**Bangalore**

**Contact Person :** Vineet Nayar
E-Mail : nayyar@bgl.vsnl.net.in

**Bareilly**

| President | Secretary | Exec. Members | Contact Person : Somesh Mohetra |
|---|---|---|---|
| Dr. Ajit Sawhney | Dr. Somesh Mehrotra | Dr. Ravi Khann, Dr.Sharad Agarwal, Dr.Sandeep Gupta, Dr.Manoj Rastogi, Dr.Amit Jauhri | |

**Bhopal**

| Chairman | Secretary | Treasurer | Exec. Members | Contact Person : Pradeep Bhattacharya |
|---|---|---|---|---|
| Dr.Ajay Goenka | Dr.Pradip Bhattacharya | Dr.Lalit Mehdiratta | Dr.Gopal Batni, Dr.Rajesh Bhagchandani, Dr.R.P.Kaushal, Dr.P.N Agarwal | Telephone : 9893181555<br>E-Mail : pradip_bhattacharya@hotmail.com |

**Bhubaneswar**

| Chairman | Vice- Chairman | Secretary | Treasurer | Contact Person : Damodar Bindhani |
|---|---|---|---|---|
| Dr.Samir Sahu<br>**Patron**<br>Dr.B.K.Mishra | Dr. Niraj Mishra | Dr.Damodar Bindhani<br>**Jt.Secretary**<br>Dr.Samir Mishra | Dr.Jagdish Rath | Plot No.3 ,Phase – III, Adarsh Vihar, Patia, Bhubaneswar, Orissa - 751024<br>Telephone : 0674-3116189<br>E-Mail : bindhanid@rediffmail.com |

**Chennai**

| Secretary | Treasurer | Exec. Members | Contact Person : Dr Babu Abraham |
|---|---|---|---|
| Dr. Babu Abraham | Dr S Mahendranan | Dr.Bala Ramchandran, Dr.Sanjay Prabhu, Dr.Indumathi Santhanam | Consultant Department of Critical Care Medicine, Apollo Hospital, Greams Lane, Greams Road, Chennai, Tamilnadu - 600006<br>Telephone : 09884286660<br>E-Mail : secretary.isccmchennai@gmail.com |

**Cochi**

**Coimbatore**

| President | Secretary | Treasurer | Exec. Members | Contact Person : N Sevalrajan |
|---|---|---|---|---|
| Dr.Y.Y.Rao | Dr.N.Selvarajan | Dr. M.N. Sivakumar | Dr. T. Mohan Kumar, Dr. M. Valliappan, Dr. C. Gopikrishnan, Dr. Kasturi Sudarshan | Old No.10 ,New No.9, 3rd Street Tatabad, Coimbatore, Karanatak - 641012<br>E-Mail : seldhana@eth.net |

**Gwalior**

| President | Secretary | Trasurer | Exec. Members | Contact Person : K. K. Tiwari |
|---|---|---|---|---|
| Dr. P.C.Mathur | Dr. K.K.Tiwari | Dr. Prashant Punekar | Dr. K.P Bhatnagar, Dr.Archana Gupta, Dr.J. Namdhari, Dr.Archana Kansal, Dr.S.Dhawale, Dr.Neelima Singh | Telephone : 9827336060 |

**Ghaziabad**

| Chairman | Secretary | Treasurer | Exec. Members | Contact Person : Ashish Agarwal |
|---|---|---|---|---|
| Dr. Deepak Agarwal | Dr. Ashish Agarwal | Dr. Vishwa Jindal | Dr. Sorabh Gupta, Dr. Ashish Prakash, Dr. Vivek Arora, Dr.Prahalad Chawla | K.B 119 ,Kavinagar, Ghaziabad, Uttarpradesh - 201001<br>Telephone : 9810307870 |

**Hyderabad**

| Chairperson | Secretary | Treasurer | Exec. Members | Contact Person : Palepu Gopal |
|---|---|---|---|---|
| Dr. Vijai Kumar | Dr. Palepu B.Gopal | Dr. Sunil Pandya | Dr. Manjunath Dinakar, Dr.R.Gopinath, Dr.Sadat Ali Kahna, Dr. Rajiv Paul | 101,Heritage Banjara, Panchavati Society, Road No3, Banjara Hills, Hyderabad, Andhra Pradesh - 500 03<br>Telephone : 9000361678<br>E-Mail : palepu_gopal@gmail.com |

**Indore**

| Chairman | Secretary | Trasurer | Exec. Members | Contact Person : Sanjay Dhanuka |
|---|---|---|---|---|
| Dr. Santosh Padhy | Dr. Sanjay Dhanuka | Dr. Sanjay Geed | Dr.Trishala Singhvi, Dr.Arun Arora, Dr.Hansmukh Gandhi, Dr. Santosh Ahuja | 201, Anu Apartment, 21-22, Shankar Nagar, Saket, Indore, Madhya Pradesh - 452018<br>Telephone : 0731-564314 |

**Jaipur**

| Chairman | Secretary | Treasurer | Exec. Members | Contact Person : Narendra Rungta |
|---|---|---|---|---|
| Dr.Narendra Rungta | Dr. Rungta Narendra | Manish Munjal | Dr. C.S.Sharma, Dr.Suresh Koolwal, Dr. Vinay Malhotra, Dr.Mukesh Sharma, Dr. S.S. Agarwal, Dr. Sudhir Khunteta | Telephone : 9829014237<br>E-Mail : drnrungta@gmail.com |

**Karamsad**

**Contact Person :** Jyoti Mannari
E-Mail : jyotigm@rediffmail.com

*Contd....*

### Kolkata

**Chairman**
Dr.Prof.Bibhukalyani Das

**Secretary**
Dr. Susruta Bandopadhyay

**Treasurer**
Dr. Animesh Dev

**Exec. Members**
Dr. Tapas Chakrabarty, Dr.Ramen Hazra, Dr.Saibal Chakraborty, Dr.Jaydip Biswas

**Contact Person :** Susruta Bandopadhyay
BA-179Salt Lake, Kolkata,
West Bengal - 700 06
Telephone : 9831079453

### Kolhapur

**Contact Person :** Vijay Karnad

### Lucknow

**Chairman**
Dr. A.K.Baronia

**Secretary**
Dr. Y.C/Govil

**Treasurer**
Dr. Afzal Azim

**Exec. Members**
Dr.Banani Poddar, Dr. B.P.Singh, Dr.Rajendra Prasad, Dr.A.Vashishtha, Dr.Ajai Kumar

**Contact Person :** A.K.Baronia
406/1, Balda Colony,New Hyderabad,
Lucknow, Uttar Pradesh - 226 00
Telephone : 0522-388461
E-Mail : ycgovil@lw1.net.in

### Mangalore

**Chairman** Dr.Chakrapani

**Secretary**
Dr.Sanjay Shastri

**Treasurer**
Dr. Damodar Shenoy

**Exec. Members**
Dr. Smitha Bhat, Dr . Vishnu Sharma, Dr. Yograj K.S, Dr.Jitendra Rai, Dr. Kishore Ubranwala

**Contact Person :** Santosh Soan
Medical Chambers, Don Bosco Cross Lane,
Kudumbi Gardens, Mangalore,
Karnataka - 575001
Telephone : 0824-442344
E-Mail : drsoans@yahoo.com

### Meerut

**Chairman** Dr.Adhip Mitra

**Secretary**
Dr.Veerottam Tomer

**Trasurer**
Dr.Sandeep Sehagal

**Exec. Members**
Dr.Y.P.Singh, Dr.Anil Kapoor Dr.Prashant Bandre, Dr.Atnu Mukherjee

**Contact Person :** Veerottam Tomer
A-4 Adarsh Nagar, Meerut-250001,
Uttar Pradesh
Telephone : 0983707182
E-Mail : manitomer@indiatimes.com

