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The incidence of pressure ulcer in patients on mechanical ventilation and effects of selected risk factors on pressure ulcer development*

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Background/aim: This study aimed to determine the incidence of pressure ulcers in patients on mechanical ventilation and selected risk factors likely to play a role in pressure ulcer development.

Materials and methods: The study included 110 patients recruited from an anesthesia critical care unit of a university hospital. Data were collected with a demographic and clinical characteristics form. The form was composed of questions about demographic characteristics and clinical features including diagnosis, duration of mechanical ventilation, general well-being, oxygenation, perfusion, and skin condition.

Results: The incidence of pressure ulcer was 15.5%. Duration of mechanical ventilation was longer and the body mass index was higher in patients developing pressure ulcers than in those without pressure ulcers. Additionally, 90.11% of patients with pressure ulcers had edema and 82.35% of patients with pressure ulcers received vasopressin. The patients with pressure ulcers had higher PH levels, lower PaO2 levels, higher PCO2 levels, lower SaO2 levels, and higher urine output.

Conclusion: It can be recommended that nurses and other health professionals should be aware of factors playing a role in pressure ulcer development and should be able to conduct appropriate interventions to prevent pressure ulcers.

Key words: Pressure ulcer, mechanical ventilation, critical care, nursing

1. Introduction

A pressure ulcer (PU) is localized tissue damage in the dermis and subdermis caused by compression, friction, shearing, and other factors (1). It is commonly encountered in all hospitalized patients, especially those in intensive care units (ICUs) (2). The incidence of PU was found to increase from 4% to 49% in Denmark and vary from 38% to 24% in Germany (3) and from 14% to 42% in the United States (4,5). Studies from Turkey showed that the incidence of PU varied between 15% and 29% (6–8).

Mobilization, sensorial perceptions, and consciousness in patients in ICUs are impaired due to the administration of sedative and anesthetic agents (9–11). It has been shown that vasopressin administered to maintain sufficient cardiac output in ICUs leads to constriction in the capillary circulation, which prevents oxygen and blood supply to the skin. This creates a risk of PU (12). Changes in metabolism resulting from such conditions as major surgery, burns, major trauma, and sepsis in ICUs increase the risk of PU development (9–13). In addition, the risk of PU is increased due to impairment of hemodynamic status, cardiovascular diseases, circulatory failure, impaired oxygenation, diabetes mellitus, anemia, infection, edema, catabolic disorders, and pressure (4,10,11,14). In a systematic review, it was reported that PU is not caused by a single factor, but rather develops due

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to a combination of factors including moisture status of the skin, age, hematological measures, nutrition, and poor general health status. It was also noted in the review that decreased mobility, perfusion, and skin status are the most important factors playing a role in the development of PU (15).

Mechanical ventilation (MV) creates a high risk of PU in ICUs. It causes immobility, which reduces venous return to the heart. This leads to hypotension and decreased perfusion, resulting in tissue necrosis (9,10,13,14,16). In studies on patients on MV in ICUs, high PH levels, high serum glucose levels, low diastolic blood pressure (17), low serum albumin levels, and prolonged length of stay in the hospital and in ICUs were found to increase the risk of PU development (7,13).

PU leads to pain and an inflammatory response, which increases the risk of systemic infection, mortality, length of stay in the hospital, and health costs and decreases the quality of life (10,18,19). Prevention of PU has been a nursing concern for many years. Many clinicians think that PU development is not simply the fault of poor nursing care, but rather a failure of the entire health care system and hence a breakdown in the cooperation and skills of the entire health care team, including nurses, physicians, physical therapists, and dietitians. Although prevention of PUs is a multidisciplinary responsibility, nurses play a major role. In a study on Japanese nurses, long work hours were found to increase the prevalence of physical restraint and PUs (20).

Nurses can enhance the quality of nursing care, decrease the length of stay in ICUs and at the hospital, reduce health costs by diagnosing risk factors of PUs completely, and use appropriate strategies for prevention of PU (21). There have been few studies on this issue in Turkey (17). Therefore, the purpose of this study is to determine the incidence of PUs in patients on MV and selected risk factors likely to play a role in PU development.

Research questions:

1. Is there an effect of general health status on PU development?
2. Is there an effect of oxygenation status on PU development?
3. Is there an effect of perfusion status on PU development?
4. Is there an effect of skin conditions on PU development?

The results of this study will contribute to the prevention of PUs and the development of effective nursing strategies.

