Bone Height Measurements Around a Dental Implant After a 6-month Space Flight: A Case Report

Claire Haigneré, DDS[1]/Pierre Jonas, DDS, DSO[2]/Philippe Khayat, DDS, MSD[3]/Gérard Girot, DDS, DSO[4]

Purpose: *In space, astronauts are subject to microgravity, which reduces skeletal loading and osteoblast function and can cause bone resorption and a decrease in bone density. No known research to date has studied the effect of microgravity on dental implants. This study evaluated peri-implant bone changes around a dental implant placed in a French astronaut who spent 6 months in Russia's Mir Space Station.* ***Materials and Methods****: Measurements were performed by 2 examiners before the flight (baseline), after the flight (stage 1), and following a recovery period (stage 2). Standardized periapical radiographs were taken, and data were recorded using a photomicroscope and a measuring scale.* ***Results:*** *Cumulatively, the implant sustained 0.43 mm of mesial bone gain and 0.31 mm of distal bone loss.* ***Discussion:*** *The observed peri-implant bone height changes were within normal limits and the implant appeared very stable during the course of this study.* ***Conclusion:*** *Peri-implant bone levels remained stable after 6 months in microgravity, and the implant continued to function without complications.* (Case Report) INT J ORAL MAXILLOFAC IMPLANTS 2006;21:450–454

Key words: *dental implants, microgravity, peri-implant bone, space flight*

In outer space, human bodies are subjected to a state of weightlessness or microgravity, which is approximately 1-millionth (10^{-6}) of the earth's gravitational pull. In that environment, weight has no action on mechanical systems, as evidenced by objects, fluids, and astronauts that float inside space vessels orbiting the earth. The immediate effects of microgravity on the human body include a shifting of fluids to the upper part of the body, loss of fine control of the musculoskeletal system, and changes in the functions of the vestibular and tactile systems.[1] Diminished gravitational forces require living organisms to adapt to a new set of environmental stimuli. Humans undergo significant physiologic and biochemical changes, including: *(1)* negative calcium balance, resulting in loss of bone; *(2)* atrophy of antigravity muscles; *(3)* fluid shifts and decreased plasma volume; and *(4)* cardiovascular deconditioning, which leads to orthostatic intolerance.[2] Humans on earth experience similar muscle and bone mass loss under certain conditions, such as the normal aging process, limb immobilization during the healing of orthopedic injuries, wheelchair confinement, and chronic bed rest required for healing.[3]

An accelerated loss of bone density, called "space osteoporosis," also occurs during prolonged space flights of 4 to 6 months. Individual bone density losses can reach as high as 14% in the femoral neck depending on the length of the mission and the bone turnover rate of the astronaut.[4] While mechanical unloading of the weight-bearing limbs is a leading cause, other risk factors for space osteoporosis include stress, nutrition imbalances, fluid shifts, dehydration, and alterations in bone perfusion (blood flow).[4] Space osteoporosis is also associated with decreased calcium absorption and increased urinary calcium excretion, which leads to a general loss of calcium.[4] This phenomenon takes place in spite of high calcium intake and vitamin D supplementation.[4] Scientists concerned that space osteoporosis could limit the ability of humans to explore the universe have developed countermeasures to help mitigate the problem, including exercise programs,[2,5] the use of vibration plates to simulate bone,[6] drugs, dietary modifications, and inertia suits (eg, the Russian "penguin" suit).[5]

[1]Private Practice, Paris, France.
[2]Expert in Dentistry, European Space Agency, Jouy en Josas, France.
[3]Affiliate Assistant Professor, Department of Restorative Dentistry, School of Dentistry, University of Washington, Seattle, Washington.
[4]Professor, University of Paris V, Paris, France.

Correspondence to: Dr Claire Haigneré, 28 rue des tanneries, 75013 Paris, France. E-mail: claire.haignere@ifrance.com

A decrease in osteoblastic activity and an increase in osteoclastic activity during long space flights have been observed to alter bone homeostasis, although no change in the hormone responsible for phosphate and calcium metabolism has been found.[7] Bone density is lost primarily from the greatest weight-bearing bone structures as compared to lower weight-bearing bones.[8,9] Similar reduced function has been simulated in human volunteers (bed rest studies) and in animal studies (hind limb unloading in rats).[10–12] This finding is in accordance with Wolff's law, which states that mechanical forces determine bone form and function.[13,14] These studies have helped to shed light on the changes in bone physiology that occur during prolonged space flight.

