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Peak strain magnitudes and rates in the tibia exceed greatly those in the skull: An in vivo study in a human subject

Richard A Hillam [1], Allen E Goodship [2], Tim M Skerry [3,*]

University of Bristol, UK

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ABSTRACT

Bone mass and architecture are the result of a genetically determined baseline structure, modified by the effect of internal hormonal/biochemical regulators and the effect of mechanical loading. Bone strain is thought to drive a feedback mechanism to regulate bone formation and resorption to maintain an optimal, but not excessive mass and organisation of material at each skeletal location. Because every site in the skeleton has different functions, we have measured bone strains induced by physiological and more unusual activities, at two different sites, the tibia and cranium of a young human male in vivo. During the most vigorous activities, tibial strains were shown to exceed 0.2%, when ground reaction exceeded 5 times body weight. However in the skull the highest strains recorded were during heading a heavy medicine/exercise ball where parietal strains were up to 0.0192%. Interestingly parietal strains during more physiological activities were much lower, often below 0.01%. Strains during biting were not dependent upon bite force, but could be induced by facial contortions of similar appearance without contact between the teeth. Rates of strain change in the two sites were also very different, where peak tibial strain rate exceeded rate in the parietal bone by more than 5 fold. These findings suggest that the skull and tibia are subject to quite different regulatory influences, as strains that would be normal in the human skull would be likely to lead to profound bone loss by disuse in the long bones.

1. Introduction

Bone responds to loads it experiences, to grow and maintain a structure adequate for function, with safety factors for moderate overload (Currey, 1984; Lanyon, 1987). Excessive mass increases costs of growth, maintenance and use, so there are evolutionary reasons why tuned skeletons are advantageous over over-engineered ones. Consequences of failure on survival differ with skeletal site, so it is reasonable to assume that different bones have different safety factors.

The effect of loading bone is deformation or strain and for most long bones, deformations during peak physiological activities are 0.2–0.3% (Rubin and Lanyon, 1984). Some information related to bone strain during activity provides feedback for control of mass and architecture. Without high habitual strains, the effect of reduced usage, and specifically, removal of high magnitude/rate strain events leads to bone loss (Uhthoff and Jaworski, 1978; Skerry and Lanyon, 1995; Sugiyama et al., 2012). Bed rest, weightlessness or immobilisation in coma patients leads to profound reductions in whole body bone density (Whedon et al., 1976; Uhthoff and Jaworski, 1978; LeBlanc et al., 1987; Lavrijsen et al., 2007; Oppl et al., 2014). Similarly, post-menopausal changes are associated with bone loss and osteoporosis (Nordin et al., 1976).

However, there is one notable exception to loss of bone in the skeleton: the cranium of the skull does not lose bone during disuse or after menopause. In spaceflight, where bone is lost from limbs, there is increased cranial bone mass (Alexandre and Vico, 1996). Prolonged bed rest and spinal cord injury both result in bone loss from the appendicular and axial skeleton but not from the skull (Leblanc et al., 1990; Garland et al., 1992). Furthermore skull bones are resistant to postmenopausal bone loss (Gallagher et al., 1987) and osteoporotic fractures do not occur there (Kleerekoper and Avioli, 1993).

While animal studies show skull strains somewhat lower than limb bone strains (Ross and Metzger, 2004; Porro et al., 2014), there are no data on in vivo strains in the human skull, and only a few reports of human tibial strain recorded in vivo summarised in

* Corresponding author. Tel.: +44 114 2712414.
E-mail address: t.skerry@sheffield.ac.uk (T. Skerry).
[1] Present address: Churchdown Veterinary Centre, Cheltenham Road East, Gloucester GL3 1HX, UK.
[2] Present address: School of Veterinary Science, University of Bristol, Southwell Street Bristol BS2 8EJ, UK.
[3] Present address: Centre for Integrated Musculoskeletal research into Ageing, Mellanby Bone Centre, Department of Human Metabolism, University of Sheffield, Beech Hill Road, Sheffield S10 2RX, UK.

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two recent review articles (Yang et al., 2011; Al Nazer et al., 2012), so we measured strains in the human tibia and cranium with simultaneous recording of bite and ground reaction forces. We show profound differences in physiological strain magnitudes and rates at the sites, suggesting either increased sensitivity to low strains in the skull, or insensitivity to effects of disuse.

2. Materials and methods

2.1. Strain gauges and subject

Three rosette strain gauges (EA-06-060RZ-120, Measurement Group, Basingstoke, UK) were prepared for implantation as described (Lanyon, 1973; Lanyon et al., 1975) and sterilised. The subject was a relatively normal healthy, 70 kg, 29 year old male (RAH). All procedures were approved by Bristol Healthcare Trust Ethical Committee to prevailing regulations and the subject gave consent for the work, and for his identity to be revealed. The gauges were applied, activities performed and gauges removed within one day.

2.1.1. Surgical implantation of the strain gauges

The right tibia and scalp were prepared for aseptic surgery. Surgery was performed under bupivacaine anaesthesia (Marcain 0.25% with 1:200,000 adrenaline, Astra Pharmaceuticals Ltd., Hertfordshire, UK).

