Intensive Care in 10 Years – Where are we going?

Introduction

On average, everyone will be admitted to an ICU once in their lifetime – some will never need ICU treatment, but others will require two or three admissions, or more. Half of us will spend time on the ICU during our last year of life, and 20% of us will die in an ICU [1]. When discussing the future of intensive care medicine, there is one thing that everyone agrees on: The demand for ICU services will grow. This increased demand in service must be accompanied by improved quality of care. Quality of ICU care is difficult to quantify and characterize but is coming under increasing scrutiny. We will consider the present situation in three key areas of intensive care medicine, structure, process, and outcome, that directly influence quality and how these areas will evolve over the next 10 years.

Structure

Neil Halpern and colleagues reviewed the evolving role, patterns of use, and costs of critical care medicine in the United States from 1985 to 2000 [2]. There was a 9% decrease in the total number of hospitals, a 16% decrease in acute care hospitals, and a 14% decrease in acute care hospitals that provide critical care medicine (these hospitals represent two thirds of all United States hospitals). As expected, the total numbers of hospital beds decreased by 26% during this time period; with medical progress and advances allowing more daycare surgery, more rapid diagnoses, ambulatory therapies, etc., hospital stays are shorter and fewer beds are needed overall. However, the number of ICU beds increased by 26%, creating a 72% increase in the ICU/hospital bed ratio! Hospital non-ICU inpatient days decreased by 31.5%, whereas ICU inpatient days increased by 29%! Interestingly, the ICU oc-
cupancy rate remained relatively constant at 65%. These findings suggest that the number of ICU beds will increase from around 5% of the total number of hospital beds today, to 10% or more in the near future. As needs increase and the population ages, the hospital of tomorrow will in fact become one large ICU with a few non-ICU beds around it. The future would therefore seem to bright for the budding intensivist!

But is it? What about the costs of this ICU expansion? In the study by Halpern et al. [2], costs per inpatient day increased by 6% per year, but by less than half this in the ICU, hence, although increasing in use and importance, critical care medicine is using less of hospital costs, national health expenses, and the gross domestic product (GDP) than perceived. Let us consider costs further: Are we, in fact, so costly in 2006? Material costs have not increased so much over time. The costs of monitoring systems and respirators are high, but have not increased markedly over the years. We have added some new pieces of equipment, e.g., hemofiltration machines, but they are not much more costly than the equipment they replace, e.g., hemodialysis machines. Costs of key, “bread and butter” ICU drugs, e.g., furosemide, vasopressors, even sedative agents, are old molecules whose costs have decreased if anything. Modern antibiotics and antifungal agents are costly, but these are not prescribed only in the ICU. Drotrecogin alfa (activated) is costly and has attracted considerable debate, but there are not many interventions that have been shown to reduce mortality [3] and costs are comparable with other widely accepted healthcare interventions [4]. In addition, other specialties also use very expensive drugs even though in many cases they do not provide a cure, but merely prolong life somewhat. For example, the rheumatologists use expensive TNF receptors; in cardiology, clopidogrel is very costly when administered for prolonged periods of time; in oncology, trastuzumab (Herceptin) costs about 34,000 euros per patient for advanced breast cancer, interferon alfa-2b (Intron) costs some 25,000 euros for advanced melanoma if the patient survives 6 months, and treatment with imatinib mesylate (Glivec) for chronic myeloid leukaemia costs around 30,000 euros. Why, therefore, should intensive care patients be deprived the possible benefits of new drugs? Importantly, too much criticism will encourage the industry to invest in other fields and yet we urgently need new drugs to improve outcomes in critically ill patients [5].

