Pressure injury: the forgotten ischaemic disorder

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Sincere congratulations to Ms Sharpe and Prof White whose letter titled "Pressure ulcer risk assessment: do we need a golden hour?" was published in the May 2015 *Journal of Wound Care*¹. It suggested that the golden hour concept be introduced to prevent pressure injury (PI). I believe this concept has great merit and its introduction to acute care hospitals might just eliminate hospital-acquired pressure injury.

The reply to their letter from a couple of members of the International Pressure Ulcer Guideline Development Group was predictably negative in that "there is no single randomised controlled double blinded trial to 'prove' that this approach is effective". However, current efforts are not stopping hospital-acquired PI and there was an increased prevalence in major intensive care units last year. Why not adopt this very logical approach to a preventable problem and get doctors involved in a 'golden hour' for pressure injury prevention?

Cardiologists adopted the 'golden hour' as the time in which restoration of coronary artery perfusion gave best clinical outcomes^{2,3}. Neurologists use the 'golden hour' in treating acute ischaemic stroke because best patient results are achieved when the time from symptom onset to treatment is within 60 minutes^{4,5}. Surely the ischaemic tissue beneath immobile patients' weight bearing bony prominences deserves restoration of blood flow before the onset of tissue necrosis — a 'golden hour' during which intervention is simple and successful.

Nurses have been burdened with sole responsibility for pressure area care ever since the days of Florence Nightingale. The involvement of doctors has mostly been after PI has developed but the introduction of a golden hour protocol for the prevention of hospital-acquired PI would necessarily involve doctors that work in acute care hospitals.

Considering the required intervention is so much simpler than that faced by cardiologists, there is no real excuse for

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patients developing PI in any health care facility. If the patient is unable to self reposition, hospital staff must manually turn the patient onto a different set of bony prominences at frequent intervals. PI develops only when this simple intervention is commenced too late!

PATHOPHYSIOLOGY OF PRESSURE INJURY

A PI is an acute wound caused by applied pressure which resulted in tissue ischaemia followed by tissue necrosis. The soft tissue squeezed between the weight bearing bony prominences and the resistant support surface is subjected to a pressure gradient with the highest internal tissue pressures found in the deep tissue surrounding the bone. Deep periosteal tissue is most at risk of ischaemic damage from the applied pressure.

Healthy tissue around the bony prominences is most probably innately resistant to tissue distortion and applied pressure so that under load, the ischaemic tissue is relatively resistant to lengthy periods of hypoxia. A lot more research is required to understand ischaemia/reperfusion injury in this tissue but it is clearly understood that PI is time-dependent. If the external pressure is applied for too long and the tissue remains ischaemic beyond the point of irreversible cellular damage, a PI develops.

The ischaemic cascade is a series of biochemical reactions that takes place in aerobic tissues when hypoxic tissue looses viability and cellular death commences. Lack of oxygen stops cells producing ATP for energy and they switch to anaerobic metabolism. This causes a series of events as the cellular calcium levels rise: the cell releases amino acids to correct those elevated levels: the calcium levels continue to peak: the cell wall breaks down and the mitochondria in the cell start to die; the cell itself dies and releases toxins into the surrounding area, damaging neighbouring cells, including cells with an adequate supply of oxygen. The extensive tissue damage seen in Stage 3 and 4 pressure ulcers is the result of the ischaemic cascade. A period of hypoxia followed by reperfusion injury has occurred in that tissue because the pressure which caused the ischaemia was not removed before cellular death occurred.

PATIENTS AT RISK

With usual daily routines, healthy tissue about the bony prominences is able to withstand lengthy hypoxic episodes without the onset of the ischaemic cascade and resulting PI.

As long as the biofeedback system protects the tissue about our bony prominences by stimulating movement, a PI will not develop.

With failure of the protective biofeedback mechanism, there comes the risk of PI. If the load bearing tissue is healthy, its tolerance to applied pressure might be two hours which is the repositioning frequency introduced by Dr Guttmann⁶ at Stoke-Mandeville Hospital when rehabilitating spinal injured service men during World War II. Nowadays, the principle of two-hourly turns is now so inculcated that it is difficult to get hospital staff to realise that sick, immobile patients require far more frequent manual repositioning.

With serious injury or illness, the tolerance of this deep tissue to applied pressure is markedly decreased and the time before cellular damage occurs is certainly much less than two hours. Therefore, when a sick, immobile patient is bought to hospital, active resuscitation must include protection from PI.

IMPLEMENTING A GOLDEN HOUR PROTOCOL

Standard 1 of the National Safety and Quality Health Service Standards (2012)⁸ advises "1.8 Adopting processes to support the early identification, early intervention and appropriate management of patients at increased risk of harm". Emergency rooms have protocols to identify patients with suspected stroke or acute coronary insufficiency and these patients receive immediate attention. Similarly, it is not difficult to triage patients and identify those at risk of PI.

Prevalence surveys in acute care hospitals⁹ show that the highest rates of pressure injuries are found in critical care units (including ER wards) which become the logical areas to introduce the golden hour concept. Identifying patients whose biofeedback mechanism has failed so they are unable to self reposition in response to pressure pain is not difficult. These are sick or injured patients lying on a trolley or bed but unable to turn from side to side when asked. The treating doctor will have examined them as a matter of high priority but afterwards they can be 'forgotten' as they await investigation.