2.1.2. Parietal bones

A 7 cm incision was made 2–3 cm lateral to the midline. Skin was retracted and haemorrhage controlled. A 1.5 cm square area of periosteum was elevated and bone scraped free of soft tissue. The bone was degreased with diethylether and chloroform. Cyanoacrylate adhesive (Histoacryl, Braun, Germany) was applied to the gauge. The gauge was placed onto the bone, so the central element was parallel to the sagittal plane. Pressure was applied for one minute to bond the gauge onto the bone. The incision was closed with lead wires passing out of the incision. The same procedure was performed on the contralateral bone. An image of a skull with a strain gauge attached in the same location and orientation is included in supplementary information.

2.1.3. Tibia

Under identical local anaesthesia, a 7 cm incision was made parallel to the long axis of the tibia. The bone was prepared as before. The gauge was bonded with the central element parallel to the bone's axis. This was confirmed by radiography. An image of a tibia with a gauge attached in the same location, and a photograph of a strain gauge in place is in Supplementary information.

2.2. Strain recording

Strain gauges were connected to amplifiers (2120A, Measurement Group, UK) whose output was fed into an A-D card (RTI-815, Analog, Norwood, USA) in a PC. Custom capture software was used ('Super', by D. McNally, University of Nottingham). Circuits were balanced with the subject relaxed (where bones were subjected to minimal stress) so that strain could be zeroed. Between sets of activities, gauges were re-zeroed. For the cranium, the relaxed position was achieved by sitting without conscious neck or face muscle activity and the head forward. For the tibia, the circuits were balanced with the subject sitting and the foot off the ground. Strains were recorded at 50 Hz for sedentary activities where no impact transients were expected and 500 Hz for more vigorous activities. No noise filtering was used

2.3. Bite force transducer

Measurements of bite force were made using a dental occlusal force meter, custom-made by staff at the Medical Physics Department, Sunderland General Hospital, UK. The device incorporated a 1000 N loadcell between dental occlusal pads. Load cell output was connected to the same amplifiers and PC as used for capture of strain data to allow simultaneous recordings. Calibration was performed by hanging known masses on it.

2.4. Ground reaction force measurements

We made force plate recordings (Kistler Instruments, Winterthur), as described previously (Dow et al., 1991; Williams et al., 1999). Output from the force plate was converted and stored as for strain, then processed to calculate the orthogonal ground reaction force parameters using in-house software, exported to Microsoft Excel for analysis and Graphpad Prism for display. For technical reasons we were unable to record GRF during walking and squat exercises. The figures display only Fz, but all GRF recordings are in supplementary information.

2.5. Activities

A range of activities were planned.

1. Biting onto a dental occlusal force meter with cheek teeth and incisors.
2. Eating a banana.
3. Grimacing/pulling facial expressions.
4. Walking at $\sim$0.7 m s^{-1}.
5. Performing squats with a 25 kg weight.
6. Jumping from 0.45 m.
7. Heading a 4.5 kg medicine/exercise ball.
8. Jumping from 1.3 m.

Except for the 1.3 m jump, all activities were performed with the feet protected by socks. For the 1.3 m jump, rubber soled, leather boots (Trader, Debenhams, UK) were worn.

2.6. Data analysis

Principal strain magnitudes were calculated from individual strain element data. Strain rates were calculated by selecting maximum rise in strain for each activity, calculated over 40 ms periods. The start time of the 40 ms period is given in results. The full set of recorded bite force, ground reaction force and strain data (and derived strain rate data) are in Supplementary information. Because the experiment had only one subject, we selected representative traces for each activity to display in figures, and we have not undertaken statistical analysis.

3. Results

3.1. Strain measurements

Where relevant, we made simultaneous recordings of parietal and tibial strains. For activities that involved no significant body movement, we recorded only parietal strains, as tibial strains were unrelated to activity. The right parietal gauge and the tibial gauge functioned well and we recorded credible strains from them. The central element of the left parietal gauge did not produce any data other than low level noise and we excluded data from that gauge in results presented. However, raw strain magnitudes from the functioning elements of that gauge were comparable with those from the right parietal gauge. The right parietal gauge worked it was detached following a direct impact from heading the medicine ball. The time this occurred was obvious because of immediate formation of a large haematoma and subsequent strain recordings that were quite different from earlier recordings. After this, parietal data were excluded. The only remaining activity to be performed was the jump from 1.3 m, so we have only tibial strain data for that. The tibial gauge functioned throughout the experiment. We recorded strains (and bite force/ground reactions) for at least 3 repetitions of each activity.

3.2. Strains during activities

3.2.1. Activities inducing parietal strain

Bite force and strain were recorded with the bite force transducer placed between molar teeth and incisors. Biting was associated in some but not all cases with apparently related increases in parietal strain (Fig. 1A and B), where peak principal compressive and tensile strains recorded were +0.0064% and −0.0059% at left molar bite force of 420 N. In the same recording, at slightly higher peak molar bite force of 449 N, strains were +0.0043% and −0.0014%, respectively. Peak parietal strain rate was 0.925% s^{-1} at 1640 ms, when bite force was being released.

Other recordings revealed no relationship between bite force and parietal strain, and what appeared to be little more than noise in traces. For example, a bite with incisors of 430 N was associated with peak principal compressive and tensile strains of +0.0029% and −0.0039% (Fig. 2A and B), although other bites had similar