2. Materials and methods

2.1. Study design, setting, and sample

This descriptive, cross-sectional, and prospective study was conducted at the anesthesia ICU of a university hospital between June 2012 and January 2013. The study included 110 patients recruited from the anesthesia ICU. The hospital is located in the province of İzmir in western Turkey. Sample inclusion criteria were: being 18 years old or older, being on MV for at least 24 h, and not having a PU at the time of admission to the ICU. Sample exclusion criteria were: having paraplegia or quadriplegia before admission to the ICU, having a PU before receiving MV, and being followed on a trauma board. There are 18 beds and 36 nurses providing care in the ICU. Every three patients are taken care of by one nurse.

2.2. Nursing interventions carried out to prevent PUs in the anesthesia ICU where the study was conducted

All patients except for those with multiple fractures and those with unstable hemodynamic status are repositioned by nurses every 2 h if they stay on air mattresses and dynamic mattresses, and every 4 h if they stay on viscoelastic mattresses. Nurses apply moisturizing cream once daily. Bed sheets are replaced by new ones every day and care is taken to avoid folds in the sheets. They are also replaced when sweating, incontinence, and leaks from wounds cause the skin to become wet. Nasogastric catheters, urinary catheters, drainage tubes, and central venous catheters are prevented from staying under the patient and from creating pressure. Air pressure status of air mattresses is checked by nurses every time they start a new work shift.

2.3. Instruments

Data were collected with a demographic and clinical characteristics form.

2.3.1. Demographic and clinical characteristics form

The form comprised questions about demographic characteristics including age and sex, and questions about clinical features, diagnosis, duration of MV, general health status, oxygenation, perfusion, and skin condition.

2.3.2. Clinical characteristics

General health status variables: These variables included serum albumin and hemoglobin levels, body mass index (BMI), nutrition, sedation and vasopressin administration, position, edema, and type of mattress.

Oxygenation status variables: These variables included power of hydrogen (PH), partial arterial oxygen pressure (PaO_2), partial arterial carbon dioxide pressure ($PaCO_2$), and oxygen saturation (SaO_2).

Perfusion status variables: These variables included systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), and urinary output (UOP).

Skin condition: Variables concerning skin condition included the mean score for the risk of PU, PU development, and PU stage on admission to the ICU and during MV. The Braden Risk Assessment Scale (BRAS) was used to measure the risk of skin breakdown. The BRAS was developed and its validity and reliability were proved

by Braden and Bergstrom in 1987 (22). Its validity and reliability in Turkish patients were tested. The study showed that the scale had high validity and reliability in evaluation of risk of PU in Turkey (23). The BRAS is composed of 6 subscales about sensory perception, moisture, activity, mobility, nutrition, friction, and shearing. Each section is scored between 1 and 4 and the lowest and the highest scores of the scale are 6 and 23, respectively. Lower scores for the scale indicate a higher risk of PU. Scores of 23–20 show low risk, scores of 19–16 show moderate risk, scores of 15–11 show high risk, and scores of 10–6 show very high risk (22).

2.4. Data collection

Data were collected by staff nurses working in the anesthesia ICU where the study was conducted and by the second, fourth, fifth, sixth, seventh, and eighth authors of this article every day. Data were obtained from computerized and noncomputerized medical records until the patients were extubated. The risk of PU was detected by nurses using the BRAS scale.

Serum hemoglobin levels were measured every day, albumin levels were measured twice a week, and BMI was determined once when the patients were first admitted to the ICU. Nutrition, sedation and vasopressin administration, positioning status, edema, and types of beds were evaluated every day. In the anesthesia ICU, oxygenation parameters are monitored depending on the patients' needs and perfusion status is monitored every hour. In this study, the mean values of the best and worst oxygenation status and the mean values of the best and the worst perfusion status were used. UOP is monitored every hour in the unit. In this study, the total amount of urine measured for 24 h was recorded on the data collection form every day.

In the anesthesia ICU unit, perfusion parameters are measured and recorded every hour. Hemograms are followed and recorded every day and albumin levels are followed and recorded 2 times a week.

2.5. Statistical analyses

Data were analyzed with SPSS 15.0 for Windows and by numbers, percentages, the Mann–Whitney U test, and the chi-square test.

