The Relationship Between Operative and Radiographic Acetabular Component Orientation

which factors influence resultant cup orientation?

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“There is great variability in acetabular component orientation following hip replacement. The aims of this study were to compare the component orientation at impaction with the orientation measured on post-operative radiographs and identify factors that influence the difference between the two. A total of 67 hip replacements (52 total hip replacements and 15 hip resurfacings) were prospectively studied.

Intra-operatively, the orientation of the acetabular component after impaction relative to the operating table was measured using a validated stereophotogrammetry protocol. Post-operatively, the radiographic orientation was measured; the mean inclination/anteversion was 43° (SD 6°)/19° (SD 7°). A simulated radiographic orientation was calculated based on how the orientation would have appeared had an on-table radiograph been taken intra-operatively.

The mean difference between radiographic and intra-operative inclination/anteversion was 5° (SD5°)/-8° (SD 8°). The mean difference between simulated radiographic and intra-operative inclination/anteversion, which quantifies the effect of the different way acetabular orientation is measured, was 3°/-6° (SD 2°). The mean difference between radiographic and simulated radiographic orientation inclination/anteversion, which is a manifestation of the change in pelvic position between component impaction and radiograph, was 1°/-2° (SD 7°).

This study demonstrated that in order to achieve a specific radiographic orientation target, surgeons should implant the acetabular component 5° less inclined and 8° more anteverted than their target. Great variability (2 SD about ±15°) in the post-operative radiographic cup orientation was seen. The two equally contributing causes for this are variability in the orientation at which the cup is implanted, and the change in pelvic position between impaction and post-operative radiograph.”
GANDER REVIEW

Effect of selenium on markers of risk of pre-eclampsia in UK pregnant women: a randomised, controlled pilot trial

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“Pre-eclampsia is a serious hypertensive condition of pregnancy associated with high maternal and fetal morbidity and mortality. Se intake or status has been linked to the occurrence of pre-eclampsia by our own work and that of others. We hypothesised that a small increase in the Se intake of UK pregnant women of inadequate Se status would protect against the risk of pre-eclampsia, as assessed by biomarkers of pre-eclampsia.

In a double-blind, placebo-controlled, pilot trial, we randomised 230 primiparous pregnant women to Se (60 mg/d, as Se-enriched yeast) or placebo treatment from 12 to 14 weeks of gestation until delivery. Whole-blood Se concentration was measured at baseline and 35 weeks, and plasma selenoprotein P (SEPP1) concentration at 35 weeks. The primary outcome measure of the present study was serum soluble vascular endothelial growth factor receptor-1 (sFlt-1), an anti-angiogenic factor linked with the risk of pre-eclampsia. Other serum/plasma components related to the risk of pre-eclampsia were also measured.

Between 12 and 35 weeks, whole-blood Se concentration increased significantly in the Se-treated group but decreased significantly in the placebo group. At 35 weeks, significantly higher concentrations of whole-blood Se and plasma SEPP1 were observed in the Se-treated group than in the placebo group.

In line with our hypothesis, the concentration of sFlt-1 was significantly lower at 35 weeks in the Se-treated group than in the placebo group in participants in the lowest quartile of Se status at baseline (P<0.039). None of the secondary outcome measures was significantly affected by treatment. The present finding that Se supplementation has the potential to reduce the risk of pre-eclampsia in pregnant women of low Se status needs to be validated in an adequately powered trial.”
GANDER REVIEW

Zoledronic Acid Prevents Decreases in Bone Mineral Density in Patients with Prostate Cancer Undergoing Combined Androgen Blockade

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The aim of this study was to evaluate the effect of zoledronic acid (ZA) on bone mineral density (BMD) in patients with prostate cancer receiving combined androgen blockade (CAB) as a first-line androgen deprivation therapy. Patients receiving CAB for prostate cancer without bone metastasis were candidates for this study. Forty-two patients were randomly assigned to receive either ZA or no treatment. BMD were measured at baseline and at 12 months. Bone-turnover markers, including cross-linked N-telopeptide of type I collagen (NTX), C-telopeptide of type I collagen (ICTP), and bone-specific alkaline phosphatase (BAP), were assessed during study periods. Patients on ZA maintained BMD after a year of treatment.

This prospective randomized trial was performed in the Department of Urology at Wakayama Medical University between July 2009 and August 2013. All patients between 60 and 80 years of age who had PCa without bone metastasis and who didn’t receive ADT previously were candidates for this study. Patients were excluded from study if they had scoliosis, osteosclerosis of the lumbar spine, any other spinal diseases. The 42 patients to meet all criteria were randomly assigned to receive either ZA (ZA-treated group; n = 21) or no treatment (control group; n = 21). At the screening visit, BMD of the posteroanterior lumbar spine (L2-L4) was determined by dual-energy x-ray absorptiometry (DXA), and the T-score was calculated.

At 1 year after treatment, patients on ZA maintained their T-score (P = 0.74), while control patients experienced a significant decrease in T-score (−3.9%, P = 0.001). The present study demonstrated that a single infusion of ZA at the time of initiation of CAB for ADT resulted in maintenance of BMD of the lumbar spine in men with PCa without bone metastasis. The effect is even more pronounced in patients with initially low T-scores.
This is the discharge nurse, she'll be able to tell you about all the services you aren't able to get any more when you leave.