Adapting fast and slow: force-related tissue damage

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- Anatomy and physiology
- Decubitus ulcer
- Force-related tissue damage
- Pressure injury
- Pressure ulcer

This paper reviews the literature surrounding force-related tissue damage (other terminology for this includes: pressure injury, pressure ulcer, decubitus ulcer) and explains the latest research behind the current understanding of the science beneath the skin; including up-to-date knowledge of the anatomy, physiology and physics that interplay within tissue when force-related tissue damage occurs. The role of force, tissue, the body's adaptation system and pathology are all explained in order to increase our understanding of the often preventable events that happen before tissue damage occurs. The article challenges the reader to consider the science of tissue damage in greater depth in order to find solutions to this distressing and costly problem. It is hoped that expanding and explaining the biology behind force-related tissue damage will stimulate discussion, research and ultimately the development of solutions that will allow the body to adapt rather than disrupt when it experiences forces that could cause damage to its structures.

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Pressure ulcers or force-related tissue damage has been a healthcare burden for many years. Current treatment and prevention algorithms are not able to reduce this burden significantly (Hahnel et al, 2017; VanGilder et al, 2017; Levine, 2018; Tomova-Simitchieva et al, 2018). To understand this profoundly disturbing lack of progress we have to take a closer look at the processes underlying force-related tissue damage.

All decubitus ulcers are a result of forces, usually referred to as pressure, on tissue. Historically, pressure injuries have been called pressure ulcers, pressure sores, bed sores/ulcers, decubitus ulcers and other names. Currently, the National Pressure Ulcer Advisory Panel (NPUAP, 2016) defines pressure injury as: *"localized damage to the skin and underlying soft tissue usually over a bony prominence or related to a medical or other device. The injury can present as intact skin or an open ulcer and may be painful. The injury occurs as a result of intense and/or prolonged pressure or pressure in combination with shear. The tolerance of soft tissue for pressure and shear may also be affected by microclimate, nutrition, perfusion, comorbidities and condition of the soft tissue."* The NPUAP present a 4 stage classification for pressure ulcers with two additional stages; unstageable pressure ulcers and deep tissue pressure ulcers.

Tissue damage is the key concept involved in the development of all wounds. More specifically, pathological damage, which is damage which is not resolved within a normal time frame or leads to a macroscopic (visual) lesion. In the case of a pressure ulcer, the causing factor is force. Therefore, we will use the term force-related tissue damage in this article.

Tissue damage as a result of force is nothing special; forces are continuously causing cell and tissue damage from the day you are born until the day you die. This damage is monitored and repaired by the body and does not lead to loss of homeostasis. This is different in an individual with a lesion caused by force-related tissue damage; here something is not working the way it should.

The first relevant observation is, as stated above, that healthy individuals do have force-related tissue damage but not to the extent of pathological force-related tissue damage or pressure ulcer. To explore

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this difference we have to take a closer look at forces, tissue and how homeostasis is maintained.

FORCES

Forces can be divided into "slow" and "fast" forces and these have differing impacts on the body. Fast or high-velocity forces have a large magnitude over a short period. These cause tissue compression, displacement and deformation. Organisms usually prevent this type of damage by not hitting hard objects, moving away from incoming objects or avoiding fast body movements. High-velocity forces cause bruises, haematomas, concussions, ruptures and other traumatic problems. Where the energy transferred by a fast force is mainly related to its magnitude; the energy transferred by a slow force is mainly related to the time it is applied. Slow or low-velocity forces are forces of limited magnitude that can cause tissue damage when applied for a longer time. Low-velocity forces cause internal stresses, poor perfusion and local necrosis (Enyedi et al, 2016). In addition, repetitive forces can and will lead to degeneration and stiffening of muscle tissue and changes in the extracellular matrix (Sanders and Goldstein, 2001; Mak et al, 2010). Currently, there is a lack of clear guidance on magnitude, direction, duration and repetition of forces in relation to the prevention and healing of tissue damage.