### Mumbai

**Chairman**
Dr.Dilip Karnad

**Secretary**
Dr. Vijaya Patil

**Traesurer**
Dr. Falguni Parikh

**Exec. Members**
Dr.Ajay Kantharia, Dr. Vatsal Kothari, Dr. Simran Singh

**Contact Person :** Vijaya Patil
Telephone : 9819883535
E-Mail : vijayappatil@yahoo.com

### Nagpur

**Chirman**
Dr.S.K.Deshpande

**Secretary**
Dr.Anand Dongre

**Treasurer**
Dr. Pratibha Deshmukh

**Exec. Members**
Dr. Rajendra Agarwal; Dr. Abhay Kelkar; Dr. Shubhangi Muley; Dr. Jayesh Timane; Dr.Girish Deshpande; Dr.Sudhir Bhutada; Dr. Deepak Jeswani

**Contact Person :** Anand Dongre
Swastik Critical Care Unit,
Near Lokmat Square,
Wardha Road, Nagpur, Maharashtra
Telephone : 9422101751
E-Mail : andydongre@hotmail.com

### Nashik

**Chairperson**
Dr.Deodatta Chafekar

**Secretary**
Dr.Suwarna Tambde

**Treasurer**
Dr.Pankaj Rane

**Exec. Members**
Dr.Yatindra Dube, Dr.Vijay Ghatge, Dr.Shrish Deo, Dr.Suderashana Patil

**Contact Person :** Suwarna Tambde
1, Sheetal Dhara, Govind Nagar, Nashik,
Maharashtra - 422009
Telephone : 9420592188
E-Mail : isccmnashik@gmail.com

### New Delhi

**Chairman**
Dr.Praveen Khilnani
**Vice Chairman**
Dr. Prakash Shastri
**Chairman , Scientific Committee**
Dr. Rajesh Chawla

**Secretary**
Dr. Suninder Arora

**Treasurer**
Dr. Deepak Govil

**Exec. Members**
Dr. Manoj Goel; Dr. Pankaj Kumar; Dr. Rajesh pande; Dr. Tarun Jhamb

**Contact Person :** Dr.Suninder Arora
Gate 10, 1st Floor, Room No. 4162,
Indraprashta Apollo Hospitals, Sarita Vihar,
Mathura Road, New Delhi - 110076
Telephone : 098101 13414
E-Mail : drssarora@gmail.com

### Pune

**Chairperson**
Dr.Kapil Zirpe

**Secretary**
Dr.Subhal Dixit

**Treasurer**
Dr.Shilpa Kulkarni

**Exec.Members**
Dr.Sameer Jog, Dr.Ajit Ambolkar, Dr.Sanjeev Gokhale, Dr.Pradeep 'D'costa, Dr.K.K.Uppadhya

**Contact Person :** Subhal Dixit
C/o Prayag Hospital, 1247, Deccan Gymkhana, Pune, Maharashtra - 411004
Telephone : 9820050240
E-Mail : subhaldixit@yahoo.com

### Sangli

**Contact Person :** Jyotsna Paranjepe
Telephone : 9423268066
E-Mail : san_sharduli@sancharnet.in

### Surat

**President**
Dr.Praful Chhasatia
**Vice President**
Dr.Nirmal Choraria

**Secretary**
Dr.Gaurish Gadbail
**Joint secretary**
Dr.Amish Shah

**Treasurer**
Dr.Vasant Panchal

**Exec. Members**
Dr.Mukul Petrolwala, Dr.Rajesh Prajapati, Dr.Ramesh Modi, Dr.Bhavin Lashkari, Dr.Pradeep Martin, Dr.Tejas Mehta, Dr.G.K.Patel

**Contact Person :** Gaurish
69,Sardarnagar Society,Somul dairy Road,
Surat, Gujarat - 395008
E-Mail : gaurish_g@hotmail.com

### Thrissur

Chairman
Dr.Jayaraj P.M

**Secretary**
Dr. Anudath K.B

**Treasurer**
Dr. J.S.Nivin

**Exec.Members**
Dr.Devaprasad S Dr.Francy Louis, Dr.Ravindran C, Dr.Elezabeth, Dr.Jyotish Gopinathan, Dr. Senoj C, Dr.Judo J.V, Dr.Benesh Krishna, Dr.Durga Prasan, Dr.Sreekumar T, Dr.Rajiv Zacharia, Dr.Pramod Mohan, Dr.Rennis Davis

**Contact Person :** Anudath K.B
5A Avenue Crest Apartment, Avenue Road,
Mudupalam, Thrissur, Kerala - 680006
Telephone : 9895899055

### Trivandrum

**Chairman**
Dr. Krishnakumar

**Secretary**
Dr.Deepak V.

**Treasurer**
Dr.Madhu

**Exec.Members**
Dr.Vivek, Dr.Suresh kumar V.K, Dr.Kesavan Nair, Dr.Sajeesh

Contact Person : **Deepak V.**
Poonilav , Bhagat Singh Road, Trivandrum,
Kerala - 274071
Telephone : 9895223210
E-Mail : dr_deepak_v@yahoo.com

### Vishkhapatnam

**Contact Person :** C Jayalaxmi
Vishkhapatnam, Andhra Pradesh

# INDIAN SOCIETY OF CRITICAL CARE MEDICINE

## NATIONAL EXECUTIVE COMMITTEE MEMBERS 2009 - 2010

| | | | |
|---|---|---|---|
| **PRESIDENT** | jdivatia@yahoo.com | **DR. J. V. DIVATIA,** *Mumbai* | 9869077435 |
| **PAST PRESIDENT** | rkmjs@vsnl.net | **DR. R.K. MANI,** *New Delhi* | 9811131719 |
| **PRESIDENT ELECT** | drchawla@vsnl.net | **DR. RAJESH CHAWLA,** *New Delhi* | 9810033395 |
| **VICE PRESIDENT** | apitxbk@vsnl.net | **DR. SIDDHARTH N. SHAH,** *Mumbai* | 9820077732 |
| | drnrungta@gmail.com | **DR. NARENDRA RUNGTA,** *Jaipur* | 9829014237 |
| | chughk2000@yahoo.co.in | **DR. KRISHAN CHUGH,** *New Delhi* | 9810608580 |
| | prajhans@gmail.com | **DR. PRASAD RAJHANS,** *Pune* | 9860077356 |
| **GENERAL SECRETARY** | cjani@vsnl.com | **DR. C. K. JANI,** *Mumbai* | 9892800883 |
| **SECRETARY** | drmmunjal@gmail.com | **DR. MANISH MUNJAL,** *Jaipur* | 9829062550 |
| **TREASURER** | kaivalyaak@yahoo.co.in | **DR. ATUL KULKARNI,** *Mumbai* | 9869077526 |
| **MEMBERS** | shyamsundert@rediffmail.com | **DR. T. SHYAM SUNDER,** *Hyderabad* | 9849038265 |
| | yatinmehta@hotmail.com | **DR. YATIN MEHTA,** *New Delhi* | 9810025816 |
| | dhruvachaudhry@yahoo.co.in | **DR. D. CHAUDHARY,** *Rohtak* | 9416051616 |
| | prakashshastri@live.in | **DR. PRAKASH SHASTRI,** *New Delhi* | 9810937295 |
| | sheila150@hotmail.com | **DR. SHEILA MYATRA,** *Mumbai* | 9820156070 |
| | drpradipkb@gmail.com | **DR. PRADIP BHATTACHARYYA,** *Bhopal* | 9893181555 |
| | drvparashar@yahoo.co.in | **DR. VINOD KUMAR PARASHAR,** *Jaipur* | 9414042057 |
| **ZONAL MEMBERS** | | | |
| **NORTH** | drdeepak@vsnl.com | **DR. DEEPAK GOVIL,** *New Delhi* | 9818056688 |
| **WEST** | subhaldixit@yahoo.com | **DR. S. DIXIT,** *Pune* | 9820050240 |
| **EAST** | drbray@tatasteel.com | **DR. B. RAY,** *Jamshedpur* | 9334800046 |
| **SOUTH** | mathewmohan@gmail.com | **DR. MOHAN MATHEW,** *Cochin* | 9447076652 |
| **CENTRAL** | drsp_critical@hotmail.com | **DR. SANTOSH PADHY,** *Indore* | 9893505906 |
| | | | |
| **CHAIRMAN PEDIATRIC SECTION** | rajivuttam@hotmail.com | **DR. RAJIV UTTAM,** *New Delhi* | 9810055670 |
| **CO-OPTED MEMBERS** | | | |
| **COURSE CO-ORDINATOR** | icudoctor@gmail.com | **DR. N.RAMKRISHNAN,** *Chennai* | 9840855115 |
| **CHAIRMAN EDUCATIONAL COMMITTEE** | ramer@vsnl.net | **DR. RAM RAJAGOPALAN** | 9841010859 |
| **EDITOR IJCCM** | drsandhya@hotmail.com | **DR. SANDHYA TALEKAR,** *Pune* | |
| **ORGANISING SECRETARY XV NATIONAL CONFERENCE, 2009** | criticareagra2009@gmail.com | **DR. B.K.SINGH,***Agra* | 9837040871 |
| **ORGNIZING SECRETARY XVI NAT. CONFERENCE** | palepu_gopal@hotmail.com | **DR. PALEPU GOPAL** | 9000361678 |
| **CHAIRMAN RESEARCH** | fnkapadia@gmail.com | **DR. FARHAD KAPADIA,** *Mumbai* | 9821031643 |