During the last decade, Russian cosmonauts involved in 4- to 6-month space flights on the Mir Space Station were examined by dual energy x-ray absorption (DEXA) to determine regional bone mineral changes along their entire skeletons. The results showed that a redistribution of density occurred from the lower extremities to the head. The greatest loss of density was observed in pelvic bones, lumbar vertebrae, and femoral necks, while bone mineral density actually increased at the head level (0.2% per month).[15,16] It is not known whether microgravity or space osteoporosis could affect endosseous dental implant function or create changes in the surrounding bone tissues.

The aim of this investigation was to evaluate bone height changes around a dental implant in a French astronaut after a 189-day space flight and a recovery period.

CASE REPORT

Background

A 48-year-old male French astronaut involved in a series of space flights on the Mir Space Station presented with a missing mandibular right first molar (Fig 1). The tooth had been extracted 10 years earlier, and an adjacent second premolar was intact and healthy. The patient rejected the option of preparing his adjacent healthy tooth to support a conventional fixed prosthesis and was unable to wear a removable prosthesis because of the nature of his work in a microgravity environment. A comprehensive diagnostic workup was performed to thoroughly evaluate the patient. His participation in the European Space Program indicated excellent health status, which was supported by a review of his medical and dental records. There was no contraindicating physical condition that might negatively affect osseointegration (eg, endocrine, autoimmune, musculoskeletal, or hematologic disease) or compromise long-term implant survival (eg, periodontal disease, immunosuppressive drug therapy, collagen disease, history of osteomyelitis, irradiated bone tissue).[17] No allergies were identified that could contraindicate the use of certain drugs or other substances associated with dental implant therapy.

A comprehensive oral examination was conducted to assess the patient for undiagnosed disease, destructive parafunctional habits, and oral pathologies that might require treatment prior to implant surgery. The 3-dimensional volume and density of available bone in the proposed implant site and the adjacent anatomical structures were evaluated through a computerized tomographic (CT) scan. A diagnostic cast was fabricated and mounted on a semi-adjustable articulator, and a face-bow and vertical registration were utilized to determine the jaw relationships, available occlusal dimension, proposed implant position, and crown-root ratio. After careful examination of the diagnostic workup and treatment plan, the medical staff of the European Space Agency authorized dental implant surgery, and the patient provided his signed informed consent.

A surgical template was fabricated from a diagnostic waxup to facilitate optimum placement of the implant relative to the proposed prosthesis. Antibiotic prophylaxis involved daily administration of amoxycillin (500 mg) beginning 1 hour before surgery and continuing for 4 days postoperatively. The patient was prepared for surgery and anesthetized (2% lidocaine and 1:100,000 epinephrine) by inferior alveolar block in the mandible. Horizontal midcrestal and terminal vertical releasing incisions were made, and full-thickness flaps were elevated to expose the alveolar process. An osteotomy was prepared by sequential cutting with internally irrigated drills, and a 4.7 × 13-mm titanium alloy (Ti6Al4V) screw implant (Screw-Vent; Zimmer Dental, Carlsbad, CA) was placed according to the manufacturer's protocol for a 2-stage surgical procedure. The mucoperiosteal flaps were approximated and closed with 4.0 vertical interrupted mattress sutures (Vicryl; Johnson & Johnson/Ethicon, Somerville, NJ). After 10 days of healing, the sutures were removed.

Healing was uneventful, and the patient was recalled 3 months after implant placement. A radiographic (periapical) evaluation was performed to assess the newly formed bone and its close adaptation to the implants. After anesthetizing the patient with local infiltration, a midcrestal incision was made, and a mucoperiosteal flap was elevated to expose the implant. Clinical osseointegration was further evaluated using manual percussion and lateral pressure. A healing collar was attached to the implant,

Table 1 Bone Height Measurements Around the Test Implant and the Mandibular Left First Molar

	Implant		Mandibular left first molar	
	Mesial (mm)	Distal (mm)	Mesial (mm)	Distal (mm)
Stage 1/baseline	+0.41	−0.20	−0.14	−0.04
Stage 1/stage 2	+0.02	−0.11	−0.04	−0.07
Stage 2/baseline	+0.43	−0.31	−0.18	−0.11

Fig 1 A French astronaut in microgravity in the MIR space station during a 180-day space flight.

and the soft tissues were sutured (3-0 Vicryl) around it according to conventional implant procedures.