2.6. Ethical considerations

Ethical approval was obtained from the Noninterventional Clinical Research Evaluation Committee of Dokuz Eylül University. Approval was also obtained from the Health Directorate of Dokuz Eylül University Hospital. Before data were collected, patients' relatives were informed about the aim and methods of the research. Verbal and written informed consent was obtained from a relative of each patient.

3. Results

3.1. Sample characteristics

The patients were aged 18–89 years with a mean age of 62.30 ± 17.20 years; 66.4% of the patients (n = 73) were male and 55% of the patients (n = 61) were admitted to the ICU due to subarachnoid bleeding, drug intoxication, or total hip prosthesis. The mean length of MV was 6.68 ± 7.12 days. The mean albumin level was 2.37 ± 0.55 mg/dL, the mean hemoglobin level was 9.82 ± 1.67 mg/dL, and the mean BMI was 26.71 ± 5.01. Furthermore, 57.3% of patients (n = 63) had enteral nutrition, 78.2% of patients (n = 86) had sedation, and 55.5% of patients (n = 61) were administered vasopressin. Positions of 76.4% of patients (n = 84) were changed and 68.2% of patients (n = 75) were found to have edema; 57.3% of patients (n = 63) stayed on air mattresses. The mean PH level was 7.42 ± 0.08, the mean PaO_2 level was 159.17 ± 36.72 mmHg, the mean $PaCO_2$ level was 35.94 ± 6.85 mmHg, and the mean SaO_2 level was 97.24 ± 4.23. The mean SBP was 124.99 ± 33.91, the mean DBP was 83.78 ± 10.85, the mean MAP was 83.78 ± 10.85 mmHg, and the mean HR was 89.89 ± 11.66/min. The mean BRAS score was 11.49 ± 1.32 on admission to the ICU and 11.6 ± 1.18 during MV. The PU incidence was 15.5% (n = 17), and 11.8% of these patients (n = 13) had second-degree PUs (Table 1).

3.2. The BRAS scores

There was no significant difference in mean BRAS scores upon admission to the ICU between the patients developing PU (mean ± SD = 11.29 ± 1.10) and those not developing PU (mean ± SD = 11.52 ± 1.36) (U = 730.51; P = 0.61). Similarly, the difference in mean BRAS scores during MV between patients developing PU (mean ± SD = 11.29 ± 1.35) and those not developing PU (mean ± SD = 11.55 ± 1.55) was not significant (U = 646.00; P = 0.23) (Table 2).

3.3. Length of MV

There was a significant difference in duration of MV between patients developing PU and those not developing PU (P < 0.05). The mean duration of MV was significantly longer in patients developing PU (mean ± SD = 13.11 ± 9.57/day) than in patients not developing PU (mean ± SD = 5.05 ± 5.92/day) (U = 330; P = 0.00) (Table 3).

3.4. General health status

BMI, presence of edema, and vasopressin administration significantly differed between patients developing PU and those not developing PU during MV (P < 0.05). Patients developing PU had a significantly higher mean BMI (mean ± SD = 29.75 ± 6.67) than those not developing PU (mean ± SD = 26.15 ± 4.47) (U = 496; P = 0.01). Furthermore, 94.11% of patients developing PU (n = 16) and 63.44% of patients not developing PU (n = 59) had edema with a significant difference (χ^2 = 4.901; P = 0.02), while 82.35%

Table 1. Demographic and clinical characteristics of patients (n = 110).

Demographic characteristics	Min	Max	Mean ± SD
Age	18	89	62.30 ± 17.20[a]
Sex			n (%)
Female			37 (33.6)
Male			73 (66.4)
Clinical characteristics			
Diagnosis			
Multiple traumas			10 (9.1)
Pneumonia			10 (9.1)
COPD			4 (3.6)
Ileus (colectomy)			19 (17.3)
Stomach cancer (gastrectomy)			6 (5.5)
Other (subarachnoid bleeding, drug intoxication, total hip prosthesis)			61 (55.5)
Length of MV (days)	2	36	6.68 ± 7.12[a]
General health status			
Albumin	1.20	4.20	2.37 ± 0.55[a]
Hb	6.4	16.30	9.82 ± 1.67[a]
BMI	16.98	43.00	26.71 ± 5.01[a]
Nutrition			n (%)
Enteral			63 (57.3)
Parenteral			47 (42.7)
Sedation			
Yes			86 (78.2)
No			24 (21.8)
Vasopressin administration			
Yes			61 (55.5)
No			49 (44.5)
Changing position			
Yes			84 (76.4)
No			26 (23.6)
Edema			
Yes			75 (68.2)
No			35 (31.8)
Type of mattress			
Air			63 (57.3)
Viscoelastic			36 (32.7)
Dynamic			11 (10.0)
Oxygenation			Mean ± SD
PH	7.14	7.56	7.42 ± 0.08[a]
PaO_2	60.50	229.50	159.17 ± 36.72[a]
$PaCO_2$	23.5	59.00	35.94 ± 6.85[a]
SaO_2	72.91	100.00	97.24 ± 4.23[a]

Table 1. (Continued).