ISCCM

# INDIAN SOCIETY OF CRITICAL CARE MEDICINE

## Updated List of Recognized Institute and teachers of Indian Diploma in Critical care (IDCC) & Indian Fellowship in Critical Care (IFCC)

| CITY | INSTITUTE | NO OF SEATS | TEACHER | E-MAIL | MOBILE NO | OFFICE |
|---|---|---|---|---|---|---|
| **Bangalore** | Manipal Hospital **- Bangalore** | 4+2 | Dr. Padmakumar A. V. | padmakumar.a@manipalhospital.org | | 080-25266646 |
| | | | Dr. Somnath Chatterjee | somnath.chatterjee@manipalhospital.org | 9945120802 | |
| | | | Dr. Jose Chacko | jose.chacko@manipalhospital.org | | 080-25023284 |
| | Narayana Hrudayalaya **- Bangalore** | 2 | Dr. Murlidhar K. | kanchirulestheworld@gmail.com | 9844013108 | 080-27835000 |
| | | | Dr. Ratankumar Gupta | ratang@sify.com | 9844138504 | 080-27835000 |
| | | | Dr. Manjunath K. | | | |
| | | | Dr. Girish TN | | | |
| | St. John's Medical College **- Bangalore** | 2 | Dr. Sriram Sampath | sriramgs@yahoo.com | 9844094443 | 080-22065336 |
| | | | Dr. Amiya Chakravarty | | | |
| **Bhubaneshwar** | Kalinga Hospital **- Bhubaneshwar** | 2 | Dr. Sameer Sahu | samirsahu_kal@yahoo.co.in | 9437005552 | 0674-2300570 |
| **Chennai** | Apollo Hospital **- Chennai** | 4+2 | Dr. N. Ramakrishnan | icudoctor@gmail.com | | 044-28293333 |
| | | | Dr. Babu Abraham | bary63@yahoo.com | 9884286660 | 044-28293333 |
| | | | Dr.Ramesh Venkataraman | | | |
| | Apollo First Med Hospital **- Chennai** | 2 | Dr. Ashwin kumar Mani | rskumar@vsnl.net | 9840480482 | 044-28296517 |
| | Apollo Specialities Chennai | 2 | Dr. R. Senthilkumar | | - | |
| | SRMC & Research Institute **- Chennai** | 2 | Dr. A. S. Arunkumar | asakumarmenon@yahoo.com | 9841172511 | 044-24765589 |
| | Sundaram Medical Foundation **- Chennai** | 3+1 | Dr. Ram Rajagopalan | ramer@vsnl.net | 9841010859 | 044-26268844 |
| | | | Dr. S. Mahendran | smahend@smfhospital.og | | 044-26268844 |
| **Coimbatore** | Kovai Medical Center & Hospital **- Coimbatore** | 2 | Dr. N. Selvarajan | drseldhana@gmail.com | | 0422-827784 |
| **Faridabad** | Escorts General Hospital **- Faridabad** | 2+1 | Dr. Sandip Bhattacharya | sandipfbd2001@yahoo.co.in | 9818590021 | 011-22543027 |
| **Guragaon** | Artemis Hospital, Gurgaon | 2 | Dr. Deepak Govil | drdeepak_govil@yahoo.co.in | 9818056688 | |
| | | | Dr. Ajay Gupta | | | |
| **Hyderabad** | Nizams Institute Of Medical Sciences **- Hyderabad** | 2 | Dr. Tirumala Suhasini | tsuhasini@hotmail.com | 9848512668 | 040-23320332 |
| | Kamineni Wockhardt Hospital **- Hyderabad** | 2 | Dr. Shyamsunder | shyamsundert@rediffmail.com | 9849038265 | 040-55924444 |
| | Care Hospital -**Hyderabad** | 2 | Dr. S. Srinivas | srinivas3271@gmail.com | 9866343632 | 30418141 |
| | | | Dr. V. Rajendra | rvajrapu@yahoo.co.uk | 9959166641 | 040-40159785 |
| | | | Dr. Ram Paparao | rampaparaodr@gmail.com | 9898042425 | |
| **Jaipur** | Santokba Durlabhji Hospital **- Jaipur** | 2 | Dr. G .R. Singhvi | sdmhos@sancharnet.in | 9829051418 | 0141-2566251 |
| | Rungta Hospital **- Jaipur** | 2 | Dr. Narendra Rungta | drnrungta@gmail.com | 9829014237 | 2522000 |
| | S. K. Soni Hospital - **Jaipur** | 2 | Dr. Suresh Bhargava | suresh3559@yahoo.com | 9829063830 | 0141-2232408 |
| | | | Dr. Brijesh Bharadwaj | brijesh2150@yahoo.com | 9829063830 | 0141-2232408 |
| | Fortis Hospital- Jaipur | 2 | Dr. Shabbar Joad | | | |
| | | | Dr. Narendra Rungta | drnrungta@gmail.com | 9829014237 | |
| **Jamshedpur** | Tata Main Hospital **- Jamshedpur** | 2 | Dr. B. Ray | bray@tmh.tatasteel.com | | 0657-423775 |
| | | | Dr. Deviprasad Samaddar | dpsamaddar@yahoo.com | 9835112931 | 0657-423775 |
| **Kochi** | Lakeshore Hospital **- Kochi** | 2 | Dr. Mohan Mathew | mathewmohan@gmail.com | 9447076652 | 0484-2701032 |
| **Kolkata** | AMRI Apollo Hospital **- Kolkata** | 2+1 | Dr. Subhash Todi | subhashtodi@vsnl.com | 9831202040 | 23557710 |
| **Mumbai** | Breach Candy Hospital **- Mumbai** | 4+2 | Dr. F. E. Udwadia | drfeudwadia@gmail.com | | 022-23871888 |
| | | | Dr. Mangal Jain | | | |
| | Bombay Hospital **- Mumbai** | 4+2 | Dr. Pravin Amin | pamin@vsnl.com | 9820121455 | 022-2067676 |
| | | | Dr. Shruti Nagarkar | madnag@gmail.com | | |
| | Lilavati Hospital **- Mumbai** | 4+2 | Dr. Prakash Jiandani | mariyapj@yahoo.com | 9820292123 | 022-26455891 |
| | | | Dr. Conrad Rui Vas | mariyapj@yahoo.com | 9820125932 | 022-26427252 |
| | Jaslok Hospital **- Mumbai** | 4+2 | Dr. Jamshed Sunavala | sunavala@rediffmail.com | | 022-66573299 |
| | | | Dr. Mayur Patel | drmayurpatel@hotmail.com | 9833645119 | 022-67570111 |