TISSUE

The second factor to examine is tissue. Under what circumstances does tissue develop damage. It does not take too much force to damage a cell, because even a distortion of a cell has an effect on its physiology, and prolonged distortion can result in tissue damage (Hoffman and Crocker, 2009; Fletcher and Mullins, 2010). Cells form an integral part of the mechanical structure of tissue. Damage to a cell reduces the mechanical structure of its surroundings leading to biomechanical and physiological alterations at the micro level (Jagannathan and Tucker-Kellogg, 2016). Larger areas of damage are a risk factor for further damage (Guz et al, 2014) and cancer development (Paszek et al, 2005).

Interestingly, there is an apparent difference at an organisational level, problems in tissue cannot occur in individual cells and vice versa. This process is called emergence, a more complex structure has more and different possibilities to solve problems but it may also have problems of its own. Cell distortion cannot lead to perfusion problems whereas, distortion of tissue can. Perfusion problems lead to problems with metabolites such as oxidative stress which can damage cells and the extracellular matrix (He et al, 2014). Secondary perfusion problems can lead to ischemia-reperfusion injury. Stressed, injured, damaged and dead cells produce a lot of signals which trigger many reactions like inflammation, dilatation, extravasation, migration, proliferation and apoptosis. This leads to the conclusion that force exerted on tissue leads to mechanical and physiological problems in cells and related structures. Secondary tissue, like skin, is not the same in all directions (Meijer et al 1999; Ito et al, 2010; Pietsch et al, 2014). That means that the direction of the force applied also plays a role, especially when pulling or pushing on tissue from an unanticipated angle (Buss et al, 1997; Kanj et al, 1998). All this applies to normal tissue. Tissue that is compromised in its biomechanical, anatomical and/or physiological state will have a different, usually reduced, capacity to handle force.

To further understand these events in tissue, it is important to realise that cells and tissues have a range of states related to damage. In force-related tissue damage, we logically distinguish six states a cell or tissue can be in: normal, adapted, stressed, injured, damaged and dead.

Normal is the normal state, where the cell is well adapted to the circumstances and it is able to fulfil its role. An adapted cell has changed its anatomy, physiology or behaviour to adapt to changes in the environment. A stressed cell is a cell where the adaptation leads to problems in its anatomy or physiology. An injured cell has reversible damage and a damaged cell has irreversible, non-lethal, damage, and a dead cell is… dead.

Only the normal state can be maintained for a long period, all the other states will aggravate over time, an adapted cell can return to a new stable normal state through proliferation. Prolonged adaptation will lead to stress and prolonged stress will lead to injury. The same applies to the force applied, the larger the magnitude the more severe the damage.

Therefore, not only the magnitude and direction

but also the time during which a force is exerted to tissue determines the outcome. Whether the tissue will adapt, stress, injure, damage, die or do nothing depends on the peculiarities of the force.

ADAPTATION

All the above is about how force and tissue interact. There is one important factor missing in the interaction between force and tissue and that is adaptation. Cells, tissues and the body do not undergo forces passively; instead, they react in two ways, anticipation and response.

To be able to react to circumstances the body (or any living organism) needs three elements:

- A sensor, which can sense the current state e.g. temperature, pH, force etc.
- A control, to assess the sensed values; is it too much or too little?
- An effector, a structure of substance to change the current state.

This rather simple system allows an organism, organ, tissue or cell to maintain the circumstances in the body at a precise level.

Interestingly, one would assume adaptation is only for a response to a changed state but it can also be used to prepare for an anticipated change, e.g. avoiding objects which are moving towards you. The relevance of this for force-related tissue damage is described here.