In fact, the largest part (some 70%) of ICU costs are for personnel, but this we cannot change. ICUs must be adequately staffed, and indeed studies demonstrate increased complications and worse outcomes when nursing ratios are low [6, 7]. Trained intensivists are also an essential component of good ICU care with studies showing worse outcomes when intensivist cover is not available [8–10]. But, how will intensivist staffing levels be able to keep pace as the numbers of ICU patients increase, and with the increased demand for 24-hour intensivist cover? Indeed, studies suggest that we will have a major shortfall of ICU doctors in the not too distant future [11]. Young doctors now prefer other specialties offering a better ‘lifestyle’ than critical care medicine. In a survey of internal medicine residents in university hospital training programs, Lorin et al. reported that although 41% had “seriously considered” pulmonary and critical care, only 3.4% actually chose the field [12]! We need to make intensive care medicine more attractive to potential intensivists. The study by Lorin et al. [12] highlighted intellectual stimulation, opportunity to manage critically ill patients, application of complex physiologic principles, number of procedures performed, and academically challenging rounds as the five most attractive features of critical care medicine. Three of the most unattractive features were overly demanding responsibilities with lack of leisure time, stress among faculty and fellows, and management responsibilities for chroni-
cally ill patients. Pay is also an important part of the equation; much as we would like to think we are all purely altruistic in our career decisions, when choosing between specialties of similar interest, we are all likely to be swayed by the promise of a higher income.

I see four possible approaches to the recruitment problem:

1. We accept it – we employ fewer doctors and rely more heavily on computerized protocols to manage patients. ICUs will become largely nurse run, with maybe one doctor for 30 beds, but many protocols, largely computer-driven.

2. We make it a primary specialty – and make it more attractive to prospective intensivists. This option is under serious consideration in the US and some European countries.

3. We regionalize the ICU – many studies have shown that centers with a greater experience in a certain field have better outcomes (PTCA, surgical procedures, trauma, recently mechanical ventilation). However, this option may not be well accepted by smaller hospitals.

4. We employ telemedicine – this should not be used to have one doctor controlling several ICUs from an office, rather to enable one doctor in a large institution to help others in smaller less well-staffed units. The telemedicine system set up by the John Hopkins Hospital in Baltimore improved clinical outcomes and hospital financial performance [13].

**Process**

The optimal process of care is difficult to define and a ‘successful’ process, therefore, difficult to quantify. It is true that care is suboptimal in some institutions – patients remain hypovolemic, hypotensive, evidence-based practices are not performed, etc. One answer to this is to standardize and control care by using protocols, and these have been widely promoted [14–19]. However, while in theory protocols seem a great idea, in practice things are never that straightforward. For example, strict control of blood glucose levels was found to improve outcomes in a single ICU composed largely of surgical ICU patients [20], but in a subsequent study in medical ICU patients there was no overall effect on mortality [21], so should this strategy be employed in all ICU patients or only in certain patient groups, and if so which? A weaning protocol that has been shown to be associated with improved outcomes in medical ICU patients [14] was not successfully applied to mechanically ventilated neurosurgical patients in whom protocol-managed patients had similar outcomes to non-protocol managed patients [22]. Care needs to be taken in determining whether any individual patient does in fact fit the group for whom the protocol was developed. And what about new evidence – how quickly should the protocol be changed and on whose authority? It is well known that physicians are relatively reluctant to adopt new practices [23], but if a study’s results show definite improved outcomes from a particular intervention, should this not be included immediately in the management protocol for that group of patients? And if it is included, but the first few patients managed with the new protocol have poor outcomes, should we revert to the old protocol again? What about sepsis? The SSC recently published evidence-based guidelines for the management of the patient with severe sepsis [24]; however, application of these guideline is less easy. Sepsis bundles (protocols) have been proposed [25], but so much is uncertain in the management of the patient with sepsis that deciding what to include in these protocols is difficult; for example, how much fluid, what kind of fluid, which vasopressors, should we give steroids, etc? Similarly in acute respiratory distress syndrome, what do we actually know for sure? We know that large tidal vol-
umes are harmful [26] so, yes, we should avoid iatrogenicity, but what can we do that really works? We can avoid fluid overload, use some positive end-expiratory pressure (we do not know how much), and that is all – prone positioning, inhaled nitric oxide, surfactant, etc., none of these other strategies has given any positive results. We are faced with the same uncertainties for blood transfusions. Exactly which of our ICU patients should be transfused and how much? Hebert et al. [27] suggested that lower transfusion triggers had the same impact on outcomes as higher triggers, and may even have improved outcomes in some patients. But this study was conducted more than 10 years ago, in an era when blood was not widely leukodepleted as it is now. Are the results still valid in today’s patients? On the surface much has changed in intensive care medicine since the specialty was born some 50 years ago [28], but we still have so much to learn. Protocolized care certainly has its benefits, but its limitations must also be realized.