| | P. D. Hinduja Hospital **- Mumbai** | 4+2 | Dr. Ashit Hegde | ashit_hegde@vsnl.com | | 022-24451515 |
| | | | Dr. Khusrav Bajan | drkhus@vsnl.com | | 022-24452222 |
| | | | Dr. Simran Singh | fnkapadia@gmail.com | | |
| | | | Dr. Farhad Kapadia | fnkapadia@gmail.com | | |
| | Saifee Hospital **- Mumbai** | 2 | Dr. C. K. Jani | cjani@vsnl.com | 9821124738 | 022-67570111 |
| | Prince Aly Khan **- Mumbai** | 2 | Dr. Kedar Toraskar | kedartoraskar@hotmail.com | 9819844451 | 022-23777800 |
| | Hurkisondas Hospital **- Mumbai** | 2 | Dr. Ajay Kantharia | ajaykantharia@hotmail.com | | 022-30095555 |
| | | | Dr. Ashit Bhagwati | ashit_bhagwati@yahoo.com | 9821172455 | 022-56660000 |
| | Wockhardt Hospitals Ltd. **- Mumbai** | 2 | Dr. Robert D'costa | robert@vsnl.com | 9821090708 | 022-67994444 |
| | Tata Memorial Hospital- **Mumbai** | 2 | Dr. Atul Kulkarni | kaivalyaak@yahoo.co.in | 9869077526 | 022-24146750 |
| | | | Dr. Vijaya Patil | vijayappatil@yahoo.com | | |
| | | | Dr.Jigi Divatia | jdivatia@vsnl.com | 9869077435 | |
| | Hiranandani Hospital - **Mumbai** | 2 | Dr. Arpita Dvivedi | | | |
| | | | Dr. Laxmi Kamat | | | |
| **Nagpur** | Dr. Tambe's Hospital & ICU - **Nagpur** | 2 | Dr. Vilas Tambe | tambe_vilas@rediffmail.com | 9823036113 | 0712-220203 |
| **New Delhi** | Escorts Heart Institute **- New Delhi** | 2+1 | Dr. Anil Karlekar | karlekar@gmail.com | 9810266905 | 011-26825000 |
| | | | Dr. Sunil Dhole | sunildhole@hotmail.com | 9818797544 | 011-26825000 |
| | Max Superspecilality **- New Delhi** | 2 | Dr. Omender Singh | omender.critical@yahoo.com | 9810734246 | 011-26525555 |
| | | | Dr. P. N. Kakkar | pn_kakar@hotmail.com | 9810853355 | 011-66115050 |
| | Indraprastha Apollo Hospital **- New Delhi** | 4+2 | Dr. Sudha Kansal | kansalsudha@yahoo.com | 9810399558 | 011-26925858 |
| | | | Dr. Rajesh Chawla | drchawla@vsnl.net | 9810033395 | 011-26925858 |
| | Sir Gangaram Hospital **- New Delhi** | 4+2 | Dr. Prakash Shastri | prakashshastri@hotmail.com | 9810937295 | 011-25868562 |
| | | | Dr. Brijendra Rao | drbkrao@yahoo.co.in | 9811177755 | 011-42252401 |
| | | | Dr. Sumit Ray | drsray67@yahoo.co.in | 9810614433 | 011-42252404 |
| | Fortis Hospital, Vasant Kunj **- New Delhi** | 2 | Dr. R. K. Mani | rkmjs@vsnl.net | 9811131719 | 011-2 6925858 |
| | | | Dr. Amit Varma | amit.varma@fortishealthcare.com | 9818355665 | 011-26325971 |
| **Noida** | Fortis Hospital **- Noida** | 2 | Dr. Mrinal Sircar | mrinalsircar@bol.net.in | 9810536268 | 120-2400222 |
| **Pune** | Deenanath Mangeshkar Hospital - **Pune** | 4+2 | Dr. Sameer Jog | drjogs@vsnl.net | | 020-25452828 |
| | | | Dr. Vinayak Desurkar | | | |
| | | | Dr. Rajhans Prasad | prajhans@gmail.com/ icu@dmhospital.org | 9860077356 | 020-4023156 |
| | Jehangir Hospital **- Pune** | 4+2 | Dr.Kayanoosh Kadapatti | drkayanoosh@gmail.com | | 020-26050550 |
| | | | Dr. Shivakumar Iyer | s_iyer@vsnl.com | 9822051719 | 020-26122551 |
| | K. E. M. Hospital **- Pune** | 4+2 | Dr. Rajesh Gadia | drbandebd@hotmail.com | | 020-625600 |
| | | | Dr. Balasaheb Bande | drbandebd@hotmail.com | 9822051593 | |
| | | | Dr. Leena Shah | l4shah@hotmail.com | 9850811659 | |
| | | | Dr. Pradeep D'costa | | | |
| | Ruby Hall Clinic **- Pune** | 4+2 | Dr. Kapil Zirpe | kapilzirpe@gmail.com | 9823166212 | 020-25454644 |
| | | | Dr. Prachee Sathe | prachee.sathe@gmail.com | 9823064906 | 020-4125243 |
| | | | Dr. Shilpa Kulkarni | shilpadeshkar@yahoo.com | 9850042103 | |
| | Shree Medical Foundation **- Pune** | 2+1 | Dr. Shirish Prayag | critical@vsnl.com | 9822012390 | 020-25532812 |
| | Prayag Hospital | | Dr. Subhal Dixit | subhaldixit@yahoo.com | 9822050240 | 020-25532812 |
| | Lokmanya Hospital **- Pune** | 2 | Dr. Lalitha Pillai | lalithapillai29@rediffmail.com | 9822478277 | 020-2765496 |
| | Inlaks & Budhrani Hospital **- Pune** | 2 | Dr Vilas Gundecha | vpgundecha@yahoo.com | 9422089990 | 020-56099999 |
| | Niramaya Hospital **- Pune** | 2 | Dr. Ajit Yadav | ajityadav@yahoo.com | 9822030711 | 020-27441860 |
| | Aditya Birla Hospital Pune | 2 | Dr. Jayant Shelgaonkar | jayant@doctors.org.uk | 9881288205 | 020-3071785 |
| **Vellore** | Christian Medical College **- Vellore** | 4+2 | Dr. Nagamani Sen | sicu@cmcvellore.ac.in | | 0416-2282693 |
| | | | Dr. George John | yokavi@yahoo.com | 9443626986 | 0416-2282039 |
| | | | Dr. Subramani | ksubramani9@hotmail.com | 9486046929 | 0416-2222102 ,319 |
| | | | Dr. J. V. Peter | peterjohnvictor@yahoo.com.au | 9840184345 | 0416-2282693 |