Demographic characteristics	Min	Max	Mean ± SD
Perfusion			
SBP	59.11	206.18	124.99 ± 33.91 [a]
DBP	40	90	64.73 ± 9.00[a]
MAP	60	114.5	83.78 ± 10.85[a]
HR	70.82	146.5	89.89 ± 11.66 [a]
Skin condition			Mean ± SD
Mean Braden Scale score on admission	9	18	11.49 ± 1.32[a]
Mean Braden Scale score during MV	9	16	11.60 ± 1.18[a]
PU			n (%)
Yes			17 (15.5)
No			93 (84.5)
PU Stage			
Stage 1			4 (3.6)
Stage 2			13 (11.8)

SD = Standard Deviation, [a] values are expressed as mean ± SD, COPD = chronic obstructive pulmonary disease, Hb = hemoglobin, PH = power of hydrogen, $PaCO_2$ = partial arterial carbon dioxide pressure, PaO_2 = partial arterial oxygen pressure, SaO_2 = oxygen saturation SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, HR = heart rate, PU = pressure ulcer, BMI = body mass index.

Table 2. Distribution and comparison of mean Braden Risk Assessment Scale scores according to pressure ulcer development status (n = 110).

	Developed a pressure ulcer n = 17 (15.5%) Mean ± SD	Did not develop a pressure ulcer n = 93 (84.5%) Mean ± SD	U	P*
Mean score for Braden scale on admission	11.29 ± 1.10[a]	11.52 ± 1.36[a]	730.51	0.61
Mean score for Braden scale during MV	11.29 ± 1.35[a]	11.5 ± 1.55[a]	646.00	0.23

SD = Standard Deviation, [a] Values are expressed as mean ± SD, U = Mann-Whitney U test; *P > 0.05

of patients developing PU (n = 14) and 50.53% of patients not developing PU (n = 47) were administered vasopressin ($\chi^2 = 4.672$; P = 0.03).

There was no significant difference in the mean albumin and hemoglobin levels, receiving sedation, changing position, nutrition status, and type of mattress between the patients developing PU and those not developing PU during MV (P > 0.05) (Table 3).

3.5. Oxygenation

There was a significant difference in all oxygenation related variables between patients with PU and those without PU during MV (P < 0.05). Patients with PU had a significantly higher mean PH (mean ± SD = 7.46 ± 0.06) than those without PU (mean ± SD = 7.42 ± 0.08) (U = 522; P = 0.02). In addition, patients with PU had a significantly lower mean PaO_2 (mean ± SD = 112.53 ± 33.61 mmHg) than those without PU (mean ± SD = 132.10 ± 32.30 mmHg) (U = 505; P = 0.01). Besides, patients with PU had a significantly higher mean PCO_2 (mean ± SD = 39.05 ± 7.19) than those without PU (mean ± SD = 35.37 ± 6.68) (U = 531; P = 0.03). The mean SaO_2 was also significantly lower in patients with PU (mean ± SD = 96.23 ± 3.26) than in those without PU (mean ± SD = 100 ± 46.69) (U = 511; P = 0.02) (Table 3).

3.6. Perfusion

There was a significant difference in the mean UOP, indicative of perfusion status, between patients with PU and those without PU (P < 0.05). Patients with PU had a significantly higher mean UOP (mean ± SD = 3088 ± 1284) than those without PU (mean ± SD = 2375 ± 1456) (U = 504.5; P = 0.01). However, there was not a significant difference in other variables related to perfusion (SBP, DBP, MAP, and HR) between the patients (P > 0.05) (Table 3).

Table 3. Comparison of general health, oxygenation, and perfusion status according to pressure ulcer development status (n = 110).