However, the doctor, now well aware that PI prevention operates under a 'golden hour' protocol, will check for existing PI and order the repositioning frequency appropriate for the support surface. Other measures to resuscitate the patient might be far more urgent or maybe just pain relief and further investigation is required. A brightly coloured, obvious turning schedule could be used to indicate the patients at risk and staff, trained to carefully reposition very sick patients, allocated to be responsible for regular turning. Unless contraindicated, there should be immediate access to a high efficiency active (dynamic) support surface to extend the repositioning frequency to practical periods of time.

A recent Cochrane Systematic Review⁹ concluded there is no reliable evidence to suggest that the use of structured,

systematic risk assessment tools reduce the incidence of PI or are superior to clinical judgment. With patient assessment within 60 minutes of admission by both doctor and nurse, there is far more chance of quickly implementing a successful PI preventative strategy in high risk patients.

As resuscitation proceeds, the ability of the tissues surrounding the vulnerable bony prominences to resist applied pressure improves and the risk of PI recedes. The acutely ill, immobile patient has been saved without having to face the consequences of PI. As soon as the patient can turn themselves in bed, the high efficiency active support surface is removed. The hospital has been saved the cost of treating a patient with a hospital-acquired PI.

I believe that the 'golden hour' concept for PI prevention should be introduced to most wards in acute care hospitals and become the responsibility of both nurses and doctors. Each ward should introduce a plan whereby patients are assessed for risk of PI within 60 minutes of admission and, if found to be at high risk, there is an immediate intervention with a repositioning regime and an active support surface. Staff must still remain alert to deterioration in patients with chronic illness and daily risk assessments may be necessary.

The 'golden hour' concept could help eradicate hospital-acquired PI and save our health budget millions of dollars. It is well accepted that a Stage 3 or 4 PI costs over \$50,000 to treat but, with many lawyers beavering away to seek retribution for this preventable disorder, health facilities are paying 10 times this amount in compensation.

THE APPROPRIATE SUPPORT SURFACE INTERVENTION

It would be helpful if there was a randomised control trial that showed the clear superiority of one support surface in relieving pressure. This is never going to occur due to the enormous number of ever changing products available, the high cost of truly effective systems and the large number of patients required to provide a statistically significant result.

However, with knowledge of basic physics principles, the pathophysiology of PI development and mechanical common sense, suitable support surfaces can be determined. The aim of the medical intervention is the frequent removal of applied pressure at the skin/support surface interface so that there is complete removal of pressure from the deep tissues about the bony prominences which provides relief of ischaemia and allows a reactive hyperaemic response in that tissue.

When patients cannot reposition themselves to remove the applied pressure, the medical intervention is manual repositioning by hospital staff. The frequency of that repositioning is determined by the characteristics of the support surface upon which the patient is lying.

Regular careful repositioning of critically ill, immobile patients being nursed upon a standard hospital foam mattress may need to be performed every 15 minutes to provide effective relief of deep tissue ischaemia. By using a high specification foam mattress, the repositioning frequency might be extended to 20 minutes. It should be obvious that maintaining these times for any length of time is impossible.

The most efficient reactive (static) support surfaces might decrease the mass of tissue subjected to ischaemia through weight redistribution over a greater surface area but because the complete relief of interface pressure only occurs when the patient is manually turned, regular repositioning every 30 minutes may still be required to provide that complete relief of deep tissue pressure. Therefore reactive (static) support surfaces do not have a role in the prevention of PI in critical care units except in the suspected spinal injured patient.

Active (dynamic) support surface are recommended for critical care units because the more efficient of these devices do relieve deep pressure at regular intervals and certainly extend the repositioning frequency to practical intervals. The "Pressure Injury Prevention for Critically III Adults, NSW Health Agency for Clinical Innovation¹⁰ (2014)" states that: "Ideally, all intensive care unit beds should have alternating pressure support mattresses; however, all patients who are classified as high risk or who have an existing or greater than grade two PI must have an active alternating pressure mattress."

There are numerous alternating pressure air mattresses (APAMs) in the medical market place but most are not suitable for use in nursing critically ill patients because they do not adequately reduce interface pressure and do not extend the repositioning frequency. In fact, some underpowered, inefficient active devices actually increase interface pressures at the foot end of the mattress and cause heel pressure injuries. The cost of intervention with a powerful APAM that actually prevents PI is so much less than the cost of treating a PI.

As the support surface of choice during the 'golden hour', I recommend a high efficiency alternating pressure air mattress with vacuum assisted deflation (APAM with VAD) because it eliminates interface pressure for sufficient time to allow for a reactive hyperaemic response in the deep tissues surrounding the weight bearing bony prominences. With a long history of proven clinical effectiveness in critical care units and a recognised 8 hour repositioning frequency, APAMs with VAD should be the first line intervention during the 'golden hour' when the patient is identified at high risk of PI.

APAMs require sufficient air in the inflated cells to lift the body off the bed base while the deflating cells retract aware from contact with the overlying body. By vacuuming all air from the deflated cell, the overlying area of the body is completely relieved of pressure which restores perfusion to the deep periosteal tissue and relieves ischaemia.

As with other life support equipment, the high efficiency APAM with VAD can be removed as the patient recovers from the acute illness or trauma. It may be replaced by another APAM if traditional risk assessment scores such as Braden, Waterlow etc suggest the patient is still at high risk. Otherwise, the patient should be suitable for nursing on a high specification foam mattress.

DISCLAIMER

I am a registered medical practitioner who has been involved in pressure area management for the past 30 years during which time I have also been a consultant to numerous manufacturers of pressure relieving devices. I have now retired from active medical practice but still have pecuniary interests in several companies that supply medical devices including APAMs.

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