**Can also visit website www.isccm.org for these details**

Society of Critical Care Medicine
The Intensive Care Professionals

# Treating Community- and Healthcare-Associated MRSA

## Is There a Difference and Does It Matter?

**Samuel Marquez,**
MD, MA
Surgical Critical Care Fellow
University of Minnesota
Minneapolis, Minnesota, USA

**Greg Beilman,**
MD
Professor and Vice Chair of Surgery,
Chief of Surgical Critical Care,
University of Minnesota Medical Center,
Minneapolis, Minnesota, USA

Methicillin-resistant *Staphylococcus aureus* (MRSA) first emerged as a pathogen in the early 1960s. It has been cultured and isolated in soft tissue infections with increasing frequency within a broad range of healthcare settings. *S. aureus* gains its resistance via a gene complex known as staphylococcal cassette chromosome *mec* (SCC*mec*), which contains the *mec*A methicillin-resistance gene.

MRSA has continued to evolve within two distinct settings with marked clinical and genetic characteristics. Healthcare-associated MRSA (HA-MRSA) first evolved as a pathogen in the 1960s and is common in patients with multiple medical comorbidities and prolonged hospitalization. Risk factors for HA-MRSA infection include immunosuppression, diabetes, and recent or frequent hospitalization. Interestingly, in the late 1990s, a new community-associated MRSA (CA-MRSA) was recognized worldwide. It was first described, and continues to be identified, in individuals without significant typical risk factors for HA-MRSA. Importantly, however, the incidence of CA-MRSA is not distributed uniformly throughout a community. Most patients with confirmed CA-MRSA come from subsets of people with low economic status, individuals in close living conditions, children and young adults, and minority groups.[1,2] Although the distinctions between CA-MRSA and HA-MRSA increasingly overlap, their biologic and genetic distinctions remain unchanged.[2] Between 1998 and 2003, MRSA isolates increased dramatically, with CA-MRSA accounting for a significant amount of the upturn.[1]

There are a number of distinctions that separate HA-MRSA and CA-MRSA. On the level of biologic behavior, CA-MRSA isolates tend to be susceptible to antimicrobial classes other than penicillin. Most CA-MRSA remains susceptible to clindamycin and trimethoprim/sulfamethoxazole. However, reports now show that some CA-MRSA clones are demonstrating resistance patterns toward these antibiotics. A key feature of many of these isolates is an inducible resistance to clindamycin, carried by the *erm* gene. It is important to be suspicious of this situation, detected with a D-test in the microbiological laboratory. In the D-test, erythromycin and clindamycin disks are dropped on a plate 15 mm apart. MRSA with the *erm* gene show and, when exposed to erythromycin, develop resistance to clindamycin and demonstrate the characteristic flattening on the erythromycin side of the clindamycin disk (thus the "D" shape).

On the genetic level, notable differences are also present. SCC*mec* typing, pulsed-field gel electrophoresis (PFGE), and the presence of the toxin Panton-Valentine leukocidin (PVL) are three commonly used methods of classifying and tracking MRSA clones.[3] SCC*mec* IV accounts for 78% of CA-MRSA cases and SCC*mec* II accounts for 78% of HA-MRSA cases. Community-acquired strains are frequently PVL positive (69%), whereas hospital-acquired strains are positive infrequently (4.8%). More than 95% of PVL-positive MRSA are SCC*mec* IVa. PFGE is used to evaluate the DNA banding pattern of MRSA; the banding pattern type USA300 accounts for nearly all CA-MRSA.[4]

It is hypothesized that it is the PVL toxin that makes CA-MRSA strains pathogenic. The mechanism of how PVL might contribute to or be responsible for the increased virulence of CA-MRSA is still not well elucidated. In nearly all published series of MRSA-associated necrotizing soft tissue infections (NSTI), the species are PVL positive.[5]

Severe and necrotizing soft tissue infections have been lumped together under various names, including gas gangrene, necrotizing fasciitis and many others. Given these variable descriptors, it would seem that these infections would best be described based on the soft tissue layer(s) involved (e.g., skin, adipose tissue, fascia or muscle) and the causative organism. NSTI traditionally is caused by group A *Streptococcus*, *Clostridium perfringens*, or a mixture of aerobic and anaerobic organisms (Table 1). The

reported mortality for NSTI historically has been in the range of 30% to 80%, with 100% morbidity due to the need for multiple disfiguring procedures.

**Table 1 : Causative Organisms of Necrotizing Soft Tissue Infections**

| Causative Organism | Frequency |
|---|---|
| Polymicrobial: Gram-negative and Gram-positive aerobes and anaerobes | 50% |
| *Streptococcus pyogenes* | 20% - 30% |
| *Clostridium perfringens* | 20% - 30% |
| CA-MRSA | 10%, increasing |

CA-MRSA as a sole causative agent in NSTI was first described widely in 2005. In a series examining soft tissue infections attributable to CA-MRSA in young military personnel, we described a case of NSTI caused by this organism.[1] Also in 2005, Miller et al described 14 cases of NSTI attributable to CA-MRSA.[6] Of the patients in that series, only five had specimens available for detailed genotype analyses. All were CA-MRSA, carried type IV SCC*mec* elements, were PVL-positive and had the USA300 DNA banding pattern. No deaths were reported in this group. The most recent series, from the University of Colorado Health Sciences Center, noted that 16.7% of their NSTI cases between 2004 and 2006 were caused by CA-MRSA clones that were USA300- and PVL-positive.[7]

Another interesting feature of NSTI caused by CA-MRSA is that up to one-third of cases originally were believed to be spider bites, even in areas where spiders typically associated with necrotic bites, such as the brown recluse spider, are not endemic. Several studies have evaluated brown recluse spiders as a potential carrier of MRSA, with an overwhelmingly negative conclusion.[6-7] It is more likely that the appearance of these two conditions is similar enough that patients, in particular, attribute the lesion to a spider bite. However, such bites typically are necrotic without purulence, allowing the astute clinician to distinguish these lesions.

One of the fundamental tenets of treating NSTI is aggressive and early surgical debridement, with many cases requiring several debridements and multiple procedures to completely eradicate infection. Because outcome is critically related to early debridement, patients with suspected NSTI should be evaluated immediately by a surgeon without significant delay for radiologic evaluation. Diagnosis is typically made in the operating room, usually after finding dishwater edema fluid and discovering the easy separation of fascia from overlying soft tissue. Confirmation of the diagnosis can be made in questionable cases if frozen section evaluation reveals polymorphonuclear leukocytes infiltrating the fascia. Debridement should include all involved fascia, muscle and short tissue, and should be repeated daily until no progression of infection is identified.

Antimicrobial coverage for NSTI historically has not included agents with activity against MRSA, but given the increasing incidence of this specific organism in multiple areas of the world, antimicrobial coverage should be broad and should include agents with activity against MRSA. Initial selection of an antibiotic regimen should include agents with coverage against Gram-negative bacteria, *Clostridium* species, β-hemolytic *Streptococcus* species, and MRSA (Table 2). Many authors suggest including agents that inhibit protein synthesis (e.g., clindamycin, daptomycin or linezolid). Antimicrobial coverage should be tailored appropriately when culture results become available.

**Table 2 : Recommended Empiric Antimicrobial Coverage for Necrotizing Soft Tissue Infections**

| Organism Suspected | Antimicrobial Agent Choices |
|---|---|
| MRSA and other Gram-positive organisms* | Vancomycin, daptomycin, clindamycin, linezolid |
| Gram-negative organisms | Carbipenem, piperacillin-tazobactam, ticarcillin-clavulanate |
| Clostridial pathogens | High-dose aqueous penicillin G |

*Daptomycin, clindamycin and linezolid all inhibit protein synthesis.

Many hospitals are isolating CA-MRSA patients, blurring the line of distinction between CA-MRSA and HA-MRSA. Despite their outlined differences, the treatment algorithm currently does not differ between these two distinct types of organisms. This may change if new agents are developed with the ability to inhibit PVL toxin activity.

In summary, CA-MRSA is an increasingly common causative organism in NSTI. Agents selected for initial empiric treatment should include those with activity against MRSA. Population susceptibility and genetic and biologic response profiles of CA-MRSA differ from those of HA-MRSA and should be noted; however, the clinical management is identical. Early and repeated surgical debridement with cultures for source control, as well as early and appropriate antimicrobial coverage, remain mandatory components for the management of these tissue infections.

**References and disclosures are available at www.sccm.org/criticalconnections.**

Reprinted with permission from the Society of Critical Care Medicine, USA: *Critical Connections,* Volume 8, Number 1, February 2009.

JOIN THE SOCIETY OF CRITICAL CARE MEDICINE (SCCM) IN

# MIAMI, FLORIDA, U.S.A.

... for the largest multiprofessional critical care event of the year. Be one of the more than 6,000 nurses, pharmacists, physicians, respiratory therapists, students, veterinarians, and other healthcare professionals from around the world to benefit from:

- CUTTING-EDGE EDUCATIONAL SESSIONS
- HANDS-ON WORKSHOPS
- THOUGHT-PROVOKING PLENARY SESSIONS
- CAPTIVATING SPONSORED SYMPOSIA
- NEARLY 200 EXHIBITORS
- UP-TO-DATE, EVIDENCE-BASED DEVELOPMENTS IN CRITICAL CARE MEDICINE

Also plan to attend the Post-Congress event in Key West, Florida, USA, for more vital information to help you take your practice of critical care to the next level.

Registration for the 39th Critical Care Congress and the Post-Congress session will open in June 2009. Please watch www.sccm.org for further details.