Allan Garland in a recent, interesting article entitled *Improving the ICU* states that “Because wide and widespread variation could not exist if most practitioners practiced optimally, such variation is evidence that suboptimal care is common.” [29]. I am not so sure. The future would have us all uniform – following the same routine patterns of care. I’m not sure that this is a vision that I find appealing or reassuring. Variation in practice may demonstrate our ignorance, but we can learn from this diversity. Those who are not at the bedside love protocols – it makes their life much easier. But as we become ‘protocolized’ we will have to think less – medicine will become ace-rebral, and then where does the intensivist fit in – after all you do not need a trained doctor or even nurse to complete a step-by-step computerized protocol!

Rounds at the bedside are a good way to check that patients are receiving the care they should be and provide a base for thought and discussion. The mnemonic, Fast Hug (Feeding, Analgesia, Sedation, Thromboembolic prophylaxis, Head-of-bed elevation, stress Ulcer prevention, and Glucose control) [30], is a useful strategy, encompassing key aspects of general care for all critically ill patients, in a simple format that can be used by every member of the ICU team every time they attend the patient for whatever reason. I believe simple checklists such as that suggested above should be more widely used in the future, rather than some of the detailed and “thoughtfree” protocols that are being promoted.

### Outcome

And, finally, outcome. Survival has for years been seen as the key outcome measure to assess the efficacy of new therapies or interventions and to judge quality of care – after all, if you are not alive, what else matters? However, increasingly, survival is no longer enough – we need also to focus on other markers of morbidity, including quality of life. This raises critical ethical questions as assessing quality of life is very subjective and crucially dependent on the individual’s circumstances, pre-morbid quality of life and expectations. This is an important area that is only just beginning to be discussed [31]. Post-intensive care survivors are beset by various difficulties related to their time on the ICU, and not just to the disease for which they were admitted. Margaret Herridge and colleagues [32] reported that most survivors of acute respiratory distress syndrome have persistent functional disability one year after discharge from the intensive care unit, largely extrapulmonary conditions with muscle wasting and weakness being most prominent. Many studies have reported mental distress and symptoms of post-traumatic stress disorder in ICU patients following discharge [33, 34].

Post-discharge ICU consultations have been suggested as a means of providing ade-
quate follow-up for these patients and trying to manage or prevent some of the post-ICU conditions. After all, patients from other departments almost always have at least one outpatient follow up with the relevant specialty – why not intensive care? Perhaps the future will also see patients groups developing, where people can exchange their experiences, as already exist for many diseases.

Conclusion

The next 10 years will see great changes in intensive care medicine as this young specialty continues to develop. The numbers of patients we treat will increase and advances in diagnostics, monitoring and therapeutics will make life simultaneously easier and more complex. Changes in structure, process, and outcome measures will combine to improve quality of care as what we do is placed under ever greater scrutiny. Intensivist-led intensive care will become the standard and a key challenge will be to attract and maintain adequate numbers of well-trained personnel. Protocols will become more widely implemented but should not be allowed to replace thoughtful individual patient care. Computerized systems and databases and telemedicine will be increasingly visual parts of daily ICU practice. Medical outreach teams will be used to assess patients before ICU admission, but should not replace adequate training of hospital staff in the management of emergency situations.

Taking care of the critically ill is a challenge, but a fascinating and worthy one – we must all work together to ensure that the ICUs of tomorrow continue to provide high quality care for future generations of patients.

References

1 Special Lecture

2 Beatmung

Why do we need an open lung

B. Lachmann | J.J. Haitsma

Adjunktive Therapie – what is proven, what is fancy?

R. Dembinski | R. Kuhlen

NIV bei der Entwöhnung vom Respirator

B. Schönhofer
**Why do we need an open lung**

**Introduction**

Every year, millions of patients worldwide receive ventilator support during surgery. Mechanical ventilation has become an important therapy in the treatment of patients with impaired pulmonary function and particularly in patients suffering from adult respiratory distress syndrome (ARDS). ARDS is caused by multiple factors and is characterized by respiratory dysfunction including hypoxemia and decreased lung compliance. It is known that the decrease in lung distensibility is due to a disturbed surfactant system with an elevated surface tension. This increase in surface tension leads to an increase in forces acting at the air-liquid interface, resulting finally in end-expiratory collapse, atelectasis, an increase in right-to-left shunt and a decrease in PaO₂.