	Developed a pressure ulcer (n = 17) Mean ± SD	Did not develop a pressure ulcer (n = 93) Mean ± SD	U	P
Length of MV (days)	13.11 ± 9.57[a]	4.05 ± 5.92[a]	330	0.00*
General health status				
Albumin	2.31 ± 0.46[a]	2.38 ± 0.56[a]	727	0.59
Hemoglobin	9.79 ± 1.55[a]	9.83 ± 1.70[a]	785	0.96
BMI	29.75 ± 6.67[a]	26.15 ± 4.47[a]	496	0.01*
Nutrition	n (%)	n (%)	χ^2	P
Enteral	11 (64.70)	52 (55.91)	0.454	1.5
TPN	6 (35.30)	41 (44.09)		
Sedation				
Yes	14 (82.35)	72 (77.44)	0.018	0.89
No	3 (17.65)	21 (22.59)		
Vasopressin				
Yes	14 (82.35)	47 (50.53)	4.672	0.03*
No	3 (17.65)	46 (49.47)		
Changing position				
Yes	14 (82.35)	70 (75.26)	0.104	0.74
No	3 (17.65)	23 (24.74)		
Edema				
Yes	16 (94.11)	59 (63.44)	4.901	0.02*
No	1 (5.89)	34 (36.56)		
Type of mattress				
Air	7 (41.17)	56 (60.2)		
Viscoelastic	10 (58.82)	26 (28.0)	7.105	2.02
Dynamic	0 (0.00)	11 (11.82)		
Oxygenation	Mean ± SD	Mean ± SD	U	P
PH	7.46 ± 0.06[a]	7.42 ± 0.08[a]	522	0.02*
PaO_2	112.53 ± 33.61[a]	132.10 ± 32.30[a]	505	0.01*
$PaCO_2$	39.05 ± 7.19[a]	35.37 ± 6.68[a]	531	0.03*
SaO_2	96.23 ± 3.26[a]	100 ± 46.69[a]	511	0.02*
Perfusion	Mean ± SD	Mean ± SD	U	P
SBP	123.47 ± 13.8[a]	83.82 ± 11.26[a]	732.5	0.63
DBP	63.58 ± 6.99[a]	64.94 ± 9.33[a]	698.5	0.44
MAP	83.54 ± 8.54[a]	83.82 ± 11.26[a]	787	0.97
HR	96.82 ± 13.64[a]	101.65 ± 17.70[a]	645	0.22
UOP	3088 ± 1284[a]	2375 ± 1456[a]	504.5	0.01*

SD = Standard deviation, [a]values are expressed as mean ± SD, Hb = hemoglobin, PH = power of hydrogen, $PaCO_2$ = partial arterial carbon dioxide pressure, PaO_2 = partial arterial oxygen pressure, SaO_2 = oxygen saturation, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, HR = heart rate, PU = pressure ulcer, BMI = body mass index, TPN = total parenteral nutrition, U = Mann–Whitney U test, χ^2 = chi-square test; *P < 0.05.

4. Discussion

The incidence of PU was 15.5% (n = 17) in patients on MV in the anesthesia ICU. It was found to be 20% (18) and 18.7% (12) in two studies from the United States, 20.1% (10) in one study from Belgium, 16.7% (19) in one study from Turkey, and 16% (13) in one study from Spain in patients on MV in ICUs. The PU rate in patients not on MV in ICUs in Turkey varies from 15% to 29% (6–8,24).

The lower rate of PUs in this study can be attributed to the fact that the nurses working in the ICU carefully implement interventions for prevention of PU and that the patients stay on air, dynamic, and viscoelastic mattresses. However, consistent with the literature, we found that the mean duration of MV was longer in patients with PU (7,11,13,16, 25). Manzano et al. reported that every day when a patient was on MV the risk of PU increased by 4.2% (12).

The BRAS scores did not significantly differ between the patients with PU and those without PU. Consistent with this finding, two studies on patients on MV in ICUs showed no significant differences between PU development, BRAS scores (16), and Norton Scale scores (17). However, unlike the present study, two other studies revealed that lower BRAS scores (8) and lower Norton Scale scores (25) increased the risk of PU development. The BRAS is commonly used in ICUs and involves sections on sensory perception, moisture, activity, mobility, nutrition, friction, and shearing. However, there are other factors that play a role in PU development such as longer duration of MV and ICU stay, low blood pressure, presence of edema, and vasopressin administration (4,9–11,13,15). In the present study, the finding that the BRAS scores did not affect PU development might have been caused by the inability of this scale to accurately measure the risk of PU due to the abovementioned factors.