39th Critical Care Congress
MIAMI BEACH
JANUARY 9-13, 2010

**RESERVE THESE DATES TODAY:**

January 9-13, 2010
39th Critical Care Congress

January 13-15, 2010
Post-Congress

Society of Critical Care Medicine
The Intensive Care Professionals

**EXPERIENCE SCCM'S COMPLETE CONGRESS PACKAGE.**

**WWW.SCCM.ORG**

# Journal Scan

**Dr. Sudha Kansal** MD
**Dr. Tarun Jhamb** MD

Dept of Critical Care Medicine,
Indraprastha Apollo Hospitals,
New Delhi

## Intensive versus Conventional Glucose Control in Critically Ill Patients

The NICE-SUGAR Study Investigators Dean R. Chittock, William R. Henderson, Daren K. Heyland, et al.

*N Engl J Med, March 2009; 360:1283-97.*

The optimal target range for blood glucose in critically ill patients remains unclear.

Within 24 hours after admission to an intensive care unit (ICU), adults who were expected to require treatment in the ICU on 3 or more consecutive days were randomly assigned to undergo either intensive glucose control, with a target blood glucose range of 81 to 108 mg per deciliter (4.5 to 6.0 mmol per liter), or conventionalglucose control, with a target of 180 mg or less per deciliter (10.0 mmol or less per liter). We defined the primary end point as death from any cause within 90 days after randomization.

Of the 6104 patients who underwent randomization, 3054 were assigned to undergo intensive control and 3050 to undergo conventional control; data with regard to the primary outcome at day 90 were available for 3010 and 3012 patients, respectively. The two groups had similar characteristics at baseline. A total of 829 patients (27.5%) in the intensive-control group and 751 (24.9%) in the conventional-control group died (odds ratio for intensive control, 1.14; 95% confidence interval, 1.02 to 1.28; P = 0.02). The treatment effect did not differ significantly between operative (surgical) patients and nonoperative (medical) patients (odds ratio for death in the intensive-control group, 1.31 and 1.07, respectively; P = 0.10). Severe hypoglycemia (blood glucose level, ≤40 mg per deciliter [2.2 mmol per liter]) was reported in 206 of 3016 patients (6.8%) in the intensive-control group and 15 of 3014 (0.5%) in the conventional-control group (P<0.001). There was no significant difference between the two treatment groups in the median number of days in the ICU (P = 0.84) or hospital (P = 0.86) or the median number of days of mechanical ventilation (P = 0.56) or renal-replacement therapy (P = 0.39).

In this large, international, randomized trial, we found that intensive glucose control increased mortality among adults in the ICU: a blood glucose target of 180 mg or less per deciliter resulted in lower mortality than did a target of 81 to 108 mg per deciliter.

## Outcomes Associated with Delirium in Older Patients in Surgical ICUs

Michele C. Balas, Mary Beth Happ, Wei Yang, et al.

*Chest, January 2009; 135:18-25*

Older adults admitted to surgical ICUs (SICUs) are at high risk for delirium. In the current study, the author describes the association between the presence of delirium and complications in older SICU patients, and describe the association between delirium occurring in the SICU and functional ability and discharge placement for older patients.

Subjects were 114 consecutive patients ≥ 65 years old admitted to a surgical critical care service. All subjects underwent daily delirium and sedation/agitation screening during hospitalization. Outcomes prospectively recorded included SICU complication development, discharge location, and functional ability (as measured by the Katz activities of daily living instrument).

Nearly one third of older adults (31.6%) admitted to an SICU had a complication during ICU stay. There was a strong association between SICU delirium and complication occurrence (p = 0.001). Complication occurrence preceded delirium diagnosis for 16 of 20 subjects. Subjects with delirium in the SICU were more likely to be discharged to a place other than home (61.3% vs 20.5%, p < 0.0001) and have greater functional decline (67.7% vs 43.6%, p = 0.023) than nondelirious subjects. After adjusting for covariates including severity of illness and mechanical ventilation use, delirium was found to be strongly and independently associated with greater odds of being discharged to a place other than home (odds ratio, 7.20; 95% confidence interval, 1.93 to 26.82).

Delirium in older surgical ICU patients is associated with complications and an increased likelihood of discharge to a place other than home.

## Radiologic Progression of Pulmonary Infiltrates Predicts a Worse Prognosis in Severe Community-Acquired Pneumonia Than Bacteremia

Thiago Lisboa, Stijn Blot, Grant W. Waterer, et al.

*Chest, January 2009; 135:165-172*

It remains unknown whether bacteremia and rapid radiologic progression of pulmonary infiltrates increase the risk of shock and mortality in ICU patients with community-acquired pneumonia (CAP). The objective of this study was to investigate the relative importance of these two factors in the outcome of patients with severe CAP (sCAP).

A secondary analysis in a multicenter observational study was conducted in 457 patients with CAP admitted to the ICU. Patients were classified into four groups: group RB, rapid radiographic spread of pulmonary infiltrates and bacteremia (n = 48); group R, rapid radiographic spread but no bacteremia (n = 183); group B, bacteremia but without rapid radiographic spread (n = 39); and group C, neither rapid radiographic spread nor bacteremia (n = 187).

Logistic regression analysis showed that group RB and group R had a greater risk for shock than group C (adjusted odds ratio [aOR], 8.9; 95% confidence interval [CI], 4.0 to 19.7; and aOR, 3.8; 95% CI, 2.5 to 5.9; respectively), while patients in group B had no increased risk. In addition, compared to group C, group RB and group R had an increased risk of ICU death, while patients in group B had none.

In this cohort of patients with severe CAP, radiologic progression of pulmonary infiltrates in the first 48 h is a significant adverse prognostic feature. In contrast, bacteremia does not affect outcomes.

## Ventilator-Associated Tracheobronchitis The Impact of Targeted Antibiotic Therapy on Patient Outcomes

Donald E. Craven, Alexandra Chroneou, Nikolaos Zias, et al.

*Chest, February 2009; 135:521-528*

Nosocomial lower respiratory tract infections are a common cause of morbidity and mortality in ICU patients receiving mechanical ventilation. Many studies have investigated the management and prevention of ventilator-associated pneumonia (VAP), but few have focused on the role of ventilator-associated tracheobronchitis (VAT). The pathogenesis of lower respiratory tract infections often begins with tracheal colonization that may progress to VAT, and in selected patients to VAP. Since there is no well-established definition of VAT, discrimination between VAT and VAP can be challenging. VAT is a localized disease with clinical signs (fever, leukocytosis, and purulent sputum), microbiologic information (Gram stain with bacteria and leukocytes, with either a positive semiquantitative or a quantitative sputum culture), and the absence of a new infiltrate on chest radiograph. Monitoring endotracheal aspirates has been used to identify and quantify pathogens colonizing the lower airway, to diagnose VAT or VAP, and to initiate early, targeted antibiotic therapy. Recent data suggest that VAT appears to be an important risk factor for VAP and that targeted antibiotic therapy for VAT may be a new paradigm for VAP prevention and better patient outcomes.

## Predicting volume responsiveness by using the end-expiratory occlusion in mechanically ventilated intensive care unit patients

Xavier Monnet, David Osman, Christophe Ridel, et al.

*Crit Care Med, Mar 2009; 37: 951-956*

During mechanical ventilation, inspiration cyclically decreases the left cardiac preload. Thus, an end-expiratory occlusion may prevent the cyclic impediment in left cardiac preload and may act like a fluid challenge. The author tested whether this could serve as a functional test for fluid responsiveness in patients with circulatory failure.

In this prospective study.

Medical intensive care unit.

Thirty-four mechanically ventilated patients with shock in whom volume expansion was planned.

A 15-second end-expiratory occlusion followed by a 500 mL saline infusion.

Arterial pressure and pulse contour-derived cardiac index (PiCCOplus) at baseline, during passive leg raising (PLR), during the 5-last seconds of the end-expiratory occlusion, and after volume expansion.