Restorative procedures were begun approximately 14 days later. A gold-palladium alloy was selected (V-Delta Metalor; Metalordental, Boulogne Billancourt, France). The ceramometal single-tooth restoration was screw-retained and connected directly to the implant. This restoration had been in function without complications for 15 months when the cosmonaut was scheduled for a 6-month mission to the Mir Space Station.

MATERIALS AND METHODS

Bone height around the implant was measured using periapical radiographs (Ektaspeed E0-02; Eastman Kodak, Rochester, NY).[18] To standardize the images, a paralleling technique involving a film holder and a beam-guiding rod were used. An acrylic resin bite block was made.[19] The film was processed immediately after exposure utilizing the manufacturer's protocol. Two radiographs were obtained: 1 of the dental implant (test) and 1 of the contralateral mandibular left first molar (control). The patient had a ceramometal crown on the mandibular left first molar. A photomicroscope with 13.5× magnification (Ultraphot; Carl Zeiss France SAS, Le Pecq, France) and a measuring scale were used to assess the distance between a reference point R on the implant (the abutment-implant junction) and the marginal bone level on both the mesial and distal aspects of the implant.[20] In the contralateral region, the distance between a second reference point C (metal-root junction) and the marginal bone level was measured.[21] Measurements were made independently by 2 examiners, and data were collected 1 month before the flight (baseline), 1 month after landing (stage 1), and after an 8-month recovery period (stage 2) (Table 1; Figs 2 and 3).

RESULTS

The test (implant) radiographs revealed an increase in mesial bone height (stage 1, +0.41 mm; stage 2, +0.2 mm) and a decrease in distal bone height (stage 1, –0.2 mm; stage 2, – 0.11 mm) throughout the observation period (baseline through stage 2). Cumulative results for the test sample after 6 months in microgravity were 0.43 mm of mesial bone gain and 0.31 mm of distal bone loss.

The control (tooth) radiographs showed bone loss in both the mesial (stage 1, –0.14 mm; stage 2, –0.04 mm) and distal (stage 1, –0.04 mm; stage 2, –0.07 mm) regions throughout the observation period. Cumulative results for the control sample after 6 months in microgravity were 0.18 mm of mesial bone loss and 0.11 mm of distal bone loss.

DISCUSSION

In a study of Russian cosmonauts who spent 1 and 6 months, respectively, in the MIR Space Station, Collet and associates[9] found that bone formation activity appeared to be suppressed after both missions, with the greatest loss of trabecular and cortical bone observed in the tibia. After 6 months of recovery, this compromise was still evident in the trabecular bone but not in the cortical bone.[9] Since no such changes occurred in the distal radius at any time, the researchers[9] concluded that the lower weight-bearing bones appeared to be more sensitive than the non–weight-bearing bones in terms of space flight–induced bone loss. This provides confirmation of the proposal formulated over a century ago by Julius Wolff that mechanical stress determines the form and function of bone.[14]

The mechanisms by which loading of bone is sensed and translated into signals controlling bone

Fig 2a Periapical radiograph of the mandibular left first molar (control) 1 month before the flight (baseline).

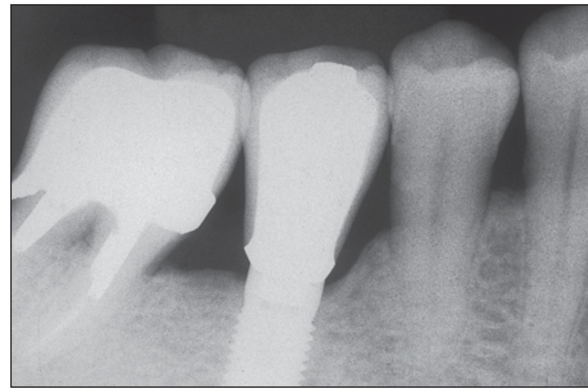

Fig 2b Periapical radiograph of the implant replacing the mandibular right first molar (test) 1 month before the flight (baseline).

Fig 3a Periapical radiograph of mandibular left first molar (control) after the recovery period (stage 2).

