

## Research in biomedical engineering: an overview of recent literature

KS Gebhardt

*Clinical Nurse Specialist Pressure Ulcer Prevention, St George's Healthcare NHS Trust, London*

### Editor's note

Every month new tissue viability publications appear in journals spread all over the world. It is a daunting task to keep abreast with all the latest information and this new occasional series aims to help readers discover and digest all that is new in tissue viability. It is intended that this Journal maintains a flow of reviewed papers over the coming months although it is not planned for each issue to contain review(s). Please let us know what you think of this new initiative.

There can be little doubt that biomedical engineers have made an enormous contribution to the knowledge base about pressure ulceration, out of all proportion to their numbers and involvement, as a profession, in pressure ulcer management. It is fair to say that they put to shame the comparatively meagre medical and physiological contributions to knowledge in this field. Indeed, in the absence of interest from the medical profession, they have undertaken seminal research such as the epidemiological surveys of Barbenel et al<sup>1</sup> not normally associated with the field of bioengineering. It can be argued that in the process, in the 1970s, they laid most of the foundation blocks of modern pressure ulcer management and research. Most of the research has been conducted on paraplegic patients, as this is the group that biomedical engineers traditionally have been most involved with, and some statements in this literature have to be read with that proviso in mind. The four papers reviewed here are some of the latest products in this long and respected line of research and development.

### Recent papers

The first of these<sup>2</sup> describes a computerised mathematical model of the human buttock. This has been used to show, first, that changes in cushion parameters and fatty layer parameters of the buttock lead to large differences in shear strain in the fat layer but relatively little change in the muscle layer; second, even with a soft cushion and effective reduction in contact interface pressure, deformation near bony prominences is high, presumably sufficiently high to create a risk of pressure damage. The

inference is that a softer cushion is not necessarily better for pressure ulcer prevention. These findings confirm those of Quillen and Spahn (unpublished report, 1991) who compared the impact of different support surfaces on interface pressure and deep tissue distortion of real buttocks using computerised tomography scanning techniques.

Breuls et al<sup>3</sup> used engineered tissue cultures to study the impact of compression on tissue viability. Discs of a construct of muscle tubules set in a gel matrix were subjected to varying regimens of compression for 8 hours:

- Control (no compression,  $n=6$ )
- Light indenter pressure ( $n=6$ )
- 30% strain ( $n=3$ )
- 50% strain ( $n=6$ ).

Little cell death occurred in the control and light indenter pressure groups. However, under 30% compression, a significant number of cell deaths occurred after 1–2 hours, earlier under 50% strain and cell death continued to occur over time. On the basis of these findings the authors hypothesise 'that sustained cell deformation is an additional mechanism that plays a role in the development of pressure ulcers'.

Breuls et al<sup>4</sup> describe the development of a computerised mathematical model to predict pressure ulcer initiation. This is based on a 'damage law' derived from the experiments described in Breuls et al<sup>3</sup>. Experiments in mathematical modelling using the model showed that it is likely that different tissues and different anatomical sites have different damage/time curves and that pressure damage prediction based on measurement of external pressures is inadequate. While at this time 'quantitative predictions on patient related simulations are still a long way ahead', the authors seem to believe that with further refinements, movement in this direction may be possible. They postulate that the minimum requirement for a useful model would be to incorporate local load history and the local tissue tolerance.

The final paper by Bouten et al<sup>5</sup> brings together the papers described above and other themes relating to the aetiology of pressure ulcers. The authors point out that the most harmful pressure ulcers develop in the deep tissues and that the current focus on the skin/support interface may be misguided as surface pressures are not necessarily representative of internal tissue loadings. They discuss the

varying theories of how mechanical loading leads to tissue necrosis – localised ischaemia, impaired lymphatic drainage, reperfusion injury and sustained deformation of cells. As a solution to the obvious knowledge gaps, the authors suggest a hierarchical approach to the study of pressure ulcer aetiology involving computer and experimental studies covering laboratory in-vitro and in-vivo studies of cells, tissues and vasculature. They believe that such an approach could lead to an understanding of pressure ulcer aetiology, allow the development of effective clinical identification and prevention, and direct available pressure-relief strategies more efficiently.