Volume expansion increased cardiac index by >15% (2.4 +/- 1.0 to 3.3 +/- 1.2 L/min/m2, p < 0.05) in 23 patients ("responders"). Before volume expansion, the end-expiratory occlusion significantly increased arterial pulse pressure by 15% +/- 15% and cardiac index by 12% +/- 11% in responders whereas arterial pulse pressure and cardiac index did not change significantly in nonresponders. Fluid responsiveness was predicted by an increase in pulse pressure >=5% during the end-expiratory occlusion with a sensitivity and a specificity of 87% and 100%, respectively, and by an increase in cardiac index >=5% during the end-expiratory occlusion with a sensitivity and a specificity of 91% and 100%, respectively. The response of pulse pressure and cardiac index to the end-expiratory occlusion predicted fluid responsiveness with an accuracy that was similar to the response of cardiac index to PLR and that was significantly better than the response of pulse pressure to PLR.

The hemodynamic response to an end-expiratory occlusion can predict volume responsiveness in mechanically ventilated patients.

## Argatroban for Anticoagulation in Continuous Renal Replacement Therapy

Andreas Link, Matthias Girndt, Simina Selejan, et al.

*Crit Care Med, Jan 2009; 37:105-110*

Argatroban, a direct thrombin inhibitor, was evaluated for anticoagulation in continuous renal replacement therapy (CRRT) in critically ill patients with heparin-induced thrombocytopenia type II and acute renal failure. The investigation focused on predictors for the maintenance doses of argatroban with efficacy and safety of argatroban being secondary outcomes.

Prospective, dose finding study.

Two intensive care units (medical and surgical) of a university hospital.

Medical and surgical patients (n = 30) with acute or histories of heparin-induced thrombocytopenia type II and acute renal failure with necessity for CRRT.

Intervention: CRRT with argatroban for anticoagulation.

Critical illness severity scores Acute Physiology and Chronic Health Evaluation (APACHE)-II, Simplified Acute Physiology Score (SAPS) II, and the indocyanine green plasma disappearance rate (ICG-PDR) were correlated to the argatroban maintenance doses. These diagnostic tools can help to identify patients with the necessity for decreased argatroban doses. The following recommendations for argatroban dosing during CRRT could be determined: a loading dose of 100 μg/kg followed by a maintenance infusion rate (μg/kg/min), which can be calculated. The efficacy and safety of anticoagulation during CRRT were determined by the steady state of blood urea nitrogen (32.16 ± 18.02 mg/dL), mean filter patency at 24 hrs (98%), and the rate of bleeding episodes. Only two patients developed minor bleeding; no patient developed severe bleeding episodes.

In critically ill patients with heparin-induced thrombocytopenia type II and necessity for CRRT critical illness scores (APACHE II, SAPS II) or ICG-PDR can help to predict the required argatroban maintenance dose for anticoagulation. These predictors identify decreased argatroban dosing requirements resulting in effective and safe CRRT.

## Saline Instillation before Tracheal Suctioning Decreases the Incidence of Ventilator-Associated Pneumonia

Pedro Caruso, Silvia Denari, Soraia A. L. Ruiz, et al.

*Crit Care Med, Jan 2009; 37:32–38*

To compare the incidence of ventilator-associated pneumonia (VAP) with or without isotonic saline instillation before tracheal suctioning. As a secondary objective, the author compared the incidence of endotracheal tube occlusion and atelectasis.

Randomized clinical trial. Setting and Patients: The study was conducted in a medical surgical intensive care unit of an oncologic hospital. We selected consecutive patients needing mechanical ventilation for >72 hrs. Patients were allocated into two groups: a saline group that received instillation of 8 mL of saline before tracheal suctioning and a control group which did not. VAP was diagnosed based on clinical suspicion and confirmed by bronchoalveolar lavage quantitative culture. The incidence of atelectasis on daily chest radiography and endotracheal tube occlusions were recorded. The sample size was calculated to a power of 80% and a type I error probability of 5%.

One hundred thirty patients were assigned to the

saline group and 132 to the control group. The baseline demographic variables were similar between groups. The rate of clinically suspected VAP was similar in both groups. The incidence of microbiological proven VAP was significantly lower in the saline group (23.5% × 10.8%; p = 0.008) (incidence density/1.000 days of ventilation 21.22 × 9.62; p < 0.01). Using the Kaplan-Meier curve analysis, the proportion of patients remaining without VAP was higher in the saline group (p = 0.02, log-rank test). The relative risk reduction of VAP in the saline instillation group was 54% (95% confidence interval, 18%-74%) and the number needed to treat was eight. The incidence of atelectases and endotracheal tube occlusion were similar between groups.

Instillation of isotonic saline before tracheal suctioning decreases the incidence of microbiological proven VAP.

## Passive leg raising for predicting fluid responsiveness: importance of the postural change

Julien Jabot, Jean-Louis Teboul, Christian Richard, et al.

*Intensive Care Med, Jan 2009; 35: 85 - 90*

Predicting fluid responsiveness by passive leg raising (PLR), the lower limbs can be elevated at 45° either from the 45° semi-recumbent position (PLRSEMIREC) or from the supine position (PLRSUPINE). PLRSUPINE could have a lower hemodynamic impact than PLRSEMIREC since it should not recruit the splanchnic venous reservoir.

A 24-bed medical intensive care unit.

A total of 35 patients with circulatory failure who responded to an initial PLRSEMIREC by an increase in cardiac index ≥ 10% were included.

PLRSEMIREC, a transfer from the semi-recumbent to the supine position and PLRSUPINE were performed in all patients in a random order before fluid expansion (500 mL saline).

Measurements and results: PLRSEMIREC, supine transfer and PLRSUPINE significantly increased the pulse-contour derived cardiac index (PiCCOplus) by 22 (17–28)%, 9 (5–15)% and 10 (7–14)% (P < 0.05 vs. PLRSEMIREC for the latter two), respectively. These maneuvers significantly increased the right ventricular end-diastolic area (echocardiography) by 20 (14–29)%, 9 (5–16)% and 10 (5–16)% (P < 0.05 vs. PLRSEMIREC for the latter two) and the central venous pressure by 33 (22–50)%, 15 (10–20)% and 20 (15–29)% (P < 0.05 vs. PLRSEMIREC for the latter two), respectively. Volume expansion significantly increased cardiac index by 27 (21–38)% and all patients were responders to volume expansion. If an increase in cardiac index ≥ 10% is considered as a positive response to PLRSUPINE, 15 (43%) patients would have been unduly predicted as non-responders to fluid administration by PLRSUPINE.

PLRSEMIREC induces larger increase in cardiac preload than PLRSUPINE and may be preferred for predicting fluid responsiveness.

## Intensive care unit-acquired neuromyopathy and corticosteroids in survivors of persistent ARDS

Catherine L. Hough, Kenneth P. Steinberg, B. Taylor Thompson, et al.

*Intensive Care Med, Jan 2009; 35:63 – 68*

To determine the incidence and outcomes of intensive care unit-acquired neuromyopathy and to investigate the role of methylprednisolone in survivors of persistent acute lung injury.

Secondary analysis of completed randomized placebo-controlled trial.

Twenty-five hospitals in the NHLBI ARDS Network.

Patients and participants: Patients enrolled in the ARDS Network study of methylprednisolone versus placebo for persistent ARDS who survived 60 days or to hospital discharge.

One hundred and twenty-eight study patients survived 60 days. Forty-three (34%) of these patients had evidence by chart review of ICU-acquired neuromyopathy, which was associated with prolonged mechanical ventilation, return to mechanical ventilation, and delayed return to home after critical illness. Treatment with methylprednisolone was not significantly associated with an increase in risk of neuromyopathy (OR 1.5; 95% CI 0.7–3.2).

ICU-acquired-neuromyopathy is common among survivors of persistent ARDS and is associated with poorer clinical outcomes. We did not find a significant association between methylprednisolone treatment and neuromyopathy. Limitations of this study preclude definitive conclusions about the causal relationship between corticosteroids and ICU-acquired neuromuscular dysfunction.

## Continuous hypertonic saline therapy and the occurrence of complications in neurocritically ill patients

Matteus Froelich, Quanhong Ni, Christian Wess, et al.