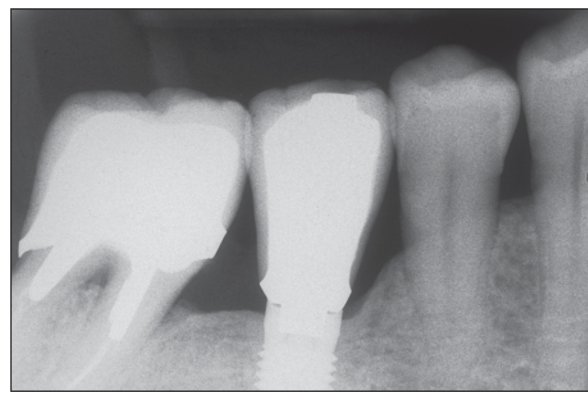

Fig 3b Periapical radiograph of the implant replacing the mandibular right first molar (test) after the recovery period (stage 2).

formation are still unknown.[22] It has been theorized that matrix/cell interactions underlie much of the mechanocoupling and that integrins are a prime mediator of such interactions.[21] It is also unclear how systemic hormones, such as parathyroid hormone (PTH), growth hormone (GH), and serum 1,25-dihydroxyvitamin D (1,25[OH]2D), compared to locally produced factors such as insulin growth factor-I (IGF-I), parathyroid hormone-related protein (PTHrP), bone morphogenetic proteins (BMPs), and transforming growth factor-β (TGF-β), modulate the cellular response to load.[22] Research indicates that skeletal unloading leads to resistance to the anabolic actions of IGF-I on bone as a result of failure of IGF-I to activate its own signaling pathways.[22] This is associated with a reduction in integrin expression, which suggests interaction between these 2 pathways.[22] The general picture is that bone resorption is unaltered or increased while bone formation is decreased partly as a result of reduced osteoblast function.[7] In vitro studies with osteoblastic cells have demonstrated that their differentiation and cell morphology were altered by prolonged exposure to microgravity, which induced the development of an adipocytic lineage phenotype.[7,23]

Data analyzed from the 1973–1974 Skylab missions disclosed that there was a rise in the systemic hormone cortisol, which may play a role in bone loss in flight.[24] In flights where bone loss was measured, the crew members experienced a significant loss of calcium accompanied by a rise in 24-hour urinary cortisol during the entire flight period.[24] In ground-based work on osteoblasts, Hughes-Fulford and colleagues[24] found that equivalent amounts of glucocorticoids inhibited osteoblast cell growth. Quiescent osteoblasts were also slower to enter the cell cycle in microgravity, which reinforces the concept that the diminished force of gravity may be a significant cause of bone loss in space.[24]

Despite these findings, only slight marginal bone changes were observed around both the implant (test sample) and the contralateral tooth (control sample) in the present investigation. Cumulative distal bone loss was only 0.2 mm greater around the implant compared to the same location around the tooth. The implant experienced +0.48 mm bone gain compared to –0.18 mm of bone loss for the tooth in the same location. In a prospective study of 238 mandibular implants monitored for 9 years, Chaytor and coworkers[25] documented considerable variation in marginal bone levels around dental implants in individual patients over time; in some years there was slight bone loss, and in other years there was slight bone gain. However, it is important to note that those mean annual bone level changes were very low (range = –0.2 mm to +0.2 mm) and rarely differed significantly from zero.[25]

In the present investigation, distal bone loss measurements for both the test and control samples were consistent with the limits of mean normal bone height variations reported by Chaytor and associates.[25] Distal bone loss on both samples was inconsequential and suggests normal variations of bone level changes rather than the effects of generalized bone loss associated with prolonged exposure to microgravity.

While the number of samples in this report was too small to draw any definitive conclusions, and the findings should be considered preliminary, this article can be considered clinically significant because it is the first known documentation of the effects of long-term exposure to microgravity on dental implants. As space exploration increases, greater research on the maintenance of osseointegration in microgravity will be needed. One area for future study would be resonance frequency analysis (RFA) of peri-implant bone density before and after long-term space flights.

CONCLUSION

Overall bone height around this dental implant appeared to be very stable during the course of the investigation and did not seem to be influenced by a 6-month stay in space. Furthermore, the implant restoration was symptom-free and fully functional during the observation period.

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