### Discussion

The laboratory and mathematical studies presented in these papers certainly expand our understanding of just how complex the pathways to distortion-induced tissue damage might be. They are clearly well-designed, well-controlled and in the case of the in-vitro studies, show a high level of repeatability. It is quite possible that they may make a significant contribution to the development of anti-pressure damage devices and treatments in the future. I would, however, argue that we should be extremely careful about extrapolating from the behaviour of cells in simple and rigid artificial constructs to the behaviour of cells in complex, flexible living tissues with vascular and other supporting structures.

However, I have considerable reservations with regard to some of the conclusions contained in these papers, stated, perhaps most explicitly, in Bouten et al<sup>5</sup>. These suggest that a solution to the problems of pressure ulcer aetiology and prevention lies within a knowledge of the impact of mechanical stresses and strains on tissues. At best, as in Breuls et al<sup>4</sup>, there is an acknowledgement that an understanding of 'local tissue tolerance' is essential to a prediction of the likelihood of pressure damage. However, even here the implication appears to be that what is needed is knowledge of how the body responds to damage already sustained rather than of why the body has allowed it at all, when normally it does not.

To use a crude analogy, if one were researching the aetiology of falls in the elderly, measuring the hardness of the floor, the susceptibility of different tissues to bruising or the effect on the brain of hitting the floor at different speeds, while of interest (not least for the design of protective devices), is unlikely to expand our knowledge of why it happens in the first place. To do that one would need to examine the psychological, physiological and pathological processes which lead to an individual falling down. I suspect that the first step in the process would have to be a good understanding of the physiological mechanisms which enable safe upright gait in healthy individuals. We know that all humans have the potential to hurt themselves if they fall over when walking. However, it is a complex of pathophysiological changes that convert

this ever-present potential danger into an acute threat. This is not related to how the impact of landing causes damage to the body or to how the condition of the body determines the amount of damage that occurs upon falling. These are separate and secondary considerations. The primary consideration is how and why some individuals lose the ability to walk safely.

Similarly with pressure ulcers. Limited knowledge of precisely how pressure damage occurs is not the most important obstacle to effective prevention. After all, it is fairly self-evident that interface pressures and shearing strains which are applied to the human body in everyday life have the potential to cause pressure damage. However, it is equally self-evident that pressure damage does not occur when the body is healthy and unconstrained. We do not understand the mechanisms by which the body achieves this. Equally we do not understand the pathology which allows some individuals to become susceptible to pressure damage, and the only tools we have to diagnose this susceptibility are very crude predictors (such as risk assessment scores, clinical judgment and intuition). I would suggest that to progress our knowledge of pressure ulcer prevention, we need to understand these biological mechanisms of physiology and pathophysiology, rather than the parameters of physical stresses and strains.

### Conclusions

The problem of pressure ulcer aetiology is not one of mechanics. It is a problem of physiology, pathophysiology and its diagnosis. I fear that until physiologists, pathophysiologicalists and clinicians – in particular the medical profession – get their act together and begin to emulate the research effort of biomedical engineers, not much progress will be made and some of the heroic effort that biomedical engineers are contributing may ultimately prove of little clinical value.

### References

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- 3 Breuls RGM, Bouten CVC, Oomens CWJ, Bader DL, Baaijens FPT. Compression induced cell damage in engineered muscle tissue: an in vitro model to study pressure ulcer aetiology. *Annals of Biomedical Engineering* 2003; 31: 1357–1364.
- 4 Breuls RGM, Bouten CVC, Oomens CWJ, Bader DL, Baaijens FPT. A theoretical analysis of damage evolution in skeletal muscle tissue with reference to pressure ulcer development. *Journal of Biomedical Engineering* 2003; 125: 902–909.
- 5 Bouten CV, Oomens CW, Baaijens FP, Bader DL. The etiology of pressure ulcers: skin deep or muscle bound. *Archives of Medical Rehabilitation* 2003; 84: 616–619.