*Crit Care Med, Apr 2009; 37:1433-1441*

To evaluate potential side effects of continuous hypertonic 3% saline (CHS) as maintenance fluid in patients with brain injury.

Retrospective chart analysis of prospectively collected data.

Patients admitted to the neurosurgical intensive care unit for >4 days with traumatic brain injury, stroke, or subarachnoid hemorrhage with a Glasgow Coma Scale <9 and elevated intracranial pressure (ICP) or at risk of developing elevated ICP were included. Based on physician preference, one group was treated with 3% CHS at a rate of 1.5 mL/kg/bw as maintenance fluid. The other group received 0.9% normal saline (NS). Two percent saline was used in the CHS group to wean patients off 3% CHS or when sodium was above 155. Data on serum sodium, blood urea nitrogen, creatinine, ICP, infection rate, length of stay, rates of deep vein thrombosis, and pulmonary emboli and dural thrombosis were collected prospectively.

One hundred seven patients in the CHS group and 80 in the NS group met the inclusion criteria. The incidence of moderate hypernatremia (Na >155 mmol/L) and severe hypernatremia (Na >160 mmol/L) was significantly higher in the CHS therapy group than in the NS group. No significant relationship between CHS infusion and renal dysfunction was found. Moderate and severe hypernatremia was associated with a higher risk of elevated blood urea nitrogen and creatinine levels. Acute renal failure was not seen in these patients. A total of 53.3% in the CHS group and in 16.3% in the NS group ($p < 0.0001$) had raised ICP (>25 mm Hg), consistent with the physicians decision to use CHS in patients with elevated ICP.

CHS therapy was not associated with an increased rate of infection, deep vein thrombosis, or renal failure. However, there was a significant risk of developing hypernatremia. We conclude that CHS administration in patients with severe injuries is safe as long as sodium levels are carefully monitored.

## The use of personal protective equipment for control of influenza among critical care clinicians: A survey study.

Elizabeth L. Daugherty, Trish M. Perl, Dale M. Needham, et al.

*Crit Care Med, Apr 2009; 37: 1210-1216*

Intensive care units (ICUs) are potential high-risk areas for transmission of viruses causing febrile respiratory illness, such as influenza. Healthcare-associated influenza is prevented through healthcare worker (HCW) vaccination and effective use of U.S. Centers for Disease Control and Prevention recommended infection control practices, including use of personal protective equipment (PPE). Although effective PPE use may significantly reduce healthcare-associated influenza transmission, PPE adherence among ICU HCWs for preventing nosocomial influenza infection has not been evaluated.

To characterize ICU HCW behavior, knowledge, and attitudes about recommended precautions for the prevention of healthcare-associated influenza infections.

A survey of 292 internal medicine housestaff, pulmonary/critical care fellows and faculty, nurses, and respiratory care professionals working in four ICUs in two hospitals in Baltimore, MD.

Of those surveyed, 88% (n = 256) completed the survey. Only 63% of respondents were able to correctly identify adequate influenza PPE, and 62% reported high adherence (>80%) with PPE use for prevention of nosocomial influenza. In multivariable modeling, odds of high adherence varied by clinician type. Respondents who believed adherence was inconvenient had lower odds of high adherence (odds ratio 0.42, 95% confidence interval 0.22-0.82), and those reporting likelihood of being reprimanded for nonadherence were more likely to adhere (odds ratio 2.40, 95% confidence interval 1.25-4.62).

ICU HCWs report suboptimal levels of influenza PPE adherence. This finding in a high-risk setting is particularly concerning, given that it likely overestimates actual behavior. Both suboptimal adherence levels and significant PPE knowledge gaps indicate that ICU HCWs may be at a substantial risk of developing and/or transmitting nosocomial respiratory viral infection. Improving respiratory virus infection control will likely require closing knowledge gaps and changing organizational factors that influence behavior.

## Interaction of vasopressin infusion, corticosteroid treatment, and mortality of septic shock.

James A. Russell, Keith R. Walley, Anthony C. Gordon, et al.

*Crit Care Med, Mar 2009; 37: 811-818*

Vasopressin and corticosteroids are often added to support cardiovascular dysfunction in patients who have septic shock that is nonresponsive to fluid resuscitation and norepinephrine infusion. However, it is unknown whether vasopressin treatment interacts with corticosteroid treatment.

Post hoc substudy of a multicenter randomized blinded controlled trial of vasopressin vs. norepinephrine in septic shock.

Twenty-seven Intensive Care Units in Canada, Australia, and the United States.

Seven hundred and seventy-nine patients who had septic shock and were ongoing hypotension requiring at least 5 [mu]g/min of norepinephrine infusion for 6 hours.

Interventions: Patients were randomized to blinded vasopressin (0.01-0.03 units/min) or norepinephrine (5-15 [mu]g/min) infusion added to open-label vasopressors. Corticosteroids were given according to clinical judgment at any time in the 28-day postrandomization period.

The primary end point was 28-day mortality. We tested for interaction between vasopressin treatment and corticosteroid treatment using logistic regression. Secondary end points were organ dysfunction, use of open-label vasopressors and vasopressin levels.

There was a statistically significant interaction between vasopressin infusion and corticosteroid treatment (p = 0.008). In patients who had septic shock and were also treated with corticosteroids, vasopressin, compared to norepinephrine, was associated with significantly decreased mortality (35.9% vs. 44.7%, respectively, p = 0.03). In contrast, in patients who did not receive corticosteroids, vasopressin was associated with increased mortality compared with norepinephrine (33.7% vs. 21.3%, respectively, p = 0.06). In patients who received vasopressin infusion, use of corticosteroids significantly increased plasma vasopressin levels by 33% at 6 hours (p = 0.006) to 67% at 24 hours (p = 0.025) compared with patients who did not receive corticosteroids.

There is a statistically significant interaction between vasopressin and corticosteroids. The combination of low-dose vasopressin and corticosteroids was associated with decreased mortality and organ dysfunction compared with norepinephrine and corticosteroids.

## Percutaneous Coronary Intervention versus Coronary-Artery Bypass Grafting for Severe Coronary Artery Disease

Patrick W. Serruys, Marie-Claude Morice, A. Pieter Kappetein, et al.

*N Engl J Med 2009;360:961-72.*

Percutaneous coronary intervention (PCI) involving drug-eluting stents is increasingly used to treat complex coronary artery disease, although coronary-artery bypass grafting (CABG) has been the treatment of choice historically. Our trial compared PCI and CABG for treating patients with previously untreated three-vessel or left main coronary artery disease (or both).

We randomly assigned 1800 patients with three-vessel or left main coronary artery disease to undergo CABG or PCI (in a 1:1 ratio). For all these patients, the local cardiac surgeon and interventional cardiologist determined that equivalent anatomical revascularization could be achieved with either treatment. A noninferiority comparison of the two groups was performed for the primary end point — a major adverse cardiac or cerebrovascular event (i.e., death from any cause, stroke, myocardial infarction, or repeat revascularization) during the 12-month period after randomization. Patients for whom only one of the two treatment options would be beneficial, because of anatomical features or clinical conditions, were entered into a parallel, nested CABG or PCI registry.

Most of the preoperative characteristics were similar in the two groups. Rates of major adverse cardiac or cerebrovascular events at 12 months were significantly higher in the PCI group (17.8%, vs. 12.4% for CABG; P = 0.002), in large part because of an increased rate of repeat revascularization (13.5% vs. 5.9%, P<0.001); as a result, the criterion for noninferiority was not met. At 12 months, the rates of death and myocardial infarction were similar between the two groups; stroke was significantly more likely to occur with CABG (2.2%, vs. 0.6% with PCI; P = 0.003).

CABG remains the standard of care for patients with three-vessel or left main coronary artery disease, since the use of CABG, as compared with PCI, resulted in lower rates of the combined end point of major adverse cardiac or cerebrovascular events at 1 year.

**EDITORIAL OFFICE** Indraprastha Apollo Hospitals, Room No. 4162, 1st Floor, Gate No. 10, New Delhi - 110076
Telefax : 011-26825586 • Emails : drchawla@vsnl.net