INTRODUCTION

Over 600,000 elderly individuals in nursing homes are regular wheelchair users (Redford, 1993). Brandeis et al. (1990) reported that 17.4% of patients had pressure ulcers upon admission to a nursing home. Ulcer development within the first year after admittance was 9.5-13.2% and by two years was 20.4-21.6%. There was greater re-hospitalization rate and death rate of residents that developed a pressure ulcer after their admission. The ischial tuberosities constituted 15% of the overall ulcer locations, while the most common site for pressure ulcer development was under the sacrum or coccyx with prolonged bed rest (Smith, 1995)

Multiple factors influence the development of pressure ulcers and there is no consensus as to the primary predisposing factors (Pang & Wong, 1998). However, several studies indicate that pressure, shear, friction, and moisture as the four main causative factors, with the magnitude and duration of pressure as the key factor among these four (Smith, 1995; Pang & Wong, 1998). Intervention efforts that have attempted to decrease the seat interface pressures of the elderly that have been institutionalized, but few objective criteria identify individuals with increased seat interface pressures. Due to the relationship between peak pressure and pressure ulcer development, it may be important to provide objective characteristics of those with high peak seat interface pressures. Garber and Krouskop (1982) was the only investigation that examined the effects of body build on seat interface pressures. The majority of participants in this study had spinal cord injuries, all with a history of pressure ulcers. Participants were divided into three categories: thin (< 90% ideal body weight), average, and obese (>110% ideal body weight). There was a trend between body build and peak seat interface pressures. Greater peak seat interface pressures were reported under the bony prominences among the thin population compared to those classified as obese.

The purpose of this study was to determine if there were differences in peak seat interface pressures of elderly who are institutionalized based on their body mass index (BMI).

METHODS

Seventy five individuals aged 65-95 residing in several skilled nursing facilities in small urban area volunteered to participate. Residents whose primary diagnosis was CVA or lower extremity amputation were excluded secondary to the likelihood of producing asymmetrical peak seat interface pressures. Residents who were no longer responsible for their own health care decisions were excluded. Each participant’s age, gender, body weight, and height were obtained from their current medical chart.

The Novel Pliance™ seat pressure-mapping system was used to collect seat interface pressure data. This system comprises 1024, 1.5 cm² capacitive sensors, enclosed in a flexible mat that is 2mm thick. This pressure mat was connected analog to digital electronics and interfaced to a personal computer. The system contains an 8-bit analog to digital converter and sensor data was captured at 15 Hz. Weekly, the sensor mat was checked for accuracy in a pressure chamber. If sensor readings were inaccurate by greater that ±1 kPa the sensor mat was recalibrated.

The pressure mat was placed in a standard folding wheelchair with a sling-type seat. Participants were asked to sit with their hands resting in their lap (not on the armrests), sitting as far back in the wheelchair as comfortable with both feet resting on the footrests. Footrests were adjusted to position the femur parallel to the floor. Seat interface pressures were collected for 30 seconds. Test-retest reliability of peak seat interface pressures of a group of 10 residents yielded an intraclass correlation coefficient of 0.84. This reliability reflects the reproducibility of the participant positioning as well as the peak seat interface pressure measurements. BMI was calculated by dividing the residents body weight in kilograms
by their height in meters squared (Kenney et al., 1995). BMI values were classified by the ACSM as follows: 20-24.9 kg/m²—desirable range for adult men and women, 25-29.9 kg/m²—grade 1 obesity, 30-39.9 kg/m²—grade 2 obesity, and > 40 kg/m²—grade 3 obesity (morbid obesity). A thin category was added to the BMI groupings by the investigators. This was defined as a BMI value of less than 20 kg/m². No data from residents in the grade 3 obesity category were obtained.

A one-way ANOVA (p<0.05) was used to compare peak seat interface pressures between the BMI groups (thin, desirable range, grade 1 obesity, and grade 2 obesity). Pair wise, post-hoc comparisons were performed using the Bonferoni procedure.

RESULTS
Peak seat interface pressures were different based on BMI groups (p=0.00). Post-hoc comparisons demonstrated differences in peak seat interface pressures between the thin, desirable range, grade 1 obesity, and grade 2 obesity groups (p=0.00, p=0.00, p=0.00, p=0.00, respectively). Differences in peak seat interface pressures diminished as BMI increased (see Figure 1). Peak seat interface pressures were different between the desirable range and the thin group (p=0.00) but no differences were between the desirable range and other groups. Differences in peak seat interface pressures were found between the grade 1 obesity and the thin group (p=0.00) but no other differences between other groups. Peak seat interface pressures were different between the grade 2 obesity group and the thin group (p=0.00) with no other differences between other groups.

DISCUSSION
Peak seat interface pressures were greatest in the thin elderly group with the lowest BMI of the groups examined. Differences in peak seat interface pressures were less as the BMI increased. Garber and Krouskop (1982) reported a similar relationship between body build and peak seat interface pressure in individuals with spinal cord injuries. The relationship between body build and peak seat interface pressures is further supported by the differences in BMI and peak seat interface pressures in the present study. An explanation for these findings may be the reduction in subcutaneous tissue. This may result in the bony prominences being less protected against localized loads (Shannon & Lehman, 1996; Maklebust, 1997).

REFERENCES