Shanin et al. (26) from Germany reported in 2009 that patients with PUs had higher BMIs and that BMI increased the risk of PUs. However, in other studies Shanin et al. (27) and Terekeci et al. from Turkey (25) reported that BMI did not affect PU development, which is conflicting with the present study. We found that a high BMI increased the risk of PU. A high body weight can increase pressure on the skin over bony prominences (28).

Congruent with the results of this study, edema was found to increase the risk of PU in one study (9). Edema decreases resistance of the skin and tissues under the skin against pressure, friction, and damage and increases the risk of PU (10,14).

Both the present study and two other studies showed that vasopressin administration affected PU development (9,25). Cox from the United States found that 49% of patients receiving norepinephrine developed PU (12). It has been reported in the literature that vasoconstrictors administered in ICUs decrease oxygenation of tissues and increase the risk of PU (4,14).

In this study we did not find a significant difference in albumin and hemoglobin levels between patients with PU and those without PU. It was reported that albumin levels were not significantly related to PU development (17). However, unlike the present study, several studies revealed that there was a negative relation between PU stage and albumin levels (24), and that lower albumin levels affected PU development (7,25).

Several studies revealed that sedation increased PU development (25,29). However, Nijs et al. reported a negative relation between receiving sedation and the risk of PU development (9). The present study also showed that patients with PU and those without PU did not differ in terms of receiving sedation. This can be explained by the fact that sedation does not affect perfusion although it decreases mobilization.

Nijs et al. (9) found that changing patients' positions fewer than 6 times a day increases the risk of PU development. Tokgöz and Demir (7) also noted that not changing patients' positions is the most important factor for PU development. The main purpose of PU prevention is to reduce pressure and degree of constant pressure. One of the most important interventions carried out to achieve this aim is changing patients' positions. Since hemodynamic status was not stable, all but 26 patients were provided with a change in their positions at certain intervals in this study. No significant differences were found between PU and changing position, conflicting with the literature. This can be ascribed to the fact that PU results from many factors (9–12,21,25).

We did not find a significant difference between nutritional status (enteral/total parenteral) and PU development, which is not consistent with the literature. İnan and Öztunç (8) reported that patients developing PU were most frequently the ones who received total parenteral nutrition (60%). In a review of 15 studies, Stratton et al. (30) from the UK suggested that enteral nutrition and a high-protein oral diet could decrease PU by 25% and that further studies on the issue were needed.

Compatible with the literature (24), we found that types of mattresses did not affect PU development. It may be that all types of mattresses can decrease and evenly distribute pressure (31).

In the present study, patients with PUs had significantly higher PH and $PaCO_2$, and significantly lower SaO_2 and PaO_2. Indeed, since insufficient oxygenation causes tissue hypoxia and necrosis, it can considerably increase the risk of PU development (9,12). However, Şenturan et al. (2009) (n = 30) reported that PaO_2, $PaCO_2$, and SaO_2 did not have an effect on PU development, although high PH was effective (17). In another study (n = 40), oxygenation

status was not found to affect PU development (16). These conflicting findings can be due to small sample sizes of the studies.

In this study, UOP, an indicator of perfusion, was significantly higher in patients with PU. As far as we know, there have not been any studies investigating the effects of UOP on PU development. However, one study revealed that hemodialysis increased the risk of PU development (9). PU development in patients with higher UOP can be ascribed to decreased tissue perfusion due to dehydration caused by excessive UOP (14). Other perfusion-related variables were not found to affect PU development in this study. Evidence for other variables of perfusion has been conflicting in the literature. Pender and Frizer (16) found no significant differences between PU, MAP, and HR, which is consistent with the results of the present study. However, Şenturan et al. (17) reported that a low DBP had an effect on PU, Terekeci et al. (25) noted that low MAP had an impact on PU, and Cox (11) revealed that low MAP, DBP, and SBP had an effect on PU.

In conclusion, pressure ulcers are a major nurse-sensitive outcome. Nurses play an important role in the prevention of PUs and PUs are indicators of insufficient nursing care. Hence, nursing care has a major effect on PU development and prevention. Therefore, it can be recommended that nurses and other health professionals be aware of these factors and develop appropriate preventive strategies to reduce the incidence of PUs. In addition, data about PU incidence and relevant risk factors in ICU patients on MV will contribute to evidence-based nursing practices and shed light on attempts to prevent and manage PU and to reduce its incidence.

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