**Ventilator associated lung injury (VALI)**

It has become clear, however, that mechanical ventilation itself can lead to lung damage and may even be the primary factor in lung injury. The recent recognition that alveolar overdistension rather than high proximal airway pressures is the primary determinant for lung injury (i.e. volutrauma instead of barotrauma) combined with shear stress evoked by repeated alveolar collapse and re-opening due to low end-expiratory volumes (1), has led to renewed interest in lung mechanics and ventilation. In the recent international consensus conference on ventilator-associated lung injury (VALI) in ARDS (2) the question was asked what non-pharmacological approaches are currently available for prevention of VALI?

Clinical studies showed that a reduction of tidal volume, reduction of peak airway pressures combined with an increase in PEEP resulted in improved outcome of ARDS patients (3). Fur-
thermore by applying a lung protective strategy pro-inflammatory mediators both in the lung as well as those circulating can be reduced (4), and reducing the circulating levels of pro-inflammatory mediators reduces the development of multi-organ failure, the major cause of mortality in ARDS patients (5).

Role of peak pressures, tidal volume and PEEP

Webb and Tierney in 1974 demonstrated the critical role that PEEP plays in preventing/reducing lung injury (6). In rats ventilated with 10 cmH$_2$O of PEEP and a peak pressure of 45 cmH$_2$O no lung injury was present but using the same peak pressure and omitting PEEP severe pulmonary edema was formed within 20 min (6). In a study by Verbrugge et al. the difference in pressure amplitude between these two groups also resulted in a difference in tidal volume, i.e. 18 ml/kg and 45 ml/kg in the 45/10 and 45/0 group, respectively (1). Dreyfuss and colleagues further explored the role of tidal volume and peak inspiratory pressures on lung injury (7). In an animal model they applied high inspiratory pressures in combination with high volumes which resulted in increased alveolar permeability (7). In a second group low pressures were combined with high volume (iron lung ventilation) again resulting in alveolar permeability (7). In the third group the effect of high pressures combined with low volume was studied, by strapping the chest wall to reduce chest excursions; the permeability of this group (high-pressure low-volume group) did not differ from the control group (7). Thus large tidal volume ventilation increases alveolar permeability, whereas peak inspiratory pressures do not seem to influence the development of this type of lung injury. Similar observations were made in rabbits ventilated with high peak pressures in which thorax excursions were limited by a plaster cast (8). In injured lungs the effect of higher volumes only aggravated the permeability, as demonstrated in animals in which the surfactant system was inactivated and which were subsequently ventilated with high tidal volumes (9, 10).

Although Webb and Tierney already demonstrated that PEEP could ameliorate lung injury (6), the mechanism is still not clearly understood. PEEP can stent alveoli at end expiration and thus prevent repetitive collapse, reducing shear forces (11, 12). The most important role of PEEP is to preserve surfactant function. Two basic mechanisms have been reported to explain the surfactant-preserving effect of PEEP during mechanical ventilation. The first mechanism is alteration of the surfactant film by surface area changes, already suggested in 1972 (13). Wyszogrodksi et al. demonstrated that PEEP could prevent collapse of the alveolar surface film due to low lung volumes in no-PEEP ventilation and thus prevent alteration of the endogenous surfactant, substantiated in this model by surface tension measurement and lung compliance (14). Later it was shown that especially large area changes result in conversion of active surfactant (large aggregates LA) into inactive surfactant (small aggregates SA), believed to be the reason for the deterioration of surfactant function (1, 15, 16). In the model first described by Webb and Tierney, 10 cmH$_2$O PEEP prevents a significant conversion of large aggregates into small aggregates compared with non-ventilated controls (1, 17). A second mechanism explaining how PEEP preserves surfactant function, is the prevention of loss of surfactant to the proximal airways. In 1976, an ex-vivo model was used to show that ventilation caused movement of surfactant to the airways from the alveoli (18). Preventing alveolar collapse and keeping the expiratory volume of alveoli at a higher level, prevents excessive loss of surfactant in the small airways by a squeeze-out mechanism during expiration (1, 18–20).