Healthy individuals prevent injury by assessing the impact of current or future forces on tissue. Anticipation involves a learning process where, for example, an organism has experienced the effect of hitting or being hit by an object and has learned to assess the resulting amount of tissue damage. Detection involves the interpretation of sensors and signals. The body recognises force, oxygenation and many other signals related to tissue quality. All these inputs combined determine the response of the body, by behaviour or by adaptation. By behaviour, we mean how the body moves and positions itself. For example, a sport injury makes people move carefully because the system has detected damage and anticipates that a brisk movement may worsen the damage. The signals are not always obvious, we reposition ourselves constantly based on signals from the body, these signals are subtle and can occur on a varying level of awareness. The system for maintaining homeostasis continuously monitors the current state of the body and its surroundings and adapts the actions of the organism to the characteristics of the force and the signals from the tissues (Wyatt et al, 2016). The combination of anticipating and detecting makes the system able to adapt to the situation depending on the amount of force, the time element and the tissue quality. At the cellular level cells are able to reorganise themselves quickly through adaptation to incoming forces (Haase and Pelling, 2013). In the case of injury, a cell can repair its membrane to a large extent (Tang and Marshall, 2017). An individual cell can, therefore, adapt. However, if the circumstances are beyond the level it can handle it will get stressed. Injury and damage will occur if the circumstances are also damaging to the cell itself. Finally, if the amount of damage becomes threatening for the cell, the system will anticipate it and order the cell to kill itself (apoptosis). Apoptosis is important because necrosis (uncontrolled cell death) is an important signaller, as it demonstrates the system is lacking control (Mariño et al, 2014; Berghe et al, 2015; Wallach et al, 2016).

The same applies to tissues where different tissues have different roles to handle incoming forces; problems in one tissue can cause problems in other tissues. Also, tissue adapts to forces (Wang and Sanders, 2003) and injury by remodelling, repair or regeneration. The difference between cell level and tissue level adaptation is that the body never decides to actively order tissue to die.

The adaptive system should have a fast sensory component, presumably neural or connective tissue, to handle acute situations and a non-acute system to handle non-acute situations, presumably local or humoral. The control system depends on the speed the signals reach the control unit, which can be local as in the cell itself or remote as in the brain. Finally, the effector system does have a fast component, the muscular-skeletal system and also a non-acute system involving homeostasis and regeneration of cells, tissues and structures. So while the fast system repositions the body, the slower system takes care of the adaptation of the tissue to the new situation.

The adaptive system is not well described in pressure injury/pressure ulcer literature. If we, apart from the focus on force, seriously assess tissue quality and the adaptive system, we will see an improvement in our understanding of this

force-related tissue damage currently known as pressure ulcer, pressure injury or decubitus ulcer. By increasing this understanding we will be in a stronger position to predict and prevent the occurrence of problems and simultaneously tailor the treatment for the resolution of the problems that have occurred.

PATHOLOGY

The difference between normal and pathological damage is that in pathological damage the body is no longer able to adapt to the situation where the force, the tissue or the adaptive system play simultaneous roles. Research needs to address all three elements of the pathology. Not addressing one of them will lead to a gap in understanding which will prevent translation from the bench to the bed (Levine, 2018).

RECOMMENDATIONS FOR PRACTICE

- Consider this improved understanding of the body's underlying anatomy and physiology as well as the complex interactions between force, tissue and the body's natural adaptive system when planning patient care to either avoid or treat force-related tissue damage.
- Acknowledge that the current practices for resting, moving and stabilizing patients in bed, either manually or mechanically may still, despite good intentions, compromise the patients innate ability to adapt in order to maintain homeostasis and optimal tissue integrity.
- Can we move and rest patients in a way that compliments rather than complicates the body's natural defence mechanisms and its desire for homeostasis? For example, this can be done by using gravity to control and move the patient on their resting surface. That requires a range of supporting layers, allowing automated orientation of the patient in 3-dimensions in time and space. This can only be achieved when all the components and the body are moved in a synergetic manner. What can be done to enable the body to adapt and heal itself when force-related damage has already occurred?
- Picture the events occurring underneath the skin as you work with your patients.
- Plan audit and research projects that take this greater understanding of the body's innate protective systems into account when trying to reduce the health burden caused by tissue damage to patients, their families and the NHS.

CONCLUSION

We may conclude that if and how much damage occurs depends not only on the force. It also depends on the quality of the tissue involved and it depends mostly on how well the body is able to adapt its behaviour, anatomy or physiology to the situation. Not taking these three factors: force, tissue and adaptation into account will reduce the usefulness of diagnosis, prevention and treatment of at-risk patients dramatically. Research into how these concepts can be applied to improve the patient experience is urgently required; this could take the form of large-scale studies or smaller audits demonstrating a test of change in care and thinking. WUK

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